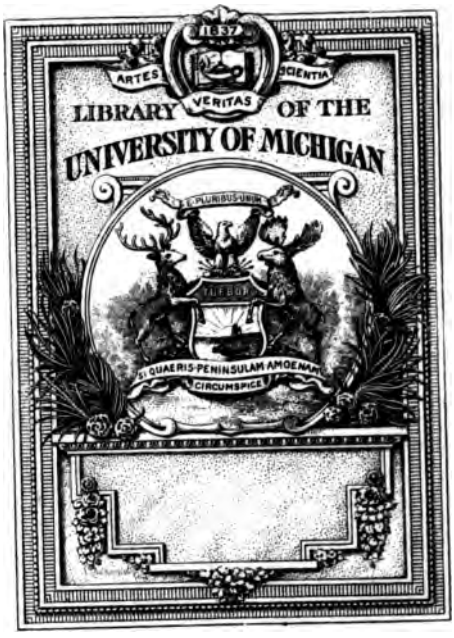


MEDICO-CHIRURGICAL TRANSACTIONS

PUBLISHED BY

THE ROYAL

MEDICAL AND CHIRURGICAL SOCIETY

OF

LONDON

VOLUME THE EIGHTY-FIFTH

(SECOND SERIES, VOLUME THE SIXTY-SEVENTH)

LONDON
LONGMANS, GREEN AND CO.
(FOR THE ROYAL MEDICAL AND CHIRURGICAL SOCIETY OF LONDON)
PATERNOSTER ROW

1902

Issued from the Society's House at 20, *Hanover Square, W.*

December, 1902.

PRINTED BY ADLARD AND SON, BARTHOLOMEW CLOSE, E.C.

CONTENTS

ROYAL
MEDICAL AND CHIRURGICAL SOCIETY
OF LONDON.

PATRON

THE KING

OFFICERS AND COUNCIL

ELECTED MARCH 1, 1902.

President

ALFRED WILLETT

VICE-PRESIDENTS	Sir R. Douglas Powell, Bart., M.D., K.C.V.O. Sir Dyce Duckworth, M.D., LL.D. N. Charles Macnamara Edgcombe Venning
HON. TREASURERS	Sir William Selby Church, Bart., M.D. J. Warrington Haward
HON. SECRETARIES	George Newton Pitt, M.D. Clinton Thomas Dent.
HON. LIBRARIANS	Norman Moore, M.D. Rickman J. Godlee, M.S.
OTHER MEMBERS OF COUNCIL	James Kingston Fowler, M.D. Archibald Edward Garrod, M.D. Francis de Havilland Hall, M.D. Sir Isambard Owen, M.D. Amand Jules McConnell Routh, M.D. Walter Hamilton Acland Jacobson Henry Edward Juler Charles R. B. Keetley Charles Barrett Lockwood Thomas Laurence Read

THE ABOVE FORM THE COUNCIL.

SECRETARY AND CONSULTING LIBRARIAN

J. Y. W. Mac Alister, F.S.A.

FELLOWS OF THE SOCIETY APPOINTED BY THE COUNCIL AS REFEREES OF PAPERS

FOR THE SESSION OF 1902–3

JOHN ABERCROMBIE, M.D.
ARTHUR E. J. BARKER
SIR WILLIAM H. BENNETT, K.C.V.O.
STANLEY BOYD, B.S.
J. ROSE BRADFORD, M.D., F.R.S.
HENRY TRENTHAM BUTLIN
THOMAS BUZZARD, M.D.
WILLIAM CAYLEY, M.D.
CHARLES J. CULLINGWORTH, M.D.
ALBAN DORAN
FREDERIC S. EVE
ALFRED LEWIS GALABIN, M.D.
S. J. GEE, M.D.
J. F. GOODHART, M.D.
W. S. A. GRIFFITH, M.D.
VINCENT D. HARRIS, M.D.
PHILIP JOHN HENSLEY, M.D.
SIR VICTOR A. HADEN HORSLEY, F.R.S.
WALTER H. H. JESSOP, M.B.
JEREMIAH MCCARTHY
G. H. MAKINS, C.B.
JOHN HAMMOND MORGAN, C.V.O.
F. W. MOTT, M.D., F.R.S.
JOSEPH FRANK PAYNE, M.D.
BERNARD PITTS, M.C.
GEORGE VIVIAN POORE, M.D.
PHILIP H. PYE-SMITH, M.D., F.R.S.
FREDERICK T. ROBERTS, M.D.
ARTHUR ERNEST SANSOM, M.D.
SEYMOUR J. SHARKEY, M.D.
A. MARMADUKE SHEILD, B.C.
HERBERT R. SPENCER, M.D.
FREDERICK TAYLOR, M.D.
HOWARD H. TOOTH, C.M.G., M.D.
AUGUSTUS WALLER, M.D., F.R.S.
WILLIAM JOHNSON WALSHAM, C.M.

TRUSTEES

TRUSTEES OF THE SOCIETY'S INVESTMENTS

WALTER BUTLER CHEADLE, M.D.
FREDERICK TAYLOR, M.D.
ALFRED WILLETT

TRUSTEES FOR THE DEBENTURE-HOLDERS

SAMUEL JONES GEE, M.D., *Chairman*
SIR THOMAS BARLOW, BART., K.C.V.O., M.D.
C. THEODORE WILLIAMS, M.D.

TRUSTEES OF THE MARSHALL HALL MEMORIAL FUND

WALTER BUTLER CHEADLE, M.D.
WILLIAM OGLE, M.D.
SIR THOMAS SMITH, BART.

COMMITTEES

COMMITTEE APPOINTED TO INVESTIGATE THE SUBJECT OF SUSPENDED ANIMATION IN THE DROWNED

EDWARD ALBERT SCHÄFER, F.R.S., *Chairman*

THE PRESIDENT
THE HON. SECRETARIES
FREDERICK W. MOTT, M.D., F.R
HENRY POWER

THOMAS PICKERING PICK, *Hon. Sec.*

LIBRARY COMMITTEE FOR 1902–3

THE PRESIDENT
THE HON. LIBRARIANS
THE HON. SECRETARIES
W. R. DAKIN, M.D.
ALBAN DORAN
WILLIAM EWART, M.D.
W. P. HERRINGHAM, M.D.
STEPHEN PAGET
JOSEPH FRANK PAYNE, M.D.
GEORGE VIVIAN POORE, M.D.
D'ARCY POWER, M.B.
P. H. PYE-SMITH, M.D., F.R.S.
A. QUARRY SILCOCK, B.S.

HOUSE COMMITTEE FOR 1902–3

THE PRESIDENT
THE HON. TREASURERS
THE HON. LIBRARIANS
THE HON. SECRETARIES
H. E. JULER
T. LAURENCE READ

PRESIDENTS OF THE SOCIETY FROM ITS FORMATION AS THE "MEDICO-CHIRURGICAL SOCIETY," 1805

ELECTED	
1805	WILLIAM SAUNDERS, M.D.
1808	MATTHEW BAILLIE, M.D.
1810	SIR HENRY HALFORD, Bart., M.D., G.C.H.
1813	SIR GILBERT BLANE, Bart., M.D.
1815	HENRY CLINE
1817	WILLIAM BABINGTON, M.D.
1819	SIR ASTLEY PASTON COOPER, Bart., K.C.H.
1821	JOHN COOKE, M.D
1823	JOHN ABERNETHY
1825	GEORGE BIRKBECK, M.D.
1827	BENJAMIN TRAVERS
1829	PETER MARK ROGET, M.D.
1831	SIR WILLIAM LAWRENCE, Bart.
1833	JOHN ELLIOTSON, M.D. (First President of the Society after its Incorporation as the Royal Medical and Chirurgical Society of London in 1834).
1835	HENRY EARLE
1837	RICHARD BRIGHT, M.D.
1839	SIR BENJAMIN COLLINS BRODIE, Bart.
1841	ROBERT WILLIAMS, M.D.
1843	EDWARD STANLEY
1845	WILLIAM FREDERICK CHAMBERS, M.D., K.C.H.
1847	JAMES MONCRIEFF ARNOTT
1849	THOMAS ADDISON, M.D.
1851	JOSEPH HODGSON
1853	JAMES COPLAND, M.D.
1855	CÆSAR HENRY HAWKINS
1857	SIR CHARLES LOCOCK, Bart., M.D.
1859	FREDERIC CARPENTER SKEY
1861	BENJAMIN GUY BABINGTON, M.D.
1863	RICHARD PARTRIDGE
1865	SIR JAMES ALDERSON, M.D.
1867	SAMUEL SOLLY
1869	SIR GEORGE BURROWS, Bart., M.D.
1871	THOMAS BLIZARD CURLING
1873	CHARLES JAMES BLASIUS WILLIAMS, M.D.
1875	SIR JAMES PAGET, Bart.
1877	CHARLES WEST, M.D.
1879	JOHN ERIC ERICHSEN
1881	ANDREW WHYTE BARCLAY, M.D.
1882	JOHN MARSHALL
1884	SIR GEORGE JOHNSON, M.D.
1886	GEORGE DAVID POLLOCK
1888	SIR EDWARD HENRY SIEVEKING, M.D.
1890	TIMOTHY HOLMES
1892	SIR ANDREW CLARK, Bart., M.D. (*Died 6th Nov.*, 1893, *and Sir. W. S. Church, Senior* [*Medical Vice-President, acted as President until 1st March*, 1894.
1894	JONATHAN HUTCHINSON
1896	WILLIAM HOWSHIP DICKINSON, M.D.
1898	THOMAS BRYANT
1900	FREDERICK WILLIAM PAVY, M.D., LL.D., F.R.S.
1902	ALFRED WILLETT

HONORARY FELLOWS

(Limited to Twelve.)

Elected

1887 FOSTER, SIR MICHAEL, K.C.B., M.D., LL.D., F.R.S., M.P., Professor of Physiology in the University of Cambridge, Nine Wells, Great Shelford, Cambridge.

1868 HOOKER, SIR JOSEPH DALTON, M.D., C.B., G.C.S.I., D.C.L., LL.D., F.R.S., Corresponding Member of the Academy of Sciences of France; The Camp, Sunningdale.

1896 KELVIN, The Right Hon. LORD, P.C., G.C.V.O., F.R.S, Pres. R.S.E., D.C.L., LL.D., &c., Glasgow.

1878 AVEBURY, The Right Hon. LORD, D.C.L., LL.D., F.R.S., High Elms, Farnborough, Kent, R.S.O.

1873 STOKES, SIR GEORGE GABRIEL, Bart., M.A., D.C.L., LL.D., Sc.D., F.R.S., Lucasian Professor of Mathematics in the University of Cambridge; Lensfield Cottage, Cambridge.

1887 TURNER, SIR WILLIAM, M.B., D.C.L., LL.D., F.R.S., Professor of Anatomy in the University of Edinburgh; 6, Eton Terrace, Edinburgh.

FOREIGN HONORARY FELLOWS

(Limited to Twenty.)

Elected

1878 Baccelli, Guido, M.D., Rome.

1896 von Bergmann, Ernst, Berlin.

1887 Billings, John S., M.D., D.C.L.Oxon., New York.

1896 Czerny, Vincent, M.D., Heidelberg.

1896 Erb, Wilhelm, M.D., Professor of Clinical Medicine, Heidelberg.

1887 von Esmarch, His Excellency Friedrich, M.D., Kiel.

1896 Fournier, Alfred, M.D., Paris.

1896 Koch, Robert, M.D., Berlin.

1896 Kocher, Theodore, M.D., Berne.

1868 Kölliker, Albert, Würzburg.

1896 Laveran, A., M.D., Paris.

1896 Marie, Pierre, M.D., Paris.

1896 Mirza-Ali, M.D., Teheran.

1896 Mitchell, Samuel Weir, M.D., Philadelphia.

FELLOWS

OF THE

ROYAL MEDICAL AND CHIRURGICAL SOCIETY

OF LONDON

EXPLANATION OF THE ABBREVIATIONS

P.—President.
V.P.—Vice-President.
T.—Treasurer.
L.—Hon. Librarian.
S.—Hon. Secretary.
C.—Member of Council.
Sci. Com.—Member of a Scientific Committee.
Ho. Com.—Member of House Committee.
Lib. Com.—Member of Library Committee.
Bldg. Com.—Member of Building Committee.
Dis. Com.—Member of Discussions Committee.

The abbreviations *Trans.* and *Pro.*, followed by figures, show the number of Papers which have been contributed to the *Transactions* or *Proceedings* by the Fellow whose name they follow. *Referee, Sci. Com., Lib. Com., Bldg. Com., Ho. Com.*, and *Dis. Com.*, with the dates of office, are attached to the names of those who have served as Referees of papers and on the Committees of the Society.

Names printed in this type are of those Fellows who have paid the Composition Fee in lieu of further annual subscriptions.

Names printed in this *type* are of those Fellows who have paid the Composition Fee entitling them to receive the *Transactions*.

RESIDENT FELLOWS

[N.B.—Fellows are reminded that they are, themselves, responsible for the correctness of the descriptions in the following lists, and it is particularly requested that any change of Title, Appointment, or Residence may be communicated to the Hon. Secretaries before the 1st of July in each year.]

Elected

1898 AARONS, S. JERVOIS, M.D., 14, Stratford place, Oxford street.

1877 **Abercrombie**, JOHN, M.D., Physician to, and Lecturer on Forensic Medicine at, Charing Cross Hospital; 23, Upper Wimpole street, Cavendish square. C. 1896-8. *Referee*, 1898—. *Trans.* 2.

Elected

1885 ABRAHAM, PHINEAS S., M.A., M.D., Dermatologist to the West London Hospital, Assistant Surgeon to Hospital for Diseases of the Skin, Blackfriars; 2, Henrietta street, Cavendish square.

1885 ACLAND, THEODORE DYKE, M.D., Physician to St. Thomas's Hospital, and Physician to the Hospital for Consumption and Diseases of the Chest, Brompton; 19, Bryanston square.

1897 ADDISON, CHRISTOPHER, M.D., Charing Cross Hospital Medical School, Chandos street.

1879 ALLCHIN, WILLIAM HENRY, M.D., F.R.S. Ed., Senior Physician to the Westminster Hospital; 5, Chandos street, Cavendish square. C. 1898-9. *Referee*, 1897.

1890 ALLINGHAM, HERBERT WILLIAM, Surgeon to His Majesty's Household, Surgeon to the Great Northern Hospital; Assistant Surgeon to St. George's Hospital; 25, Grosvenor street, Grosvenor square.

1888 ANDERSON, JOHN, M.D., C.I.E., Physician to the Seamen's Hospital, Greenwich; Lecturer on Tropical Medicine at St. Mary's Hospital Medical School; 9, Harley street, Cavendish square.

1891 ANDREWES, FREDERICK WILLIAM, M.D., Highwood, Hampstead lane, Highgate.

1902 ARMOUR, DONALD JOHN, M.B., Bentinck street.

1893 BAILEY, ROBERT COZENS, M.S., 21, Welbeck street, Cavendish square.

1891 BAKER, CHARLES ERNEST, M.B., 5, Gledhow gardens, South Kensington.

1900 BALDWIN, ASLETT, 6, Manchester square.

1887 BALL, JAMES BARRY, M.D., Physician to the West London Hospital; 12, Upper Wimpole street, Cavendish square.

Elected

1885 BALLANCE, CHARLES ALFRED, M.S., Assistant Surgeon to St. Thomas's Hospital and to the Hospital for Sick Children, Great Ormond street; Surgeon to the National Hospital for the Paralysed and Epileptic, Queen square; 106, Harley street, Cavendish square. *Trans.* 6.

1879 BARKER, ARTHUR EDWARD JAMES, Professor of the Principles and Practice of Surgery and Professor of Clinical Surgery at University College, and Surgeon to University College Hospital, London; 87, Harley street, Cavendish square. C. 1895-7. *Referee*, 1897—. *Trans.* 7.

1876 **Barlow**, SIR THOMAS, Bart., K.C.V.O., M.D., B.S., *Trustee for Debenture-holders;* Physician to His Majesty's Household; Physician to University College Hospital; 10, Wimpole street, Cavendish square. C. 1892. S. 1899-1902. *Referee*, 1896-9. *Trans.* 2.

1893 BARRETT, HOWARD, 49, Gordon square.

1880 BARROW, A. BOYCE, Surgeon to King's College Hospital; 8, Upper Wimpole street, Cavendish square.

1896 BARTON, JAMES KINGSTON, 14, Ashburn place, Courtfield road, South Kensington.

1859 BARWELL, RICHARD, Consulting Surgeon to the Charing Cross Hospital; 55, Wimpole street. C. 1876-77. V.P. 1883-4. *Referee*, 1868-75, 1879-82. *Trans.* 12. *Pro.* 1.

1868 **Bastian**, HENRY CHARLTON, M.A., M.D., F.R.S., Emeritus Professor of the Principles and Practice of Medicine and of Clinical Medicine in University College, London; Consulting Physician to University College Hospital and Physician to the National Hospital for the Paralysed and Epileptic; 8A, Manchester square. C. 1885. *Referee*, 1886-96. *Trans.* 3.

1890 BATEMAN, WILLIAM A. F., Bridge House, Richmond, Surrey.

1891 BATTEN, FREDERICK E., M.D., B.C., 33, Harley street.

Elected

1875 BEACH, FLETCHER, M.B., Physician to the West End Hospital for Nervous Diseases, Winchester House, Kingston Hill [79, Wimpole street].

1883 BEALE, EDWIN CLIFFORD, M.A., M.B., Physician to the City of London Hospital for Diseases of the Chest, and Physician to the Great Northern Central Hospital; 23, Upper Berkeley street.

1862 BEALE, LIONEL SMITH, M.B., F.R.S., Professor of the Principles and Practice of Medicine in King's College, London, and Physician to King's College Hospital; 61, Grosvenor street. C. 1876-7. *Referee*, 1873-5. *Trans.* 1.

1897 BEDDARD, A. P., M.B., Assistant Physician to Guy's Hospital; 44, Seymour street.

1880 BEEVOR, CHARLES EDWARD, M.D., Physician for Out-patients to the National Hospital for the Paralysed and Epileptic, and to the Great Northern Hospital; 135, Harley street, Cavendish square. C. 1900-2. *Referee*, 1896-1900. *Trans.* 1.

1901 BEEVOR, SIR HUGH REEVE, Bart., M.D., 17, Wimpole street, Cavendish square.

1877 BENNETT, SIR WILLIAM HENRY, K.C.V.O., Surgeon to St. George's Hospital; 1, Chesterfield street, Mayfair. C. 1893-4. *Referee*, 1892-93, 1899—. *Trans.* 4.

1897 BERKELEY, COMYNS, M.B., B.C., Physician to Out-Patients, Chelsea Hospital for Women; 53, Wimpole street.

1885 BERRY, JAMES, B.S., Surgeon to the Royal Free Hospital, and Lecturer on Surgery at the London School of Medicine for Women; Demonstrator of Practical Surgery, St. Bartholomew's Hospital; 21, Wimpole street, Cavendish square.

1893 BIDWELL, LEONARD A., Senior Assistant Surgeon to the West London Hospital; 15, Upper Wimpole street, Cavendish square.

Elected

1851 **Birkett,** JOHN, F.L.S., Consulting Surgeon to Guy's Hospital; Corresponding Member of the Société de Chirurgie of Paris; 1, Sussex gardens. L. 1856-7. S. 1863-5. C. 1867-8. T. 1870-78. V.P. 1879-80. *Referee*, 1851-5, 1866, 1869. *Sci. Com.* 1863. *Lib. Com.* 1852. *Trans.* 8.

1897 BLACKER, G. F., M.D., Obstetric Physician to University College Hospital and to the Great Northern Central Hospital; 11, Wimpole street, Cavendish square.

1901 BLAIKIE, J. BRUNTON, M.D., C.M., 22, Grosvenor street Grosvenor square.

1883 BLAND-SUTTON, JOHN, Assistant Surgeon to the Middlesex Hospital; Surgeon to the Chelsea Hospital for Women; 47, Brook street, Grosvenor square. *Trans.* 6.

1865 **Blandford,** GEORGE FIELDING, M.D., Lecturer on Psychological Medicine at St. George's Hospital; 48, Wimpole street, Cavendish square. C. 1883-4. V.P. 1898-1900.

1891 BOKENHAM, THOMAS JESSOPP, 10, Devonshire street, Portland place.

1882 BOWLBY, ANTHONY ALFRED, C.M.G., Assistant Surgeon to St. Bartholomew's Hospital; 24, Manchester square. *Trans.* 8.

1870 *Bowles,* ROBERT LEAMON, M.D., 16, Upper Brook street, Grosvenor square. C. 1897-9. *Sci. Com.* 1896-1902. *Trans.* 3.

1886 BOXALL, ROBERT, M.D., Obstetric Physician to Out-patients, and Lecturer on Midwifery and Diseases of Women, at the Middlesex Hospital; 40, Portland place.

1884 BOYD, STANLEY, B.S., Surgeon to, and Lecturer on Surgery at, the Charing Cross Hospital; Surgeon to the Paddington Green Children's Hospital; Consulting Surgeon to the New Hospital for Women; 134, Harley street, Cavendish square. *Referee*, 1895—. *Trans.* 1.

Elected

1890 BRADFORD, JOHN ROSE, M.D., D.Sc., F.R.S., Physician to University College Hospital; 8, Manchester square. *Referee*, 1899—. *Trans.* 1.

1897 BRAILEY, WILLIAM ARTHUR, M.D., 11, Old Burlington street.

1899 BREMRIDGE, RICHARD HARDING, 72, Great Russell street.

1901 BREWERTON, ELMORE WRIGHT, 45, Weymouth street, Portland place.

1898 **Broadbent**, J. F. H., M.D., 35, Seymour street.

1868 **Broadbent**, SIR WILLIAM HENRY, Bart., K.C.V.O., M.D., F.R.S., LL.D., Physician in Ordinary to H.M. the King; Consulting Physician to St. Mary's Hospital; Consulting Physician to the London Fever Hospital; 84, Brook street, Grosvenor square. C. 1885. *Referee*, 1881-4, 1891-7. *Trans.* 5.

1872 BRODIE, GEORGE BERNARD, M.D., Consulting Physician-Accoucheur to Queen Charlotte's Hospital; 3, Carlos place, Grosvenor square. *Trans.* 1.

1880 BROWNE, JAMES WILLIAM, M.B., 37, Holland Park avenue. C. 1900-1.

1881 BROWNE, OSWALD AUCHINLECK, M.A., M.D., Physician to the Royal Hospital for Diseases of the Chest and to the Metropolitan Hospital; 7, Upper Wimpole street.

1874 BRUCE, JOHN MITCHELL, M.D., Physician to, and Lecturer on Medicine at, the Charing Cross Hospital; Consulting Physician to the Hospital for Consumption, Brompton; 23, Harley street. C. 1892, 1897-9. S. 1893-6. *Sci. Com.* 1889-1902. *Ho. Com.* 1898-9. *Referee*, 1886-91. *Lib. Com.* 1888-91. *Trans.* 3.

1898 BRUCE, SAMUEL NOBLE, 15, Queensborough terrace, Hyde Park.

1871 BRUNTON, SIR THOMAS LAUDER, M.D., D.Sc. LL.D., F.R.S., Physician to, and Lecturer on Pharmacology and Therapeutics at, St. Bartholomew's Hospital; 10, Stratford place, Oxford street. C. 1888-9. *Referee*, 1880-87. *Lib. Com.* 1882-7. *Trans.* 2.

Elected

1898 BRYANT, J. H., M.D., Assistant Physician to Guy's Hospital; 4, St. Thomas's street, London bridge.

1860 **Bryant,** THOMAS, M.Ch., Honorary Surgeon-in-Ordinary to H.M. the King, Consulting Surgeon to Guy's Hospital; Member of the Société de Chirurgie, Paris; 27, Grosvenor street, Grosvenor square. P. 1898-1900. C. 1873-4. V.P. 1885-6. *Sci. Com.* 1863. *Referee,* 1882-4. *Lib. Com.* 1868-71. *Trans.* 17. *Pro.* 1.

1901 BUCKNALL, THOMAS RUPERT HAMPDEN, M.S., M.D.; 35, Harley street, Cavendish square.

1889 BULL, WILLIAM CHARLES, M.B., Aural Surgeon to, and Lecturer on Aural Surgery at, St. George's Hospital; 5, Clarges street, Piccadilly.

1893 BURGHARD, FRÉDÉRIC FRANÇOIS, M.D., M.S., Surgeon to King's College Hospital and Paddington Green Children's Hospital; 86, Harley street, Cavendish square.

1885 BUTLER-SMYTHE, ALBERT CHARLES, Senior Out-Patient Surgeon, Samaritan Free Hospital for Women and Children, Soho; Senior Surgeon to the Grosvenor Hospital for Women and Children; 76, Brook street, Grosvenor square.

1873 **Butlin,** HENRY TRENTHAM, D.C.L., Surgeon to St. Bartholomew's Hospital; 82, Harley street, Cavendish square. C. 1887-8. *Referee,* 1893—. *Trans.* 4. *Pro.* 1.

1896 BUTTAR, CHARLES, M.D., 10, Kensington gardens square, Bayswater. *Pro.* 1.

1883 BUXTON, DUDLEY WILMOT, M.D., B.S., Administrator, and Teacher of the Use, of Anæsthetics, in University College Hospital; Consulting Anæsthetist to the National Hospital for the Paralysed and Epileptic, Queen square, and Anæsthetist to the London Dental Hospital; 82, Mortimer street, Cavendish square.

1899 BUZZARD, EDWARD FARQUHAR, M.B., 33, Harley street, Cavendish square.

Elected

1868 **Buzzard,** THOMAS, M.D., Physician to the National Hospital for the Paralysed and Epileptic; 74, Grosvenor street, Grosvenor square. C. 1885-6. *Referee*, 1887—.

1885 CAHILL, JOHN, M.D., Surgeon to the Hospital of St. John and St. Elizabeth; 12, Seville street, Lowndes square.

1893 CALEY, HENRY ALBERT, M.D., Physician in charge of Out-patients, Lecturer on Materia Medica and Therapeutics, and Dean of the Medical School, St. Mary's Hospital; 24, Upper Berkeley street, Portman square.

1887 CALVERT, JAMES, M.D., 113, Harley street. *Trans.* 1.

1897 CANTLIE, JAMES, M.B., 46, Devonshire street.

1901 CARGILL, LIONEL VERNON, 31, Harley street, Cavendish square.

1888 CARLESS, ALBERT, M.S., Professor of Surgery in King's College, London; Surgeon to King's College Hospital; 10, Welbeck street.

1896 CARR, J. WALTER, M.D., Physician to the Royal Free Hospital; Physician to the Victoria Hospital for Children; 19, Cavendish place. *Trans.* 1.

1898 CARTER, H. RONALD, 11, Leonard place, Kensington.

1853 CARTER, ROBERT BRUDENELL, Consulting Ophthalmic Surgeon to St. George's Hospital; 31, Harley street, Cavendish square, and Kenilworth, Clapham common. *Trans.* 1.

1888 CAUTLEY, EDMUND, M.D., B.C., 15, Upper Brook street. *Trans.* 1.

1871 **Cayley,** WILLIAM, M.D., Consulting Physician to the Middlesex Hospital, Consulting Physician to the London Fever Hospital, and to the North-Eastern Hospital for Children; 27, Wimpole street, Cavendish square. C. 1888. *Referee*, 1886-7, 1899—. *Lib. Com.* 1886-7. *Trans.* 2.

1879 CHAMPNEYS, FRANCIS HENRY, M.D., Physician-Accoucheur and Lecturer on Obstetric Medicine at St. Bartholomew's Hospital; 42, Upper Brook street, Grosvenor square. C. 1898-1900. *Referee*, 1891-8. *Lib. Com.* 1885-98. *Trans.* 8.

Elected

1868 **Cheadle,** WALTER BUTLER, M.D., *Trustee;* Physician to, and Lecturer on Clinical Medicine at, St. Mary's Hospital; Consulting Physician to the Hospital for Sick Children; 19, Portman street, Portman square. S. 1886-8. C. 1890-91. *Sci. Com.* 1889-95. *Bldg. Com.* 1889-92. *Referee,* 1885. *Trans.* 1.

1879 CHEYNE, WILLIAM WATSON, M.B., F.R.S., Surgeon to King's College Hospital, and Professor of Clinical Surgery in King's College, London; 75, Harley street, Cavendish square. C. 1897-9. *Referee,* 1894-7. *Lib. Com.* 1886-8, 1891-6. *Trans.* 1.

1890 CHILDS, CHRISTOPHER, M.D., 10, Manchester square.

1866 **Church,** SIR WILLIAM SELBY, Bart., K.C.B., M.D., *Hon. Treasurer,* President of the Royal College of Physicians of London, Physician to, and Lecturer on Clinical Medicine at, St. Bartholomew's Hospital; 130, Harley street, Cavendish square. C. 1885-6. V.P. 1892-4. T. 1894—. *Referee,* 1874-81. *Ho. Com.* 1898—.

1879 CLARK, ANDREW, Surgeon to, and Lecturer on Surgery at, the Middlesex Hospital; 71, Harley street, Cavendish square.

1882 CLARKE, ERNEST, M.D., B.S., Surgeon to the Central London Ophthalmic Hospital; Ophthalmic Surgeon to the Miller Hospital; 3, Chandos street, Cavendish square.

1890 CLARKE, JAMES JACKSON, M.B., Assistant Surgeon to the North-West London and City Orthopædic Hospitals; 18, Portland Place.

1848 **Clarke,** JOHN, M.D., 48, Carlisle place, Victoria street. C. 1866.

1888 CLARKE, ROBERT HENRY, M.B., 80, Hamlet Gardens, Ravenscourt Park.

Elected

1881 CLARKE, W. BRUCE, M.B., Assistant Surgeon to, and Lecturer on Anatomy at, St. Bartholomew's Hospital; Surgeon to the West London Hospital, 51, Harley street, Cavendish square. C. 1899-1901. *Trans.* 1.

1879 **Clutton,** HENRY HUGH, M.B., M.C., Surgeon to St. Thomas's Hospital; 2, Portland place. C. 1897-9. *Dis. Com.* 1897-8. *Referee,* 1896-7. *Trans.* 3.

1888 COCK, FREDERICK WILLIAM, M.D., 1, Porchester Houses, Porchester square.

1902 COLLIER, JAMES STANSFIELD, M.D., B.Sc., 57A, Wimpole street.

1897 COLMAN, W. S., M.D., Assistant Physician to St. Thomas's Hospital; 9, Wimpole street.

1865 COOPER, SIR ALFRED, Surgeon in Ordinary to H.R.H. the Duke of Saxe-Coburg-Gotha; Consulting Surgeon to the West London Hospital and to St. Mark's Hospital; 9, Henrietta street, Cavendish square.

1898 CORFIELD, W. H., M.D., Professor of Hygiene and Public Health at University College, London; Medical Officer of Health for St. George's, Hanover square; 19, Savile row, and Whindown, Bexhill, Sussex.

1889 COSENS, CHARLES HENRY, 49, Oxford terrace, Hyde Park.

1902 COTTON, HOLLAND JOHN, M.D., C.M., 33, Lowndes street.

1860 **Couper,** JOHN, Consulting Surgeon to the Royal London Ophthalmic Hospital and to the London Hospital; 80, Grosvenor street. C. 1876. *Referee,* 1882-3.

1877 COUPLAND, SIDNEY, M.D., Commissioner in Lunacy; late Physician to, and Lecturer on Medicine at, the Middlesex Hospital; 16, Queen Anne street, Cavendish square. C. 1893-4. *Referee,* 1892-3. *Ho. Com.* 1895-8.

1862 **Cowell,** GEORGE, Consulting Surgeon to the Westminster Hospital and to the Royal Westminster Ophthalmic Hospital; 24, Harrington gardens, South Kensington. C. 1882-3.

Elected

1897 CRAWFURD, RAYMOND H. PAYNE, M.D., 71, Harley street.

1878 CRICHTON-BROWNE, SIR JAMES, M.D., LL.D., F.R.S., Lord Chancellor's Visitor in Lunacy; 61, Carlisle place Mansions, Victoria street.

1874 CRIPPS, WILLIAM HARRISON, Surgeon to St. Bartholomew's Hospital; 2, Stratford place, Oxford street. C. 1890-91. *Trans.* 1.

1882 CROCKER, HENRY RADCLIFFE, M.D., Physician to the Skin Department, University College Hospital; 121, Harley street, Cavendish square. *Trans.* 3.

1898 CROMBIE, ALEXANDER, M.D., 3, Bickenhall Mansions, Gloucester place.

1899 CROSSE, W. H., M.D., 37, Albemarle street, Piccadilly.

1890 CROWLE, THOMAS HENRY RICKARD, 35, St. James's place.

1888 CULLINGWORTH, CHARLES JAMES, M.D., D.C.L., Obstetric Physician and Lecturer on Diseases of Women to St. Thomas's Hospital; 14, Manchester square. *Referee*, 1896—.

1879 CUMBERBATCH, A. ELKIN, M.B., Aural Surgeon to St. Bartholomew's Hospital, and to the National Hospital for the Paralysed and Epileptic; 80, Portland place. *Trans.* 1.

1898 CURRIE, A. STARK, M.D., 81, Queen's road, Finsbury park.

1886 DAKIN, WILLIAM RADFORD, M.D., Obstetric Physician to, and Lecturer in Midwifery at, St. George's Hospital, and Physician to the General Lying-in Hospital; 8, Grosvenor street, Grosvenor square. *Lib. Com.* 1902—.

1872 DALBY, SIR WILLIAM BARTLETT, M.B., Consulting Aural Surgeon to St. George's Hospital; 18, Savile row. C. 1896-7. V.P. 1901-2. *Trans.* 4.

Elected

1891 DALTON, NORMAN, M.D., Physician to King's College Hospital; Professor of Pathological Anatomy in King's College, London; 4, Mansfield street, Cavendish square.

1896 DAUBER, JOHN HENRY, M.B., B.Ch., Assistant Physician to the Hospital for Women, Soho square; 29, Charles street, Berkeley square.

1889 DEAN, HENRY PERCY, M.S., Surgeon to the London Hospital; 69, Harley street, Cavendish square.

1878 DENT, CLINTON THOMAS, *Hon. Secretary*, Surgeon to, and Lecturer on Surgery at, St. George's Hospital; 61, Brook street. C. 1890. S. 1901—. *Bldg. Com.* 1890-2. *Referee*, 1892—1901. *Trans.* 6.

1891 DE SANTI, PHILIP ROBERT WILLIAM, Assistant Surgeon and Aural Surgeon to the Westminster Hospital; 15, Stratford place.

1894 DICKINSON, THOMAS VINCENT, M.D., Physician to the Italian Hospital, Queen square; 33, Sloane street.

1859 **Dickinson,** WILLIAM HOWSHIP, M.D., Consulting Physician to St. George's Hospital, and Consulting Physician to the Hospital for Sick Children; Honorary Fellow of Caius College, Cambridge; 9, Chesterfield street, Mayfair. P. 1896-8. C. 1874-5. V. P. 1887. *Referee*, 1869-73. 1882-6. *Sci. Com.* 1867, 1879, 1889-96. *Trans.* 16.

1891 **Dickinson,** WILLIAM LEE, M.D., Assistant Physician to St. George's Hospital and to the Hospital for Sick Children; 9, Chesterfield street, Mayfair.

1889 DODD, HENRY WORK, Surgeon to the Royal Westminster Ophthalmic Hospital; Ophthalmic Surgeon to the Royal Free Hospital and to the West-End Hospital for Nervous Diseases; 136, Harley street, Cavendish square.

Elected

1888 DONELAN, JAMES, M.B., M.C., Physician to the Italian Hospital, Queen square; 6, Manchester square.

1877 DORAN, ALBAN HENRY GRIFFITHS, Surgeon to the Samaritan Free Hospital; 9, Granville place, Portman square. C. 1893-4. *Lib. Com.* 1891-3, 1899—. *Referee*, 1898—. *Trans.* 3.

1891 DOVE, PERCY W., M.B., 80, Crouch hill.

1896 DOWNES, JOSEPH LOCKHART, M.B., C.M., 269, Romford road.

1893 DRYSDALE, JOHN H., M.B., 11, Devonshire place.

1865 **Duckworth**, SIR DYCE, M.D., LL.D., Physician to, and Lecturer on Clinical Medicine at, St. Bartholomew's Hospital; 11, Grafton street, Bond street. C. 1883-4. V.P. 1902—. *Referee*, 1885-97. *Trans.* 2.

1880 DUNBAR, JAMES JOHN MACWHIRTER, M.D., Hedingham House, Clapham Common.

1884 DUNCAN, WILLIAM, M.D., Obstetric Physician to, and Lecturer on Midwifery at, the Middlesex Hospital; 6, Harley street, Cavendish square.

1887 DUNN, HUGH PERCY, Ophthalmic Surgeon to the West London Hospital; 54, Wimpole street, Cavendish square.

1898 DUNN, L. A., M.S., 51, Devonshire street, Portland place.

1874 DURHAM, FREDERIC, M.B., Senior Surgeon to the North-West London Hospital; 52, Brook street, Grosvenor square.

1894 DURHAM, HERBERT EDWARD, M.B., 52, Brook street, Grosvenor square. *Trans.* 2.

1868 **Eastes**, GEORGE, M.B.Lond., 35, Gloucester terrace, Hyde Park. C. 1892-3.

Elected

1893 Eccles, William McAdam, M.S., Assistant Surgeon to the West London Hospital and to the City of London Truss Society; 124, Harley street.

1891 Eddowes, Alfred, M.D., 28, Wimpole street.

1898 Edkins, J. S., Brambles, Watford road, Northwood.

1898 Edmunds, P. J., M.B., 5, Great Marlborough street, Regent street.

1883 Edmunds, Walter, M.C., 2, Devonshire place, Portland place. *Trans.* 3.

1884 Edwards, Frederick Swinford, Surgeon to the West London Hospital, and to St. Peter's Hospital; Senior Assistant Surgeon to St. Mark's Hospital; 55, Harley street, Cavendish square.

1902 Evans, Arthur, M.S., 53, Queen Anne street, Cavendish square.

1898 Evans, Willmott H., M.D., B.S., B.Sc., Assistant Surgeon and Surgeon in charge of Skin Department, Royal Free Hospital; 2, Upper Wimpole street.

1879 Eve, Frederic S., Surgeon to the London Hospital; Surgeon to the Evelina Hospital for Sick Children; 125, Harley street, Cavendish square. C. 1897-9. *Referee*, 1902—. *Trans.* 4.

1877 Ewart, William, M.D., Physician to St. George's Hospital and to the Belgrave Hospital for Children; 33, Curzon street, Mayfair. C. 1895-7. *Lib. Com.* 1897—. *Sci. Com.* 1889-1902. *Trans.* 2. *Pro.* 1.

1900 Fairbairn, John Shields, M.B., 60, Wimpole street.

1872 Fayrer, Sir Joseph, Bart., K.C.S.I., LL.D., M.D., F.R.S., Surgeon-General; Physician Extraordinary to H.M. the King; late Physician to the Secretary of State for India in Council, and President of the Medical Board at the India Office; 16, Devonshire street, Portland place. C. 1888. *Referee*, 1881-7.

Elected

1898 FENWICK, E. HURRY, Surgeon to the London Hospital and to St. Peter's Hospital; 14, Savile row.

1880 FERRIER, DAVID, M.D., LL.D., F.R.S., Professor of Neuropathology in King's College, London, and Physician to King's College Hospital; Physician to the National Hospital for the Paralysed and Epileptic; 34, Cavendish square. *Referee*, 1891-6. C. 1896-8. *Dis. Com.* 1896—. *Trans.* 2.

1889 FIELD, GEORGE P., Aural Surgeon to, and Lecturer on Aural Surgery at, St. Mary's Hospital; 34, Wimpole street, Cavendish square.

1900 FLEMMING, PERCY, M.D., B.S., Assistant Ophthalmic Surgeon to University College Hospital; Assistant Surgeon to the Royal London Ophthalmic Hospital, City road; 31, Wimpole street.

1891 FLETCHER, HERBERT MORLEY, M.D., Assistant Physician, East London Hospital for Children; 98, Harley street, Cavendish square.

1892 FORSBROOK, WILLIAM HENRY RUSSELL, M.D., 40, Lower Belgrave street, Eaton square.

1896 FOULERTON, ALEXANDER GRANT RUSSELL, Middlesex Hospital. *Trans.* 1.

1883 FOWLER, JAMES KINGSTON, M.D., Physician to, and Lecturer on Medicine at, the Middlesex Hospital; Physician to the Hospital for Consumption, Brompton, 35, Clarges street, Piccadilly. C. 1902—. *Trans.* 1.

1880 FOX, THOMAS COLCOTT, B.A., M.B., Physician for Diseases of the Skin to the Westminster Hospital, and Physician to the Skin Department of the Paddington Green Hospital for Children; 14, Harley street, Cavendish square. *Trans.* 1.

Elected

1871 FRANK, PHILIP, M.D., 3, Elvaston place, South Kensington.

1902 FRENCH, HERBERT, M.B., 26, St. Thomas's street.

1896 FREYER, P. J., M.D., M.Ch., Surgeon to St. Peter's Hospital; 46, Harley street, Cavendish square. *Trans.* 1.

1898 FRIPP, A. DOWNING, C.B., M.V.O., M.S., Honorary Surgeon in Ordinary to H.M. the King; Assistant Surgeon to Guy's Hospital; 19, Portland place.

1898 FROST, WILLIAM ADAMS, Ophthalmic Surgeon to St. George's Hospital, and Surgeon to Royal Westminster Ophthalmic Hospital; 30, Cavendish square.

1884 FULLER, CHARLES CHINNER, 10, St. Andrew's place, Regent's Park.

1883 FULLER, HENRY ROXBURGH, M.D., 45, Curzon street, Mayfair.

1894 FURNIVALL, PERCY, Assistant Surgeon, London Hospital; Assistant Surgeon, St. Mark's Hospital; 28, Weymouth street, Portland place.

1899 FÜRTH, KARL, M.D., 94, Harley Street.

1874 **Galabin**, ALFRED LEWIS, M.D., Obstetric Physician to, and Lecturer on Midwifery and the Diseases of Women at, Guy's Hospital; 49, Wimpole st., Cavendish square. C. 1892. *Referee*, 1882-91, 1896—. *Lib. Com.* 1883-4. *Trans.* 2.

1895 GALLOWAY, JAMES, M.D., Physician, Skin Department, and Joint Lecturer on Practical Medicine, Charing Cross Hospital; 54, Harley street, Cavendish square.

1883 GALTON, JOHN CHARLES, M.A., F.L.S., 10, Upper Cheyne row, Chelsea.

1865 **Gant**, FREDERICK JAMES, Consulting Surgeon to the Royal Free Hospital; 16, Connaught square, Hyde Park. C. 1880-81. V.P. 1897-9. *Referee*, 1886-97. *Lib. Com.* 1882-5. *Trans.* 3.

Elected

1854 **Garrod,** Sir Alfred Baring, M.D., F.R.S., Physician Extraordinary to Her late Majesty Queen Victoria; Consulting Physician to King's College Hospital; 10, Harley street, Cavendish square. C. 1867. V.P. 1880-81. *Referee*, 1855-65. *Trans.* 9.

1886 Garrod, Archibald Edward, M.D., Medical Registrar and Demonstrator of Morbid Anatomy, St. Bartholomew's Hospital; Physician to the Hospital for Sick Children, Great Ormond street; 9, Chandos street, Cavendish square. C. 1902—. *Sci. Com.* 1889-1902. *Lib. Com.* 1896-1902. *Trans.* 7.

1887 Gay, John, 119, Upper Richmond road, Putney.

1866 **Gee,** Samuel Jones, M.D., *Chairman of Trustees for Debenture-holders;* Honorary Physician to H.R.H. the Prince of Wales; Physician to St. Bartholomew's Hospital; 31, Upper Brook street, Grosvenor square. C. 1883-4. L. (June) 1887-99. V.P. 1899-1900. *Sci. Com.* 1879. *Bldg. Com.* 1889-92. *Referee*, 1885-7, 1900—. *Lib. Com.* 1871-6. *Ho. Com.* 1898-1900. *Trans.* 1.

1898 Gibbes, Cuthbert Chapman, M.D., 89, Harley street.

1880 Gibbons, Robert Alexander, M.D., Physician to the Grosvenor Hospital for Women and Children; 29, Cadogan place. C. 1896-7. *Trans.* 1.

1893 Giles, Arthur Edward, M.D., B.Sc., Assistant Surgeon, Chelsea Hospital for Women; 10, Upper Wimpole street.

1894 Gill, Richard, 72, Wimpole street.

1877 Godlee, Rickman John, M.S., *Hon. Librarian;* Honorary Surgeon-in-Ordinary to H.M. the King; Surgeon to University College Hospital, and Professor of Clinical Surgery in University College, London; Surgeon to the Hospital for Consumption, Brompton; 19, Wimpole street, Cavendish square. S. 1892-4. L. 1895—. *Referee*, 1886-91. *Ho. Com.* 1898—. *Trans.* 11.

Elected

1870 **Godson,** CLEMENT, M.D., Consulting Physician to the City of London Lying-in Hospital; 82, Brook street, Grosvenor square.

1886 GOLDING-BIRD, CUTHBERT HILTON, M.B., Surgeon to, and Lecturer on Clinical Surgery at, Guy's Hospital; 12, Queen Anne street, Cavendish square. *Trans.* 1.

1897 GOODBODY, F. W., M.D., 6, Chandos street, Cavendish square.

1896 GOODALL, EDWARD WILBERFORCE, M.D., B.S., Eastern Hospital, Homerton.

1883 GOODHART, JAMES FREDERIC, M.D., Physician to Guy's Hospital; Consulting Physician to the Evelina Hospital for Sick Children; 25, Portland place. *Referee*, 1900—. *Lib. Com.* 1893-6.

1889 GOODSALL, DAVID HENRY, Surgeon to the Metropolitan Hospital; Surgeon to St. Mark's Hospital; 17, Devonshire place, Upper Wimpole street.

1895 GOSSAGE, ALFRED MILNE, M.B., 54, Upper Berkeley street.

1877 GOULD, ALFRED PEARCE, M.S., Surgeon to, and Lecturer on Surgical Pathology at, the Middlesex Hospital; 10, Queen Anne street, Cavendish square. C. 1892-3. S. 1898—1901. *Referee*, 1895-8. *Ho. Com.* 1892-8. *Lib. Com.* 1891. *Trans.* 3.

1891 GOW, WILLIAM J., M.D., Assistant Obstetric Physician to St. Mary's Hospital; Obstetric Physician to the Royal Hospital for Women and Children; Physician to Out-Patients, Queen Charlotte's Lying-in Hospital; 27, Weymouth street, Portland place.

1873 **Gowers,** SIR WILLIAM RICHARD, M.D., F.R.S., Consulting Physician to University College Hospital; Physician to the National Hospital for the Paralysed and Epileptic; 50, Queen Anne street, Cavendish square. C. 1891. *Referee*, 1888-90. *Lib. Com.* 1884-6. *Trans.* 7.

1892 GRANT, J. DUNDAS, M.A., M.D., 18, Cavendish square.

Elected

1868 GREEN, T. HENRY, M.D., Physician to the Charing Cross Hospital, and to the Hospital for Consumption, Brompton; 74, Wimpole street, Cavendish square. C. 1886. *Referee*, 1882-5.

1885 GRIFFITH, WALTER SPENCER ANDERSON, M.D., Assistant Physician-Accoucheur, St. Bartholomew's Hospital; Physician to Queen Charlotte's Lying-in Hospital; 96, Harley street, Cavendish square. *Referee*, 1902—.

1889 GUBB, ALFRED S., M.D., 29, Gower street.

1883 GUNN, ROBERT MARCUS, M.B., Surgeon to the Royal London Ophthalmic Hospital, Moorfields; Ophthalmic Surgeon to the National Hospital for the Paralysed and Epileptic; 54, Queen Anne street, Cavendish square.

1890 GUTHRIE, LEONARD GEORGE, M.D., B.Ch., Physician to the Regent's Park Hospital for Epilepsy and Paralysis; Assistant Physician to the North-West London Hospital; Assistant Physician to the Children's Hospital, Paddington Green; 15, Upper Berkeley street, Portman square.

1886 HABERSHON, SAMUEL HERBERT, M.D., Assistant Physician to the Hospital for Consumption, Brompton; 88, Harley street, Cavendish square.

1885 HAIG, ALEXANDER, M.D., Physician to the Metropolitan Hospital, and to the Royal Hospital for Children and Women; 7, Brook street, Grosvenor square. *Trans.* 6.

1890 HALE, CHARLES DOUGLAS BOWDICH, M.D., 3, Sussex place, Hyde Park.

1881 **Hall**, FRANCIS DE HAVILLAND, M.D., Physician to, and Joint Lecturer on Medicine at, the Westminster Hospital; 47, Wimpole street, Cavendish square. C. 1901. *Referee*, 1893-7.

Elected

1891 HAMER, WILLIAM HEATON, M.D., 1A, Bramshill gardens, Dartmouth park hill, Highgate.

1889 HANDFIELD-JONES, MONTAGU, M.D., Obstetric Physician to, and Lecturer on Midwifery and Diseases of Women at, St. Mary's Hospital; Physician to the British Lying-in Hospital; 35, Cavendish square.

1893 HARLEY, VAUGHAN, M.D., 25, Harley street, Cavendish square.

1901 HARMER, WILLIAM DOUGLAS, M.B., 45, Weymouth street.

1892 HAROLD, JOHN, M.B., 91, Harley street, Cavendish square.

1880 HARRIS, VINCENT DORMER, M.D., Physician to the City of London Hospital for Diseases of the Chest, Victoria Park; 22, Queen Anne street, Cavendish square. *Referee*, 1899—.

1870 HARRISON, REGINALD, Surgeon to St. Peter's Hospital; 6, Lower Berkeley street, Portman square. C. 1894-5. V.-P. 1898-1900. *Trans.* 4.

1870 **Haward**, J. WARRINGTON, *Hon. Treasurer;* Consulting Surgeon to, and Lecturer on Clinical Surgery at, St. George's Hospital; 57, Green street, Grosvenor Square. C.1885. S. 1888-91. V.P. 1894-5. T. (June) 1895—. *Lib. Com.* 1881-4. *Sci. Com.* 1889-91. *Bldg. Com.* (*Sec.*) 1889-92. *Ho. Com.* 1892—. *Trans.* 3.

1891 HAWKINS, HERBERT PENNELL, M.D., B.Ch., Physician to St. Thomas's Hospital; 56, Portland place.

1875 HAYES, THOMAS CRAWFORD, M.D., Physician-Accoucheur and Physician for Diseases of Women and Children to King's College Hospital, and Professor of Midwifery in King's College; Physician for Diseases of Women to the Royal Free Hospital; 17, Clarges street, Piccadilly.

1891 HAYWARD, JOHN ARTHUR, M.D., 17, Lingfield road, Wimbledon. *Pro.* 1.

Elected

1865 **Heath,** CHRISTOPHER, Emeritus Professor of Clinical Surgery in University College, London; and Consulting Surgeon to University College Hospital; 36, Cavendish square. C. 1880. V.P. 1889. *Lib. Com.* 1870-3. *Trans.* 4.

1895 HENDERSON, EDWARD ERSKINE, B.A., M.B., B.C., 20, Queen Anne street, Cavendish square.

1901 HENRY, JOHN PATRICK, M.D., B.Ch., Ophthalmic Surgeon to the Italian Hospital, Queen square; Oculist to the London School Board; 41, Welbeck street, Cavendish square.

1882 HENSLEY, PHILIP JOHN, M.D., Physician to, and Lecturer on Forensic Medicine at, St. Bartholomew's Hospital; 4, Henrietta street, Cavendish square. *Referee*, 1897—.

1877 HERMAN, GEORGE ERNEST, M.B., Obstetric Physician to, and Lecturer on Midwifery at, the London Hospital; 20, Harley street, Cavendish square. C. 1900-2. *Referee*, 1892-1900. *Lib. Com.* 1898-1900. *Trans.* 1.

1900 HERN, WILLIAM, 7, Stratford place.

1877 HERON, GEORGE ALLAN, M.D., Physician to the City of London Hospital for Diseases of the Chest, Victoria Park; 57, Harley street, Cavendish square.

1891 HERRING, HERBERT T., M.B., B.S., 50, Harley street, Cavendish square.

1883 HERRINGHAM, WILMOT PARKER, M.D., Assistant Physician, St. Bartholomew's Hospital; 40, Wimpole street, Cavendish square. *Lib. Com.* 1902—. *Trans.* 2.

1893 HERSCHELL, GEORGE, M.D., 36, Harley street, Cavendish square.

1887 HEWITT, FREDERIC WILLIAM, M.V.O., M.D., Honorary Anæsthetist to H.M. the King; Anæsthetist to, and Instructor in Anæsthetics at, the London Hospital; Anæsthetist at the Dental Hospital of London; 14, Queen Anne street, Cavendish square. *Trans.* 3.

Elected

1873 HIGGENS, CHARLES, Ophthalmic Surgeon to, and Lecturer on Ophthalmic Surgery at, Guy's Hospital; 52, Brook street, Grosvenor square. C. 1894-5. *Trans.* 2.

1890 HILL, G. WILLIAM, M.D., B.Sc., 26, Weymouth street, Portland place.

1899 HILLIER, ALFRED P., M.D., 30, Wimpole street.

1856 **Holmes**, TIMOTHY, M.A., Consulting Surgeon to St. George's Hospital; Corresponding Member of the Société de Chirurgie, Paris; 6, Sussex place, Hyde Park. C. 1869-70. L. 1873-7. S. 1878-80. V.P. 1881-2. T. 1885-7. P. 1890-92. *Bldg.Com.* (*Chairman*) 1889-92. *Referee*, 1866-8, 1872, 1883-4. *Sci. Com.* 1867. *Lib. Com.* 1863-5, 1892-5. *Ho. Com.* 1892-8. *Trans.* 8.

1878 HOOD, DONALD WILLIAM CHARLES, C.V.O., M.D., Senior Physician to the West London Hospital; Examining Physician for King's Messengers, Foreign Office; 43, Green street, Park lane.

1898 HORDER, THOMAS J., M.D., 141, Harley street.

1883 HORSLEY, SIR VICTOR ALEXANDER HADEN, F.R.S., Surgeon to University College Hospital, Surgeon to the National Hospital for the Paralysed and Epileptic; 25, Cavendish square. *Referee*, 1897—. *Trans.* 1.

1896 HORTON-SMITH, PERCIVAL, M.D., 19, Devonshire street, Portland place. *Sci. Com.* 1897-1902. *Trans.* 1.

1892 HOWARD, R. J. BLISS, M.D., 31, Queen Anne street, Cavendish square.

1874 HOWSE, SIR HENRY GREENWAY, M.S., Surgeon to, and Lecturer on Surgery at, Guy's Hospital; Consulting Surgeon to the Evelina Hospital for Sick Children; 59, Brook street, Grosvenor square. C. 1890. V.P. 1899-1901. *Sci. Com.* 1879. *Referee*, 1887-9. *Trans.* 3.

Elected

1889 HUNTER, WILLIAM, M.D., Senior Assistant Physician to the London Fever Hospital; Curator and Pathologist, Charing Cross Hospital; 103, Harley street.

1856 **Hutchinson,** JONATHAN, F.R.S., Consulting Surgeon to, and Emeritus Professor of Surgery at, the London Hospital; Consulting Surgeon to the Royal London Ophthalmic Hospital, Moorfields, and Senior Surgeon to the Hospital for Diseases of the Skin; 15, Cavendish square. C. 1870. V.P. 1882. P. 1894-5. *Referee,* 1876-81, 1883-94. *Lib. Com.* 1864-5. *Trans.* 15. *Pro.* 2.

1888 HUTCHINSON, JONATHAN, Jun., Surgeon to the London Hospital; 1, Park crescent. *Trans.* 3.

1897 HUTCHISON, ROBERT, M.D., 22, Queen Anne street, Cavendish square.

1871 JACKSON, J. HUGHLINGS, M.D., LL.D., F.R.S., Consulting Physician to the London Hospital; Physician to the National Hospital for the Paralysed and Epileptic; 3, Manchester square. C. 1889.

1883 JACOBSON, WALTER HAMILTON ACLAND, M.Ch.Oxon., Assistant Surgeon to Guy's Hospital; Surgeon to the Royal Hospital for Children and Women; 66, Great Cumberland place, Hyde Park. C. 1902—. *Referee,* 1895-1902. *Lib. Com.* 1896-1902. *Trans.* 2.

1897 JENNER, LOUIS, M.B., 4A, Bloomsbury square.

1883 JESSOP, WALTER H. H., M.B., Ophthalmic Surgeon to St. Bartholomew's Hospital; 73, Harley street. *Referee,* 1901.

1881 JOHNSON, GEORGE LINDSAY, M.D., Cortina, Netherhall gardens, South Hampstead, and 36, Finsbury pavement.

1889 JOHNSON, RAYMOND, M.B., B.S., Assistant Surgeon to University College Hospital; Surgeon to the Victoria Hospital for Children; 11, Wimpole street, Cavendish square. *Trans.* 1.

Elected

1884 JOHNSTON, JAMES, M.D., 53, Prince's square, Bayswater.

1899 **Jones**, GEORGE, M.B., 8, Church terrace, Lee.

1887 JONES, HENRY LEWIS, M.D., Medical Officer in charge of Electrical Department at St. Bartholomew's Hospital; 61, Wimpole street, Cavendish square.

1896 JONES, L. VERNON, B.A., M.D., B.Ch., 7, Arlington street, St. James's.

1881 JULER, HENRY EDWARD, Ophthalmic Surgeon to St. Mary's Hospital; Surgeon to the Royal Westminster Ophthalmic Hospital; Consulting Ophthalmic Surgeon to the London Lock Hospital; 23, Cavendish square. C. 1901—. *Ho. Com.* 1902—.

1898 KEEP, A. CORRIE, M.D., C.M., Surgeon to out-patients Samaritan Free Hospital for Women and Children; 14, Gloucester place, Portman square.

1882 KEETLEY, CHARLES R. B., Senior Surgeon to the West London Hospital; 56, Grosvenor street, Grosvenor square. C. 1901—. *Trans.* 1.

1898 KELLOCK, THOMAS HERBERT, M.D., B.C., Assistant Surgeon to Middlesex Hospital and to the Hospital for Sick Children; 8, Queen Anne street, Cavendish square.

1901 KELYNACK, T. N., M.D., 53, Harley street.

1902 KERR, JAMES, M.D., D.P.H., School Board for London, Victoria Embankment.

1857 **Kiallmark**, HENRY WALTER, 5, Pembridge gardens. C. 1890–91.

1881 KIDD, PERCY, M.D., Physician to the Hospital for Consumption, Brompton; Physician to the London Hospital; 60, Brook street, Grosvenor square. C. 1900-2. *Trans.* 4.

Elected

1851 **Kingdon**, JOHN ABERNETHY, Consulting Surgeon to the Bank of England, Threadneedle street. C. 1866-7. V.P. 1872-3. *Sci. Com.* 1867. *Trans.* 1.

1900 LAKE, RICHARD, 19, Harley street. *Trans.* 1.

1896 LANE, JAMES ERNEST, Surgeon to Out-patients, St. Mary's Hospital; 46, Queen Anne Street, Cavendish square.

1884 LANE, WILLIAM ARBUTHNOT, M.S., Surgeon to Guy's Hospital and to the Hospital for Sick Children, 21, Cavendish square. *Trans.* 4.

1882 LANG, WILLIAM, Ophthalmic Surgeon to, and Lecturer on Ophthalmic Surgery at, the Middlesex Hospital; Surgeon to the Royal London Ophthalmic Hospital, Moorfields; 22, Cavendish square.

1894 LANGDON-DOWN, REGINALD LANGDON, M.B., B.C., 47, Welbeck street.

1865 **Langton**, JOHN, Surgeon to, and Lecturer on Clinical Surgery at, St. Bartholomew's Hospital; Surgeon to the City of London Truss Society; 62, Harley street, Cavendish square. C. 1881–2. V.P. 1895-7, *Referee*, 1885-95. *Lib. Com.* 1879-80, 1888-95, *Trans.* 2.

1898 LATHAM, A. C., M.D., 44, Brook street, Grosvenor square.

1890 LAW, EDWARD, M.D., C.M., 8, Wimpole street, Cavendish square.

1898 LAWFORD, J. B., Ophthalmic Surgeon and Lecturer on Ophthalmology, St. Thomas's Hospital; Surgeon to Royal London Ophthalmic Hospital; 99, Harley street.

1888 LAWRENCE, LAURIE ASHER, 9, Upper Wimpole street.

1890 LAWRIE, EDWARD, M.B., Surgeon Lieutenant-Colonel, Indian Medical Department; late Residency Surgeon, Hyderabad, Deccan; Harley Lodge, 115A, Harley street.

Elected

1893 LAWSON, ARNOLD, Ophthalmic Surgeon to the Children's Hospital, Paddington Green; 12, Harley street, Cavendish square.

1884 LAWSON, GEORGE, Consulting Surgeon to the Royal London Ophthalmic Hospital; Consulting Surgeon to the Middlesex Hospital; 12, Harley street, Cavendish square.

1900 LEAF, CECIL HUNTINGTON, M.A., M.B.; 75, Wimpole street, Cavendish square.

1896 LEE, WILLIAM EDWARD, M.D., 36, Finsbury pavement.

1895 LEES, DAVID BRIDGE, M.D., Physician to, and Lecturer on Medicine at, St. Mary's Hospital, and Physician to the Hospital for Sick Children; 22, Weymouth street, Portland place. *Trans.* 2.

1899 LEGGE, THOMAS MORISON, M.D., 2, Mitre court buildings, Temple.

1900 LENDON, EDWIN HARDING, M.D., 162, Holland park avenue.

1895 LESLIE, ROBERT MURRAY, M.B., Assistant Physician to Royal Hospital for Diseases of the Chest; 26, Harley street, Cavendish square.

1897 LEVY, ALFRED G., M.D., 41, Devonshire street, Portland place.

1886 LEWERS, ARTHUR HAMILTON NICHOLSON, M.D., Obstetric Physician to the London Hospital; 72, Harley street, Cavendish square. *Trans.* 1.

1878 LISTER, RIGHT HON. LORD, P.C., O.M., D.C.L., LL.D., F.R.S., Sergeant-Surgeon in Ordinary to H.M. the King; Emeritus Professor of Clinical Surgery in King's College, London; and Consulting Surgeon to King's College Hospital; 12, Park crescent, Regent's Park. C. 1892.

1891 LITTLE, ERNEST MUIRHEAD, Surgeon to the National Orthopædic Hospital; 40, Seymour street, Portman square.

Elected

1889 LITTLE, JOHN FLETCHER, M.B., 32, Harley street, Cavendish square.

1881 LOCKWOOD, CHARLES BARRETT, Surgeon to the Great Northern Central Hospital; Assistant Surgeon to, and Lecturer on Surgical and Descriptive Anatomy at, St. Bartholomew's Hospital; 19, Upper Berkeley street, Portman square. C. 1901—. *Trans.* 4.

1897 LOW, HAROLD, 10, Evelyn gardens.

1881 LUCAS, RICHARD CLEMENT, B.S., M.B., Surgeon to, and Lecturer on Surgery, late Lecturer on Anatomy at, Guy's Hospital; Consulting Surgeon to the Evelina Hospital for Sick Children; 50, Wimpole street, Cavendish square. C. 1900-2. *Ho. Com.* 1901-2. *Trans.* 3.

1888 LUFF, ARTHUR PEARSON, M.D., B.Sc., Physician to Out-patients and Lecturer on Medical Jurisprudence at St. Mary's Hospital; 9, Queen Anne street, Cavendish square. *Trans.* 1.

1887 LUSH, PERCY J. F., M.B., 4, Maresfield gardens, Hampstead.

1898 LYSTER, C. R. C., Bolingbroke Hospital, Wandsworth common.

1873 **MacCarthy**, JEREMIAH, M.A., Consulting Surgeon to the London Hospital, late Lecturer on Surgery at the London Hospital Medical College; 1, Cambridge place, Victoria road, Kensington. C. 1886-7. *Lib. Com.* 1882-5. *Referee*, 1890—.

1899 MACDONALD, GREVILLE, M.D., 85, Harley street.

1898 MCFADYEAN, JOHN, The Royal Veterinary College, Camden Town.

1894 MACFADYEN, ALLAN, M.D., C.M., Jenner Institute of Preventive Medicine, Chelsea bridge.

Elected

1880 McHARDY, MALCOLM MACDONALD, Ophthalmic Surgeon to King's College Hospital, and Professor of Ophthalmic Surgery in King's College, London; Senior Surgeon to the Royal Eye Hospital, Southwark; 5, Savile row.

1873 MACKELLAR, ALEXANDER OBERLIN, M.Ch., Surgeon to St. Thomas's Hospital; Surgeon-in-Chief to the Metropolitan Police Force; 79, Wimpole street, Cavendish square.

1881 MACKENZIE, STEPHEN, M.D., Physician to the London Hospital; Physician to the Royal London Ophthalmic Hospital; 18, Cavendish square. C. 1899-1900. *Referee*, 1890-9. *Trans.* 1.

1879 MACLAGAN, THOMAS JOHN, M.D., Physician-in-Ordinary to their R.H. the Prince and Princess Christian of Schleswig-Holstein; 9, Cadogan place, Belgrave square.

1881 MACREADY, JONATHAN FORSTER CHRISTIAN HORACE, Surgeon to the Great Northern Hospital; 42, Devonshire street.

1880 MADDICK, EDMUND DISTIN, 31, Cavendish square.

1886 MAGUIRE, ROBERT, M.D., Physician to Out-patients and Joint Lecturer on Pathology at St. Mary's Hospital; Physician to the Hospital for Consumption, Brompton; 4, Seymour street, Portman square. *Sci. Com.* 1889-1902.

1880 MAKINS, GEORGE HENRY, C.B., Surgeon to St. Thomas's Hospital; Consulting Surgeon to the Evelina Hospital for Children; 47, Charles street, Berkeley square. C. 1899-1900. *Referee*, 1898-9, 1902—. *Trans.* 2.

1885 MALCOLM, JOHN DAVID, M.B., C.M., Surgeon to the Samaritan Free Hospital; 13, Portman street, Portman square. *Trans.* 2.

Elected

1890 MANSON, PATRICK, C.M.G., M.D., C.M., LL.D., F.R.S., Physician to the Seamen's Hospital, Albert Docks; Lecturer on Tropical Medicine at St. George's Hospital; 21, Queen Anne street, Cavendish square.

1867 MARSH, F. HOWARD, Surgeon to, and Lecturer on Surgery at, St. Bartholomew's Hospital; 30, Bruton street, Berkeley square. C. 1882-3, 1889. S. 1885-7. V.P. 1891-3. *Lib. Com.* 1880-1. *Trans.* 4.

1891 MARTIN, HENRY CHARRINGTON, M.D., 27, Oxford square.

1884 MARTIN, SIDNEY HARRIS COX, M.D., F.R.S., Assistant Physician to University College Hospital, and to the Hospital for Consumption, Brompton; Professor of Pathology, University College, London; 10, Mansfield street, Portland place.

1892 MASTERS, JOHN ALFRED, M.D., 31, Albert gate, Hyde Park.

1891 MAY, WILLIAM PAGE, M.D., B.Sc., 9, Manchester square.

1880 MEREDITH, WILLIAM APPLETON, M.B., C.M., Surgeon to the Samaritan Free Hospital for Women and Children; 21, Manchester square. C. 1897-9. *Ho. Com.* 1898-9. *Trans.* 1.

1894 MICHELS, ERNST, M.D., Surgeon to the German Hospital; 48, Finsbury square. *Trans.* 2.

1893 MILEY, MILES, M.B., 21, Belsize avenue, Hampstead.

1887 MIVART, FREDERICK ST. GEORGE, M.D., Local Government Board Inspector; 13, Stafford terrace, Kensington.

1891 MOLINE, PAUL, M.B., 42, Walton street, Chelsea.

1873 MOORE, NORMAN, M.D., *Hon. Librarian*, Assistant Physician and Lecturer on Medicine to St. Bartholomew's Hospital; 94, Gloucester place, Portman square. C. 1891-2. L. 1899—. S. 1896-9. *Referee*, 1886-90. *Ho. Com.* 1898—. *Sci. Com.* 1889-1902.

Elected

1878 MORGAN, JOHN HAMMOND, C.V.O., M.A., Surgeon to the Charing Cross Hospital, and to the Hospital for Sick Children, Great Ormond street; 68, Grosvenor street. C. 1895-7. *Dis. Com.* 1896-7. *Referee*, 1901—. *Trans.* 2.

1894 MORISON, ALEXANDER, M.D., 14, Upper Berkeley street.

1874 **Morris**, HENRY, M.A., Surgeon to the Middlesex Hospital; 8, Cavendish square. C. 1888-9. V.P. 1900-2. *Referee*, 1882-7. *Lib. Com.* 1895-6. *Trans.* 10.

1879 MORRIS, MALCOLM ALEXANDER, Surgeon to the Skin Department of, and Lecturer on Dermatology at, St. Mary's Hospital; 8, Harley street, Cavendish square. *Sci. Com.* 1889-1902. *Trans.* 1.

1898 MORRISON, JAMES, M.D., 11, Brook street, Grosvenor square.

1885 MOTT, FREDERICK WALKER, M.D., F.R.S., Assistant Physician, Charing Cross Hospital; Pathologist to the London County Council; 25, Nottingham place. *Referee*, 1900—. *Sci. Com.* 1899—. *Trans.* 1.

1902 MUMMERY, JOHN PERCY LOCKHART, B.A., 10, Cavendish place.

1899 MUNDY, HERBERT, St. Bartholomew's Hospital.

1900 MURPHY, WILLIAM REID, D.S.O., Lieutenant-Colonel I.M.S.; East India and Colonial Club, 16, St. James's street.

1896 **Murphy**, JAMES KEOGH, M.B., St. Bartholomew's Hospital.

1888 **Murray**, HUBERT MONTAGUE, M.D., Physician to Out-patients, and Joint Lecturer on Medicine at, the Charing Cross Hospital; Physician to the Victoria Hospital for Children; 25, Manchester square.

Elected

1898 MURRAY, JOHN, Assistant Surgeon to the Middlesex Hospital and to the Paddington Green Children's Hospital; 110, Harley street.

1880 MURRELL, WILLIAM, M.D., Physician to, and Lecturer on Materia Medica, Pharmacology, and Therapeutics at, the Westminster Hospital; 17, Welbeck street, Cavendish square. *Sci. Com.* 1889-1902. *Trans.* 1.

1863 **Myers**, ARTHUR BOWEN RICHARDS, late Brigade-Surgeon, Brigade of Guards; 43, Gloucester street, Warwick square. C. 1878-9. *Lib. Com.* 1877.

1864 **Nunn**, THOMAS WILLIAM, Consulting Surgeon to the Middlesex Hospital; 27, York terrace, York gate.

1880 OGILVIE, GEORGE, M.B., B.Sc., Physician to the Hospital for Epilepsy and Paralysis, Regent's Park; 22, Welbeck street, Cavendish square. *Trans.* 1.

1891 OGLE, CYRIL, M.A., M.B., Assistant Physician to St. George's Hospital; 96, Gloucester place, Portman square.

1858 **Ogle**, JOHN WILLIAM, M.D., Consulting Physician to St. George's Hospital; 96, Gloucester place, Portman square. C. 1873. V.P. 1886. *Referee*, 1864-72. *Trans.* 4.

1860 OGLE, WILLIAM, M.D., late Superintendent of Statistics in the Registrar-General's Department, Somerset House; 10, Gordon street, Gordon square. S. 1868-70. C. 1876-7. V.P. 1887. *Lib. Com.* 1871-5. *Trans.* 5.

1892 OPENSHAW, T. HORROCKS, M.B., M.S., Surgeon to, and Lecturer on Anatomy at, the London Hospital; 16, Wimpole street, Cavendish square.

1877 ORMEROD, JOSEPH ARDERNE, M.D., Assistant Physician to St. Bartholomew's Hospital; Physician to the National Hospital for the Paralysed and Epileptic, Queen square; 25, Upper Wimpole street. C. 1897. *Lib. Com.* 1896-7. *Trans.* 1.

Elected

1875 OSBORN, SAMUEL, 1A, Devonshire street, Portland place, and Maisonnette, Datchet, Bucks.

1879 OWEN, EDMUND, M.B., Senior Surgeon to, and Lecturer on Clinical Surgery at, St. Mary's Hospital; Senior Surgeon to the Hospital for Sick Children, Great Ormond street; 64, Great Cumberland place, Hyde park. C. 1896-7. *Trans.* 4.

1882 OWEN, SIR ISAMBARD, M.D., Deputy-Chancellor of the University of Wales; Physician to, and Lecturer on Forensic Medicine at, St. George's Hospital: 40, Curzon street, Mayfair. C. 1902—. *Bldg. Com.* 1889-92. *Referee*, 1893, 1895-1902.

1892 PAGE, H. MARMADUKE, 14. Grenville place, South Kensington.

1874 PAGE, HERBERT WILLIAM, M.A., M.C., Surgeon to, and Joint Lecturer on Surgery at, St. Mary's Hospital; 146, Harley street, Cavendish square. C. 1890-91. *Referee*, 1884-9. *Lib. Com.* 1886-8. *Trans.* 4.

1886 PAGET, STEPHEN, Surgeon to the West London Hospital; Surgeon to the Throat and Ear Department of the Middlesex Hospital; 70, Harley street. *Lib. Com.* 1902—.

1895 PARKER, CHARLES ARTHUR, 141, Harley street, Cavendish square.

1889 PARSONS, J. INGLIS, M.D., Physician to the Chelsea Hospital for Women; 3, Queen street, Mayfair.

1883 PASTEUR, WILLIAM, M.D., Physician to the Middlesex Hospital; Consulting Physician to the North-Eastern Hospital for Children; 4, Chandos street, Cavendish square.

1901 PATERSON, HERBERT JOHN, 9, Upper Wimpole street.

1891 PATERSON, WILLIAM BROMFIELD, 7A, Manchester square.

Elected

1891 PATON, EDWARD PERCY, M.D., M.S., 84, Park street, Grosvenor square.

1865 **Pavy,** FREDERICK WILLIAM, M.D., LL.D., F.R.S., Consulting Physician to Guy's Hospital; 35, Grosvenor street. P. 1900-2. C. 1883-4. V.P. 1893-4. *Referee*, 1871-82. *Trans.* 1.

1869 PAYNE, JOSEPH FRANK, M.D., Physician to, and Lecturer on Medicine at, St. Thomas's Hospital; 78, Wimpole street, Cavendish square. C. 1887. *Referee*, 1890—. *Sci. Com.* 1879. *Lib. Com.* 1878-85, 1889—.

1894 PEGLER, L. HEMINGTON, M.D., 2, Henrietta street, Cavendish square.

1898 PENDLEBURY, HERBERT STRINGFELLOW, M.B., B.C., 44, Brook street, Grosvenor square.

1887 PENROSE, FRANCIS GEORGE, M.D., Physician to St. George's Hospital and to the Hospital for Sick Children, Great Ormond street; 84, Wimpole street, Cavendish square. *Sci. Com.* 1889-1902.

1890 PERRY, EDWIN COOPER, M.D., Physician to, and Demonstrator of Pathology at, Guy's Hospital; The Superintendent's House, Guy's Hospital.

1895 PHEAR, ARTHUR G., M.D., Assistant Physician and Pathologist to the Metropolitan Hospital; 47, Weymouth street, Portland place. *Trans.* 1.

1883 PHILLIPS, CHARLES DOUGLAS F., M.D., LL.D., 10, Henrietta street, Cavendish square.

1884 PHILLIPS, GEORGE RICHARD TURNER, J.P., 28, Palace Court, Bayswater hill.

Elected

1889 PHILLIPS, SIDNEY, M.D., Physician and Lecturer on Medicine at St. Mary's Hospital; Senior Physician to the London Fever Hospital, and to the Lock Hospital; 62, Upper Berkeley street, Portman square. *Trans.* 1.

1867 **Pick**, THOMAS PICKERING, Consulting Surgeon to St. George's Hospital; 18, Portman street, Portman square. C. 1884-5. V.P. 1893-4. *Referee*, 1882-3, 1891-93. *Sci. Com.* 1870, 1889—. *Lib. Com.* 1879-81.

1884 PITT, GEORGE NEWTON, M.D., *Hon. Secretary*, Physician to, and Pathologist at, Guy's Hospital; 15, Portland place. S. 1902—. *Referee*, 1897-1902. *Trans.* 1.

1889 PITTS, BERNARD, M.A., M.C., Surgeon to St. Thomas's Hospital and Lecturer on Surgery; Surgeon to the Hospital for Sick Children, Great Ormond street; 109, Harley street, Cavendish square. *Referee*, 1897—.

1899 PLAYFAIR, ERNEST, M.B., 57, Gloucester terrace, Hyde Park.

1901 PLIMMER, HARRY GEORGE, 28, St. John's Wood road.

1885 POLAND, JOHN, Surgeon to the City Orthopædic Hospital and Miller Hospital, Greenwich; 2, Mansfield street, Cavendish square.

1884 POLLARD, BILTON, B.S., Surgeon to University College Hospital; Consulting Surgeon to the North-Eastern Hospital for Children; 24, Harley street, Cavendish square. *Trans.* 1.

1865 **Pollock**, JAMES EDWARD, M.D., Consulting Physician to the Hospital for Consumption, Brompton; 37, Collingham place. C. 1882-3. V.P. 1896-7. *Referee*, 1872-81.

1894 POLLOCK, WILLIAM RIVERS, M.B., B.C., Assistant Obstetric Physician to the Westminster Hospital; 56, Park street, Grosvenor square.

Elected

1871 POORE, GEORGE VIVIAN, M.D., Professor of Medical Jurisprudence and Clinical Medicine in University College, London; Physician to University College Hospital; 24A, Portland place. C. 1890-91. *Referee*, 1887-9, 1892—. *Lib. Com.* 1895—. *Trans.* 2.

1867 POWELL, SIR RICHARD DOUGLAS, Bart., K.C.V.O., M.D., Physician Extraordinary to H.M. the King; Physician to, and Lecturer on Medicine at, the Middlesex Hospital; Consulting Physician to the Hospital for Consumption, Brompton; 62, Wimpole street, Cavendish square. S. (Oct.) 1883-5. C. 1887-8. V.P. 1902—. *Referee*, 1879-83, 1886. *Trans.* 3.

1887 POWER, D'ARCY, M.A., M.B., Assistant Surgeon at St. Bartholomew's Hospital; Surgeon to the Victoria Hospital for Children, Chelsea; 10A, Chandos street, Cavendish Square. *Lib. Com.* 1896—. *Trans.* 2.

1867 **Power**, HENRY, Consulting Ophthalmic Surgeon to St. Bartholomew's Hospital; 10A, Chandos street, Cavendish square, and Bagdale Hall, Whitby. C. 1882-3. V.P. 1892-3. *Referee*, 1870-81, 1891-2. *Sci. Com.* 1870, 1889—. *Lib. Com.* 1872-8.

1883 PRINGLE, JOHN JAMES, M.B., C.M., Physician in Charge of Skin Department at the Middlesex Hospital; 23, Lower Seymour street, Portman square. *Trans.* 2.

1874 PURVES, WILLIAM LAIDLAW, Aural Surgeon to Guy's Hospital; 20, Stratford place, Oxford street. *Trans.* 2.

1877 PYE-SMITH, PHILIP HENRY, M.D., F.R.S., Physician to and Lecturer on Medicine at, Guy's Hospital; 48, Brook street, Grosvenor square. C. 1893-4. *Lib. Com.* 1887-93, 1899—. *Referee*, 1897—. *Trans.* 1.

1898 RAMSAY, HERBERT MURRAY, 35A, Hertford street.

1893 RANKIN, GUTHRIE, 4, Chesham street, Belgrave square.

1899 RAWLING, LOUIS BATHE, M.B., B.C., 16, Montagu street, Portman square.

Elected

1892 RAYNER, HENRY, M.D., Lecturer on Psychological Medicine to St. Thomas's Hospital; 16, Queen Anne street, Cavendish square.

1869 READ, THOMAS LAURENCE, 11, Petersham terrace, Queen's gate. C. 1901—. *Ho. Com.* 1902—.

1891 REECE, RICHARD JAMES, 62, Addison gardens.

1882 REID, SIR JAMES, Bart., G.C.V.O., K.C.B., M.D., Physician-in-Ordinary to H.M. the King; 72, Grosvenor street, Grosvenor square.

1891 RENDEL, ARTHUR BOWEN, M.A., M.B., B.C., 43, Albion street, Hyde Park.

1887 RICHARDSON, GILBERT, M.A., M.D., Hillside, Putney hill.

1863 RINGER, SYDNEY, M.D., F.R.S., Holme Professor of Clinical Medicine in University College, London, and Physician to University College Hospital; 15, Cavendish place, Cavendish square. C. 1881-2. V.P. 1900-2. *Referee*, 1873-80, 1889-97. *Trans.* 6.

1900 RIVIERE, CLIVE, M.D., 19, Devonshire street.

1896 ROBERTS, CHARLES HUBERT, M.D., Physician to Out-Patients, Samaritan Hospital for Women; Physician to Out-patients, Queen Charlotte's Lying-in Hospital, London; 21, Welbeck street.

1893 ROBERTS, D. WATKIN, M.D., 56, Manchester street, Manchester square.

1878 ROBERTS, FREDERICK THOMAS, M.D., Professor of Medicine, and of Clinical Medicine, in University College, London; Physician to University College Hospital; Consulting Physician to the Hospital for Consumption, Brompton; 102, Harley street, Cavendish square. C. 1894-5. *Referee*, 1899—. *Sci. Com.* 1889-1902.

1898 ROBERTSON, F. W., M.D., "Ravenstone," Lingfield road, Wimbledon, Surrey.

1901 ROBINSON, GEORGE HENKELL DRUMMOND, M.D., 84, Park street, Grosvenor square.

Elected

1896 ROBINSON, HENRY BETHAM, M.S., Assistant Surgeon to, and Surgeon in Charge of the Throat Department, St. Thomas's Hospital; Assistant Surgeon to the East London Hospital for Children, Shadwell; 1, Upper Wimpole street.

1889 ROBSON, ARTHUR WILLIAM MAYO, Professor of Surgery, Yorkshire College, Leeds; Senior Surgeon, Leeds General Infirmary; 8, Park crescent, London, and 7, Park square, Leeds. *Trans.* 5. *Pro.* 1.

1890 ROLLESTON, HUMPHRY DAVY, M.D., Physician to, and Lecturer on Pathology at, St. George's Hospital; Senior Physician to Out-patients, Victoria Hospital for Children; 55, Upper Brook street, Grosvenor square.

1857 **Rose,** HENRY COOPER, M.D., 16, Warwick road, Maida Vale. C. 1886-7. *Trans.* 1.

1888 ROUGHTON, EDMUND WILKINSON, B.S., M.D., Surgeon and Surgical Tutor to the Royal Free Hospital; 38, Queen Anne street. *Trans.* 1.

1882 ROUTH, AMAND JULES MCCONNEL, M.D., B.S., Obstetric Physician to, and Lecturer on Midwifery at, the Charing Cross Hospital; Physician to the Samaritan Free Hospital for Women and Children; 14A, Manchester square. C. 1902—. *Lib. Com.* 1900-2. *Referee,* 1900-2.

1849 **Routh,** CHARLES HENRY FELIX, M.D., Consulting Physician to the Samaritan Free Hospital for Women and Children; 52, Montagu square. *Lib. Com.* 1854-5. *Trans.* 1.

1891 RUSSELL, J. S. RISIEN, M.D., Assistant Physician to University College Hospital, and Pathologist to the National Hospital for the Paralysed and Epileptic, Queen square; 44, Wimpole street, Cavendish square. *Trans.* 1.

Elected

1900 RYALL, CHARLES, 51, Queen Anne street.

1886 SAINSBURY, HARRINGTON, M.D., Physician to the Royal Free Hospital; Physician to the City of London Hospital for Diseases of the Chest; 52, Wimpole street, Cavendish square. *Trans.* 1.

1899 SANDILAND, JOHN EDWARD, M.B., 1, Montague square.

1869 **Sansom,** ARTHUR ERNEST, M.D., Physician to the London Hospital; Consulting Physician, North-Eastern Hospital for Children; 84, Harley street, Cavendish square. C. 1887-8. *Referee,* 1889—. *Trans.* 3.

1902 SAUNDERS, EDWARD ARTHUR, M.B., B.Ch., 49, Harley street, Cavendish square.

1879 SAVAGE, GEORGE HENRY, M.D., Lecturer on Mental Diseases at Guy's Hospital; 3, Henrietta street, Cavendish square. C. 1898-9.

1892 SCHORSTEIN, GUSTAVE M.A., M.B., B.Ch., D.P.H., Assistant Physician to the London Hospital, and to the Hospital for Consumption, Brompton; 11, Portland place.

1899 SCOTT, LINDLEY MARCROFT, M.D., 98, Sloane street.

1863 **Sedgwick,** WILLIAM, 101, Gloucester place, Portman square. C. 1884-5. *Trans.* 3.

1892 SEGUNDO, CHARLES SEMPILL DE, M.B., B.S., 6, Brook street, Hanover square.

1892 SELWYN-HARVEY, JOHN STEPHENSON, M.D., 1, Astwood road, Cromwell road.

1877 SEMON, SIR FELIX, M.D., Physician Extraordinary to H.M. the King; Physician for Diseases of the Throat to the National Hospital for Epilepsy and Paralysis, Queen square; 39, Wimpole street, Cavendish square. C. 1895-7. *Lib. Com.* 1894-5. *Trans.* 3.

Elected

1900 SEQUEIRA, JAMES HARRY, M.D., 13, Welbeck street.

1894 SEWILL, JOSEPH SEFTON, 9A, Cavendish square.

1882 SHARKEY, SEYMOUR JOHN, M.D., Physician to, and Joint Lecturer on Medicine at, St. Thomas's Hospital; 22, Harley street, Cavendish square. C. 1899-1900. *Referee*, 1897-9, 1902—. *Trans.* 2.

1900 SHAW, HAROLD BATTY, M.D., 7, Devonshire street, Portland place.

1886 SHAW, LAURISTON ELGIE, M.D., Physician to Guy's Hospital; 64, Harley street, Cavendish square.

1884 SHEILD, ARTHUR MARMADUKE, M.B., B.C., Assistant Surgeon to St. George's Hospital; 4, Cavendish place. *Referee*, 1897—. *Trans.* 6.

1896 SHORE, THOMAS WILLIAM, M.D., Heathfield, Alleyn park, Dulwich.

848 **Sieveking**, SIR EDWARD HENRY, M.D., LL.D., F.S.A., Physician Extraordinary to H.M. the King; Consulting Physician to St. Mary's and the Lock Hospitals; 17, Manchester square. C. 1859-60. S. 1861-3. V.P. 1873-4. L. 1881-2. P. 1888-9. *Referee*, 1855-8, 1864-72, 1875-80. *Sci. Com.* 1862. *Trans.* 2.

1886 SILCOCK, ARTHUR QUARRY, B.S., Surgeon in charge of Out-patients, and Teacher of Operative Surgery, St. Mary's Hospital; Surgeon to the Royal London Ophthalmic Hospital; 52, Harley street, Cavendish square. *Lib. Com.* 1895—.

1842 **Simon**, SIR JOHN, K.C.B., F.R.S., Hon. M.D.Dublin, 1887, Consulting Surgeon to St. Thomas's Hospital; 40, Kensington square. C. 1854-5. V.P. 1865. *Referee*, 1851-3, 1866-81. *Trans.* 1.

1899 SIMPSON, WILLIAM JOHN RITCHIE, M.D., 12, Gloucester place, Portman square.

Elected

1894 SLATER, CHARLES, M.B., 81, St. Ermin's mansions, Westminster.

1890 SMALE, MORTON, Surgeon Dentist to St. Mary's Hospital; 22A, Cavendish square.

1879 SMITH, E. NOBLE, Surgeon to the City Orthopædic Hospital; Surgeon to All Saints' Children's Hospital; Orthopædic Surgeon to the British Home for Incurables; 24, Queen Anne street, Cavendish square.

1881 SMITH, EUSTACE, M.D., Physician to H.M. the King of the Belgians; Physician to the East London Children's Hospital, and to the Victoria Park Hospital for Diseases of the Chest; 15, Queen Anne street, Cavendish square. C. 1899-1900.

1866 SMITH, HEYWOOD, M.A., M.D., 18, Harley street, Cavendish square.

1889 SMITH, ROBERT PERCY, M.D., B.S., Lecturer on Psychological Medicine, Charing Cross Hospital; 36, Queen Anne street.

1863 **Smith**, SIR THOMAS, Bart., Honorary Sergeant-Surgeon to H.M. the King; Consulting Surgeon to St. Bartholomew's Hospital; 5, Stratford place, Oxford street. S. 1870-2. C. 1875-6. V.P. 1887-8. *Referee*, 1873-4, 1880-6. *Sci. Com.* 1867. *Trans.* 4.

1872 SMITH, THOMAS GILBART, M.D., Physician to the London Hospital; Physician to the Royal Hospital for Diseases of the Chest, City road; 68, Harley street, Cavendish square. C. 1890. *Trans.* 1.

1873 SMITH, W. JOHNSON, Surgeon to the Seamen's Hospital Society, Greenwich.

1874 *Smith*, WILLIAM ROBERT, M.D., D.Sc., F.R.S.Edin., Barrister-at-Law, Professor of Forensic Medicine, and Director of the Laboratories of State Medicine in King's College, London; 74, Great Russell street. *Trans.* 1.

Elected

1889 SPENCER, HERBERT R., M.D., B.S., Professor of Midwifery in University College; Obstetric Physician to University College Hospital; 104, Harley street. *Referee*, 1894—.

1887 SPENCER, WALTER GEORGE, M.B., M.S., Surgeon to, and Lecturer on Physiology at, the Westminster Hospital; 35, Brook street, Grosvenor square. *Trans.* 2.

1888 SPICER, ROBERT HENRY SCANES, M.D., Surgeon to the Department for Diseases of the Throat, St. Mary's Hospital; 28, Welbeck street, Cavendish square.

1890 SPICER, WILLIAM THOMAS HOLMES, M.B., 5, Wimpole street, Cavendish square.

1875 SPITTA, EDMUND JOHNSON, Ivy House, 31, South Side, Clapham Common, Surrey.

1885 SQUIRE, JOHN EDWARD, M.D., Physician to the North London Hospital for Consumption; 2, Harley street, Cavendish square. *Trans.* 2.

1897 STAINER, EDWARD, M.A., M.B., 60, Wimpole street.

1896 STEPHENS, JOHN WILLIAM WATSON, M.B., B.C., 8, Fopstone road, Earl's Court.

1899 STEWART, PURVES, M.D., 7, Harley street. *Trans.* 1.

1856 **Stocker**, ALONZO HENRY, M.D., Peckham House, Peckham.

1884 STONHAM, CHARLES, Surgeon to, and Lecturer on Surgery and Teacher of Operative Surgery at, the Westminster Hospital; Surgeon to the Poplar Hospital for Accidents; 4, Harley street, Cavendish square.

1896 SUTHERLAND, GEORGE ALEXANDER, M.D., Physician to Paddington Green Children's Hospital; Assistant Physician to the North-West London Hospital; 73, Wimpole street, Cavendish square.

Elected

1896 SWAN, CHARLES ROBERT JOHN ATKIN, M.B., B.Ch., 4, Devonport street, Hyde Park.

1890 SYERS, HENRY WALTER, M.D., 75, Wimpole street.

1886 SYMONDS, CHARTERS JAMES, M.S., M.D., Surgeon to, and Surgeon in charge of the Throat Department at, Guy's Hospital; 58, Portland place.

1875 TAY, WARREN, Senior Surgeon to the London Hospital, to the Royal London Ophthalmic Hospital, and to the Hospital for Diseases of the Skin, Blackfriars; Consulting Surgeon to the North-Eastern Hospital for Children; 4, Finsbury square.

1873 TAYLOR, FREDERICK, M.D., *Trustee;* Physician to, and Lecturer on Medicine at, Guy's Hospital; Consulting Physician to the Evelina Hospital for Sick Children; 20, Wimpole street, Cavendish square. S. 1889-93. C. 1894-6. *Sci. Com.* 1889-1902. *Referee,* 1887-8, 1899—. *Trans.* 3.

1893 TAYLOR, JAMES, M.D., Assistant Physician to the National Hospital for the Paralysed and Epileptic; Physician to the North-Eastern Hospital for Children, and to the National Orthopædic Hospital; 49, Welbeck street, Cavendish square. *Trans.* 1.

1890 TAYLOR, SEYMOUR, M.D., Assistant Physician, West London Hospital; 16, Seymour street, Portman square.

1859 TEGART, EDWARD, 60, Scarsdale Villas, Kensington. C. 1888-9.

1874 THIN, GEORGE, M.D., 63, Harley street, Cavendish square. C. 1893-4. *Trans.* 14.

1900 THOMPSON, CHARLES HERBERT, M.D., 133, Harley street, Cavendish square.

1862 **Thompson,** EDMUND SYMES, M.D., Consulting Physician to the Hospital for Consumption, Brompton; Gresham Professor of Medicine; 33, Cavendish square. S. 1871-4. C. 1878-9. *Sci. Com.* 1889-1902. *Referee,* 1876-7. *Trans.* 1.

Elected

1852 **Thompson,** Sir Henry, Bart., Surgeon-Extraordinary to H.M. the King of the Belgians ; Emeritus Professor of Clinical Surgery in University College, London ; and Consulting Surgeon to University College Hospital ; 35, Wimpole street, Cavendish square. V.P. 1888. C. 1869. *Trans.* 8.

1862 **Thompson,** Reginald Edward, M.D., Consulting Physician to the Hospital for Consumption, Brompton ; 13, Cheyne gardens, Chelsea. C. 1879. S. 1880-82. V.P. 1883-4. *Referee*, 1873-8. *Sci. Com.* 1867. *Trans.* 2.

1899 Thomson, Herbert Campbell, M.D., 34, Queen Anne street. *Trans.* 2.

1892 Thomson, StClair, M.D., Physician to the Throat Hospital, Golden Square ; Surgeon to the Royal Ear Hospital, London ; 28, Queen Anne street, Cavendish square. *Trans.* 1.

1900 Thomson-Walker, John William, M.B., 8, Cavendish place.

1892 Thorne, William Bezly, M.D., 53, Upper Brook street.

1899 Thursfield, James Hugh, M.D., 10, Bentinck street, Manchester square. *Trans.* 1.

1889 Tirard, Nestor Isidore Charles, M.D., Professor of the Principles and Practice of Medicine, King's College ; Physician to King's College Hospital, and Physician to the Evelina Hospital for Sick Children ; 74, Harley street, Cavendish square.

1872 Tomes, Charles Sissmore, M.A., F.R.S., 9, Park crescent, Portland place. C. 1887. V.P. 1897-99. *Lib. Com.* 1879.

1882 Tooth, Howard Henry, C.M.G., M.D., Physician to the National Hospital for the Paralysed and Epileptic, Queen square ; Assistant Physician to St. Bartholomew's Hospital ; 34, Harley street, Cavendish square. *Referee*, 1902—. *Sci. Com.* 1896-1902.

Elected

1879 TREVES, SIR FREDERICK, Bart., C.B., K.C.V.O., Honorary Sergeant-Surgeon to H.M. the King; Surgeon-in-Ordinary to H.R.H. the Prince of Wales; Consulting Surgeon to the London Hospital; 6, Wimpole street, Cavendish square. C. 1895-6. *Referee*, 1890-95. *Sci. Com.* 1889-95. *Trans.* 6.

1902 TREVOR, ROBERT SALUSBURY, M.B., B.C., 21, FitzGeorge avenue, West Kensington.

1859 **Truman**, EDWIN THOMAS, Surgeon-Dentist to His Majesty's Household; 23, Old Burlington street.

1897 TUNNICLIFFE, FRANCIS WHITTAKER, M.D., 6, Devonshire street, Portland place.

1889 TURNBULL, GEORGE LINDSAY, M.D., 47, Ladbroke square.

1882 TURNER, GEORGE ROBERTSON, Surgeon to, and Joint Lecturer on Surgery at, St. George's Hospital; Visiting Surgeon to the Seamen's Hospital, Greenwich; 41, Half Moon street, Piccadilly. *Trans.* 1.

1898 TURNER, WILLIAM, M.B., B.S., Assistant Surgeon, Westminster Hospital; 53, Queen Anne street, Cavendish square.

1896 TURNER, WILLIAM ALDREN, M.D., Assistant Physician to King's College Hospital and to the National Hospital for the Paralysed and Epileptic, Queen Square; 13, Queen Anne street, Cavendish square.

1896 TURNEY, HORACE GEORGE, M.D., Joint Lecturer on Pathology and Assistant Physician to St. Thomas's Hospital; 68, Portland place. *Trans.* 1.

1892 TWEEDY, JOHN, Professor of Ophthalmic Medicine and Surgery in University College, Ophthalmic Surgeon to University College Hospital, and Surgeon to the Royal London Ophthalmic Hospital; 100, Harley street, Cavendish square.

Elected

1876 VENN, ALBERT JOHN, M.D., 63, Grosvenor street.

1870 VENNING, EDGCOMBE, 30, Cadogan place. C. 1898-1900. V.P. 1902—.

1902 VINCENT, RALPH, M.D., B.S., 1, Harley street.

1891 VOELCKER, ARTHUR FRANCIS, M.D., B.S., Assistant Physician to, and Lecturer on Pathology at, the Middlesex Hospital; Assistant Physician, Hospital for Sick Children, Great Ormond street; 101, Harley street.

1896 WAGGETT, ERNEST, M.B., B.C., Surgeon, London Throat Hospital; Surgeon to Out Patient Throat and Ear Department, Great Northern Central Hospital; 45, Upper Brook street.

1884 WAKLEY, THOMAS, jun., 5, Queen's Gate, South Kensington.

1896 WALDO, FREDERICK JOSEPH, M.D., City Coroner, 40, Lansdowne road, Holland park.

1900 WALKER, H. ROE, 8, Harley street, Cavendish square.

1887 WALLACE, EDWARD JAMES, M.D., 22, Hans crescent, Chelsea.

1883 WALLER, AUGUSTUS, M.D., F.R.S., Lecturer on Physiology, St. Mary's Hospital; Weston Lodge, 16, Grove End road, St. John's Wood. *Referee*, 1895—.

1888 WALLIS, FREDERICK CHARLES, M.B., B.C., Assistant Surgeon to the Charing Cross Hospital; 107, Harley street, Cavendish square.

1896 WALSHAM, HUGH, M.A., M.D., Assistant Physician to the City of London Hospital for Diseases of the Chest; Assistant Medical Officer in Electrical Department, St. Bartholomew's Hospital; 114, Harley street, Cavendish square.

1873 **Walsham**, WILLIAM JOHNSON, C.M., Surgeon to, and Lecturer on Surgery at, St. Bartholomew's Hospital; Consulting Surgeon to the Metropolitan Hospital; 77, Harley Street, Cavendish square. C. 1888-9. *Referee*, 1895—. *Lib. Com.* 1882-5. *Trans.* 8.

Elected

1886 WARD, ALLAN OGIER, M.D., 73, Cheapside.

1890 WARD, ARTHUR HENRY, Surgeon to Out-patients, Lock Hospital; 31, Grosvenor street.

1894 WARD-HUMPHREYS, GEORGE HERBERT, 7, Cavendish place, Cavendish square.

1891 WARING, H. J., M.B., M.S., B.Sc., Assistant Surgeon and Demonstrator of Operative Surgery, St. Bartholomew's Hospital; Surgeon, Metropolitan Hospital; 37, Wimpole street.

1877 WARNER, FRANCIS, M.D., Physician to, and Lecturer on Materia Medica and Therapeutics at, the London Hospital; 5, Prince of Wales terrace, Kensington Palace. C. 1899-1901. *Trans.* 3.

1894 WATERHOUSE, HERBERT FURNIVALL, C.M., Senior Assistant Surgeon and Lecturer on Anatomy, Charing Cross Hospital; Surgeon, Victoria Hospital for Children; 81, Wimpole street.

1861 **Watson**, WILLIAM SPENCER, M.B., 61, Bedford gardens, Campden hill, Kensington. C. 1883-4. *Trans.* 1.

1891 **Weber**, FREDERIC PARKES, M.D., Physician to the German Hospital, Dalston; 19, Harley street. *Trans.* 1. *Pro.* 1.

1857 WEBER, SIR HERMANN, M.D., Consulting Physician to the German Hospital; 10, Grosvenor street, Grosvenor square. C. 1874-5. V.P. 1885-6. *Sci. Com.* 1889-1902. *Referee*, 1869-73, 1878-84. *Lib. Com.* 1864-73. *Trans.* 6.

1895 WELLS, SYDNEY RUSSELL, M.D., 24, Somerset street, Portman square.

1877 WEST, SAMUEL, M.D., Assistant Physician to St. Bartholomew's Hospital; Senior Physician to the Royal Free Hospital; 15, Wimpole street, Cavendish square. C. 1894-5. *Lib. Com.* 1892-4. *Trans.* 7.

Elected

1888 WETHERED, FRANK JOSEPH, M.D., Assistant Physician to the Hospital for Consumption, Brompton; 83, Harley street, Cavendish square. *Trans.* 1.

1881 WHARRY, ROBERT, M.D., 7, Cambridge gate, Regent's park.

1875 WHIPHAM, THOMAS TILLYER, M.D., Consulting Physician to St. George's Hospital; 11, Grosvenor street, Grosvenor square. C. 1892-3.

1891 WHITE, CHARLES PERCIVAL, M.B., B.C., 22, Cadogan gardens.

1881 WHITE, WILLIAM HALE, M.D., Physician to, and Lecturer on Materia Medica at, Guy's Hospital; 65, Harley street, Cavendish square. C. 1900-2. *Referee*, 1888-97, 1899-1900. *Trans.* 4.

1890 WHITE-COOPER, W. G. O., M.B., 5, Courtfield road, Gloucester road.

1897 WHITFIELD, ARTHUR, M.D., 12, Upper Berkeley street.

1899 WHITING, ARTHUR J., M.D., 142, Harley street.

1863 **Wilks**, SIR SAMUEL, Bart., M.D., LL.D., F.R.S., Physician Extraordinary to Her late Majesty Queen Victoria, Physician in Ordinary to their Royal Highnesses the Duke and Duchess of Connaught; Consulting Physician to Guy's Hospital; 8, Prince Arthur road, Hampstead. *Referee*, 1872–81.

1890 WILLCOCKS, FREDERICK, M.D., Physician to Out-Patients, and Lecturer on Materia Medica and Therapeutics, at the Charing Cross Hospital; Physician to the Evelina Hospital for Sick Children; 14, Mandeville place, Manchester square.

1865 **Willett**, ALFRED, *President; Trustee;* Surgeon to St. Bartholomew's Hospital; Surgeon to St. Luke's Hospital; 36, Wimpole street, Cavendish square. C. 1880-1. V.P. 1890-1. P. 1902—. *Referee*, 1882-9, 1892-1902. *Bldg. Com.* 1889-92. *Ho. Com.* 1892-8. *Trans.* 2.

Elected

1887 WILLETT, EDGAR, M.B., 22, Queen Anne street, Cavendish square.

1888 WILLIAMS, CAMPBELL, 18, Queen Anne street.

1866 **Williams**, CHARLES THEODORE, M.A., M.D., *Trustee for Debenture-holders;* Consulting Physician to the Hospital for Consumption and Diseases of the Chest, Brompton; 2, Upper Brook street, Grosvenor square. C. 1884-5. V.P. 1900-2. *Referee*, 1888-1900. *Lib. Com.* 1880-3. *Ho. Com.* 1900-2. *Sci. Com.* 1889-1902. *Trans.* 6.

1881 WILLIAMS, DAWSON, M.D., Physician to the East London Hospital for Children; 2, Wyndham place, Bryanston square. *Trans.* 1.

1900 WILLIAMS, HUGH LLOYD, 2, Upper Wimpole street.

1872 WILLIAMS, SIR JOHN, Bart., M.D., Physician-Accoucheur to H.R.H. the Princess of Wales, Physician to H.R.H. the Princess Beatrice; Emeritus Professor of Obstetric Medicine, University College, London; Consulting Obstetric Physician to University College Hospital; 63, Brook street, Grosvenor square. C. 1891. *Referee*, 1878-90. *Lib. Com.* 1876-82.

1901 WILLIAMS, LEONARD, M.D., 8, York street, Portman square.

1890 WILLS, WILLIAM ALFRED, M.D., Assistant Physician to the Westminster Hospital; Senior Physician to the North-Eastern Hospital for Children; 29, Lower Seymour street, Portman square.

1879 WOAKES, EDWARD, M.D., Senior Aural Surgeon to the London Hospital; 78, Harley street, Cavendish square.

1887 WOOD, THOMAS OUTTERSON, M.D., Senior Physician to the West End Hospital for Nervous Diseases; 40, Margaret street, Cavendish square.

Elected

1891 WOODFORDE, ALFRED POWNALL, 160, Goldhawk road, Shepherd's Bush.

1890 WYNTER, WALTER ESSEX, M.D., Physician to the Middlesex Hospital; 30, Upper Berkeley street, Portman square.

LIST OF RESIDENT FELLOWS

ARRANGED ACCORDING TO

DATE OF ELECTION

1842 Sir John Simon, K.C.B., F.R.S.
1848 Sir Edward H. Sieveking, M.D.
John Clarke, M.D.
1849 C. H. F. Routh, M.D.
1851 John Birkett.
John A. Kingdon.
1852 Sir Henry Thompson, Bart.
1853 Robert Brudenell Carter.
1854 Sir Alfred B. Garrod, M.D., F.R.S.
1856 Jonathan Hutchinson, F.R.S.
Timothy Holmes.
Alonzo H. Stocker, M.D.
1857 Sir Hermann Weber, M.D.
Henry Cooper Rose, M.D.
Henry Walter Kiallmark.
1858 John William Ogle, M.D.
1859 Wm. Howship Dickinson, M.D.
Edwin Thomas Truman.
Richard Barwell.
Edward Tegart.
1860 William Ogle, M.D.
Thomas Bryant.
John Couper.
Henry Howard Hayward.
1861 William Spencer Watson.
1862 Lionel Smith Beale, M.B., F.R.S.
Edmund Symes Thompson, M.D.
Reginald Edward Thompson, M.D.
George Cowell.
1863 Sir Samuel Wilks, Bt., M.D., F.R.S.
Samuel Fenwick, M.D.
Sydney Ringer, M.D., F.R.S.
Sir Thomas Smith, Bart.
Arthur B. R. Myers.
William Sedgwick.
1864 Thomas William Nunn.
1865 James Edward Pollock, M.D.
George Fielding Blandford, M.D.
Sir Dyce Duckworth, M.D.
Frederick W. Pavy, M.D., F.R.S.
John Langton.
Frederick James Gant.
Alfred Willett.
Sir Alfred Cooper.
Christopher Heath.
1866 Samuel Jones Gee, M.D.
Charles Theodore Williams, M.D.
Heywood Smith, M.D.
Sir William Selby Church, Bart., K.C.B., M.D.
1867 Sir R. Douglas Powell, Bart., M.D.
F. Howard Marsh.
Henry Power.
Thomas Pickering Pick.
1868 H. Charlton Bastian, M.D., F.R.S.
Sir W. H. Broadbent, Bart., M.D.
Thomas Buzzard, M.D.
Walter Butler Cheadle, M.D.

1868 T. Henry Green, M.D.
George Eastes.
1869 Joseph Frank Payne, M.D.
Arthur E. Sansom, M.D.
Thomas Laurence Read.
1870 J. Warrington Haward.
Edgcombe Venning.
Clement Godson, M.D.
Reginald Harrison.
Robert Leamon Bowles, M.D.
1871 William Cayley, M.D.
Sir T. Lauder Brunton, M.D., F.R.S.
J. Hughlings Jackson, M.D., F.R.S.
George Vivian Poore, M.D.
Philip Frank, M.D.
1872 T. Gilbart-Smith, M.D.
George B. Brodie, M.D.
Sir John Williams, Bart., M.D.
Sir J. Fayrer, M.D., F.R.S.
Charles S. Tomes, M.A., F.R.S.
Sir William Bartlett Dalby.
1873 Frederick Taylor, M.D.
Norman Moore, M.D.
Sir William R. Gowers, M.D., F.R.S.
Jeremiah McCarthy.
Wm. Johnson Smith.
Alex. O. MacKellar.
Henry T. Butlin.
Charles Higgens.
William J. Walsham.
1874 Alfred Lewis Galabin, M.D.
George Thin, M.D.
John Mitchell Bruce, M.D.
Henry Morris.
William Laidlaw Purves.
William Harrison Cripps.
Sir Henry G. Howse, M.S.
Herbert William Page.
Frederic Durham.
William Robert Smith, M.D.
1875 Thomas T. Whipham, M.D.
Thomas Crawford Hayes, M.D.
Waren Tay.
Edmund J. Spitta.
Samuel C. Osborn.
Fletcher Beach, M.B.
1876 Sir Thomas Barlow, Bart., K.C.V.O., M.D.
Albert J. Venn, M.D.
1877 Sir Felix Semon, M.D.
Sidney Coupland, M.D.
Francis Warner, M.D.
William Ewart, M.D.
1877 Alfred Pearce Gould, M.S.
Rickman J. Godlee, M.S.
Alban H. G. Doran.
George Ernest Herman, M.B.
Samuel West, M.D.
John Abercrombie, M.D.
George Allan Heron, M.D.
Joseph A. Ormerod, M.D.
P. Henry Pye-Smith, M.D., F.R.S.
Sir William Henry Bennett.
1878 Sir Jas. Crichton-Browne, M.D.
Fred. T. Roberts, M.D.
Lord Lister, P.C., O.M., F.R.S.
Clinton T. Dent.
John H. Morgan, C.V.O.
Donald W. Charles Hood, M.D.
1879 Edward Woakes, M.D.
Malcolm A. Morris.
A. E. Cumberbatch.
Edmund Owen.
Arthur E. J. Barker.
Sir Fredk. Treves, Bart., C.B., K.C.V.O.
Thomas John Maclagan, M.D.
Andrew Clark.
Francis Henry Champneys, M.D.
William Watson Cheyne, F.R.S.
George Henry Savage, M.D.
H. H. Clutton, M.A.
Frederic S. Eve.
E. Noble Smith.
William Henry Allchin, M.D.
1880 Robert Alex. Gibbons, M.D.
David Ferrier, M.D., F.R.S.
Vincent Dormer Harris, M.D.
Edmund Distin Maddick.
Jas. John MacWhirter Dunbar, M.D.
James William Browne, M.B.
William Appleton Meredith, M.B.
Malcolm Macdonald McHardy.
A. Boyce Barrow.
William Murrell, M.D.
George Ogilvie, M.B.
Charles Edward Beevor, M.D.
Thomas Colcott Fox, M.B.
George Henry Makins, C.B.
1881 Francis de Havilland Hall, M.D.
Robert Wharry, M.D.
Richard Clement Lucas, B.S.
Stephen Mackenzie, M.D.
William Hale White, M.D.
Eustace Smith, M.D.
Percy Kidd, M.D.
Oswald A. Browne, M.D.

1881 W. Bruce Clarke, M.B.
Dawson Williams, M.D.
George Lindsay Johnson, M.D.
Henry Edward Juler.
Jonathan F. C. H. Macready.
C. B. Lockwood.
1882 Philip J. Hensley, M.D.
Ernest Clarke, M.D., B.S.
George Robertson Turner.
Howard Henry Tooth, C.M.G., M.D.
Sir Herbert Isambard Owen, M.D.
Charles R. B. Keetley.
Anthony A. Bowlby, C.M.G.
Amand J. McC. Routh, M.D.
Seymour J. Sharkey, M.D.
William Lang.
Henry Radcliffe Crocker, M.D.
Sir James Reid, Bart., G.C.V.O.
1883 Edwin Clifford Beale, M.A., M.B.
James Kingston Fowler, M.D.
James Frederic Goodhart, M.D.
John Charles Galton, M.A.
W. Hamilton A. Jacobson, M.Ch.
Walter H. Jessop, M.B.
Walter Edmunds, M.C.
Sir Victor A. Horsley, F.R.S.
Dudley Wilmot Buxton, M.D.
Charles Douglas F. Phillips, M.D.
John James Pringle, M.B.
Henry Roxburgh Fuller, M.D.
Wilmot Parker Herringham, M.D.
Augustus Waller, M.D.
William Pasteur, M.D.
John Bland-Sutton.
Robert Marcus Gunn, M.B.
1884 George Newton Pitt, M.D.
Charles Stonham.
Stanley Boyd, B.S.
William Arbuthnot Lane, M.S.
Arthur Marmaduke Sheild, M.B.
Sidney Harris Cox Martin, M.D., F.R.S.
George Lawson.
Thomas Wakley, jun.
F. Swinford Edwards.
James Johnston, M.D.
William Duncan, M.D.
Charles Chinner Fuller.
George Richard Turner Phillips.
Bilton Pollard.
1885 Alexander Haig, M.D.
Theodore Dyke Acland, M.D.
1885 Frederick Walker Mott, M.D., F.R.S.
James Berry.
John Cahill, M.D.
John Poland.
A. C. Butler-Smythe.
Charles Alfred Ballance, M.S.
Walter S. A. Griffith, M.D.
John Edward Squire, M.D.
John D. Malcolm, M.B., C.M.
Phineas S. Abraham, M.D.
1886 Robert Maguire, M.D.
Harrington Sainsbury, M.D.
Cuthbert Hilton Golding-Bird, M.B.
Lauriston Elgie Shaw, M.D.
Charters James Symonds, M.S.
Robert Boxall, M.D.
Allan Ogier Ward, M.D.
Archibald Edward Garrod, M.D.
Stephen Paget.
William Radford Dakin, M.D.
Samuel Herbert Habershon, M.D.
Arthur Quarry Silcock.
Arthur H. N. Lewers, M.D.
1887 Walter George Spencer.
Thomas Outterson Wood, M.D.
Edgar William Willett, M.B.
Henry Lewis Jones, M.D.
Francis George Penrose, M.D.
Hugh Percy Dunn.
Frederic William Hewitt, M.D.
James Barry Ball, M.D.
Gilbert Richardson, M.D.
D'Arcy Power, M.B.
John Gay.
James Calvert, M.D.
Percy J. F. Lush, M.B.
Edward James Wallace, M.D.
1888 Robert Henry Scanes Spicer, M.D.
Jonathan Hutchinson, jun.
Campbell Williams.
James Donelan, M.B., C.M.
John Anderson, M.D., C.I.E.
Laurie Asher Lawrence.
Arthur Pearson Luff, M.D., B.Sc.
Albert Carless, M.S.
Frederick C. Wallis, M.B., B.C.
Charles James Cullingworth, M.D.
Edmund Cautley, M.D., B.C.
H. Montague Murray, M.D.
Frank Joseph Wethered, M.D.
Edmund Wilkinson Roughton, B.S.
Frederick William Cock, M.D.
Robert Henry Clarke, M.B.
1889 Montagu Handfield-Jones, M.D.

1889 David Henry Goodsall.
Raymond Johnson, M.B.
John Fletcher Little, M.B.
Henry Work Dodd.
George Lindsay Turnbull, M.D.
Sidney Phillips, M.D.
William Charles Bull, M.B.
George P. Field.
Charles Henry Cosens.
Henry Percy Dean, M.B., M.S.
Alfred Samuel Gubb, M.D.
William Hunter, M.D.
J. Inglis Parsons, M.D.
Bernard Pitts, M.B., M.C.
Robert Percy Smith, M.D., B.S.
Herbert R. Spencer, M.D., B.S.
Nestor Isidore Chas. Tirard, M.D.
Arthur William Mayo Robson.

1890 John Rose Bradford, M.D., F.R.S.
Roland Danvers Brinton, M.D.
Charles D. B. Hale, M.D.
Edwin Cooper Perry, M.D.
Morton Smale.
Frederick Willcocks, M.D.
William T. Holmes Spicer, M.B.
Thomas Henry Crowle.
Henry Walter Syers, M.D.
Seymour Taylor, M.D.
William Alfred Wills, M.D.
G. O. White-Cooper, M.B.
Herbert William Allingham.
William A. F. Bateman.
James Jackson Clarke, M.B.
Leonard G. Guthrie, M.D., B.Ch.
G. William Hill, M.D., B.Sc.
Edward Law, M.D., C.M.
Patrick Manson, C.M.G., M.D., C.M., F.R.S.
Humphry D. Rolleston, M.D.
Arthur Henry Ward.
Walter Essex Wynter, M.D., B.S
Edward Lawrie, M.B.
Christopher Childs, M.D.

1891 William Lee Dickinson, M.D.
Herbert P. Hawkins, M.D., B.Ch.
Cyril Ogle, M.A., M.B.
Arthur F. Voelcker, M.D., B.S.
Alfred Pownall Woodforde.
Herbert T. Herring, M.B., B.S.
Ernest Muirhead Little.
Henry Charrington Martin, M.D.
Frederick William Andrewes, M.D.
Alfred Eddowes, M.D.
Herbert Morley Fletcher, M.D.

1891 William Heaton Hamer, M.D.
William Bromfield Paterson.
Holburt Jacob Waring.
Frederic Parkes Weber, M.D.
F. E. Batten, M.D.
Thomas Jessopp Bokenham.
Norman Dalton, M.D.
P. R. W. De Santi.
P. W. Dove.
William J. Gow, M.D.
Paul Frank Moline, M.B.
Edward Percy Paton, M.D.
Arthur Bowen Rendel, M.B., B.C.
James Samuel Risien Russell, M.D.
Charles Percival White, M.B., B.C.
W. Page May, M.D.
Richard J. Reece.

1892 J. Dundas Grant, M.D.
R. J. Bliss Howard, M.D.
Thomas Horrocks Openshaw, M.B.
William Bezly Thorne, M.D.
W. H. Russell Forsbrook, M.D.
John Harold, M.B.
John Alfred Masters, M.D.
Gustave Schorstein, M.B.
Charles Sempill de Segundo, M.B.
John Tweedy.
J. S. Selwyn-Harvey, M.D.
StClair Thomson, M.D.
Henry Rayner, M.D.
H. Marmaduke Page.

1893 James Taylor, M.D.
Howard Barrett.
Robert Cozens Bailey, M.B.
Henry Albert Caley, M.D.
Arthur Edward Giles, M.D.
Miles Miley, M.B.
D. Watkin Roberts, M.D.
Leonard A. Bidwell.
Frédéric F. Burghard, M.D., M.S.
J. H. Drysdale, M.B.
William McAdam Eccles, M.S.
Vaughan Harley, M.D.
George Herschell, M.D.
Arnold Lawson.
Guthrie Rankin.

1894 Richard Gill.
Joseph Sefton Sewill.
Thomas Vincent Dickinson, M.D.
Herbert Edward Durham, M.B.
Alexander Morison, M.D.
L. Hemington Pegler, M.D.
Herbt. Furnivall Waterhouse, C.M.

1894 Percy Furnivall.
R. L. Langdon-Down, M.B.,B.C.
Allan Macfadyen, M.D., B.S.
Ernst Michels, M.D.
Wm. Rivers Pollock, M.B., B.C.
Charles Slater, M.B.
G. H. Ward-Humphreys.
1895 Charles Arthur Parker.
Sydney Russell Wells, M.D.
Alfred Milne Gossage, M.B.
Robert Murray Leslie, M.B.
James Galloway, M.D.
David Bridge Lees, M.D.
Arthur G. Phear, M.D.
1896 Joseph Lockhart Downes, M.B.
Edward Wilberforce Goodall, M.D.
James Ernest Lane.
George Alex. Sutherland, M.D.
Charles Buttar, M.D.
P. J. Freyer, M.D., I.M.S., M.A.
Percival Horton-Smith, M.D.
Thomas William Shore, M.D.
William Aldren Turner, M.D.
Charles Hubert Roberts, M.D.
Charles R. J. Atkin Swan, M.B.
James Kingston Barton.
J. Walter Carr, M.D.
John H. Dauber, M.A., M.B., B.Ch.
Alexander Grant Russell Foulerton.
L. Vernon Jones, B.A., M.D., B.Ch.
Henry Betham Robinson. M.S.
Horace George Turney, M.D.
Ernest Waggett, M.B., B.C.
Frederick Joseph Waldo, M.D.
Hugh Walsham, M.D.
J. W. W. Stephens, M.D.
1897 Comyns Berkeley, M.B., B.C.
William Arthur Brailey, M.D.
James Cantlie, M.B.
Raymond H. Payne Crawfurd, M.D.
Louis Jenner, M.B.
Francis Whittaker Tunnicliffe, M.D.
Arthur Whitfield, M.D.
Edward Stainer, M.A., M.B.
Alfred G. Levy, M.D.
A. P. Beddard, M.B.
G. F. Blacker, M.D.
W. S. Colman, M.D.
F. W. Goodbody, M.D.
R. Hutchison, M.D.
Harold Low.
Christopher Addison, M.D.
1898 J. H. Bryant, M.D.
W. H. Corfield, M.D.
1898 L. A. Dunn, M.S.
E. Hurry Fenwick.
A. Downing Fripp, C.B., M.V.O., M.S.
A. Corrie Keep, M.D.
A. C. Latham, M.D.
J. B. Lawford.
John McFadyean.
H. Murray Ramsay.
J. F. H. Broadbent, M.D.
H. Ronald Carter.
A. Stark Currie, M.D.
P. J. Edmunds, M.B.
James Morrison, M.D.
J. S. Edkins.
Thomas J. Horder, M.D.
F. W. Robertson.
S. Jervois Aarons, M.D.
Willmott Evans, M.D., B.S., B.Sc.
John Murray.
W. Adams Frost.
C. R. C. Lyster.
Samuel Noble Bruce.
Cuthbert Chapman Gibbes, M.D.
H. Stringfellow Pendlebury, M.B.
William Turner, M.B.
Alexander Crombie, M.D.
Thomas Herbert Kellock, M.D.
1899 James Hugh Thursfield, M.D.
Lindley Marcroft Scott, M.D.
Alfred P. Hillier, M.D.
Louis Bathe Rawling, M.B.
John Edward Sandiland, M.B.
Herbert Mundy.
Arthur J. Whiting, M.D.
W. H. Crosse, M.D.
Edward Farquhar Buzzard, M.B.
Greville Macdonald, M.D.
George Jones, M.B.
Richard Harding Bremridge.
Herbert Campbell Thomson, M.D.
Thomas Morison Legge, M.D
William John Ritchie Simpson, M.D.
Ernest Playfair, M.B.
Karl Fürth, M.D.
Purves Stewart, M.D.
1900 Clive Riviere, M.B.
H. Roe Walker.
Richard Lake.
Percy Flemming, M.D., B.S.
John Shields Fairbairn, M.B., B.Ch.

1900 Hugh Lloyd Williams.
Aslett Baldwin.
Charles Ryall.
William Hern.
Cecil Huntington Leaf, M.B.
Edwin Harding Lendon, M.D.
Lieut.-Col. William Reid Murphy, D.S.O., I.M.S.
James Harry Sequeira, M.D.
Harold Batty Shaw, M.D.
Charles Herbert Thompson, M.D.
John William Thomson-Walker.

1901 Sir Hugh Reeve Beevor, Bart., M.D.
J. Brunton Blaikie, M.D.
John Patrick Henry, M.D.
Herbert John Paterson.
George Henkell Drummond Robinson, M.D.
Elmore Wright Brewerton.

1901 Thomas Rupert Hampden Bucknall. M.S., M.D.
William Douglas Harmer.
Harry George Plimmer.
Lionel Vernon Cargill.
T. N. Kelynack, M.D.
Leonard Williams, M.D.

1902 J. P. L. Mummery, B.A.
James Stansfield Collier, M.D., B.Sc.
Robert Salusbury Trevor, M.B., B.C.
Edward Arthur Saunders, M.B., B.Ch.
Ralph Vincent, M.D., B.C.
Herbert French, M.B.
Holland John Cotton, M.D., C.M.
Arthur Evans, M.S.
James Kerr, M.D., D.P.H.
Donald John Armour, M.B.

The following Non-resident Fellows pay an annual subscription of £3 3s., and are thereby entitled to all the privileges of Resident Fellows.

Elected

1900 BLAKE, WILLIAM HENRY, M.D.Brux., Bedford Lodge, West Wickham, Kent.

1884 DRAGE, LOVELL, M.D., B.Ch.Oxon., Burleigh Mead, Hatfield, Herts.

1897 GILFORD, HASTINGS, Norwood House, King's road, Reading. *Trans.* 1.

1873 PARKER, ROBERT WILLIAM, Senior Surgeon to the East London Hospital for Children; Senior Surgeon to the German Hospital; Caryll Hurst, West Grinstead, Sussex. C. 1888-9, 1899—1901. S. 1895-8. *Bldg. Com.* 1889-92. *Referee*, 1891-5. *Lib. Com.* 1885-87. 1892-5, 1898-9. *Ho. Com.* 1892-5, 1899—1901. *Trans.* 4.

1900 PRICE-JONES, CECIL, M.B., 7, Claremont road, Surbiton, Surrey.

1882 REID, THOMAS WHITEHEAD, M.D., Surgeon to the Kent and Canterbury Hospital; St. George's House, Canterbury.

1891 RUFFER, MARC ARMAND, M.D., The Quarantine Board, Alexandria.

1898 THOMAS, J. LYNN, C.B., Surgeon to the Cardiff Infirmary; Consulting Surgeon to the Hamadryad Hospital, Green-lawn, Pen-y-Lan, Cardiff.

NON-RESIDENT FELLOWS

Elected

1866 *Allbutt, Thomas Clifford*, M.D., LL.D.Glasgow, F.R.S., Regius Professor of Physic, University of Cambridge; Consulting Physician to the Leeds General Infirmary; St. Rhadegund's, Cambridge. *Trans.* 3.

1884 *Anderson, Alexander Richard*, Surgeon to the General Hospital, 5, East Circus Street, Nottingham. *Trans.* 1.

1880 *Appleton, Henry*, M.D. (Address uncommunicated.)

1896 *Bagshawe, Frederic*, M.D., J.P., 35, Warrior Square, St Leonard's-on-Sea.

1902 *Bailey, William Henry*, M.B., Featherstone Hall, Southall, Middlesex.

1895 *Baldwin, Gerald R.*, 166, Victoria street, Melbourne, Australia.

1891 *Balgarnie, Wilfred*, M.B., The Dutch House, Hartley Wintney, Winchfield.

1896 *Ball, Charles Bent*, M.D., Ch.M., 24, Merrion square North, Dublin.

1866 *Banks, Sir John*, K.C.B., M.D., LL.D., D.Sc., Physician in Ordinary to H.M. the King in Ireland; Physician to Richmond, Whitworth, and Hardwicke Hospitals; Regius Professor of Physic in the University of Dublin; 45, Merrion square, Dublin.

1886 *Banks, Sir William Mitchell*, M.D., Surgeon to the Liverpool Royal Infirmary; 28, Rodney street, Liverpool.

Elected

1900 *Bardswell, Noel Dean*, M.D., The Sanatorium, Mundesley, Norfolk.

1882 *Barker, Frederick Charles*, M.D., Surgeon-Major, Bombay Medical Service.

1881 *Barnes, Henry*, M.D., LL.D., F.R.S. Ed., Physician to the Cumberland Infirmary; 6, Portland square, Carlisle.

1861 **Barnes**, *Robert*, M.D., Bernersmede, Eastbourne. C. 1877-8. V.P. 1889-90. *Referee*, 1867-76, 1891—. *Lib. Com.* 1869-73. *Sci. Com.* 1889—1902. *Trans.* 4.

1860 *Bealey, Adam*, M.D., M.A., Felsham Lodge, Felsham road, St. Leonard's-on-Sea, Sussex.

1896 *Belben, Frank*, M.B., Endsleigh, Suffolk road, Bournemouth.

1880 *Bennett, Alexander Hughes*, M.D. (Travelling.)

1889 *Bentley, Arthur J. M.*, M.D., Mena House, Pyramids, Cairo, Egypt.

1872 *Beverley, Michael*, M.D., Consulting Surgeon to the Norfolk and Norwich Hospital; 54, Prince of Wales road, Norwich.

1865 *Bickersteth, Edward Robert*, Consulting Surgeon to the Liverpool Royal Infirmary; 2, Rodney street, Liverpool. *Trans.* 1.

1892 *Bickersteth, Robert Alexander*, M.A., M.B., Assistant Surgeon to the Liverpool Royal Infirmary: 2, Rodney street, Liverpool.

1849 **Birkett**, *Edmund Lloyd*, M.D., Consulting Physician to the City of London Hospital for Diseases of the Chest; Westbourne Rectory, Emsworth, Hampshire. C. 1865-6. *Referee*, 1851-9.

1901 *Bisshopp, Francis R. B.*, M.D., Belle Vue, Mount Pleasant, Tunbridge Wells.

1900 *Blake, William Henry*, M.D.Brux., Bedford Lodge, West Wickham, Kent.

1865 *Blanchet, Hilarion*, 35, Conillard street, Quebec, Canada.

Elected

1890 *Bostock, R. Ashton*, Surgeon, Scots Guards, Cefn Mor, Penmaen, Glamorganshire.

1869 *Bourne, Walter*, M.D. (Travelling.)

1874 *Bradshaw, A. F.*, C.B., Surgeon Major-General, 111, Banbury road, Oxford.

1899 *Bradshaw, Thomas Robert*, M.D., 51, Rodney street, Liverpool. *Trans.* 2.

1900 *Braine-Hartnell, James Christopher Reginald*, Cotswold Sanatorium, Stroud, Glos.

1876 *Bridges, Robert*, M.B., Manor House, Yattendon, Newbury, Berks.

1867 *Bridgewater, Thomas*, M.B., LL.D., Harrow-on-the-Hill, Middlesex.

1891 *Brodie, Charles Gordon*, Fernhill, Wootton Bridge, Isle of Wight.

1892 *Bronner, Adolph*, M.D., Senior Surgeon to Bradford Eye and Ear Hospital; Laryngologist to Bradford Royal Infirmary; 33, Manor row, Bradford.

1894 *Brook, William Henry Breffit*, M.D., B.S., 8, Eastgate, Lincoln.

1899 *Brooksbank, Hugh Lamplugh*, M.B., B.C., 5, College road, Windermere.

1888 *Browne, Henry Langley*, Moor House, West Bromwich.

1881 *Browne, John Walton*, M.D., Surgeon to the Belfast Royal Hospital; Surgeon to the Belfast Ophthalmic Hospital; 10, College square N., Belfast.

1864 *Buckle, Fleetwood*, M.D., Merton Lodge, Merton road, Southsea.

1901 *Byrne, William Samuel*, M.D., Anne street, Brisbane, Queensland.

1851 *Cadge, William*, Consulting Surgeon to the Norfolk and Norwich Hospital; 49, St. Giles's street, Norwich. *Trans.* 1.

1891 *Campbell, Henry Johnstone*, M.D., 36, Manningham lane, Bradford.

Elected

1900 *Carlyon, Thomas Baxter.*

1875 *Carter, Charles Henry*, M.D., Consulting Physician to the Hospital for Women, Soho Square; 5, Homefield road, Bromley, Kent.

1888 *Carter, William Jeffreys Becher*, Aliwal North, Cape Colony.

1898 *Cave, Edward John*, M.D., Bath.

1884 *Chaffey, Wayland Charles*, M.D., Physician to the Royal Alexandra Hospital for Children; 13, Montpellier road, Brighton.

1885 *Chapman, Paul Morgan*, M.D., Physician to the Hereford General Infirmary, 1, St. John street, Hereford. *Trans.* 1.

1881 *Chavasse, Thomas Frederick*, M.D., C.M., Senior Surgeon to the Birmingham General Hospital; 22, Temple row, Birmingham. *Trans.* 3.

1873 *Chisholm, Edwin*, M.D., 44, Rosslyn gardens, Darlinghurst, Sydney, New South Wales.

1896 *Christopherson, John Brian*, M.D., B.C., Assistant Demonstrator of Anatomy at St. Bartholomew's Hospital; Surgeon to Seamen's Hospital, Albert Dock; c/o P.M.O., Egyptian Army, Cairo.

1892 *Clark, James Charles*, 35, Castle road, Bedford.

1897 *Clark, W. Gladstone*, Civil Service Club, Capetown.

1857 *Coates, Charles*, M.D., Consulting Physician to the Bath Royal United Hospital; 10, Circus, Bath.

1893 *Cole, Robert Henry*, M.D., Moorcroft, Hillingdon, Uxbridge.

1891 *Cook, Herbert George*, M.D., B.S., 22, Newport road, Cardiff.

1899 *Corrigan, William Jenkinson*, Cloughmore, Splott avenue, Cardiff.

1891 *Coumbe, John Batten*, M.D., 64, Caeran road, Newport, Mon.

1869 *Cresswell, Pearson R.*, C.B., Senior Surgeon to the Merthyr General Hospital; Dowlais, Merthyr Tydfil.

Elected

1892 *Cross, Francis Richardson*, M.B., Ophthalmic Surgeon to the Bristol Royal Infirmary, and Surgeon to the Bristol Eye Hospital; Worcester House, Clifton, Bristol.

1895 *Dardel, Jean*, M.D., Aix-les-Bains, Savoy.

1879 *Darwin Francis*, M.B., F.R.S., Wychfield, Huntingdon road, Cambridge.

1874 *Davidson, Alexander*, M.D., Consulting Physician to the Liverpool Royal Infirmary; Emeritus Professor, University College, Liverpool; 2, Gambier terrace, Liverpool.

1878 *Davy, Richard*, Consulting Surgeon to the Westminster Hospital; Burstone House, Bow, North Devon. *Trans.* 1.

1882 *Dawson, Yelverton*, M.D., Heathlands, Southbourne-on-Sea, Hants.

1889 *Delépine, Sheridan*, B.Sc., M.B., C.M., Professor of Pathology, Owens College, Manchester. *Trans.* 1.

1899 *Douglas, Archibald Robert John*, M.B., B.S., c/o Watson & Co., 7, Waterloo place, S.W.

1867 *Drage, Charles*, M.D., Hatfield, Herts.

1884 *Drage, Lovell*, M.D. Oxon., Burleigh Mead, Hatfield, Herts.

1898 *Dreschfeld, Julius*, Farndon House, Rusholme, Manchester.

1885 *Drummond, David*, M.D., 7, Saville place, Newcastle-on-Tyne.

1880 *Drury, Charles Dennis Hill*, M.D., Bondgate, Darlington.

1899 *Drury, Edward Guy Dru*, M.B., B.S., Grahamstown, South Africa.

1871 *Dukes, Clement*, M.D., B.S., Physician to Rugby School, and Senior Physician to the Hospital of St. Cross, Rugby; Sunnyside, Rugby, Warwickshire.

1867 *Dukes, Major Charles*, M.D., Clarence Villa, Torrs park, Ilfracombe, North Devon.

Elected

1889 *Duncan, John*, M.D., St. Petersburg, Russia.

1872 *Eager, Reginald*, M.D., Northwoods, near Bristol.

1887 *Easmon, John Farrell*, M.D., Assistant Colonial Surgeon, Gold Coast Colony, and Acting Chief Medical Officer of the Colony; Accra, Gold Coast, West Africa.

1887 *Elliott, John*, 24, Nicholas street, Chester.

1868 *Ellis, James*, M.D., The Sanatorium, Anaheim, Los Angeles County, California.

1889 *Elliston, William Alfred*, M.D., Stoke Hall, Ipswich.

1875 *Fagan, John*, Consulting Surgeon to the Belfast Royal Hospital; 20, Fitzwilliam place, Dublin.

1897 *Fagge, Thomas Henry*, M.D., Villa de la Porte Rouge, Monte Carlo.

1869 *Fairbank, Frederick Royston*, M.D., Westcott, Dorking.

1902 *Fennell, Charles Henry*, M.A., M.D., Darenth Asylum, Dartford, Kent.

1872 *Fenwick, John C. J.*, M.D., Physician to the Durham County Hospital; Long Framlington, Morpeth.

1879 *Finlay, David White*, M.D., Professor of the Practice of Medicine in the University of Aberdeen; Physician and Lecturer on Clinical Medicine to the Aberdeen Royal Infirmary; Consulting Physician to the Royal Hospital for Diseases of the Chest, London; 2, Queen's terrace, Aberdeen. *Referee*, 1891-3. *Trans.* 2.

1864 *Folker, William Henry*, Consulting and late Hon. Surgeon to the North Staffordshire Infirmary; Bedford House, Hanley, Staffordshire.

1896 *Forestier, Henri*, M.D., Aix-les-Bains, Savoie, France.

1892 *Foster, Michael George*, M.A., M.B., Villa Camilla, San Remo.

1884 *Franks, Kendal*, M.D., c/o J. H. Franks, Esq., C.B., Dalriada, Blackrock, co. Dublin. *Trans.* 2.

1876 *Furner, Willoughby*, M.D., Surgeon to the Sussex County Hospital; Brunswick square, Brighton.

Elected

1864 *Gairdner, Sir William Tennant*, M.D., K.C.B., LL.D., F.R.S., Honorary Physician in Ordinary to H.M. the King in Scotland; formerly Professor of the Practice of Medicine in the University of Glasgow; Honorary Consulting Physician to the Western Infirmary, Glasgow; 32, George square, Edinburgh. *Trans.* 1.

1885 *Gamgee, Arthur*, M.D., F.R.S., Emeritus Professor of Physiology in the Owens College, Victoria University, Manchester; Montreux, Switzerland.

1867 *Garland, Edward Charles*, Yeovil, Somerset.

1879 *Garstang, Thomas Walter Harropp*, Englefield, Delamer road, Bowdon, Cheshire.

1889 *Gaskell, Walter Holbrook*, M.D., F.R.S., Lecturer on Physiology, University of Cambridge; The Uplands, Great Shelford, Cambs.

1884 *Gibbes, Heneage*, M.D., Health Officer, Detroit, Michigan, U.S.A.

1897 *Gibson, George Alexander*, M.D., D.Sc., 3, Drumsheugh Gardens, Edinburgh.

1897 *Gilford, Hastings*, Norwood House, King's road, Reading. *Trans.* 2.

1893 *Gordon, William*, M.B., M.C., The Old Rectory, Goring-on-Thames, Oxon.

1890 *Gordon, William*, M.D., Barnfield Lodge, Exeter.

1898 *Granville, Alexander*, Turf Club, Cairo.

1898 *Gray, J. A.*, M.B., Wadham Lodge, Uxbridge road, Ealing.

1889 *Greene, George Edward Joseph*, M.A., D.Sc., F.L.S., Monte Vista, Ferns, County Wexford.

1875 *Greenfield, William Smith*, M.D., Professor of Pathology and Clinical Medicine in the University of Edinburgh; 7, Heriot row, Edinburgh. *Sci. Com.* 1879. *Referee*, 1881.

1900 *Greer, William Jones*, 2, Chepstow road, Newport, Mon.

1882 *Gresswell, Dan Astley*, M.A., M.D., D.P.H., Chairman, Board of Public Health, Melbourne, Victoria.

Elected

1889 *Griffiths, Joseph*, M.A., M.D., C.M., Reader in Surgery in the University of Cambridge; Surgeon to Addenbrooke's Hospital; 63, Trumpington street, Cambridge. *Pro.* 1.

1870 *Hamilton, Robert*, Consulting Surgeon to the Royal Southern Hospital, Liverpool; Magherabuoy, Portrush, co. Antrim, Ireland.

1864 **Harley,** *John*, M.D., F.L.S., Hon. Physician to St. Thomas's Hospital; Consulting Physician to the London Fever Hospital; Beeding, Pulborough, Sussex. S. 1875-7. C. 1879-80. V.P. 1895-7. *Referee*, 1871-4, 1882-95. *Sci. Com.* 1879. *Trans.* 10.

1901 *Hartigan, T. J. P.*, "Heathcote," East Grinstead, Sussex.

1854 *Haviland, Alfred*, Ridgemount, Frimley Green, Surrey.

1890 *Haviland, Frank Papillon*, M.D., B.C., 57, Warrior square, St. Leonard's-on-Sea.

1885 *Hawkins, Francis Henry*, M.D., Physician to the Royal Berkshire Hospital; 73, London street, Reading. *Trans.* 1.

1900 *Hayford, Ernest James*, M.D., c/o The Agent, Claude's Ashanti Goldfields, Limited, Cape Coast Castle, Gold Coast.

1860 **Hayward,** *Henry Howard*, Consulting Surgeon Dentist to St. Mary's Hospital; Harbledown, 120, Queen's road, Richmond. C. 1878-9.

1861 *Hayward, William Henry*, Oxford road, Burnley, Lancashire.

1899 *Hind, Henry*, Harrogate.

1900 *Hobhouse, Edmund*, M.D., 36, Brunswick place, Brighton.

1843 **Holden,** *Luther*, Consulting Surgeon to St. Bartholomew's Hospital, Pinetoft, Ipswich. C. 1859. L. 1865. V.P. 1874. *Referee*, 1866-7. *Lib. Com.* 1858.

Elected

1894 *Holland, James Frank*, M.D., St. Moritz, Engadine, Switzerland.

1868 *Hollis, William Ainslie*, M.D., Physician to the Sussex County Hospital ; 1, Palmeira avenue, Hove. *Trans.* 1.

1881 *Howard, Henry*, M.B., Medical Officer of Health, Williamstown, Melbourne, Victoria.

1898 *Hulke, S. Backhouse*, Ivy House, Walmer, Kent.

1882 *Humphry, Laurence*, M.D., 3, Trinity street, Cambridge.

1847 *Image, William Edmund*, Herringswell House, Mildenhall, Suffolk. *Trans.* 1.

1883 *Jenkins, Edward Johnstone*, M.D., The Australian Club, Sydney, New South Wales.

1881 *Jennings, William Oscar*, M.D., 74, Avenue Marceau, Paris.

1901 *Johnson, Edward Angas*, M.B., St. Catharine's, Prospect, South Australia.

1889 *Johnson, Harold J.*, Senior Assistant, Gloucester County Asylum, Gloucester.

1876 *Jones, Leslie Hudson*, M.D., Limefield House, Cheetham hill, Manchester.

1875 *Jones, Philip Sydney*, M.D., Consulting Surgeon to the Sydney Infirmary; 10, College street, Sydney, New South Wales. [Agents: Messrs. D. Jones & Co., Wool Exchange, Coleman Street, E.C.]

1865 *Jordan, Furneaux*, Consulting Surgeon to the Queen's Hospital, Birmingham ; Harborne, near Birmingham.

1872 *Kelly, Charles*, M.D., Ellesmere, Gratwicke road, Worthing, Sussex.

1890 *Kerr, J. G. Douglas*, M.B., C.M., 6, The Circus, Bath.

1884 *Keser, Jean Samuel*, M.D., Villa St. Martin, Vevey, Switzerland.

Elected

1877 *Khory, Rustomjee Naserwanjee,* M.D.Brux., Hormazd Villa, Khumballa hill, Bombay.

1898 *Klefstad-Sillonville, O.,* M.D., Aix-les-Bains, Savoie.

1888 *Kynsey, Sir William Raymond,* C.M.G., Westfield, Catherine road, Surbiton. (Travelling.)

1889 *Lancaster, Ernest le Cronier,* M.B., B.Ch., Assistant Physician to the Swansea Hospital; Hon. Physician to the Swansea and South Wales Institution for the Blind; Winchester House, Swansea, S. Wales.

1873 *Larcher, O.,* M.D., Laureate of the Institute of France, of the Medical Faculty, and Academy of Paris, &c.; 97, Rue de Passy, Passy, Paris.

1862 *Latham, Peter Wallwork,* M.D., Downing Professor of Medicine, Cambridge University, 1874–94; Senior Physician to Addenbrooke's Hospital, Cambridge; 17, Trumpington street, Cambridge.

1880 *Laycock, George Lockwood,* M.B., C.M., Melbourne, Victoria, Australia.

1892 *Lazarus-Barlow, Walter Sydney,* M.D., Cecil House, Cavendish road, Sutton, Surrey. *Sci. Com.* 1892—1902.

1886 *Lediard, Henry Ambrose,* M.D., Surgeon to the Cumberland Infirmary; 35, Lowther street, Carlisle. *Trans.* 1.

1882 *Ledwich, Edward l'Estrange,* Anatomist to the Royal College of Surgeons, Ireland; 30, Upper Fitzwilliam street, Dublin.

1883 *Leeson, John Rudd,* M.D., C.M., Clifden House, Twickenham.

1869 *Legg, John Wickham,* M.D. C. 1886. *Referee,* 1882-5. *Lib. Com.* 1878-85. *Trans.* 2.

1898 *Lindsay, James,* M.A., M.D., 13, College square East, Belfast.

Elected

1889 *Little, James*, M.D., Physician to the Adelaide Hospital; 14, Stephen's Green North, Dublin.

1894 *Lowe, Thomas Pagan*, 16, The Circus, Bath.

1889 *MacAlister, Donald*, M.A., B.Sc., M.D., Physician to Addenbrooke's Hospital; Linacre Lecturer and Tutor, St. John's College; University Lecturer in Medicine; St. John's College, Cambridge.

1887 *Macdonald, George Childs*, M.D. (Address uncommunicated.)

1866 *Macgowan, Alexander Thorburn*, M.D., Vyvian House, Clifton park, Bristol.

1859 *M'Intyre, John*, M.D., LL.D., Odiham, Hants.

1876 *Mackey, Edward*, M.D., Physician to the Sussex County Hospital; Senior Physician to the Royal Alexandra Hospital for Sick Children; 56, Lansdowne place, Brighton.

1854 *Mackinder, Draper*, M.D., 12, Park View Villas, Hove, Sussex.

1893 *MacLeod, Surgeon-Colonel Kenneth*, M.D., The Towers, Woolston, S. Hants.

1876 *Macnamara, N. Charles*, Consulting Surgeon to the Westminster Hospital, and to the Royal Westminster Ophthalmic Hospital; The Lodge, Chorley Wood. C. 1891-2. V.P. 1902—. *Referee*, 1884-90, 1895-7. *Lib. Com.* 1886-90.

1891 *Manby, Alan Reeve*, M.V.O., M.D., Surgeon Apothecary to His Majesty's Household at Sandringham and to T.R.H. the Prince and Princess of Wales at Sandringham; East Rudham, Norfolk.

1894 *Marriott, Charles William*, M.D., Aubrey House, Bath road, Reading.

1892 *Martin, Christopher*, M.B., C.M., Surgeon to the Birmingham and Midland Hospital for Women; 35. George road, Edgbaston, Birmingham.

Elected

1899 *Martyn, Gilbert John King*, M.D., 8, Gay street, Bath.

1883 *Maudsley, Henry Carr*, M.D., 22, Collins street, Melbourne, Victoria.

1839 *Meade, Richard Henry*, Consulting Surgeon to the Bradford Infirmary; Bradford, Yorkshire. *Trans.* 1.

1897 *Merry, William Joseph Collings*, M.D., B.Ch., 2, Chiswick place, Eastbourne.

1898 *Millard, William Joseph Kelson*, M.D., 7, Bayshill villas, Cheltenham.

1895 *Mills-Roberts, Robert Herbert*, Hafod-ty, Llanberis, North Wales.

1896 *Moore, Sir John*, M.D., 40, Fitzwilliam square west, Dublin.

1891 *Morris, Graham*, Wallington, Surrey.

1894 *Morse, Thomas Herbert*, All Saints' Green, Norwich. *Trans.* 1.

1902 *Moynihan, Berkeley George Andrew*, M.S., 33, Park square, Leeds.

1892 *Myddelton-Gavey, E. Herbert*, 16, Broadwater Down, Tunbridge Wells.

1881 *Nall, Samuel*, M.B., Dryhurst Lodge, Disley, Stockport.

1889 *Napier, Francis Horatio*, M.B., Cape Town.

1870 *Neild, James Edward*, M.D., Lecturer on Forensic Medicine and Psychological Medicine in the University of Melbourne; 21, Spring street, Melbourne, Victoria.

1895 *Newsholme, Arthur*, M.D., 11, Gloucester place, Brighton.

1868 *Nicholls, James*, M.D., Trekenning House, St. Columb, Cornwall.

1847 *Nourse, William Edward Charles*, Norfolk Lodge, Thurloe road, Torquay.

1884 *Oakes, Arthur*, M.D., Narrabri, Cole Park road, Twickenham.

1880 *O'Connor, Bernard*, A.B., M.D., Senior Physician to the North London Hospital for Consumption; 25 Hamilton road, Ealing.

Elected

1855 *Ogle, William*, M.A., M.D., Consulting Physician to the Royal Derbyshire Infirmary; The Elms, Duffield road, Derby.

1870 *Oldham, Charles Frederic*, India [Agents: Messrs. Grindlay and Co., 55, Parliament street].

1896 *Oliver, George*, M.D., Riversleigh, Farnham, Surrey, and Harrogate.

1883 *Oliver, Thomas*, M.A., M.D., Professor of Physiology, University of Durham; and Physician to the Newcastle-on-Tyne Infirmary; 7, Ellison place, Newcastle-on-Tyne. *Trans.* 1.

1871 *O'Neill, William*, M.D., C.M., late Physician to the Lincoln Lunatic Hospital, and Physician, Lincoln General Dispensary, &c.; 2, Lindum road, Lincoln.

1890 *Ord, William Wallis*, M.D., The Hall, Salisbury.

1885 *Ormsby, L. Hepenstal*, M.D., Lecturer on Clinical and Operative Surgery and Surgeon to the Meath Hospital and County Dublin Infirmary; Surgeon to the Children's Hospital, Dublin; 92, Merrion square West, Dublin.

1887 *Paget, Charles Edward*, Medical Officer of Health to the County Council of Northamptonshire; County Hall, Northampton.

1858 *Paley, William*, M.D., Physician to the Ripon Dispensary; Yore Bank, Ripon, Yorkshire.

1887 *Pardington, George Lucas*, M.D., 47, Mount Pleasant road, Tunbridge Wells.

1873 *Parker, Robert William*, Senior Surgeon to the East London Hospital for Children; Senior Surgeon to the German Hospital; Caryll Hurst, West Grinstead, Sussex. C. 1888-9, 1899—1901. S. 1895-8. *Bldg. Com.* 1889-92. *Referee*, 1891-5. *Lib. Com.* 1885-87, 1892-5, 1898-9. *Ho. Com.* 1892-5, 1899—1901. *Trans.* 4.

1885 *Parker, Rushton*, M.B., B.S., Professor of Surgery, University College, Liverpool (Victoria University); Surgeon to the Liverpool Royal Infirmary; 59, Rodney street, Liverpool.

Elected

1891 *Parkin, Alfred*, M.S., M.D., 24, Albion street, Hull. *Trans.* 1.

1879 *Peel, Robert*, 120, Collins street East, Melbourne, Victoria.

1874 *Penhall, John Thomas*, The Cedars, Broadwas-on-Teme, Worcester.

1897 *Perram, Charles Herbert*, M.D., 55, Bromham Road, Bedford.

1879 *Pesikaka, Hormasji Dosabhai*, 43, Hornby road, Bombay.

1878 *Philipson, Sir George Hare*, M.D., D.C.L., Professor of Medicine in Durham University; Consulting Physician to the Newcastle-upon-Tyne Royal Infirmary; 7, Eldon square, Newcastle-upon-Tyne.

1898 *Phillips, L. C. Powell*, Kasr-el-Aini Hospital, Cairo.

1891 *Pierce, Bedford*, M.D., The Retreat, York.

1897 *Pigg, T. Strangeways*, St. John's College, Cambridge.

1841 **Pitman**, *Sir Henry Alfred*, M.D., Consulting Physician to St. George's Hospital; Cranbrook, Bycullah park, Enfield. L. 1851-3. C. 1861-2. T. 1863-8. V.P. 1870-1. *Referee*, 1849-50. *Lib. Com.* 1847.

1892 *Powell, Herbert Andrews*, M.A., M.D., M.Ch., Piccards Rough, Guildford.

1900 *Price-Jones, Cecil*, M.B., 7, Claremont road, Surbiton, Surrey.

1897 *Quartey-Papafio, Benjamin William*, M.D., Accra, Gold Coast, West Africa.

1857 *von Ranke, Henry*, M.D., 3, Sophienstrasse, Munich.

1890 *Ransom, William Bramwell*, M.D., Physician to the Nottingham General Hospital; The Pavement, Nottingham. *Trans.* 1.

1854 *Ransom, William Henry*, M.D., F.R.S., Consulting Physician to the Nottingham General Hospital; 17, Park Valley, Nottingham. *Trans.* 1.

1902 *Raw, Nathan*, M.D., B.S., 66, Rodney street, Liverpool.

Elected

1884 *Reid, Thomas Whitehead*, M.D., Surgeon to the Kent and Canterbury Hospital; St. George's House, Canterbury, Kent.

1901 *Reissmann, Charles Henry*, M.D., B.C., B.Sc., St. Peter's, College Green, Adelaide, South Australia.

1881 *Rice, George*, M.B., C.M., Sutton, Surrey.

1889 *Rivers, W. H. Rivers*, M.D., St. John's College, Cambridge.

1871 *Roberts, David Lloyd*, M.D., F.R.S.E., Consulting Obstetric Physician to the Manchester Royal Infirmary; Physician to St. Mary's Hospital, and Lecturer on Clinical Obstetrics and Gynæcology at the Owens College, Manchester; 11, St. John street, Manchester.

1889 *Roberts, Leslie*, M.D., 46, Rodney street, Liverpool.

1873 *Robertson, William Henry*, M.D., Consulting Physician to the Buxton Bath Charity and Devonshire Hospital; Buxton, Derbyshire.

1888 *Robinson, Frederick William*, M.D., C.M., Huddersfield.

1885 *Rockwood, William Gabriel*, M.D., Colombo, Ceylon.

1898 *Rogers, Leonard*, I.M.S. [care of Messrs. Watson & Co., Calcutta.] *Trans.* 2.

1863 *Rowe, Thomas Smith*, M.D., Consulting Surgeon to the Royal Sea-Bathing Infirmary; Union crescent, Margate, Kent.

1891 *Ruffer, Marc Armand*, M.D., The Quarantine Board, Alexandria.

1898 *Salter, A.*, M.D.

1855 **Sanderson**, *Sir John Burdon, Bart.*, M.D., LL.D., D.C.L.Durham, D.Sc., F.R.S., Regius Professor of Medicine in the University of Oxford; 64, Banbury road, Oxford. C. 1869-70. V.P. 1882. *Referee*, 1867-8, 1876-81. *Sci. Com.* 1862, 1870. *Lib. Com.* 1876-81. *Trans.* 2.

Elected

1867 *Sandford, Folliott James*, M.D., V.D., late Surgeon-Major, 2nd Batt. S.V.L.Infy., now Hon. Surgeon-Major; Surgeon to the Market Drayton Dispensary, and Consulting Physician to the Market Drayton Cottage Hospital ; Market Drayton, Shropshire.

1886 *Saundby, Robert*, M.D., LL.D., Physician to the General Hospital, and Consulting Physician to the Hospital for Women, and to the Eye Hospital, Birmingham ; Professor of Medicine, Mason University College; 140B, Great Charles street, Birmingham.

1891 *Saunders, Frederick William*, M.B., B.C., Chieveley House, near Newbury, Berks.

1883 *Schäfer, Edward Albert*, LL.D., F.R.S., Professor of Physiology in the University of Edinburgh. C. 1899-1900. *Referee*, 1888-99. *Sci. Com.* 1889—.

1861 *Scott, William*, M.D., Senior Physician to the Huddersfield Infirmary ; Waverley House, Huddersfield.

1897 *Semple, Edward*, M.D., Grove house, Fenstanton, Hunts.

1897 *Seymour, Surg.-Major Charles*, Bareilly, North-West Provinces, India.

1899 *Shuttleworth, George Edward*, M.D., Ancaster House, Richmond Hill.

1887 *Sidebotham, Edward John*, M.B., Erlesdene, Bowdon, Cheshire.

1857 *Siordet, James Lewis*, M.B., Villa Cabrolles, Mentone, Alpes Maritimes, France.

1896 *Sloane, John Stretton*, M.B., B.S., B.Sc., 7, Highfield street, Leicester.

1891 *Smith, G. Cockburn*, M.D., 29, Lansdown crescent, Cheltenham.

1886 *Smith, Howard Lyon*, Buckland House, Buckland Newton, Dorchester.

1894 *Smith, Robert Shingleton*, M.D., B.Sc., Deepholm, Clifton Park, Clifton, Bristol.

1894 *Smith, Thomas Rudolph*, M.B., B.C., Blytheholm, Stockton-on-Tees.

Elected

1868 *Solly, Samuel Edwin,* Colorado Springs, Colorado, U.S.A.

1899 *Stephen, Guy Neville,* Foreign Office Medical Staff.

1891 *Stevens, Surg.-Capt. Cecil Robert,* M.B., B.S., I.M.S., Eden Hospital, Calcutta.

1854 *Stevens, Henry,* M.D., late Inspector, Medical Department, Local Government Board, Whitehall; Durham Lodge, St. Margaret's road, Twickenham.

1884 *Stewart, Edward,* M.D., Brook House, East Grinstead.

1879 *Stirling, Edward Charles,* M.D., Senior Surgeon to the Adelaide Hospital; Lecturer on Physiology in the University of Adelaide, South Australia [care of Messrs. Elder and Co., 7, St. Helen's place].

1871 *Strong, Henry John,* M.D., J.P., Consulting Surgeon to the Croydon General Hospital; Colonnade House, The Steyne, Worthing.

1890 *Sympson, E. Mansel,* M.D., B.C., Surgeon to the Lincoln County Hospital; Deloraine Court, Lincoln.

1886 *Teale, Thomas Pridgin,* M.B., F.R.S., Consulting Surgeon to the Leeds General Infirmary; 38, Cookridge street, Leeds.

1898 *Thomas, J. Lynn,* C.B., Surgeon to the Cardiff Infirmary; Consulting Surgeon to the Hamadryad Hospital; Green Lawn, Pen-y-lan, Cardiff.

1890 *Thomas, William Robert,* M.D., Little Forest, Bath road, Bournemouth.

1891 *Thomson, John Roberts,* M.D., Monkchester, Bournemouth.

1876 *Thornton, J. Knowsley,* M.B., C.M., Consulting Surgeon to the Samaritan Free Hospital for Women and Children; Hildersham Hall, Cambridge. C. 1891. *Lib. Com.* 1886-90, 1893-95. *Trans.* 5.

1883 *Thursfield, Thomas William,* M.D., Physician to the Warneford and South Warwickshire General Hospital; Selwood, Beauchamp square, Leamington.

Elected

1880 *Tivy, William James*, 8, Lansdowne place, Clifton, Bristol.

1871 *Trend, Theophilus W.*, M.D., 1, Grosvenor square, Southampton.

1881 *Treves, William Knight*, Surgeon to the National Hospital for Scrofula; 31, Dalby square, Cliftonville, Margate.

1867 *Trotter, John William*, formerly Surgeon-Major, Coldstream Guards; 4, St. Peter's terrace, York.

1873 *Turner, George Brown*, M.D., Camden House, Hemel Hempsted, Herts.

1894 *Turner, Philip Dymock*, M.D., Sudbury, Isle of Wight.

1891 *Tweed, Reginald*, M.D., Hembury Fort Cross, Honiton, Devon.

1881 *Tyson, William Joseph*, M.D., Senior Medical Officer of the Victoria Hospital, Folkestone; 10, Langhorne Gardens, Folkestone.

1900 *Uhthoff, John Caldwell*, M.D., Wavertree House, Hove, Brighton.

1867 **Vintras**, *Achille*, M.D., late Physician to the French Embassy and Senior Physician to the French Hospital and Dispensary, Shaftesbury avenue; De Courcel road, Brighton.

1854 *Waddington, Edward*, Hamilton, Auckland, New Zealand.

1868 *Walker, Robert*, Clovelly, Bideford.

1867 *Wallis, George*, Consulting Surgeon to Addenbrooke's Hospital; 6, Hills road, Cambridge.

1899 *Walters, Frederick Rufenacht*, M.D., Crooksbury Sanatorium, Farnham, Surrey.

1883 *Walters, James Hopkins*, Surgeon to the Royal Berkshire Hospital; 15, Friar street, Reading.

1899 *Warde, Wilfred Brougham*, M.D., 13, Lonsdale Gardens, Tunbridge Wells.

1846 *Ware, James Thomas*, Tilford House, near Farnham, Surrey.

Elected

1861 *Waters, A. T. Houghton*, M.D., Consulting Physician to the Royal Infirmary; 69, Bedford street, Liverpool. *Trans.* 3.

1874 *Wells, Harry*, M.D., San Ysidro, Buenos Ayres, S. America.

1882 *Wharry, Charles John*, M.D., 14, Ewell road, Surbiton, Surrey.

1897 *White, Charles Powell*, 2, Blandford Gardens, Woodhouse lane, Leeds.

1881 *Whitehead, Walter*, F.R.S. Ed., Senior Surgeon to the Manchester Royal Infirmary, Manchester and Salford Lock Hospital, and Manchester and Salford Skin Hospital; Professor of Clinical Surgery, Owens College, Victoria University; 499, Oxford road, Manchester. *Trans.* 1.

1885 *Whitla, Sir William*, M.A., M.D., Professor of Materia Medica and Therapeutics, Queen's College, Belfast; Physician to, and Lecturer in Medicine at, the Belfast Royal Hospital; Consulting Physician to the Ulster Hospital for Women and Children; Consulting Physician to the Belfast Ophthalmic Hospital; 8, College square north, Belfast.

1870 *Wilkin, John F.*, M.D., Rose Ash Court, South Molton, Devon.

1883 *Willans, William Blundell*, Much Hadham, Herts.

1896 *Williams, Alfred Henry*, M.D., Rotorua, Harrow.

1859 *Williams, Charles*, Senior Surgeon to the Norfolk and Norwich Hospital; 48, Prince of Wales road, Norwich.

1887 *Wilson, Arthur Hervey*, M.D., 504, Broadway, Boston, U.S.A.

1889 *Wise, A. Tucker*, M.D., Montreux, Switzerland.

1850 *Wise, Robert Stanton*, M.D., Consulting Physician to the Southam Eye and Ear Infirmary; Beech Lawn, Banbury.

1885 *Wolfenden, Richard Norris*, M.D., Rangemont, Seaford. Sussex.

Elected

1892 *Woodhead, German Sims*, M.D., Professor of Pathology in the University of Cambridge; 6, Scrope terrace, Cambridge.

1879 *Woodward, G. P. M.*, M.D., Deputy Surgeon-General; 157, Liverpool street, Hyde Park, Sydney, New South Wales.

1892 *Wright, Almroth Edward*, M.D., Ch.B., Oakhurst, Netley, Hants.

1899 *Wynter, Andrew Ellis*, M.D., Corner House, Beckenham, Kent.

ANNUAL MEETING.

March 1st, 1902, at 5 p.m.

Present—F. W. PAVY, M.D., LL.D., F.R.S., President;

Sir THOMAS BARLOW, Bart., M.D., } Hon. Secs.;
CLINTON T. DENT, }

and 32 Fellows.

The President nominated Drs. Horton-Smith and Goodall as Scrutineers, and declared the Ballot open until six o'clock.

The Report of the Council, including the Treasurers' Report, was read by the Senior Honorary Secretary.

REPORT OF THE COUNCIL.

The Council has pleasure in reporting that the position of the Society, both as regards its financial stability and the promotion of its principal objects, is entirely satisfactory.

The number of new Fellows added during the past year is not so large as usual, but this is probably the result of the exceptionally large increase which has taken place during the four or five preceding years.

During the past year there have been elected 12 Resident Fellows and 5 Non-resident Fellows, while 9

Fellows have been lost by death and 6 by resignation. The Roll of Fellows now stands as follows:

Honorary Fellows—English	6	
Foreign	16	
	—	22
Fellows—Resident	526	
Non-resident	289	
	—	815
Total		837

In view of the termination in 1904 of the Berners Street lease, which produces a profit rental of about £435, and of the fact that the income of the Society would then be insufficient, without interference with the vigorous working of the Society, to meet the annually increasing expenditure and the repayment of the debentures, the Council felt compelled to take into serious consideration the utilisation of other resources. They came to the conclusion that the best and, as it appeared, the only adequate means of increasing the income of the Society, to meet the prospective loss of rent, was to let off the rooms hitherto occupied by the Resident Librarian. These rooms were estimated to be of the value of from £400 to £500 per annum. On receiving an offer, which had to be dealt with immediately, of £270 per annum for the use of the meeting room, and of three of the ten rooms occupied by their resident officer, they decided to accept it, and to make a suitable arrangement with the resident officer to meet the altered circumstances. There are still seven rooms to be let, and the offers now before the House Committee justify the belief that the income of the Society will be substantially and permanently increased by the change.

The arrangement made led the Council to consider the whole question of the service of the Society.

For some years it has been apparent that the business operations of the Society could not be carried on satisfac-

torily by the officer, who was responsible also for the daily work of the Library, and that a division of duties had become absolutely necessary. After earnest consideration the Council decided that the work of the Society would be better done—and responsibility more fairly apportioned—by a Secretary and a Librarian. They therefore appointed Mr. MacAlister to be Secretary and Mr. Clarke to be Librarian, with a clear definition of their respective duties. Mr. MacAlister's long experience as Resident Librarian will still be at the service of the Fellows who may desire to consult him on matters connected with the Library.

The changes in the Bye-laws rendered necessary by these decisions are submitted for confirmation, and the Council have confidence in looking to the Fellows to indemnify them for the action that was taken under the exigency of the circumstances that existed.

The new arrangement as to papers works extremely well, and most authors have availed themselves of the privilege of printing their papers in the journals immediately after being read.

The following Reports have been received:

Report of the Honorary Librarians.

"The Honorary Librarians have pleasure in reporting on the steady growth and increased usefulness of the Library.

"There have been added to the Library during 1901 a total of 686 volumes, 355 of which have been received as gifts from Fellows and others. In addition to these the use of 258 volumes of new books, much in demand, has been obtained from Lewis's Library.

"The total number of books issued to Fellows was 3683: in addition to these a very large number of books has been used by Fellows in the Library.

"The question of increased accommodation for books has become rather pressing, and is now

under the serious consideration of the Library Committee. Along with this question will be considered the advisability of new shelf notation to make the reference from the catalogue to books more direct than is possible under the present arrangement."

NORMAN MOORE.
R. J. GODLEE.

Report of Committee on Climates and Baths.

"I beg to state that during the past year the publication of the concluding volume of 'The Climates and Baths of Great Britain and Ireland' has been taken in hand by the Committee with the sanction of the Council.

"This is now being vigorously pushed forwards, and Messrs. Macmillan have promised that an advanced copy shall be in the hands of the President at the Annual General Meeting on March 1st.

"The publication of the volume will complete the work of the Committee."

P. HORTON-SMITH.

Report of the Honorary Treasurers.

"The Honorary Treasurers report that the financial position of the Society is in a satisfactory condition. The surplus of assets over liabilities amounts to £27,919 3*s.* 4*d.*, showing an increase in the assets during the year of £769 13*s.* The income has been well maintained, the total for the year amounting to £4319 10*s.* 9*d.*, showing £8 4*s.* 2*d.* less than in 1900; this difference is more than accounted for by the diminution in the number of entrance fees paid during the year."

W. S. CHURCH,
WARRINGTON HAWARD.

The Council has passed the following resolution:

"The Council desires to express its warm appreciation of the valuable work performed by the members of the Committee appointed to investigate the Medical Climatology and Balneology of Great Britain and Ireland, and to tender its best thanks to those who have aided the Society in this work."

The President moved—

"That the Report of the Council, together with the Treasurers' audited Statement of Accounts, be adopted."

After some discussion this was carried.

The President moved—

"That the following alterations in the Bye-laws, which have been made by the Council under the powers conferred on them by the Charter, be and are hereby confirmed,—that is to say: BYE-LAWS—CHAP. XI, including Sections I, II, III, and IV, are rescinded, and the following substituted therefor:

I. The Secretary shall either not be a Fellow of the Society; or, if a Fellow, shall cease to be so on his election to and acceptance of that office.

II. The Secretary shall give such security as may be required by the Council.

III. The Secretary shall transact the general business of the Society and conduct its correspondence under the direction of the Council and of the House Committee; he shall receive all papers submitted for reading, send them to the appointed referees, enter the referees' reports in the proper book, and pass the Society's publications through the press; he shall supervise the servants of the Society in their work, and be responsible for their orderly conduct; he shall supervise and take care of the Society's premises and other property, report on defects, and deal with emergencies; he shall be in attendance during

all meetings of the Society, of the Council, and of the House Committee, and shall attend daily at the Society's House for such time as shall be fixed by the Council in the "Standing Orders." He shall further generally assist the Hon. Secretaries in the non-scientific part of their work.

IV. The Librarian shall be responsible for the arranging and cataloguing of the books, and generally for the proper conduct of the Library; he shall attend daily between the hours of 10 a.m. and 6.30 p.m.; he shall be in attendance at the meetings of the Library Committee, and shall submit to the Hon. Librarians and the Library Committee all books recommended to be added to the Library or offered for purchase, and be responsible for the keeping in proper order of the books and other contents of the Library.

CHAP. XII, Sect. I, line 2.—The word 'resident' is deleted.

CHAP. XV, Sect. III, lines 3 and 4.—The word 'Secretary' is substituted for the words 'Resident Librarian.'"

Carried *nem. con.*

The President read the Annual Address (see p. xcix).

The usual votes of thanks to the retiring President and other officers, and members of Council, were carried unanimously.

At six o'clock the President called upon the Scrutineers to close the ballot, and to report the result.

The Scrutineers announced the result of the ballot to be as follows:

President.—Alfred Willett.

Vice-Presidents.—Sir Richard Douglas-Powell, Bart., M.D., K.C.V.O.; Sir Dyce Duckworth, M.D., LL.D.; N. Charles Macnamara; Edgcombe Venning.

Honorary Treasurers.—Sir William Selby Church, Bart., M.D.; J. Warrington Haward.

Honorary Secretaries.—George Newton Pitt, M.D.; Clinton Thomas Dent.

Honorary Librarians.—Norman Moore, M.D.; Rickman J. Godlee, M.S.

Members of Council.—James Kingston Fowler, M.D.; Archibald Edward Garrod, M.D.; Francis de Havilland Hall, M.D.; Isambard Owen, M.D.; Amand Jules McConnell Routh, M.D.; Walter Hamilton Acland Jacobson; Henry Edward Juler; Charles R. B. Keetley; Charles Barrett Lockwood; Thomas Laurence Read.

The President then installed the President elect, Mr. ALFRED WILLETT, in the chair, and invested him with the President's badge and master-key.

The President elect briefly thanked the Fellows for electing him, and declared the meeting closed.

(A) Income and Expenditure Account for the Year ending 31st December, 1901

Expenditure

	£	s.	d.	£	s.	d.
Rent, Rates, and Taxes	215	12	0			
Salaries of Library Staff and Accountant	594	5	0			
House Servants, Cleaners, etc. ..	240	17	0			
Lighting, Warming, and Cleaning ..	280	6	11			
Printing and Stationery, Stamps and Telegrams	173	4	0			
Meeting Expenses	36	2	6			
Miscellaneous Disbursements ..	79	15	3			
				1620	2	8
Repairs, Alterations, Furniture, etc. ..				127	17	10
Interest on Debentures . ..				1032	10	11
Depreciation of Library Purchases ..	256	7	5			
Depreciation of Fixtures, Fittings, etc. ..	58	2	7			
				314	10	0
Architect's Fees				29	13	0
Scientific Committee				12	14	6
'Transactions'				294	13	8
Law Charges				25	17	0
Audit Fee				10	10	0
Cost of Addresses to the King and Professor Virchow				16	2	6
Balance, being excess of Income over Expenditure during the year ..				834	18	8
				£4319	10	9

Income

	£	s.	d.	£	s.	d.
448 *Annual Subscriptions at £3 3s.* ..	1411	4	0			
94 *do. £1 1s.* ..	98	14	0			
				1509	18	0
Composition Fees				88	4	0
Entrance Fees				91	7	0
Rents Receivable				2549	17	0
Sale of 'Transactions'				61	5	3
Interest on New South Wales Stock ..				12	6	11
Miscellaneous Receipts				6	12	7
				£4319	10	9

Audited and approved,
TOM MUNDY,
Chartered Accountant.

February, 1902.

LIABILITIES.		£ s. d.
3 per Cent. First Mortgage Debentures . .		34,400 0 0
[The Debenture Debt has been reduced by £100 since the previous statement, by cash paid in respect to Debentures redeemed.]		
Sundry Creditors		765 13 4
Debenture Interest not claimed		15 11 0
Endowment Fund Account		5 5 0
[Surplus 31st December, 1900	£27,084 4 8	
Add—		
Excess of Income over Expenditure, 1901 . .	834 18 8	
	£27,919 3 4]	
Balance, being Surplus of Assets over Liabilities		27,919 3 4
		£63,105 12 8

ASSETS.			£ s. d.
Freehold and Leasehold Property (as per valuation of Messrs. Giddy & Giddy, 2 June, 1896).			51,150 0 0
Fixtures, Fittings, and Furniture	£1162 11 9		
Less 5 per cent. written off for depreciation . . .	58 2 7		1104 9 2
Engravings . . . . (as per valuation of Mr. F. B. Daniell, 19 August, 1896).			555 0 0
Contents of Library as on December 31st, 1900 . .	8536 7 2		
Added in 1901 . £512 14 10			
Less depreciation (50 per cent.) 256 7 5			
	256 7 5		8792 14 7
Stock of 'Climates and Baths' .			128 0 7
Investment—"Permanent Endowment Fund" . . . . (New South Wales 4 per Cent. Inscribed Stock).			326 7 3
Sundry Debtors for Rents and Outstanding Subscriptions .			633 7 6
Cash at Bank (Debenture Account)			15 11 0
Cash at Bank and in hand . .			400 2 7
			£63,105 12 8

NOTE.—The Society is also possessed of £640 11*s.* 11*d.* Consols, but as the sum in question is held in trust for a specific purpose, viz. the Marshall Hall Memorial Prize Fund, the capital sum has not been included amongst the assets of the Society.

Audited and approved,
TOM MUNDY,
Chartered Accountant.

February, 1902.

LIST OF PAPERS.

N.B.—The Council of the Royal Medical and Chirurgical Society deem it proper to state that the Society does not hold itself in any way responsible for the statements, reasonings, or opinions set forth in the various papers which, on grounds of general merit, are thought worthy of being published in the *Transactions*.

ADDRESS

OF

FREDERICK WILLIAM PAVY, M.D., LL.D., F.R.S., F.R.C.P.,

PRESIDENT,

AT THE

ANNUAL MEETING, MARCH 1ST, 1902.

GENTLEMEN,—By the onward march of time, which stops for no one, we are brought to another Annual Meeting. Last year we met under mournful circumstances, arising from the death, but a short time previously, of the good Queen Victoria, who had passed through a record reign—regarded alike from its duration, the happy social conditions that existed, and the great strides of advance that took place in knowledge. A tie of many years as our Patron was severed. All have to bow to the inexorable laws of nature; and fortunate are we now in finding that our tie with royalty has been renewed by the gracious assent of His Majesty King Edward VII to become our Patron, thus creating a line of succession of patronage through three Sovereigns.

Satisfaction will, I am sure, be felt at the honour that

was conferred upon our Society by the King in receiving our address of condolence on the death of Her Majesty the late Queen at an audience granted to a deputation from the Society for the purpose.

I referred last year to the altered procedure that has been adopted with regard to the publication of papers. With the surrounding changes effected by the advance of time, the restrictive character of the traditions of the Society stood as a bar to communications being presented for reading. There seemed to be a growing prospect of the usefulness of the Society being checked by dearth of material. To meet these circumstances it was decreed in the Standing Orders that "after a paper has been read before the Society, the author, or authors, shall be entitled to publish it in one or more medical or scientific periodicals—provided that in the heading of such paper it shall be stated that it belongs to and was read before this Society."

As the result of the year's working under the new regulation, it is noticeable that authors have extensively availed themselves of the opportunity afforded them of promptly placing their communications before the profession; and if speedy diffusion of knowledge is to be considered advantageous, benefit has been conferred upon both profession and author. Quality has always been looked upon as a primary consideration in the acceptance of papers; and, with the alteration that has been made, there can be no doubt that the Society has greatly improved its position for attaining the object desired.

Our death roll at the last Annual Meeting was a heavy one, comprising as it did twenty-one Fellows, and including an exceptional number of men of mark in the profession. This year death has dealt more lightly with us, ten being the number of obituary records to place before the meeting. Of the ten deaths one occurred at 90, one at 89, two at 87, one at 85, two at 65, one at 63, one at 59, and one at 32. I will take the records in the order in which the deaths occurred.

Benjamin Barrow, F.R.C.S., J.P., who died at Ryde, Isle of Wight, on March 7th, 1901, was born at Bath in 1814. Two of his brothers became Generals in the British Army, and each gained the distinction of K.C.B.

Mr. Barrow was articled to Mr. Stanley, Surgeon to St. Bartholomew's Hospital, and resided in his house, where Mr. Luther Holden was one of Mr. Barrow's fellow-pupils. He became M.R.C.S. in 1836, and F.R.C.S. in 1862, and served as Surgeon in the Army until he settled in practice in Ryde in 1848.

At St. Bartholomew's, we have it on Mr. Holden's authority, Mr. Barrow was "quite an example to the other students in the way in which he did his work. He was an excellent talker, but not a good listener. At the Abernethian Debating Society he always commanded attention. He was the original Founder and Secretary of the Contemporary Club, which consisted of St. Bartholomew's men during the period between 1830 and 1840," and numbered among its members Sir Richard Owen, Sir Charles Locock, Sir James Paget, Dr. Jeaffreson, Dr. Bostock, and other notabilities.

He was a good man of business, and played a foremost part in the public affairs of Ryde, especially in the crusade against the polluted surface wells from which the inhabitants drank. In connection with this matter he allowed himself no rest until the water from the neighbouring chalk downs was brought to supply the town. Similarly he fought for the proper sewerage of the place and the drainage of the marshes, and succeeded in carrying his point in face of strenuous opposition. He was Chairman of the Water Committee and of other sanitary committees, and was nine times Mayor of Ryde. The Esplanade and other public works of the town were largely due to his advocacy. He was one of the founders of the Infirmary, and was one of its honorary surgeons, and subsequently its consulting surgeon. Some six other local institutions were also founded during his residence in Ryde, and obtained his powerful support. In 1881 he was President of the British

Medical Association when the annual meeting was held at Ryde.

Mr. Barrow was twice married; firstly to Miss Stanley, and secondly to Miss Arnold, who has survived him. He had no child.

In the autumn of 1900, whilst staying in London, he fell in the street, and sustained a comminuted fracture of the left wrist, of which he characteristically made light. Sinuses formed, however, and the arm was amputated above the elbow in January, 1901, by his friend Mr. Alfred Willett, who attended him in conjunction with Mr. Ingleby Mackenzie. Notwithstanding Mr. Barrow's great age—over 86—the stump was entirely healed at the end of three weeks; but a little later he became restless, and gradually sank. He was followed to the grave by a large concourse of all ranks of people, amidst every manifestation of sorrow, as was only, it may be said, his due, for he had unceasingly worked with enthusiasm for the public good.

Sir Edwin Saunders, F.R.C.S.Eng., whose death took place after a short illness at his residence, Fairlawn, Wimbledon Common, on March 15th, 1901, at the patriarchal age of 87, was born in 1814 in London, where his father was a book publisher. Early in life he showed great aptitude in connection with mechanical appliances and inventions. He became articled as a dental pupil to Mr. Lemaile, and gave some lectures on elementary mechanics and anatomy before a mechanics' institute. At one of these Mr. Tyrrell, Surgeon to St. Thomas's Hospital, was present, and was so "favourably impressed by the young lecturer that, after a consultation with his colleagues, he invited him to give a course of lectures at the hospital." After his admission to the Membership of the Royal College of Surgeons, in 1839, Sir Edwin became Dental Surgeon and Lecturer on Dental Surgery to St. Thomas's Hospital. He became a Fellow of his College in 1855. In 1840 he investigated the date of eruption of the various teeth, and published the results under the title of "The Teeth a Test of Age." In 1846, at the earnest desire of

Mr. Nasmyth, when attacked with illness, Sir Edwin took his large dental practice, succeeding also to the appointment of Dental Surgeon to Queen Victoria, the Prince Consort, and other members of the Royal Family. In 1883 he received the honour of knighthood.

He was amongst the earliest to seek to obtain from the Royal College of Surgeons an examination and diploma for dental practitioners, and Parliament was eventually induced to give authority to the College thus to extend its powers. In 1859 he and others established the Dental Hospital and School in Soho Square. The institution prospered, and became so successful that in 1874 a larger establishment became necessary; and chiefly through Sir Edwin Saunders' energy and liberality, and his influence over others, the hospital in Leicester Square was equipped and handed over free of debt. In commemoration of these services his colleagues and friends established the Saunders Scholarship at the School. He subsequently remained on the management committee until it was decided to build the new hospital recently inaugurated; when, as he did not agree with the majority of his colleagues, he resigned.

At Sir Edwin Saunders' house the Odontological Society was started in 1857: he was its first Treasurer, and twice, viz. in 1864 and 1879, he held office as its President. In 1881 he occupied the chair of the dental section at the meeting of the International Medical Congress in London. He was also President of the Metropolitan Counties Branch of the British Medical Association in the same year, and was a constant attendant at the meetings of the British Dental Association, over which he presided in London in 1886. He was always anxious to associate the dental with the medical profession; and recognised the necessity of a special training in dentistry, which he regarded as a branch of medicine. It is also largely due to his efforts and example that the dental profession owes its present high position in this country.

He married Maria, daughter of Mr. E. Burgess; and at

their golden wedding his friends presented him with an illuminated address, bound in vellum, and to Lady Saunders a diamond brooch. He relinquished practice several years before his death, and enjoyed the ensuing leisure. In his beautiful garden at Wimbledon he attained great success in the cultivation of chrysanthemums and other flowers. Altogether he possessed keen and wide sympathies, was hospitable to a degree, and a good conversationalist. He also possessed an inexhaustible fund of knowledge of art and travel. It is easy, therefore, to perceive how such a man became the head of his branch of the profession, and how wide a gap was created by his retirement from practice. His funeral at Putney was attended by very many medical and dental practitioners.

Christopher Mercer Durrant, M.D.Edin., F.R.C.P.Lond., who was Consulting Physician to the East Suffolk and Ipswich Hospital, died on April 6th, 1901, in his eighty-eighth year. He was born in Lewes in 1814, and was apprenticed for many years to a medical practitioner at Maidstone. He next spent six months in Berlin, that he might study German; and then proceeded to Edinburgh, where he took the M.D. degree in 1839. In the same year he commenced practice as a physician in Ipswich. Soon afterwards he was appointed Physician to the East Suffolk Hospital, in which institution he henceforward took a very warm interest, and to which he attracted many patients anxious to obtain his advice. He further assisted the Ipswich Nurses' Home and the Convalescent Home at Felixstowe, and was a member of the Acting Committee of both institutions. He was a J.P. for East Suffolk.

Dr. Durrant became M.R.C.P. in 1859, and was elected F.R.C.P. in 1873. He was also one of the oldest Fellows of our Society, having joined in 1843.

In 1879 a serious illness compelled him to discontinue his work on the acting staff of the East Suffolk Hospital, and on his resignation he was appointed Consulting Physician, which post he retained until his death.

He married, in 1839, the daughter of William Rawes,

M.D. The marriage was a happy one, and they lived to celebrate their golden wedding surrounded by a numerous family.

He was the first President of the East Anglian Branch of the British Medical Association after its foundation in 1843, and again occupied the chair when it celebrated its jubilee in 1893. He contributed several papers to the 'British Medical Journal.' He was a kind-hearted physician, greatly esteemed in East Anglia, both socially and professionally; and in middle life he had a large practice as a consulting physician.

John Cavafy, M.D., F.R.C.P., Consulting Physician to St. George's Hospital, died suddenly on April 28th, 1901. He was of Greek descent, and was born at Tulse Hill in June, 1838. He was educated at Brighton and University College, and then worked for four years in the office of his father, a well-known merchant in the City of London. Young Cavafy's desires were, however, towards medicine; and eventually he "entered" at St. George's Hospital, in October, 1861, when he was twenty-three years of age. He graduated M.B.Lond. in 1867, and M.D. two years later. In 1868 he became M.R.C.P., and was elected a Fellow in 1876. He was subsequently a Councillor of the College, and Examiner in Medicine both for the College and for the University of London.

At St. George's Hospital Medical School he held the several offices of Demonstrator of Histology, Lecturer on Comparative Anatomy, Medical Registrar, Lecturer on Physiology, and, finally, Lecturer on Medicine. He was appointed Assistant Physician to the Hospital in 1875, and Physician in 1882, when he also took charge of the skin department. During the International Medical Congress held in London in 1881, he was Honorary Secretary to the Section of Dermatology. Early in 1896 he suffered severely from enteric fever, had a protracted convalescence, and never regained his previous strength. He subsequently gave an admirable lecture on his own case, which was published in 'The Clinical Journal.' In 1898, feeling

that his strength was failing, he resigned his Physiciancy to St. George's Hospital, and was elected Consulting Physician. He was also at one time Physician to the Victoria Hospital for Children.

Dr. Cavafy was always a lucid lecturer and teacher, and possessed a very wide acquaintance with medical literature. He contributed many papers to Heath's 'Dictionary of Surgery,' to the 'St. George's Hospital Reports,' the medical journals, and the Transactions of various societies, amongst the latter being a paper on "Amœboid Movements of the Colourless Blood-corpuscles in Leuchæmia," read before this Society in 1880. His papers chiefly related to diseases of the skin, though he also wrote on the "Education of the General Practitioner," "Diabetes," "Myxœdema," "Yellow Atrophy of the Liver," "Rheumatism treated by Salicylate of Soda," and "Rheumatic Nodules."

From his retirement in 1898, Dr. Cavafy lived at Hove, Sussex, and after an attack of influenza had a remarkably slow pulse. Whilst visiting London in April last he died suddenly and painlessly from cardiac syncope. His wife, by whom he has left one daughter, was the youngest daughter of Mr. Antonio Ralli.

Dr. Cavafy was a born artist with the brush, a good musician, and excelled as a linguist; had a ready wit, and was fluent in conversation; was a genial companion, a keen humorist, and had a true love of nature, especially of botany and ornithology. In his school and college he was courteous and loyal to those with whom he came in contact. He possessed great ability as a teacher, was highly sensitive and sympathetic with the patients, and conscientiously devoted to his duties, both to them and to the students. Altogether, as an intimate acquaintance of more than forty years' standing wrote in the 'Lancet,' "he was a learned physician, he was a just and honourable man. No man ever made firmer friends or fewer foes."

Carston Holthouse, F.R.C.S., who died on July 18th, 1901, was within three months of completing his ninety-

first year, and was probably the senior member of this Society. He was born at Edmonton in October, 1810; and at the age of fourteen was apprenticed to his uncle, Mr. Le Gay Brewerton, at Bawtry, Yorks. He studied medicine at St. Bartholomew's Hospital, was dresser under Sir William Lawrence, and clinical clerk under Dr. Latham. He became L.S.A. in 1832, and M.R.C.S. in 1833, and studied for a time in Paris. Returning to London, he worked in the out-patient department of St. Bartholomew's Hospital, and published three papers on *Acarus scabiei,* which attracted considerable attention.

In 1843 he was appointed Lecturer on Anatomy and Physiology at the Aldersgate School of Medicine upon Mr. Skey's promotion to the Lectureship on Anatomy at St. Bartholomew's. He worked strenuously at the subject of his lectures, and soon established for himself a reputation as an anatomist; and in the same year, 1843, he was elected one of the 150 original Fellows of the Royal College of Surgeons.

Westminster Hospital started a medical school in 1841, but it came to an end in 1846. In 1849, however, a new staff of lecturers was appointed, and to Mr. Holthouse was assigned the Chair of Anatomy. But the school was not at first successful; and, after lecturing for five years without any pecuniary reward, Mr. Holthouse resigned. He was then appointed sole manager of the school, and eventually placed it on a substantial foundation, so that he was virtually the founder of the Westminster Hospital Medical School of to-day. In 1853 he was appointed Assistant Surgeon to the Hospital, and in 1857 became Surgeon, having meanwhile served on the staff of the British Hospital at Smyrna during the Crimean war. Before this episode in his career he had studied ophthalmology; and in 1854 he published six lectures on the 'Pathology of Strabismus and its Treatment by Operation.' In 1857 he assisted to found the Surrey Ophthalmic Dispensary, now well known as the Royal Eye Hospital, Southwark. In the following year he published a work "On Squinting,

Paralytic Affections of the Eye, and Certain Forms of Impaired Vision.' He wrote a book 'On Hernial and other Tumours of the Groin,' and an article for Holmes' 'System of Surgery' on "The Lower Extremity."

On his retirement from the Surgeoncy to the Westminster Hospital, in 1875, Mr. Holthouse was appointed its Consulting Surgeon. He subsequently started an institution for the reception and treatment of inebriates. The venture, however, brought him anxiety and loss, though, as an object lesson, it probably assisted in the passage of the Inebriates Act, which soon ensued.

As a surgeon he excelled in diagnosis. In operative surgery he was not so brilliant. As a writer in the 'British Medical Journal' truly remarks, "in spite of real ability and devotion to his profession, Mr. Holthouse never reaped the reward that he might have looked for. Whilst his work in each branch of surgery that he took up was thorough so far as it went, he allowed his energies to range over too many subjects, and when success in one seemed close at hand he had already turned his attention to another. Confident also, and justly so, in his own powers of diagnosis and his judgment as to treatment, he was scarcely ready enough to make allowance for the views which others might take of a case. These characteristics were naturally a serious bar to success in practice."

After a long and vigorous life, he was seized about two years before his death with right-sided hemiplegia and aphasia, from which he quickly recovered. But other attacks followed, rendering him quite helpless during several of the closing weeks of life.

Mr. Holthouse was twice married, and has left three sons by his first wife. His second wife, who also died before him, was Martha, the daughter of Dr. John Nicol, of Inverness. By her he had no children.

Thomas Vincent Jackson, F.R.C.S.Edin., M.R.C.S.Eng., J.P., Surgeon to the Wolverhampton and Staffordshire General Hospital, died at his house in Wolverhampton on October 12th, 1901, at the age of 65. He was born in

London, and educated at Brighton and King's College School. He studied medicine at University College, London; and, after a distinguished career, became President of the College Medical Society. He obtained the diploma of M.R.C.S. in 1857, and that of L.S.A. in 1858. He was elected Demonstrator of Anatomy in University College School, and House Surgeon in the hospital; and acted as private assistant to Mr. Richard Quain. For reasons of health he quitted London; first assisted Dr. Quinton, of Willenhall, and was appointed in 1861 House Surgeon to the Wolverhampton and Staffordshire General Hospital. Subsequently, joining Dr. Gatis in practice, he was appointed Honorary Surgeon to the hospital. He became F.R.C.S.Edin. in 1883, and was for many years Consulting Surgeon to the Hospital for Women and Surgeon to the Orphan Asylum at Wolverhampton. He was also Life Governor of Birmingham University, and a member of several medical societies.

Besides holding these several hospital appointments, he was for nearly forty years engaged in general practice. This did not, however, prevent him from achieving a high repute as a surgeon and successful operator in South Staffordshire. He contributed articles to the medical press on perinæal operations, lithotomy, and colotomy; published, in 1889, an 'Essay on the Medical Profession and Public Life;' and in 1898 an 'Address on the Medical Craft in Britain, from the Earliest Period to the Victorian Era.' He was a Justice of the Peace for the town of Wolverhampton and for the county of Stafford.

Early in his career he joined the British Medical Association; was one of the founders of the Staffordshire branch, served as its Secretary from 1874 to 1888, and filled the Presidential Chair in 1889. He also represented his branch on the Central Council of the Association for the last twenty years of his life, and was a most constant attendant at the Council meetings.

He gave a large amount of time to the municipal work of Wolverhampton; was elected Town Councillor in 1876,

served on several standing committees of the Council, and was for many years Chairman of the Public Works Committee. He became Mayor of the borough in 1887, the year of Queen Victoria's Jubilee; and largely through his exertions the Queen Victoria Nursing Home for Wolverhampton, which cost £5000, was erected as a memorial of the Jubilee. He was an Alderman of the borough from 1887 onwards. He discharged the duties of all these posts with zeal and ability, and always evinced a keen desire to promote the welfare of the people among whom he lived. In 1896 he assisted largely in remodelling the operating theatre of the Wolverhampton and Staffordshire General Hospital in accordance with modern requirements.

He was twice married: first to the daughter of his early partner, Dr. Gatis; secondly to the daughter of Dr. Symonds, of Southampton. This lady survived him.

Mr. Jackson's health had lately failed, but his final illness was of brief duration. On October 6th he felt unwell, but gave evidence in a police case next day. On October 9th pneumonia was detected, and he died on the 12th. The first part of the funeral service was conducted at Wolverhampton on October 17th, and was attended by the Mayor and Corporation, by a large body of Mr. Jackson's medical colleagues, and by the officials of the many institutions with which he was connected. The interment took place on the same day at Highgate Cemetery, London.

Throughout life Mr. Jackson won the sincere respect and regard of his colleagues, his patients, and his fellow-citizens of all shades of political opinion. One of his friends in the Midlands wrote thus of him:—"As a surgeon Vincent Jackson was a bold and enterprising operator, a keen observer, quiet in his work, but considerate and careful. To those who knew little of him his manners often seemed nervously excitable, not to say fussy, but this was all upon the surface. His old friends and patients found beneath the superficial mannerism sound judgment, wide knowledge, and generous self-sacrifice, and so learned to prize him as a faithful friend and a devoted doctor."

Henry Spencer Smith, F.R.C.S.Eng., who was the *doyen* of this Society, to which he was elected as long ago as 1838, died on October 29th, 1901, aged 89, at his residence in Oxford Terrace, Paddington. He received his professional education chiefly at St. Bartholomew's Hospital, where he was a pupil of Mr. (afterwards Sir William) Lawrence. He also studied at Berlin and Paris, and became M.R.C.S.Eng. in 1837. Subsequently he was House Surgeon to St. Bartholomew's Hospital and Surgeon to the Royal General Dispensary, Aldersgate Street. He was appointed one of the three Assistant Surgeons to St. Mary's Hospital upon its foundation in 1851. When a medical school in connection with that hospital was originated, in 1854, he was appointed its Dean, and for six years laboured unremittingly at the work. When he resigned the post in 1860 he was presented by the students with a piece of plate, and by the lecturers with a silver-gilt inkstand. He lectured for many years in St. Mary's Medical School on Surgery; and upon his retirement from the Surgeoncy to the Hospital was elected Consulting Surgeon. He was also a member, and the Honorary Secretary, of the Government Commission of Inquiry into the question of Venereal Disease as occurring in the Navy and Army. He was one of the original Fellows of the Royal College of Surgeons of England who were chosen in 1843, and, after the death of Mr. Carston Holthouse in July last, became the sole survivor of those original Fellows. He held office as a member of the Council of the College from 1867 to 1875, and had been a member of the Court of Examiners. He had at various times held office in this Society, as Councillor, Secretary, Vice-President, and Treasurer, and served as Referee and member of the Library Committee, but never contributed to the Society's 'Transactions.'

He translated into English Schwann's 'Microscopic Researches into the Accordance in the Structure and Growth of Animals and Plants,' which was published by the Sydenham Society. He also translated Bischoff's

memoir 'On the Periodical Maturation and Extrusion of Ova, independently of Coitus, in Mammalia and Man;' and he contributed various clinical lectures to the medical journals.

Mr. Smith retired from practice about fifteen years ago. He was twice married, and has left a widow, with a son and daughter from his first marriage.

Henry Sutherland, M.D., M.A.Oxon., M.R.C.P.Lond., died on November 19th, 1901, aged 59. He was the second of the six sons of Dr. Alexander John Sutherland, F.R.C.P., F.R.S. Both father and son were Physicians to St. Luke's Hospital for the Insane.

Henry Sutherland was educated at Westminster and Radley Schools, and at both Oxford and Cambridge Universities. He took the B.A.Cantab. in 1867, the M.A. and M.B.Oxon. in 1869, and the M.D.Oxon. in 1872. His medical studies were pursued at St. George's Hospital, London, and Addenbrooke's Hospital, Cambridge; and he became M.R.C.P.Lond. in 1870. He studied insanity at Bethlem Royal Hospital and at the West Riding Asylum, Wakefield, under Dr. (now Sir James) Crichton-Browne. Returning to London to practise, he was, in 1872, appointed Lecturer on Psychological Medicine to the Westminster Hospital, a post which he retained for about fifteen years. His work lay henceforth in this special branch of practice, though he was also for some thirty years Physician to the St. George's, Hanover Square, Dispensary. That post he highly valued, as it kept him in touch with general medicine.

Dr. Sutherland was a Fellow or Member of eight of the chief medical societies of London, and the author of several articles on subjects connected with his own speciality, very many of which appeared in the columns of the 'Lancet.'

In early manhood he excelled as a fencer, gaining the "prize foils" both at Oxford and at Cambridge; he also excelled in running. He was an enthusiastic Freemason, and had held the Mastership of several Lodges, besides being a Past Grand Deacon. He was industrious and

painstaking, a generous and genial host, and always widely considerate for others; so that he naturally made and retained a large circle of friends. He married somewhat late in life, and has left a widow to mourn his death.

Sir William MacCormac, Bart., K.C.B., K.C.V.O., F.R.C.S.Eng. and Ireland, D.Sc., M.Ch., R.U.I., Honorary Sergeant-Surgeon to H.M. the King; Consulting Surgeon to, and Emeritus Lecturer in Surgery at, St. Thomas's Hospital; ex-President of the Royal College of Surgeons of England.

The death of Sir William MacCormac, on December 3rd, 1901, deprived surgery of one of its foremost exponents. He had been a prominent figure in every professional event for thirty years, so that his life-work covered a peculiarly wide field.

William MacCormac was the eldest son of Dr. Henry MacCormac of Belfast, and was born there in 1836. Dr. MacCormac was a cultured physician, an expert in tropical diseases, and a strenuous advocate of the value of fresh air in the treatment of phthisis. Young MacCormac received his early education at the Royal Belfast Institution, and studied medicine at Dublin and at Paris. He became successively B.A. in 1856, M.A. in 1858, M.Ch. in 1879, and D.Sc. (honoris causâ) in 1882 in the Queen's University in Ireland, and in 1882 he also received its gold medal.

After graduation he studied surgery for a considerable time at Berlin, where he made the firm and lasting friendship of von Langenbeck, Billroth, and von Esmarch. He subsequently became a member of the Senate, and Examiner in Surgery, of the Queen's University; Honorary M.D. and M.Ch. of the University of Dublin; and Fellow of the Royal College of Surgeons of Ireland. He early commenced practice in Belfast, where he was for six years Surgeon, and subsequently Consulting Surgeon, to the Royal Hospital.

Not being entirely satisfied with the prospects open to him in his native city, he decided to seek his fortune in London; and had just established himself here in 1870

when the Franco-German war began. Hastening to Paris, he joined Sir John Farley, the representative of the British Red Cross Society, Dr. Franks (of Cannes), who had been an English Army Surgeon, and Dr. Marion Sims, all desirous to succour the wounded. They decided to establish an Anglo-American Ambulance, which proceeded first to Mézières and Sedan with Mr. MacCormac as its chief surgeon. Sedan was reached on August 30th, just in time for the fighting. This foreign ambulance was so placed and so well equipped in the matter of surgical appliances and comforts as to be able to render services such as no other ambulance in either army was in a position to do during the war. For a hospital they were assigned an infantry barracks on the ramparts of Sedan, overlooking the Meuse, in which 384 beds were set up. On August 31st MacCormac, with several colleagues, went on to Balan, where no fewer than 260 wounded were attended to the same evening. On the next morning, September 1st, he was back in Sedan. The French wounded were about 12,000; and of these 274 were received in the course of that day into the beds of the Anglo-American Ambulance, while many more were treated there and sent away. Each day considerably more than a hundred major operations were performed, and MacCormac's share of these was a large one. A day of his work at the Caserne d'Asfeld is well described in his own words: "I did not succeed in keeping a record of all the work that was done that day. Indeed, I only wonder I kept any record at all. I find, however, that I performed several amputations of the leg, the thigh, the forearm, and the arm; that I excised the shoulder and the elbow joints, and also performed partial resections of the upper and lower maxillæ, and of nearly the whole ulna. The number of bullets and pieces of shell that were extracted from various parts of the body are too numerous to reckon." His energy, decision of character, and robust constitution enabled him to withstand enormous vicissitudes and fatigue, including a febrile attack, the result of inoculation with infective discharge. In spite of his mul-

tifarious duties, however, he kept a diary of his experiences in the field, the publication of which in weekly instalments brought his name prominently before the profession and the public, not only of his own country, but all over the civilised world. He remained at Sedan until the wounded had been dealt with, discharging all the duties of his post with zeal and ability.

Returning to London just when the staff was being appointed to the new St. Thomas's Hospital, he obtained the post of Assistant Surgeon against strong competition, being greatly assisted in his election by the London Committee of the Red Cross Society, who entertained a high opinion of the work which he had accomplished under their direction.

At St. Thomas's he became full Surgeon in 1873 and Lecturer on Surgery, and after several years of service retired as Consulting Surgeon and Emeritus Lecturer on Clinical Surgery. He was earnest and zealous in his work at both hospital and school. He educated his pupils thoroughly in the art and science of surgery, brought the work of the hospital under the eyes of the profession at home and abroad, and materially advanced his own professional reputation.

In 1876 he acted as Chief Surgeon to the National Aid Society for the Sick and Wounded in War during the Turco-Servian campaign, and was present at the Battle of Alexinatz; but he did not remain long away from England. As one result of his experience on several battlefields he became deeply impressed with the value of female nurses in the stationary hospitals at the seat of war, though not within the zone of fire. He also made the well-known collection of gunshot fractures now to be found in St. Thomas's Museum.

Besides his appointment at St. Thomas's he was Consulting Surgeon to the French, the Italian, and Queen Charlotte's Hospitals; and examined in surgery at the University of London, and for Her Majesty's Naval, Army, and Indian Medical Services.

In 1881 he was Honorary General Secretary to the International Medical Congress held that year in London, and fulfilled the duties so efficiently that the late Queen Victoria subsequently conferred upon him the honour of knighthood. This Congress was, in fact, one of the great events of his life. Its success depended very largely upon the broad lines on which it was organised, and the energy with which it was administered by its Secretary-General. Henceforth private professional work came to him in abundance. He was elected President of the Medical Society of London in 1888, and held the similar post in the Metropolitan Counties Branch of the British Medical Association in 1890.

Sir William MacCormac became a Member of the Royal College of Surgeons of England in 1857, and was admitted a Fellow (*ad eundem*) in 1871. In 1883 he was elected a member of the Council, and in 1887 became a member of the Court of Examiners. In 1893 he delivered the Bradshaw Lecture, choosing as his subject "Sir Astley Cooper and his Surgical Work." In 1897 Sir William MacCormac was elected President of the College, and in the four succeeding years he was re-elected to the same high office. The last of these occasions was memorable, inasmuch as it conferred upon him the distinguished honour of occupying the Presidential Chair during the celebration of the centenary of the College, when again his great organising powers were brought into play with marked success.

In 1897 Sir William MacCormac was created a baronet on the occasion of Queen Victoria's Diamond Jubilee; and was appointed Surgeon-in-Ordinary to the Prince of Wales, whom he attended in the following year after the accident to the Prince's patella. In recognition of his services in connection with the repair of the fracture, the Queen made him a Knight of the Royal Victorian Order; and soon after his present Majesty's accession he was appointed honorary Sergeant-Surgeon to the King. On February 14th, 1899, Sir William delivered the Hunterian Oration in the presence of the Prince of Wales.

Honours from foreign countries, too, fell thick upon Sir William MacCormac. He was appointed in 1898 an Honorary Member of the Imperial Military Academy of Medicine of St. Petersburg, and was Honorary Fellow or Member of various other Foreign Medical and Surgical Societies; Officer of the Legion of Honour, Commander of the Order of Dannebrog of Denmark, of the Crown of Italy, and of the Tahovo of Servia. He held also Orders given by the rulers of Prussia, Portugal, Sweden, Bavaria, Spain, and Turkey; and in this country was a Knight of Grace of the Order of the Hospital of St. John of Jerusalem in England, and was Chief Surgeon to the ambulance department of the Order.

He published several papers on surgical subjects in the medical journals, and read others before the different medical societies with which he was connected. He read two such papers before this Society, viz. one on "A Case of Resection of the Shoulder and Elbow Joints in the same Arm for Gunshot Injury," read March 12th, 1872; and the other on "The Wounded in the Transvaal War," read on May 22nd, 1900. He also wrote in 1871 a graphic account of his experiences in war, under the title of 'Work under the Red Cross,' which was translated into German, French, Spanish, Dutch, Italian, Russian, and Japanese. He published in 1880 'Antiseptic Surgery,' which gave rise to one of the most interesting debates of modern times. It was introduced by him, and in the subsequent spirited discussion most of the chief surgeons of the day took part. Listerism, it may be noted, was not at that time accepted by all. The first part of his work on 'Surgical Operations' appeared in 1885; but although he worked constantly upon it he did not live to complete it. He also wrote on "Abdominal Surgery."

At the beginning of the present war in South Africa he volunteered for service, and was appointed by the Government Consulting Surgeon to the Field Force. On November 3rd, 1899, at a week's notice, he and his old pupil, Mr. Makins, sailed for Capetown. He had a grand "send-off"

at Waterloo Station, Sir John Furley, Dr. P. Frank, and Dr. Blewitt, three of the colleagues with whom he had worked in the Franco-German campaign, being present to wish him "God-speed and a safe return." He soon found himself amongst the wounded of Sir Redvers Buller's army, after the unsuccessful attempt to cross the Tugela; and then returned to Cape Colony and the Orange River to take charge of the arrangements for the wounded from Lord Methuen's force advancing to the relief of Kimberley. He was at the seat of war for some four and a half months. Whilst in South Africa he visited all the hospitals in Natal and in Cape Colony, and was at the front four times. He was also in hospital himself for a short interval, suffering from dysentery. Upon his return home the Knighthood of the Order of the Bath was conferred upon him, and he was honoured with an invitation to dine with the Queen at Osborne.

He was not particularly strong, and in the course of life suffered from several illnesses. After an attack of erysipelas, in 1879, his hair rapidly turned grey; and in 1896 pneumonia and empyema brought him nearly to death's door. But he recovered, and it was only a few months before his death that he was known to be indisposed. Since his return from South Africa he had suffered from dysenteric symptoms, lumbar pain, and abdominal tenderness. His appetite had failed, and he had lost flesh and suffered from sleeplessness. The fogs of October and November last in London tried him severely; and a trip to Bath in conjunction with Lady MacCormac was undertaken, in the hope that it would expedite his recovery. He had slept only at fitful intervals, and chiefly in his chair; but after a deep hot mineral bath, on the morning of his arrival, he slept well in bed the greater part of the day and following night. Next morning, however, whilst he was arranging for a second bath he died suddenly in his bed.

Sir William MacCormac married, in 1861, Miss Charters, of Belfast, who survives him, and of whom it may be said

that she charmed and cheered his life, and was to him throughout his career a devoted helpmate. Sir William left no family.

The first part of the funeral service took place at St. Peter's, Vere Street, where the body of the church was filled to its utmost capacity by friends and *confrères* of the deceased, and by representatives of the many public bodies to which he was affiliated. The King was represented, as also were the French and German Ambassadors, the Army Medical Department, the Naval Medical Service, the British Medical Association, the two Royal Colleges, and the hospitals to which he was attached. The interment took place at Kensal Green, and was also very largely attended.

Such are the chief facts in the professional career of Sir William MacCormac; it still remains to notice some of the characteristics of his many-sided nature.

He was so well known to the Fellows of this Society that it is not necessary to do more than just allude to his massive frame, fine open countenance, genial smile, and charm of manner that endeared him to all. His hospitality, kindliness, and generosity were unbounded; and his professional success enabled him to exercise these faculties without stint of any kind. This open-handed hospitality was exercised towards pupils, colleagues, and members of the profession from all parts of the world, and with a cordiality not easily forgotten. Beneath his roof, indeed, the most distinguished members of the profession from Europe and America were constantly assembled.

He was an early riser, and ofttimes an inordinate worker. A proof of his capacity for work was furnished by the fact that immediately after the meeting of the International Medical Congress of 1881 he started to edit the 2370 pages of the 'Transactions,' which, in their three languages, appeared complete within a period of six months from the termination of the meeting.

He was a most popular teacher, and in 1899 a dinner was given in his honour by the practitioners scattered over

England who had held office as House Surgeons at St. Thomas's Hospital during his tenure of office as Surgeon, from which only five were absent, and they unavoidably so. Until the last he kept himself thoroughly abreast of English, American, and Continental literature. In his lectures he furnished his class with the most recent and approved views on the subject under discussion, and a printed summary of the same was given to each of the class. He was also an efficient, considerate, and fair examiner.

As a surgeon he disliked over-specialisation. He adopted with avidity each advance in operative surgery and surgical technique, and was ready to make trial of any appliance or new instrument that promised well.

Sir William MacCormac had travelled in most countries of Europe and twice in America, and was a charming travelling companion; but he was usually impatient of holidays and anxious to return to his work. He was a good draughtsman. In his youth he had loved athletics; later in life was fond of walking; and, finally, becoming an enthusiastic golfer, he and his dog were familiar figures on the links at Mitcham and Deal. A dog, indeed, was his constant companion through life. Fishing was apparently the only form of sport in which he indulged.

He had innumerable friends, amongst whom were some of the highest in the land; but to others who were less successful his kindness and generosity were no less marked. He deserved good fortune, and when it came he was not spoilt by it. He was ambitious, and rigidly determined to see his own schemes succeed. He was especially proud of his profession, his school, and his college. In 1898, when the Lord Mayor of London received the Presidents of the two Colleges and the other heads of the profession, the occasion gave our deceased *confrère* great satisfaction, especially as Lord Lansdowne announced on the occasion that "all the reforms asked for by the Army Medical Department and the profession had been granted, and that

the Queen herself had graciously consented to the new corps being styled the Royal Army Medical Corps."

Arthur Nesham Weir, M.D., B.Sc.Lond., F.R.C.S.Eng., died on January 24th of the present year from a railway accident, at the early age of 32. After an extended training at Merchant Taylors' School he entered, in 1887, at St. Bartholomew's Hospital, where he had a distinguished career. On entrance he gained an open scholarship in Science, in the following year a junior scholarship, and in 1892 the Brackenbury Scholarship in Surgery. He took the degree of B.Sc.Lond. in 1888, that of M.B. in 1894, and M.D. (in State Medicine) in 1899; became a Member of the Royal College of Surgeons in 1892, and Fellow of that College in 1894.

At St. Bartholomew's he acted as Sir Thomas Smith's House Surgeon, and afterwards Dr. Champneys appointed him his midwifery assistant, which post he held for three months. He was also senior Assistant Demonstrator of Anatomy.

On leaving the hospital he worked for nine months as Medical Inspector for the Home Office (Burial Acts Department), and then went to South Africa as senior Civil Surgeon to Princess Charlotte's Hospital in Natal. When that hospital was disbanded he went to No. 19 Stationary Hospital at Harrissmith, Orange River Colony, where the work was very heavy, and his professional skill obtained for him a considerable reputation. He came home in July of last year.

For three months before his death Dr. Weir acted temporarily as Medical Officer of Health at Tottenham, but had decided not to remain on. He became a Fellow of this Society in 1896, and was also a member of the Anatomical Society. Altogether he was a man of considerable ability and of sterling qualities, and a highly promising member of our profession. Moreover, not only did he excel in medical science and art, he was also an enthusiastic athlete. In his student days he was captain of the St. Bartholomew's Hospital football team; more than held

his own at boxing, fives, and water-polo; and was captain of the Stanmore Golf Club in 1899-1900. For ten years, too, he was a member of the Old Merchant Taylors' Football Club.

The funeral took place at Kensal Green Cemetery, and was attended by many medical men, and by representatives of the many clubs of which Dr. Weir was a member.

ACUTE DILATATION OF THE STOMACH

WITH ILLUSTRATIVE CASES

BY

H. CAMPBELL THOMSON, M.D., F.R.C.P.

ASSISTANT PHYSICIAN; PATHOLOGIST AND CURATOR OF THE MUSEUM TO THE MIDDLESEX HOSPITAL; MEDICAL TUTOR IN THE MEDICAL SCHOOL.

Received 5th July—Read October 22nd, 1901.

Acute dilatation of the stomach is characterised by its sudden onset, by the vomiting of enormous quantities of fluid, and by very severe general symptoms, which, in the recorded cases, have generally ended fatally within a few days after the first onset of the disease.

The condition was first fully described by Dr. Hilton Fagge,[1] who recorded four cases, two of which had come under his own personal observation. Since then, although a number of cases have been recorded, the subject does not appear to have attracted much attention. I have during the past three years made post-mortem examinations upon four cases in which death was immediately due to this condition, and I believe that the disease, though of course very uncommon, is not so rare as has generally been supposed, and that probably the difference between the very serious cases and the less severe forms of dilata-

[1] 'Guy's Hosp. Reports,' 1872-3.

tion, also acute, which not infrequently accompany severe illnesses, is one of degree rather than of kind.

Before referring to the cases recorded by others I will give a brief account of those which have come under my own notice, and I must here acknowledge my indebtedness to Mr. Henry Morris and Dr. Kingston Fowler, who have kindly allowed me to make use of the clinical notes of cases which have been under their care. The first case is one in which acute dilatation suddenly supervened upon chronic dilatation, the latter being due to a growth of the pylorus.

The patient was a man aged 48, and was admitted into the Middlesex Hospital on October 31st, 1899, under the care of Dr. Kingston Fowler. Symptoms had existed for three months before admission, the chief being discomfort after food and frequent vomiting.

On admission the patient was found to be considerably emaciated; the stomach was dilated, and extended downwards to about an inch above the umbilicus; no splash was obtained. An indefinite tumour could be felt in the epigastrium. The pulse was 84, regular, and the patient, considering the disease he was suffering from, did not appear to be unduly ill, and certainly presented no immediate symptoms of an alarming character. On November 3rd, *i. e.* three days after admission, the stomach was washed out in order to relieve the vomiting, which occurred at intervals, and which in no way differed from that which usually takes place in cases of pyloric cancer. On this occasion 38 oz. were drawn off with a soft syphon tube. The patient expressed himself as feeling relieved by the washing, and the process was repeated on the following morning (November 4th), no vomiting having occurred in the interval. Relief was again obtained, and the patient was able to take about half a pint of beef tea and a very little pudding at mid-day.

Suddenly, during the afternoon, a change for the worse took place, which was ushered in by slight hiccough,

accompanied by abdominal pain and uneasiness, which the patient attributed to flatulence; at 6 p.m. the pain was considerable, at 8 p.m. he vomited about 8 oz. of thick, dark brown fluid, and an hour later (9.15 p.m.) he was very collapsed, with a feeble pulse of 120, a subnormal temperature, and cold extremities.

There was now severe abdominal pain, the outline of the stomach was easily seen, and appeared to cover a greater area than formerly; there was no muscular rigidity, but the abdominal walls did not appear to move with respiration. In the face of these acute symptoms it was thought possible that a perforation of the stomach might have taken place; a ¼ gr. of morphia was given hypodermically, and hot bottles were put to the feet; no food was given by the mouth. About midnight the patient was very wakeful, but slept after a second injection of morphia. The next day (November 5th) the patient was drowsy, but said he had no pain, the pulse was 120, and the abdomen moved slightly with respiration; the lower border of the stomach now reached the umbilicus, and a tympanitic percussion note could be obtained in the left axilla as high as the fourth rib. Nourishment was given by nutrient enemata and suppositories, which were retained.

The general condition remained about the same all day, but towards evening the patient became more collapsed, and at 6 p.m. a hypodermic injection of strychnine was given. The abdomen, however, became more distended. Death took place at 2.55 a.m. on the morning of November 7th.

During the acute illness the urine became very scanty, none at all was passed from 1 a.m. till midnight on November 5th, when a catheter was passed, but only one ounce was drawn off.

At the post-mortem examination the contents of the abdomen were almost entirely obscured by the dilated stomach, which was tightly distended with gas, and also contained a considerable quantity of dark brown fluid.

In shape the stomach was cylindrical, the lesser curvature making a sharp curve, while the greater curvature was rounded and reached a point just below the level of the iliac crest. There was a growth of the pylorus which considerably narrowed the orifice.

This case, then, is an example of acute dilatation suddenly supervening upon a chronic one; owing to the stricture the stomach had, no doubt, for some time had a considerable strain put upon it, and then suddenly acute dilatation set in. Possibly the slight irritation produced by washing out the stomach may have upset the balance; or, and what I think is more likely, the growth may have implicated some of the nervous structures in the neighbourhood.

The second case occurred after an exploration of the kidney.

The patient, a man aged 26, was admitted under the care of Mr. Henry Morris with symptoms pointing to the presence of renal calculus, and on these grounds the right kidney was explored by a lumbar incision on July 30th, 1900; there was nothing of special note connected with the operation. Vomiting commenced a few hours after the operation was performed, and persisted almost incessantly up to the time of death, which took place on the evening of August 4th. The temperature fluctuated a little but kept low, and reached 98° before death; the pulse was very rapid, varying from 120 to 140 per minute. There was no suppression of urine, on the day of death 33 oz. were passed.

At the post-mortem examination, made on August 5th, I found the stomach to be enormously distended and of cylindrical shape, the lower end being on a level with the iliac crest; the first part of the duodenum was also distended. The contents of the stomach consisted of gas and a considerable quantity of thick green fluid. The intestines—with the exception of the first part of the duodenum—were all somewhat collapsed. There was no obstruction of the pylorus, and no definite change of any

kind to be observed in the stomach walls. The recent incision into the right kidney was in process of healing, and all the structures around it appeared healthy; there was some chronic nephritis of both kidneys.

The third case was that of a female aged 40, who was admitted into the hospital under my care (in the absence of Dr. Fowler), suffering with deep jaundice, which had come on suddenly with severe pain a few weeks previously.

As the diagnosis between gall-stones and malignant disease was somewhat uncertain, it was thought advisable to explore the abdomen; this was accordingly done by Mr. Murray on April 30th, 1901, the condition proving to be a growth of the head of the pancreas, and a distended gall-bladder; the gall-bladder was drained and the wound sutured.

All went perfectly well till May 4th, when the patient passed a restless night, and vomited early in the morning of May 5th. She also became very collapsed, but this may have been partially due to some hæmorrhage which occurred in the wound. The vomiting, however, persisted, and large quantities of brownish fluid were thrown up; the urine became scanty, and the temperature was subnormal before death, which took place on May 9th.

The post-mortem examination showed the stomach to be greatly distended, but chiefly with gas, there being only a small quantity of greenish fluid present. The stomach had the same cylindrical appearances as in the other cases, but was not quite so large as any of the other three which I have met with.

There was a hard tumour of the head of the pancreas, but it was not very prominent, and as far as could be seen it had not caused any definite obstruction to the pylorus, nor was any obvious dilatation of the stomach noted when the abdomen was explored.

The next case is one in which acute dilatation occurred as a complication of pleurisy and pneumonia.

The patient, a female aged 24, was admitted into the

hospital under the care of Dr. Fowler on June 26th, 1901. She was first taken ill on June 24th, and previous to that she had been in good health.

On admission there were signs of consolidation over the lower lobe of the right lung, and also well-marked signs of pleurisy on the same side. The next morning (June 27th) there was some improvement, and no signs of any extension of the disease; later on in the day, however, there was pain and friction in the left side, and at 1 p.m. the patient suddenly vomited. The vomiting continued incessantly from 1 p.m., June 27th, till 6.30 a.m., June 28th, and then ceased till 1.5 p.m. the same day, when it recommenced and continued till death, which took place at 2 a.m., June 29th, *i.e.* about thirty-six hours after the vomiting first began. The vomit was of a dark greenish colour, and large quantities were brought up without any violent effort. The abdomen was very carefully examined on the 28th, but no distension was observed until the afternoon of that day (3 p.m.).

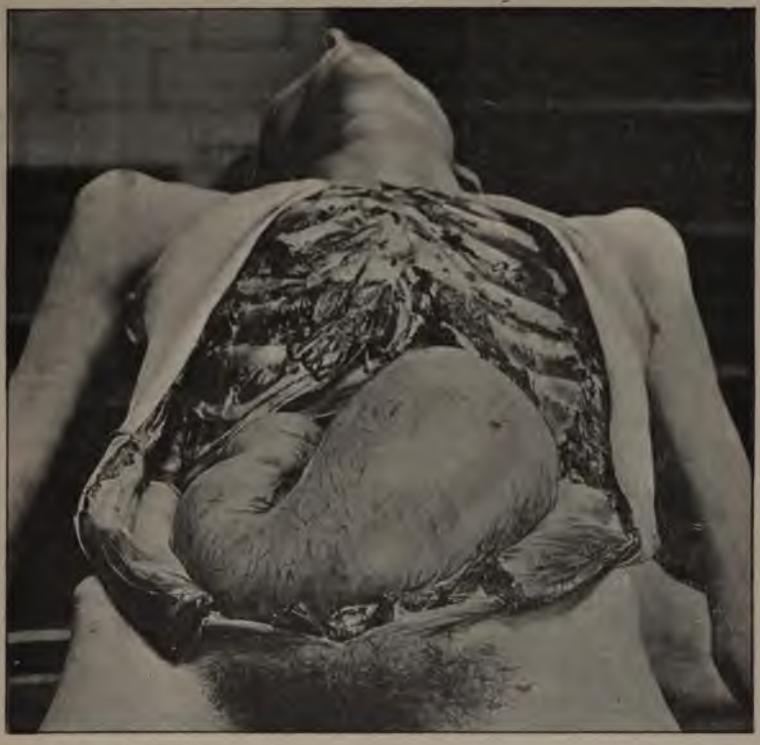
The urine was passed in usual quantities throughout the illness, but it may here be mentioned that at the post-mortem examination the bladder was perfectly empty.

At the post-mortem examination the stomach was enormously distended, and reached down to the pubes. Its appearance is well shown by the accompanying photographs, which were taken at the time.

The stomach contained about 35 oz. of dark greenish fluid, and the mortuary attendant informed me that a large quantity had escaped by the mouth when the body was being moved. On relieving the stomach of its contents it rapidly shrank, and in a few minutes it had the appearance of being but little larger than normal, and no one seeing it would have thought it could have been so enormously dilated only a few minutes before; the stomach walls appeared perfectly healthy, and microscopically there were no changes to be detected.

The intestines were collapsed, apparently from compression; there were no other abnormalities in any of the

FIG. 1.



(A) Acute dilatation of stomach complicating pneumonia and pleurisy.

(B) Side view of the same case (A).

other abdominal organs, except that the liver was rather larger than normal.

In the thorax the lower lobe of the right lung was consolidated and in a condition of red hepatisation; there was no pneumonia elsewhere. Both pleuræ were extensively covered with a thick yellow exudation, which on the left side was particularly marked over the base of the lung, where it rested on the diaphragm.

Acute dilatation of the stomach may arise without any apparent cause whatever, the patient being, as far as one can tell, in ordinary health up to the time of the onset of acute symptoms. This was so in Fagge's second case, in which the patient died after three days' acute illness, and after death no other morbid condition was found except that of the stomach. Fagge considered that the actual process of enlargement of the stomach is more gradual, and is in the end succeeded by sudden symptoms of great severity; but although this is sometimes so—as, for instance, in the case I have recorded, where there was obstruction to the pylorus, and possibly also in the case in which there was a tumour of the pancreas,—there is no reason to believe that there was any slow dilatation previous to the acute symptoms in the other two cases.

In many cases (*v.* Table) some other morbid condition is found in addition to the dilated stomach, and in other instances the dilatation appears to follow immediately upon some surgical operation, which may or may not be connected with the abdomen.

In the case recorded by Mr. Morris the operation consisted in the removal of some necrosed bone from the foot; the patient began to vomit about an hour after the conclusion of the operation, and brought up quantities of thin, greenish fluid almost continuously until death took place, two days afterwards.

The accompanying table of 10 cases shows some of the associations which have been observed between acute dilatation of the stomach and other lesions, and surgical operations.

Table of Ten Cases of Acute Dilatation of Stomach.[1]

No.	Author.	Reference.	Sex.	Age.	Morbid conditions found in addition to dilated stomach.	Operation (if any) prior to the onset of symptoms.
1	Hilton Fagge	'Guy's Hosp. Reports,' 1872–3	M.	18	Retro-peritoneal abscess communicating with duodenum	—
2	„	„	M.	30	Nil	—
3	Miller and Humby	'Trans. Path. Soc.,' vol. iv; also quoted by Fagge	F.	48	Nil	—
4	Hughes-Bennett	'Principles and Practice of Med.;' also quoted by Fagge	M.	26	Empyema	—
5	Henry Morris	'Trans. Path. Soc.,' vol. xxxiv	M.	37	Nil	Operation upon foot.
6	Goodhart	„	M.	29	Nil, except some œdema of lungs	Excision of knee.
7	Campbell Thomson	—	M.	48	Carcinoma of pylorus	Passage of soft tube into stomach.
8	„	—	M.	—	Nil	Exploration of right kidney (extra-peritoneal method).
9	„	—	F.	40	Carcinoma of pancreas	Abdominal exploration.
10	„	—	F.	24	Pneumonia and extensive diaphragmatic pleurisy, the latter chiefly on the left side	—

From this table the cases may be conveniently arranged in the following groups :

[1] For a fuller collection of published cases see the author's book on 'Acute Dilatation of the Stomach.'

A. Those in which the dilatation occurred without any apparent cause, and in which, after death, no other lesion was found. (Nos. 2 and 3.)

B. Those in which after death some other lesion has been found. (Nos. 1, 4, 7, 9, and 10.)

C. Those in which the dilatation has followed some surgical operation, and in which after death no other lesion has been found. (Nos. 5, 6, and 8.)

In two cases there was some surgical interference as well as another lesion found after death, viz. in No. 7, in which a tube was passed into the stomach, and in No. 9, in which the abdomen was explored; and although these operations may have had a certain amount of influence in determining the onset of the condition, there can, I think, be little doubt that the predominant factor in each was the growth, which, as will be seen later on, probably produced the effects by implicating surrounding nerve structures.

I may here mention another case where the dilatation followed an operation, of which Mr. Henry Morris has kindly given me the notes. The patient, a thin weakly man, underwent nephrectomy for polycystic disease of the kidney, after which all the symptoms of acute dilatation set in: the abdomen became unsymmetrically distended, and great quantities of fluid were vomited until the time of death. Although there can be no doubt as to the nature of the disease, as no post-mortem examination was obtained, I have not included it in the table on the preceding page.

There is yet another group of cases, in which in debilitated subjects the ingestion of a large quantity of badly masticated food appears to have been the exciting cause. In a case mentioned by Dr. Walter Broadbent,[1] a man after tramping about the country for two days without food, and who was therefore very exhausted, partook of a large meal of roast pork, after which he was seized with abdominal pain and vomiting, which in spite

[1] 'Medical Magazine,' July, 1901.

of treatment terminated fatally in two days. After death the stomach was found to be enormously dilated, the lower border reaching nearly to the pubes.

In a case recorded by Dr. W. H. Dickinson,[1] dilatation occurred in a child suffering from fatty degeneration of the heart, and after death the stomach, which was greatly distended, was inflated with gas and contained a large quantity of meat and potatoes, which were in lumps with sharp angles and edges, just as they had been cut by the nurse.

A case has also been recorded by Box and Wallace which followed an injury to the abdomen.[2]

Summary of symptoms and post-mortem appearances.

Distension of abdomen.—As might be expected, the distended stomach gives rise to a swelling of the abdomen; the swelling is not uniform, but fills chiefly the left half and lower part of the abdomen, the right hypochondrium sometimes appearing to be flattened. This swelling, which is of diagnostic value, is not, however, quite constant, for in the case recorded by Mr. Morris it is stated that the abdomen was retracted, and after death, although the stomach was enormously dilated and occupied almost the whole of the abdomen, its anterior surface was said to be flattened. No doubt the abdominal swelling varies with the vomiting, especially in those cases where there is a large quantity of fluid in the stomach, as in one of Fagge's cases, where the swelling disappeared after a quantity of fluid had been removed by the stomach-pump.

Peristaltic waves of contraction, with one exception, do not seem to have ever been observed in these cases, which, I think, rather opposes the theory suggested by Pepper and Stengel, that spasmodic contraction of the pylorus is the cause of the dilatation.

[1] 'Trans. Path. Soc. Lond.,' vol. xiii.

[2] 'Trans. Clin. Soc.,' 1898.

Vomiting.—Vomiting appears to be a constant symptom, and usually large quantities of brownish or greenish fluid are brought up. The fluid is usually thin and watery, and is generally vomited without causing the patient any great effort or distress.

Urine.—As a rule the urine becomes very scanty, and almost entirely suppressed for the last twenty-four hours before death. In seven cases in which the condition of the urine is mentioned, there was more or less suppression in five, and in three of these it was almost absolute during the last twenty-four hours of life; in two it is mentioned that a catheter was passed under the idea that there might be retention, but only a few drops of water were drawn off.

General symptoms.—The general symptoms are those of collapse: the pulse is small and very rapid, the respirations are frequent, and the temperature low, usually subnormal. There is also great thirst, which is probably accounted for by the excessive vomiting of fluid.

FIG. 2.

Diagram of shape of stomach in acute dilatation.

Condition of the stomach.—The appearance of the stomach as seen after death is very characteristic; it is

like a tightly distended cylinder, shaped like a V with one limb shorter than the other. The angle between the two limbs formed by the lesser curvature is a very sharp one.

The walls of the stomach, though so much distended, do not after the stomach has collapsed usually appear to be much thinned, and moreover they retain their elasticity, as shown by the contraction which occurs after death as soon as the contents are let out. There are, in fact, no definite abnormalities to be observed in connection with the stomach walls.

The intestines.—The condition of the intestines varies: usually they are collapsed and have the appearance of having been compressed by the distended stomach; sometimes parts of them may be distended, especially the duodenum, and in Fagge's first case there was some distension of the cæcum and ascending colon.

The immediate cause of acute dilatation of the stomach probably depends upon some disturbance of the nervous system, which gives rise to paralysis of the muscular walls, and which also frequently causes excessive secretion into the stomach cavity. Another explanation which seems within the bounds of possibility is that the distension might be caused by a rapid production of gas within the stomach. This mode of origin was actually suggested in one case, in which the patient was known to have drunk two bottles of effervescing lemonade not very long before the acute symptoms began; but Hughes-Bennett, under whose care the case was, rejected the idea, and preferred to leave the cause unexplained rather than suppose that gas sufficient to distend the stomach so enormously could have been generated by two bottles of lemonade. Neither is there, as far as I can find, any evidence whatever that there has been any undue putrefaction taking place in the stomach in any of these cases; and, moreover, this view of the causation would not explain the occurrence of excessive secretion.

In considering the part which the nervous system may

take in the production of acute dilatation there are two processes to be taken into account, viz. (1) the dilatation and (2) the increased secretion; and the question at once arises concerning the relationship of these two processes to each other: do they take place independently, or is one dependent in some way upon the other?

The inclination hitherto seems to have been to look upon the increase of secretion as the primary condition, and to regard the dilatation as secondary and immediately dependent upon it. This appears to have been the view taken by Fagge, when, in speaking of his first case, he says that the stomach was paralysed from over-distension and unable to rid itself of its burden.

Mr. Henry Morris also took this view; he considered that both dilatation and vomiting were due to excessive secretion, and on these grounds proposed that the disease should be called "acute gastrorrhœa."

In support of this view Mr. Morris quoted Moreau's experiments, which showed that after a loop of intestine had been isolated by ligatures, and all the nerves passing to it along the mesentery cut, a paralytic secretion took place, and the intestine was found to contain a quantity of fluid which on chemical examination proved to be a very dilute intestinal secretion. Dr. Pye-Smith and Sir T. Lauder Brunton[1] have shown that the regulating influences conveyed by the nerves divided in Moreau's experiments arise from some of the ganglia in the solar plexus.

As a result of Mr. Henry Morris's paper, Dr. J. F. Goodhart[2] brought forward notes of all the cases of dilated stomach not due to pyloric obstruction observed in the post-mortem room of Guy's Hospital from 1875 to 1882, and in the light of general information obtained from these Dr. Goodhart concluded that "paralysis of the viscus is, if not the determining cause, at any rate an accompanying condition."

[1] 'Report of Brit. Assoc. for Advancement of Science,' 1874 and 1875.

[2] 'Trans. Path. Soc. Lond.,' vol. xxxiv.

It is, of course, naturally very difficult to establish the exact relationship between the two conditions, but although they are so often present together, and produced by the same underlying cause, I think the available evidence shows that they are at any rate distinctly separate processes, and that the dilatation is not the mere mechanical result of excessive secretion. In some cases, for instance, there is very little fluid present, the stomach being in such cases almost entirely blown out by gas.

I have recently had an opportunity of observing an interesting case in which the stomach appeared to be in an early stage of acute dilatation, and in this instance there was no fluid at all, and only a very slight trace of semi-solid, almost completely digested food. The patient was an old woman who died almost immediately after the conclusion of a severe operation upon the lower jaw, and at the post-mortem examination the stomach was distended with gas, and had the cylindrical sausage-shaped appearance which is so typical in the more advanced cases.

The appearance of the stomach in this case is seen in the accompanying figure, which is taken from a sketch made at the time, for which I am indebted to Mr. W. T. Hillier.

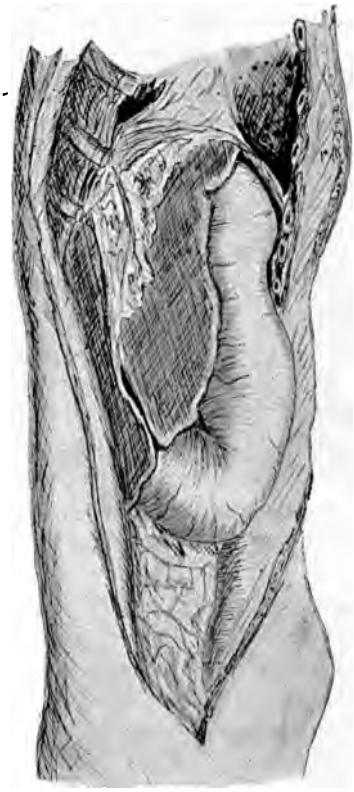
The stomach, though much distended (it measured about nine inches in length in the longer limb, and about seven inches at its greatest circumference), was nothing like the size which the others I have met with reached; but naturally there must be an early stage of the condition, and this case, I believe, is an example of such.

This case, then, as far as it goes, tends to show that the distension may take place independently of the secretion, and some information regarding the relationship between the two processes may, I think, be obtained from the consideration of cases of chronic dilatation which depend upon pyloric obstruction. In many of these there is a very great secretion, just as there is in the acute cases. Osler and Macrae,[1] for instance, mention a case of dilatation

[1] 'Cancer of the Stomach,' p. 81.

of the stomach due to malignant growth of the pylorus, in which on two occasions the stomach was washed and emptied as thoroughly as possible, and for forty-eight hours afterwards the patient was fed by the rectum, and

FIG. 3.



Appearance of a stomach in a stage of commencing acute dilatation.

all nourishment by the mouth stopped. At the expiration of this time, on the first occasion 545 c.c. of fluid were drawn off, and on the second occasion 500 c.c. In such cases as this the increased secretion is obviously secondary to the dilatation, as it also frequently is in cases of chronic

dilatation which are not due to obstruction; and it seems, therefore, that the increased secretion is an accompaniment of, or a result of, the dilatation rather than a cause of it. The two conditions, there is little doubt, are independent processes which come into action separately or in combination, and it is most likely that their relative importance varies. There can be no doubt that excessive secretion, when present, adds greatly to the gravity of the situation, but there does not seem to be any clear proof that excessive secretion can act as the dilating force, unless there is at the same time some paralysis of the stomach walls.

The stomach derives its nerve supply from the vagi and the splanchnic nerves; stimulation of the vagi gives rise to peristaltic movements, while stimulation of the splanchnic nerves brings the movements to a standstill. Sir Michael Foster,[1] in speaking of the nervous mechanism of the alimentary canal, says, "We may, therefore, speak of fibres inhibitory of peristaltic movements of the stomach and intestines as passing from the spinal cord through the splanchnic nerves, and reaching those organs through the abdominal plexuses." With regard to the nervous mechanism of secretion Sir Michael Foster says, "It has been suggested that while impulses reaching the stomach along the vagi excite secretion, those reaching the stomach along the sympathetic nerves inhibit it; but this has not been satisfactorily proved."

Dilatation of the stomach can probably be produced by the local interference of nerves of the stomach,[2] or it may arise after a shock affecting the general nervous system.

Examples of the latter are seen in cases such as that

[1] 'Text-book of Phys.,' part ii, p. 491.

[2] Paralysis of the muscular coat of the stomach limited to the pyloric portion, and preventing the propulsion of food into the duodenum, has been stated to be a cause of dilatation, and Wilson Fox ('Diseases of the Stomach,' p. 215) quotes a case recorded by Andral, where there was extensive ulceration of the pyloric region without obstruction, and yet extreme dilatation of the stomach. Traube attributes such dilatation to destruction of the branches of the pneumogastric nerve.

recorded by Mr. Henry Morris, where the dilatation followed an operation upon the foot; while examples of local interference are shown where the disease has followed some lesion in the neighbourhood of the stomach, and it is interesting to note how, in No. 10 of the preceding table, the onset of the condition appeared to coincide with the spreading of an acute pleurisy to the base of the left lung, which must be in close relationship to the nervous system of the stomach.

As cases of acute dilatation have followed closely upon operations, it is necessary to inquire into the possibility of the anæsthetic having some influence in their causation.

Mr. Morris considered this question fully with regard to his case, and came to the conclusion that the anæsthetic was not to blame, and examination of the other cases does not show any direct evidence that the condition can be traced to this cause; but it would seem quite possible that under certain conditions an anæsthetic might influence the dilatation through its wide-spread effects upon the nervous system.

The theory that there may be an obstruction of the pylorus must be given up, at any rate in many cases, since there is so frequently dilatation of the duodenum, and also since biliary contents have been noted in the vomit.

The idea that there is some obstruction lower down has received a considerable amount of support, and the fact that the distended bowel often terminates in collapsed bowel, at about the point where the superior mesenteric artery is situated, has suggested the theory that, under certain conditions, a traction of the mesentery takes place which pulls upon the artery and converts it into a constricting cord. The conditions which are thought to be necessary to bring this about are collapse of the intestines and their prolapse into the pelvis. While constriction by the mesenteric artery is probably mechanically possible, it is very doubtful whether the conditions necessary to produce it are often present during life, and

certainly in many autopsies the intestines, although collapsed, have not been found prolapsed into the pelvis.[1]

On the other hand, it seems very likely that after the stomach has become paralysed, and distension taken place, some secondary obstruction may take place, either through kinks in the pyloric region, or, as Box and Wallace [2] suggest, through pressure of the distended organ upon the duodenum. As above stated, the theory of obstruction seems to have largely arisen in order to account for the sudden termination of distended intestine in collapse, as is found in other forms of obstruction; but it must be remembered that if it be allowed that there is a primary paresis of the stomach wall, there is no reason why that paresis should not extend along the first few inches of the intestine as well. Against any constant cause of obstruction, such as the mesenteric artery, is the fact that the point where the distension ends varies; in some cases only the first part of the duodenum is involved, while in others the distension has extended some way along the jejunum. Also the fact that in many cases the bowels have acted freely (in some cases there has been severe diarrhœa) is against any marked obstruction by an external cause.

Acute dilatation of the stomach, though of course very much more rare, is probably closely allied in its causation and nature to the paralytic distension of intestines which frequently occurs after severe abdominal operations, and also in inflammatory conditions of the peritoneum. At present there seems no adequate explanation as to why the intestines should be distended in some cases and the stomach in others, though most likely this difference depends upon differences in reaction to stimulation of different nerve ganglia.

I think that acute dilatation of the stomach, to some

[1] The paragraph dealing with pressure of the mesenteric artery as a possible cause has been inserted since the paper was read. Further information on this subject, and also on the whole question of duodenal obstruction, will be found by Dr. William Ewart in the 'Lancet,' October 28th, 1899, and November 2nd, 1901.

[2] 'Lancet,' November 6th, 1901.

extent, is not so rare as supposed, and that if looked for all degrees of severity may be found between the slighter forms of dilatation—such as, for instance, are not infrequently noted in acute specific fevers—and the most severe and rapidly fatal cases, such as I have described to-night. Treatment of the recorded cases seldom seems to have been of any avail in checking the disease, but a few cases have been recorded as ending in recovery. Box and Wallace quote five, and Mayo Robson and Moynihan, in their recent work on 'Diseases of the Stomach,'[1] give an account of two cases which came under their care, and recovered after exhibiting all the typical symptoms of the disease. The most obvious indication in these very severe cases is to relieve the distension of the stomach by means of a tube, and this seems to have been an important factor, if not the chief one, in some of the cases which have recovered.

All nutrition should be administered by the rectum, and the tendency to collapse met by hypodermic injections of strychnine. Some of the more serious symptoms are probably produced by the loss of the large quantities of fluid which are secreted, and this loss should be counteracted by injection of saline solution into the rectum or by transfusion.

Lastly, it must be remembered that possibly on some occasions the condition may be a more general one than seems at first sight, and that the dilatation of the stomach may be one of the local manifestations of general collapse.

[1] 'Diseases of the Stomach and their Surgical Treatment.'

DISCUSSION.

Dr. T. R. BRADSHAW (Liverpool) thought the most obvious explanation of this condition was pyloric obstruction. If nerve disturbance leading to paralysis were the cause, how could the excessive vomiting be explained? For the stomach contents to be expelled active contraction of the stomach wall was necessary. If there were no obstruction at the pyloric orifice, it would have been expected that a portion of the gastric contents would have passed into the duodenum. The *post-mortem* examination, moreover, showed the intestines contracted, which also supported the theory of obstruction. A case was referred to of old gastric ulcer in which enormous dilatation of the stomach was present; on three occasions there was sudden dilatation with vomiting, and later sudden relief by the bowel. At one time an attack came on after eating carrot, and within the last year, when only liquid food had been taken, no attack had occurred, suggesting narrowing and obstruction at the pylorus. He suggested that the exciting cause of the dilatation was some kink of the pyloric region of the stomach.

Dr. W. P. HERRINGHAM could not understand how the excessive secretion could follow the acute active dilatation, as was apparently maintained in the paper, seeing that the organ was under constant outside pressure in the abdominal cavity. It seemed to be a necessary supposition that there must be a distending force. The gas described in the paper as being present in the stomach could not have been aspirated into the viscus from the outside; it must have been formed within the stomach, and this associated with the paralysis of its muscular wall was, he suggested, the cause of its dilatation.

Dr. ARTHUR VOELCKER referred to the case described in the paper in which the dilatation occurred in association with right-sided pleurisy. This, he thought, threw some light on the physical sign seen in left-sided pleurisy of a high stomach resonance. The vomiting of bile-stained fluid seemed to negative the obstruction theory and to favour the paralytic theory. In regard to treatment, he would hesitate to recommend lavage of the stomach, especially in such cases as were critical.

The PRESIDENT (Dr. F. W. PAVY) considered that this acute dilatation was comparable to the dilatation that might occur in the case of the bladder. Under atony or paralysis of the muscular wall the stomach would yield to a distending influence from within, but an active dilatation of the stomach was inconceivable. Either the secretion of fluid or the formation of gas or the presence of food was necessary for the dilatation. He had in former times seen the experimental division of the vagi

in a dog lead to paralysis of the muscular walls of the œsophagus and an enormous dilatation of it from the accumulation of the food that the animal had afterwards swallowed.

Dr. CAMPBELL THOMSON, in reply, said that peristaltic contraction had never been observed in these cases, and there was no *post-mortem* evidence in the cases he had described of any mechanical displacement of the stomach such as might lead to kinking. He did not mean that there was any active dilatation, but that paralysis was the primary factor, and the distending force of air or secretion the secondary factor.

ULCERATION OF THE ŒSOPHAGUS AND STOMACH

DUE TO SWALLOWING STRONG HYDROCHLORIC ACID

LESSONS OF TREATMENT DEDUCED FROM THREE CASES

BY

C. B. KEETLEY, F.R.C.S.

SURGEON TO THE WEST LONDON HOSPITAL

Received September 26th—Read November 12th, 1901

THE main conclusion I draw from a study of these three cases is that, in cases of poisoning by the more powerful corrosive acids, surgical intervention should be almost immediate, and that it is a mistake to postpone resort to surgery until there is no other alternative except that of letting the patient die of inanition. I will give the histories briefly, and then discuss the question of treatment.

CASE 1.—Enormous dilatation of stomach developed after accidental poisoning by strong hydrochloric acid. Loreta's operation. Complete relief, apparently lasting. Rapid recovery of flesh and strength.

In the middle of October, 1897, I was asked by my colleague, Dr. J. B. Ball, to see a patient of his, Alice M—,

aged 32, who had eight months previously swallowed pure hydrochloric acid by mistake. This was the most striking case of chronic dilatation of the stomach I have seen, and it is much to be regretted that no photograph was taken. When she stood up, whether she was viewed from the front or from the side, the whole anterior abdominal wall, except in the left iliac region and the extreme right of the right lumbar region, could be seen pushed forward by the stomach. The patient was very emaciated, and the shape and movements of the organ could be seen with ease.

There was a difficulty of swallowing, and immediate vomiting, which, together with the history of corrosive acid poisoning, suggested either stricture or spasm of the œsophagus as well as of the pylorus. In fact, at this time it seemed almost equally difficult to get food into and out of the stomach.

October 18th, 1897, *operation.* Incision in middle line above umbilicus. Stomach presented. Pylorus could be felt some inches away in the right iliac or lower part of the right lumbar region. No adhesions or signs of thickening of the stomach wall were found. The pyloric portion of the stomach was now "hauled" outside. The word "haul" gives a better idea of the length and size of the organ than would the usual word "pull." Protective gauze packing. One and a half inch incision into stomach. Pylorus thickened and so contracted that it would only just admit the closed blades of a pair of polypus forceps. Gradual dilatation was made, first with the forceps, next with the little finger, lastly with a three-bladed rectal dilator. With the latter the pyloric opening was stretched to a circumference of four and a half inches, and a slight sensation of tearing was felt. Closure and removal of dilator. Stomach washed out with hot water through wound. Suture of opening in stomach and of wound in abdominal wall.

Improvement began at once and progressed rapidly. Indeed, as soon as the patient had fully recovered consciousness after the anæsthetic she felt well, and com-

plained of nothing afterwards but an inordinate appetite. Vomiting ceased. The patient rapidly put on flesh. A year afterwards she was in good health and strength. The stomach never quite returned to its normal size. Last year I heard she had not been so well recently, but did not learn what was the matter. She has left her former address, and I cannot find her, so I cannot report on her present state. The patient was shown at the 1897 December meeting of the West London Medico-Chirurgical Society. I cannot remember who was the medical friend who saw her last year; but, if he sees this, I hope he will communicate with me.

Case 2.—Suicidal poisoning by strong hydrochloric acid. Rapid development of bronchitis and obstruction to breathing. Extreme weakness. Abdominal incision, but stomach not opened on account of sudden collapse. Great temporary improvement for ten days. Death three days later. Contraction of pylorus and pneumonia.

October 19th, 1897.—The day after the operation on Case 1, Case 2 was readmitted into hospital. Thomas P—, aged 25, had been first admitted under Dr. Hood.

History.—Thirty-two days before, he had swallowed strong hydrochloric acid with suicidal intent. After eighteen days in hospital he was discharged. He had then no pains and no physical signs of illness, and he "could swallow thin foods and milk." Very shortly after leaving he began to lose flesh, found difficulty in swallowing even liquids, and at last "what food he did swallow was vomited. Now there is a constant feeling of sickness."

On admission.—Emaciation. No ulceration or cicatrisation of mouth or fauces. Cannot swallow saliva. "Attempts to pass œsophageal bougies cause much distress and induce vomiting." Signs of pyloric obstruction. Greater curvature of stomach descends an inch below umbilicus. Palpation causes slight pain and excites spasmodic contraction. No thickening can be felt. Vomit for the most part liquid and very dark brown. Constipa-

tion. October 21st, rectal feeding commenced. October 28th, patient has been getting more and more emaciated. Mr. Keetley saw him and recommended operation. I noticed that he was then suffering from some bronchial or pneumonic affection with expectoration of copious blackish phlegm.

October 29th, *operation.* Made an incision to the right of the middle line. The pylorus was exposed, but before further steps could be taken, patient became collapsed and blue. It was considered necessary to postpone opening stomach. Iodoform gauze (wrung out in 1—2000 sublimate lotion) was placed in wound so as to prepare for a future second stage of operation, without anæsthetic, in a day or two. A few ounces of neutral saline were injected subcutaneously into the axilla, and fourteen ounces of warm milk into the rectum. A urethral bougie was passed down œsophagus.

My intention, as may have been inferred, was to enlarge the pylorus without a general anæsthetic about forty-eight hours after the unfinished operation; but a curious change in the patient prevented me. "He was much better in the night, and for the first time for eight days was able to take fluid by mouth." For the next ten days he took milk freely and easily, and improved in strength and spirits every day. But, unfortunately, on the eleventh day a good deal of pain was complained of in the right side. On the thirteenth day the note is: "Better night. Kept expectorating dark-coloured phlegm. Very collapsed in morning. Very little pulse. Gradually sank and died."

The temperature had ranged from 97° to 98·4°, rising only one degree the day before death.

Post-mortem.—The only observations noted are pneumonia of the base of the lung, congestion, and possibly a stricture of the upper part of the œsophagus, thickening and a very tight stricture of the pylorus, enormous distension of stomach.

It is difficult to be sure of what occurred in this case after the incomplete operation. Possibly the passage of the

urethral bougie down the œsophagus restored the power of swallowing, but as the pyloric stricture remained unrelieved, less fluid passed out of than into the stomach, and so the gastric dilatation increased and the general condition became more dangerous. I was lulled into a false sense of security by the improvement in the patient's spirits and appearance.

Notes by Mr. Flavelle and Mr. Granville.

CASE 3.—Suicidal poisoning by strong hydrochloric acid. Stricture and progressive ulceration of œsophagus and of pyloric part of stomach as well as of pylorus. Great emaciation and depression. Gastro-enterostomy with Murphy's button. Immediate relief and continued improvement for nearly six weeks, then death from bronchitis and pneumonia. Murphy's button found in stomach, and ulceration of œsophagus unhealed.

Emily B—, aged 46, admitted under Dr. Hood, September 13th, 1900. Patient got drunk and attempted suicide with strong HCl. "Spat out most of it." Mouth and fauces burnt by acid. Pain all down throat and in stomach. Great thirst. An emetic and then Pot. Bicarb. and calcined magnesia. Pulse 104, good volume and tension.

For ten days she had pain, and the vomit occasionally contained blood. Garg. Pot. Chlor. and Mist. Bismuthi Co. + glyc. acid. carbol. ℥x. The mixture seemed to remove both pain and vomiting. Nutrient enemata. September 30th (eighteenth day).—Mouth and lips healed. October 1st.—Milk by mouth. October 9th.—Nutrient enemata stopped. Takes more by mouth; great hunger.

Swallowing, unfortunately, became more and more difficult. Before October 27th (forty-third day) scarcely even the smallest quantity of liquid could be swallowed. Emaciation and weakness were extreme. Nutrient enemata had been renewed on the 23rd. October 27th, *operation*. Median incision. Pylorus presented at once in middle line, but could not be turned out, owing to extensive and tough adhesions. Incision prolonged up to xiphoid. Left rectus

and superjacent skin cut through transversely. Extensive strong adhesions of stomach to omentum, abdominal wall, and transverse colon, partly clamped and all divided or separated. Stomach could then be moved. The pyloric portion was contracted to the shape of a small sausage; the cardiac end was smaller than natural, and almost entirely under the left costal margin. An anterior gastro-jejunostomy with a Murphy's button was performed. The bowel and stomach apertures were tightened round the halves of the buttons by two or three interrupted fine silk sero-muscular sutures.

The contracted pyloric part of the stomach was three or four inches long. Its lumen would not admit the tip of the little finger,—in fact, seemed almost impervious. Parietal wound closed in layers. No drain.

Patient very collapsed after operation. Pulse in evening 176. Nutrient enemata not retained.

Milk and hot water (in equal parts) were therefore given by the mouth at once, ℥v (℥j every quarter of an hour). This was repeated in the evening; no vomiting. Patient's condition improved.

October 28th (day after operation).—Pulse 112. Liquid food retained both by stomach and rectum. October 30th. —Better still; pulse 90. Patient vomited altogether three times in the course of the first ten days. On the tenth night she retched a great deal. There was no abdominal tenderness. Did the Murphy's button fall back into the stomach at this time and cause the retching? Feeding by the mouth was stopped for twenty-four hours and then resumed cautiously with milk and "valentin." No more vomiting.

The wound healed. The patient increased in strength and cheerfulness. The temperature was normal till November 20th (twenty-fifth day after operation), when it began to rise gradually, and on the twenty-ninth day reached 101·6°; it only once reached 102° (four-hourly chart). With the rising temperature we noticed a slight cough with mucous expectoration. No pain or tenderness in epi-

gastrium. Some pain over base of right lung, and occasional paroxysms of pain in "left iliac region." Was this caused by the button, which X rays had, a week before, shown to have probably fallen back into the stomach? *Râles* on coughing, but no dulness at right base. November 24th.—Mucus slightly rusty. Chest tender when percussed. November 29th.—Sputum offensive, muco-purulent, more copious. December 2nd (thirty-ninth day after operation). —Cannot swallow solid food. December 4th.—Diarrhœa for last three days. December 5th.—Died collapsed.

Post-mortem (œsophagus).—At upper extremity a stricture two inches long, scarcely admitting a lead pencil. At cardiac end a second stricture, less tight, but with ulceration still active. Stomach:—Murphy's button free in the cavity. Ulceration quite healed. The cicatrised and contracted pyloric end has further contracted longitudinally to about half its length at the date of the gastro-enterostomy (six weeks before). A narrow curved or sinuous passage leads through it into the duodenum.

The gastro-enterostomy was perfect, with a free passage into distal loop of jejunum, and a narrower one into proximal.

The extensive adhesions observed at the operation had nearly all disappeared, and nothing remained to interfere with free movements of the stomach.

Lungs.—Large bronchi ulcerated and containing foul purulent secretion. Grey hepatisation of left lung throughout. Some pneumonia at base of right lung.

Notes by Mr. Bennett and Mr. O. Inchley.

Remarks on the three cases.—That which was least injured and non-suicidal recovered, but passed through a period of illness, which if left unrelieved must have had serious consequences.

Both suicidal cases were much worse than Case 1 at the time of operation. Besides, in both cases the bronchial trouble began before operation. In Case 2 the following note was recorded the day after the acid was swallowed:—

"Large mucous râles all over chest, back and front." Two days later there is the note, "Respiration, especially at night, is very noisy, and sounds as if it was obstructed." The note on the day of his discharge is, "Lungs practically clear;" it continues, "no sickness, no dysphagia, no" etc., etc. Nevertheless this patient had to be readmitted in fourteen days worse than ever. There is no note about his respiratory organs on readmission, probably because attention was concentrated on his serious œsophageal and gastric trouble. Cases 2 and 3 did not suffer to anything like the same extent as Case 1 from gastric dilatation; indeed, Case 3 had a stomach much smaller than normal. But they were more seriously injured in the œsophagus. A careful post-mortem examination of that organ from Case 3 persuades me that its ulceration was still progressive rather than healing, although her death occurred forty days after operation and eighty-three days after swallowing the corrosive acid. Both fatal cases died of septic broncho-pneumonia; I see no reason for attributing this to the operations. In Case 2 nothing was done but the making a small incision in the abdominal wall. In Case 3 healing was rapid. A post-mortem examination showed the gastro-enterostomy to be perfect, and not only was there no peritonitis, but most of the adhesions seen at the operation, forty days before, had been absorbed.

I believe the ulceration of the œsophagus or of the pharynx leads to the infection of the air-passages. This may occur directly through the lymphatics, or indirectly through the passage of muco-purulent discharge downwards through a glottis, perhaps itself œdematous or thickened, or otherwise impaired by the action of the acid, not necessarily on the glottis itself, but on parts closely adjacent to it.

At the same time the physical strength, and mental and moral state of such patients, are lowered extremely, by both the causes and the results of the accident. The utmost conceivable depth of "lowness" is reached by a patient who, as a consequence of swallowing a corrosive

acid suicidally, is for a long period neither able to pass food through the œsophagus nor chyme through the pylorus.

The question of treatment.—These cases seem to me to teach certain lessons. Conclusions should be drawn cautiously from a short series of only three cases; but the rules I am going to lay down are indicated by these cases, not only collectively, but individually. It is not, therefore, a mere matter of statistics.

1. The patient should receive no food (either liquid or solid) by the mouth for several weeks, *i. e.* he should not be fed by the mouth as soon as he can swallow with little or no pain; but *oral feeding should be postponed until there is good reason to believe that the injuries have completely healed.*

2. *When the injuries are serious (and they generally are so), an operation should be performed within a few days of the date of the poisoning, the sooner the better.*

It must always be a matter of conjecture to determine whether the injuries have healed or not. The only parts of the injured tract visible are the mouth and pharynx. But the pylorus, or even the middle of the stomach may be much worse, as, *e. g.*, in Case 3, not to mention the œsophagus. In this Case 3, the mouth and lips are noted as healed on the 18th day; but active ulceration of the gullet was found after death, on the eighty-fourth day (three months after the accident).

I am afraid that it is rarely safe to assume that a case of this kind is not serious, unless it is positively known that only a minute quantity of acid has been swallowed. Case 3 was scarcely a truly suicidal one. The patient got drunk on the "rent money," was scolded by her husband, drank the acid, but spat most of it out again, was brought to the hospital and made to swallow calcined lime and an emetic, and yet her injuries were terrible.

Even when swallowed by pure accident, an ounce or more is easily taken into the gullet before the mistake is discovered, and most of it passes into the stomach, run-

ning along the lesser curvature till it is stopped by the pylorus or by food already in the stomach.

All the three cases were treated according to what seems to be the usual practice, that is they were allowed to swallow food when they could do it without much difficulty or pain. Case 2 was fed by the mouth from the very first day. Case 3 began with milk on the 6th day, and took puddings on the 15th. She never got as far as fish or meat.

Granting that the patient should not be fed at all by the mouth for several weeks, and that we should only be satisfied with rectal feeding in trivial cases, the severe cases remain to raise the *question of Operative Treatment.*

The region most seriously injured is usually the pyloric part of the stomach. The problem for the surgeon's solution is not simple.

The choice of operations apparently lies between gastrostomy, duodenostomy, jejunostomy, gastro-enterostomy, and a combination of gastrostomy with gastro-enterostomy.

Gastrostomy does not give rest to the most injured part, namely the pylorus. Gastro-enterostomy does not rest the œsophagus. Jejunostomy, when properly done, is practically a double operation. Duodenostomy would seem to be the simplest and most straightforward procedure, although it is liable to permit bile to leak out and irritate the skin.

All these methods are open to the objection that, in the by no means unlikely event of an œsophageal or a pyloric contraction taking place after all, a secondary operation may have to be done, in addition to one for undoing the first operation.

Therefore the indications would most likely be best met by *combining a gastrostomy with a gastro-enterostomy,* and carrying the gastrostomy tube through the gastro-enterostomy wound for some distance down the efferent loop of the jejunum. This is, practically, the method recommended by Witzel for an ordinary gastro-enterostomy, except that

in the cases I am writing about something more is desirable, viz. an arrangement for washing out and draining the stomach through the gastrostomy wound. This could be obtained either by using a double tube, specially constructed so that the shorter channel opened into the stomach; or, more readily, by passing the long, narrow gastro-jejunal tube through a short, wide gastric tube.

If in spite of treatment pyloric contraction should take place, this plan provides a gastro-enterostomy ready made. The gastrostomy wound could be closed or kept open, according to the final condition of the œsophagus.

In the hands of careful and experienced operators the method would probably be found very safe, as the patient would be operated on while in fair physical condition, and the gastro-jejunal tube should resist the dangers of the "vicious circle." These dangers could be further minimised by using Murphy's button, and, should that contrivance fall into the stomach, the gastrostomy wound itself could be enlarged to permit its extraction.

Further, the treatment above recommended would reduce to a minimum the danger of infection of the air-passages through the swallowing or the regurgitation of septic discharge, or of food, either of which might easily find its way through a glottis œdematous and stiffened either by direct injury or by injury to neighbouring parts.

In a long series of cases of poisoning by corrosives, now and then the glottis, etc., is likely to be so severely injured as to demand prompt tracheotomy. This would make it more than ever desirable not to feed by the mouth.

At the post-mortem examination of Case 3 I was struck by the resemblance of the ulcerated bronchi to those of a case in which bronchial infection and gangrene had been caused by a tracheo-œsophageal fistula.

After-treatment.—Feeding by the gastro-jejunal tube would be commenced at once, in spite even of moderate ether or chloroform vomiting, should those anæsthetics be used. But gas alone, or with oxygen, would suffice for the operation described, or even local anæsthesia.

Local treatment should be given to (1) the mouth and nose, (2) the pharynx and œsophagus, and (3) the stomach.

1. *The mouth.*—This should be frequently washed out with warm solution of chlorate of potash or warm boracic lotion, or both. Dirty teeth should be cleaned, diseased teeth treated with pure carbolic acid or by extraction, and suppurating alveoli attended to. If the process is very painful owing to the burning, then gas, cocaine, or eucaine should be used.

The nasal passages should be attended to if unhealthy. If healthy they should be left untouched.

2. The pharynx is said not to be reached by gargles. It should be sprayed frequently with hot boracic lotion, and twice a day dusted with a little, not much, iodoform powder through a puff.

As soon as the patient can swallow without pain, he should be allowed hot water or hot neutral saline *ad lib.*, and be encouraged to take it.

The stomach.—The hot water swallowed should be allowed to escape by the short gastrostomy tube, so that it would tend to wash out the stomach also. In addition, after each meal given by the gastro-jejunal tube, the stomach should be washed out with hot water by the gastric tube.

The application, in some such way as that above sketched, of the principles of surgery to this distressing class of cases would, I believe, greatly reduce their mortality, and lessen the permanent injury done to those who more or less recover.

DISCUSSION

Mr. E. Percy Paton referred to a case which came under his care four weeks after swallowing hydrochloric acid with suicidal intention, a woman aged 30, in whom the chief injury seemed to be to the œsophagus; but an œsophageal tube was fairly easily passed, and she improved for a time under washing out of the stomach. A few days later, however, great distension of the stomach supervened; it was not dilated but very tense, and felt about the size of a fœtal head. He ascribed this distension to obstruction both at the œsophageal and pyloric openings of the stomach. An incision was then made into the stomach, and the first part of the duodenum was so ulcerated that gentle pressure with the finger through the pylorus caused rupture of its wall. The rupture was closed by suturing transversely and by an omental graft; this held well, but the patient died in forty-eight hours. At the necropsy it was found that there was only a small portion of the gastric mucous membrane unaffected by the acid. A gastro-enterostomy might have given more chance of recovery, and he regretted that he had not performed that operation.

Mr. Clinton Dent believed that any operation performed in the dark, such as Loreta's operation, was unsatisfactory. A pyloroplasty was, in his opinion, much more satisfactory, as in that operation a good view was obtained of the mucous membrane of the stomach and duodenum, and the extent of the injury done by the acid could be estimated. Where there was dilatation of the stomach a gastro-enterostomy was not a good method, and with a Murphy button still more unsuitable. Senn's plates or stitching were methods to be preferred. The avoiding of feeding by the mouth for a long time was questionable. A broad rule after gastric operations was to feed by the stomach as soon as possible, providing the food were hot. Suicidal cases, it had seemed to him, did just as well as non-suicidal cases, the depressed psychical condition, as far as he had been able to observe, in no way retarding healing and recovery.

The Chairman (Mr. J. Warrington Haward) thought it was rare for the œsophagus to be so injured in these cases that a tube could not be passed; the difficulty in swallowing arose largely from spasm and painfulness. For stricture of the pylorus pyloroplasty seemed to be the best method, but if that were contra-indicated by any condition such as duodenal ulceration, a gastro-enterostomy could be performed. For this he preferred Senn's plates to Murphy's button on account of the

probability of the latter falling back into the stomach. He did not think it probable that any contraction would occur around the gastro-enterostomy opening if the edge were sewn round with a fine continuous suture. The healing of any part of the body depended on the general nutrition, and feeding by the mouth helped this much more than feeding by other channels. He had never regretted feeding a patient by the mouth too soon after the operation, but he had had cause to regret not having done so earlier.

Mr. KEETLEY, in reply, said that the case described by Mr. Percy Paton was another instance of the need for early operation. When the pylorus was not seriously injured, *e. g.* as in Case 1, Loreta's operation might suffice, and it was safer than either pyloroplasty or gastro-enterostomy. Comparative statistics of the operation were at present misleading, because those of Loreta's operation were collected from an early period in which all these operations were more dangerous than now. In recent years the mortality of gastric operations in general had diminished, but Loreta's operation had gone out of fashion. He also favoured the use of a Murphy button; as Miculicz had pointed out, it prevented the development of a vicious circle, in the way of the contents of the stomach passing through the gastro-enterostomy opening back into the duodenum and into the stomach again. Supposing that the Murphy button did fall back into the cavity of the stomach, it could easily be removed.

CASE OF INTESTINAL OBSTRUCTION

DUE TO THE

PRESSURE OF A VESICAL SACCULUS UPON A COIL OF SMALL INTESTINE

BY

THOMAS BRYANT, F.R.C.S.

Received August 6th—Read November 26th, 1901

On April 8th, 1901, I was asked by Dr. M. Biggs, of New Wandsworth, to see Mr. F—, aged 67, who had been suffering for five days from intestinal obstruction, and had been vomiting brown fœtid fluid for twelve hours.

Dr. Biggs had been attending Mr. F— for four days previously for what he regarded as angina pectoris; but at my visit the symptoms of this affection had been much relieved, and the attacks had become less frequent and severe.

It was during these early days that the bowel complication had appeared, and persisted in spite of the use of such medicines as had previously given relief; and it was owing to the investigation by Dr. Biggs into the cause of the obstruction that he found in his patient's abdomen the enlargement of a tumour the existence of which he had

recognised in the autumn of 1899, Dr. Biggs having at that time been called in for some passing bladder trouble associated with a difficulty of micturition, which was successfully treated by the passage of a catheter on a single occasion.

At that time Dr. Biggs had, however, made out that there was some enlargement of the prostate gland, and had discovered the presence of a small firm tumour on the right side of the median line of the abdomen over the region of the bladder. There were then no special symptoms, and up to the time of my being called into consultation, the patient had been practically free from all bladder complications—indeed, Dr. Biggs had not been consulted by his patient from June, 1900, until April, 1901, the date of his present illness.

When I saw the patient on April 8th, he was evidently very ill and feeble. His angina symptoms were not in evidence, but those of obstruction were well marked; he had not passed a motion for five days, and had but recently brought up some fœtid brown fluid. His abdomen was somewhat swollen but not tense, and the swelling occupied the left central abdominal region below the umbilicus, which suggested small intestine obstruction, and over this region the percussion note was mostly resonant.

On the right of the median line of the abdomen a tense sausage-like swelling was however made out, which extended upwards as high as, if not above, the umbilicus and to the right beyond the semilunar line, and over this area there was distinct dulness and much resistance.

The prostate gland was examined and found to be somewhat enlarged, and with the finger in the rectum it was thought that some resisting growth at the brim of the pelvis could be felt. There was no difficulty in micturition, and the urine passed was clear and sweet. In my presence the patient passed several ounces, and he was sure that he could quite empty his bladder. No change in the size or tension of the tumour followed micturition. The tumour when the bladder was emptied seemed to be

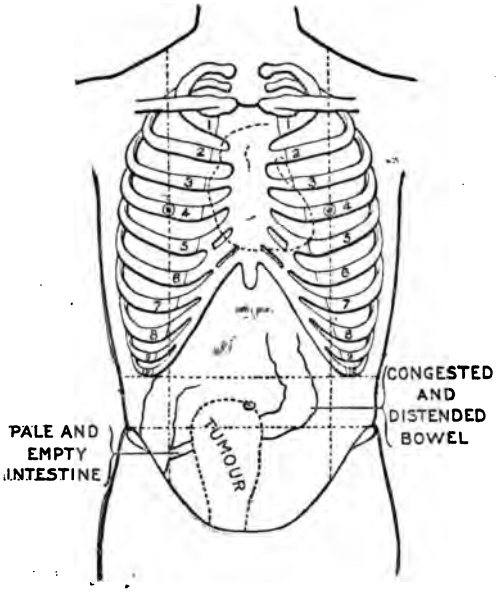
slightly movable from side to side, its manipulation was not painful, nor did external pressure appear to make any change in its condition.

On this visit I advised the use of full enemata, and on these failing, and the symptoms of obstruction persisting, an abdominal exploration.

The means suggested, although well applied, were not successful; the fluid thrown into the bowel returned hardly more than discoloured, and the vomiting not only persisted, but by the 10th had become fæcal. Under these circumstances an exploratory operation was decided upon.

This was carried out on the early morning of April 11th, with the patient under the influence of ether, which he took well.

I made an incision over the abdominal tumour in the right semilunar line, and came down upon an empty



cæcum and some pale empty coils of small intestines (*vide* diagram), situated on the right of a tense elastic sausage-

like tumour, one coil of empty bowel being found emerging from between the tumour and the bodies of the lumbar vertebræ (*vide* diagram). There were no signs of local inflammation. I passed my finger in front of the tumour, which was in contact with the abdominal parietes, and also behind the tumour, which pressed backwards upon the spine, and in so doing found that the tumour had so pressed upon the small intestine as to occlude it, for, as already described, the small intestine to the right of the tumour was flaccid and empty, whereas that on the left was much distended, congested, and full.

The upper end of the tumour was rounded and unattached, the lower end seemed to be attached to the bladder. A catheter was then passed and a quantity of clear limpid urine drawn off, but this action had no influence upon the shape of the tumour. I then, with the catheter in the bladder, pressed with my fingers—which were grasping the tumour—upon its body, when slowly and surely the tumour was emptied, and the conclusion was forced upon us that we were dealing with a vesical sacculus which had a very small orifice of communication with the bladder. The contents of the sacculus were like clear urine. The parts were then readjusted and sutured, and the patient put to bed. For some hours after the operation Dr. Biggs reported the patient seemed to be under the anæsthetic and slept peacefully. His pulse was good; some flatus had passed downwards, but no motion. At 8 p.m. he had hiccough, which was so sudden and severe as to jerk the whole body, and seemed momentarily to lift the body from the bed; at 9.30 p.m. some ounces of urine were drawn off. During the night he was restless, and at 2 a.m. his breathing became bad; three or four breaths were taken and then a long pause. Shortly after dawn he had a rigor, and the temperature ran up to 104°. From this time he gradually sank, and died about 8 a.m. on April 12th, or about twenty-two hours after the operation. No change in the abdomen was observed.

No post-mortem examination could be obtained.

Remarks.—This case is published as an unusual one—for it seems certain that the cause of the patient's intestinal obstruction was due to the pressure of the sausage-shaped vesical sacculus upon a coil of small intestine which passed behind it, and between it and the spinal column; and this view is supported by the fact that on opening the abdomen in our operation the colon and small intestine on the right of the tumour were found pale and empty, whereas the small intestine on the left side was found full and congested, the seat of pressure upon the bowel by the tumour being very evident.

It is likewise clear that the opening of communication between the sacculus and the bladder must have been very minute, for the bladder seemed to have performed its functions during the formation of the sacculus in apparently a satisfactory way, and even at the time of operation, when the bladder was emptied by means of a catheter, the tension in the sacculus was not materially affected, for it was only upon my manually compressing the sacculus that it was emptied, and then but slowly.

When I first felt the sacculus during the operation, I thought of the possibility of its being a urachal cyst; but when I found its upper end was free, unattached, and rounded, I dismissed the thought, and from the position of its base upon the upper right half of the bladder the question of its having any connection with the ureter was not entertained. The conclusion therefore remains: That as a cause of intestinal obstruction, a vesical sacculus must not be forgotten as a possible one.

DISCUSSION

Mr. Reginald Harrison referred to two cases in some respects similar. In one, occurring in connection with a large posterior vesical sacculus, there were long bouts of obstinate constipation, for which no explanation could be given. An operation was performed, and the sacculus was found to be in contact with the rectum, and pressing upon it so that it was evident the constipation was due to this cause. After it was drained the constipation, which had been serious, disappeared. In the other there was a large suppurating vesical sacculus, the apex of which at the necropsy was found adherent to and constricting a coil of small intestine. The lumen of the gut was at the point of adhesion constricted to about half its normal diameter.

Mr. Clinton Dent asked as to the actual condition of the gut, whether diseased or otherwise, particularly if there were any stenosis. In the absence of inflammatory adhesion, the obstruction of the bowel from mere pressure was extraordinary.

Mr. Bryant, in reply, remarked that Mr. Reginald Harrison's first case was comparable with that which he had described, but the second differed, inasmuch as the sacculus was adherent to the intestine and had caused actual narrowing. In his own cases it was quite obvious that the pressure of the sacculus was the cause of the obstruction, for there was no organic stricture, and as soon as the sacculus was raised the contents of the distended intestine passed into the empty intestine below. The intestine above the sacculus was congested, while that below it was absolutely white and empty. There was no evidence whatever of adhesion or organic disease.

AN ANALYSIS OF FORTY-SIX CASES

OF

CANCER OF THE BREAST

WHICH HAVE BEEN OPERATED UPON AND SURVIVED THE OPERATION FROM FIVE TO THIRTY-FIVE YEARS

With Remarks upon the Treatment of Recurrent Growths, including the Disease of the Second Breast, Operative and otherwise

BY

THOMAS BRYANT, M.Ch., F.R.C.S.

CONSULTING SURGEON TO GUY'S HOSPITAL.

Received March 10th—Read May 13th, 1902

THE paper I ask your attention to this evening should be regarded as a sequel to a communication made by Mr. Marmaduke Sheild, on January 25th, 1898, to this Society, when I had the honour to occupy the presidential chair; as it was from the interesting collection of facts which he had gathered from varied sources, and analysed, that I was led to search my own note-books, and to extract from them such material as might throw some light upon—(1) the prospects of life after primary operations for cancer of the breast; (2) the question of recurrence of the disease at the seat of the primary operation and second breast; and (3)

the nature and effects of operation upon the progress of the disease.

It must, however, be steadily borne in mind by the readers of this paper, that the cases tabulated include only such examples of cancer of the breast as have been under my care, and have been operated upon, and have survived the primary operation five years and upwards; for I have always felt that the three years' freedom from recurrent disease after a primary operation, which has been so dogmatically laid down as a significant indication of a cure of cancerous disease, was not only unreliable but misleading.

In my book on 'Diseases of the Breast,' published in 1887, I satisfactorily showed (page 152) the inaccuracy of such a view, and pointed out that if, after the primary operation for cancer of the breast, forty patients out of sixty there tabulated died within this three years limit, there were at least twenty patients who had survived the primary operation from five to ten years, for four of these twenty instances lived for eight or nine years, and six for ten years.

In the tables I now bring before you, many instances of much longer survival after the primary operation will be found recorded, and likewise many instances of recurrence of disease after prolonged periods of immunity which are very striking.

Group I

includes seventeen cases of cancer of the breast relieved by operation which are now alive, or have died without evidence of recurrent disease, five or more years after operation.

Of this group four have died, and thirteen are living and in good health.

Of the four which died—

Case 15 died from an accident, aged 62, five years after the primary operation.

Case 16 from old age, aged 80, twenty years after operation.

Case 14 from acute jaundice, aged 63, fourteen years after operation, and

Case 13 from intestinal obstruction due to gall-stones, aged 79, thirteen years after operation. In both of these cases a necropsy was performed, and no evidence of recurrent disease was found.

Of the thirteen cases which are now alive and well, one has remained free from recurrence for five years, one for six years, three for eight years, three for nine years, two for ten years, two for fourteen years, and one for sixteen years.

Taking the whole group of seventeen cases together, there was an absence of any evidence of recurrent disease from five to ten years after the primary operation in nine cases or in more than half, and from ten to twenty years in eight cases, thirteen of these patients being now alive and apparently well.

I should like here to say that the operation I now do, and have done for many years, is neither the one I was originally taught and had seen practised by my senior colleagues—which was certainly inadequate—where lymphatic glands were rarely removed, or anything more than the diseased breast itself, with the skin covering it when involved; nor is it the more modern operation known as "Halsted's," and made public in 1894, but which should be known as "Moore's," or Banks's, who advocated the principle of free removal in 1882, not only of the diseased breast with the fat and skin over it in every case, but also of the pectoral muscle, fascia and lymphoid tissue from the axillary vessels, and which is now known as the complete or adequate operation.

The Operation adopted.

My operation is something between the two, but nearer the latter than the former, and I hold with the results before me that it is a complete and adequate measure under the most favourable conditions.

My routine operation is to remove the whole gland that

is diseased with the skin and fat over the diseased area; when the axillary glands are enlarged to dissect out the axilla and subpectoral spaces, and in every case, for examination purposes, to cut into the axilla, and to take away glands or lymphoid tissue which appear to be suspicious, but otherwise not to dissect it out, my incision into the axilla skirting the axillary border of the pectoral muscle. I invariably drain the wound through the axilla for the first two or three days.

The pectoral muscle I dissect clean, but do not remove it, although, should disease be found to have invaded the muscle, the diseased muscle must be freely taken away. I regard the removal of the muscle as a routine measure to be unnecessary, and the facts I now bring before you tend to support this view,—for I am more impressed by accumulating experience that successful results in operations for cancer are more certainly to be secured by an early operation than by "performing tremendous operations upon practically hopeless cases."[1]

I may say at once that it was from the careful study of Moore's memorable paper on "Inadequate Operations on Cancer," published in 1867 in the fiftieth volume of the 'Transactions' of this Society, that I was led to deviate from the practice I had been taught, and to follow, as far as I thought right, in the lines of Moore's suggestions, which have been, without question, the basis of all recent operative procedures.

In more recent times the principle of free removal of cancerous disease has been well brought before the profession by Sir W. Mitchell Banks in papers of great importance published in 1877, 1882, and 1900 in the 'British Medical Journal,' and it is through him more than any other writer that Moore's views have become established.

What I regard as a point of more importance than so-called complete or adequate operations is early interference, and in my sanguine hours I have imagined with Sir

[1] E. Banks, 'Brit. Med. Journ.,' Jan. 4th, 1902, p. 5.

Mitchell Banks what the results would be if all cancers were thoroughly excised when they were no bigger than peas, or, as I would prefer to say, when the disease is in its very early stage.

Indeed, I am fairly sure that it has been from my acting upon this principle that I am enabled to bring before you to-day the satisfactory results of treatment which my tables indicate, for in Group I, in which there are seventeen cases tabulated, the disease was in most of them in an early stage of development when submitted to operation. The disease appeared, when I first saw the cases, as a lump in the breast without skin implication or lymphatic glandular enlargement, and in which the question arose as to the lump being due either to the presence of a cyst or early cancerous infiltration, for at this stage of the tumour's growth the question could only be settled by an exploratory incision.

Under such circumstances an exploratory incision was made into the lump, and when cancer was recognised the gland was removed. Under these circumstances the good results which have been recorded are to be explained, and they are certainly satisfactory. They are, moreover, what I expected they would be when I operated, for in 1900 I wrote a paper for a sister society [1] on 'Cysts of the Breast: their Relation, Frequency, Diagnosis, and Treatment,' and in composing it I analysed 242 consecutive cases of breast disease, as they had recently appeared before me in private practice; 163 of these cases were registered as solid tumours or examples of cancer or sarcoma, and 67 as cases of cystic disease.

Of these 163 diagnosed as solid tumours, 126 were operated upon, and out of the 67 examples of cyst disease 44 were operated upon, the percentage of cyst disease to cancerous disease being 25 to 74; the conclusion becoming clear that out of every four cases of breast disease, more or less simulating cancer, one will prove to be an example of cyst disease.

[1] Medical Society of London, vol. xxiii; 'Lancet,' April 28th, 1900.

I went, however, much further, and was able to show that if we eliminate from our consideration all such examples of cancerous tumours of the breast as are so well marked as to forbid an error in diagnosis being made, and apply our argument to those alone in which there is only a lump in the breast gland without any collateral symptoms to support a diagnosis of cancer, it would not be wrong to conclude that in every two cases of this kind one will be cystic and the other cancerous.

In these cases of early cancer an exploratory operation was undertaken, and when the tumour was found to be cancerous the gland was removed by the mode of operation I have described, the operation having been undertaken at the period of the tumour's growth after which the most favourable result might be expected. In all of these the axilla was explored, but not dissected, the incision I adopt allowing the finger to explore the subpectoral spaces.

In all of these cases the whole gland was removed with the fat over it and integument. In all of these the pectoral muscle was well cleaned, but *not* removed. In a few of these only were enlarged lymphatic glands found. In all of those early cases microscopic evidence was sought, and found to correspond with that which the naked-eye appearances had suggested. In fact, in the majority of the cases of this group, as well as in many in Group II, the same remarks are applicable. The disease in all was palpably cancer, and the success recorded is due to its complete and early removal.

Group II.

This group includes nineteen cases of cancer of the breast relieved by operation and followed by recurrence in the seat of the primary operation.

In three of the cases (Nos. 12, 17, 18) recurrence took place in the scar of the primary operation, and a second

operation was performed one year after the primary; two of these three cases were well and in good health four years later, and in the third case ten years later.

In nine cases recurrence occurred from three to seven years after the primary operation. In three of these no second operation was called for.

In one of the three cases in which no operation was performed (Case 4) the recurrent disease appeared as tubercles in the flaps three years after the primary operation and spread slowly for nine years, when bladder disease appeared. In another (Case 1) the recurrence showed itself as a sternal growth six years after operation. In the third case (No. 2) chest symptoms appeared seven years after operation.

In the six other cases second operations were undertaken three, three, four, five, five, and seven years respectively after the primary. In one (19), three years after the first operation a tumour was removed from the axilla, and the patient died six years later from lung disease, aged sixty-five, having survived the first operation nine years. In Case 14, where a second operation was called for three years after the first, the patient was well six years later. In Case 15, where an interval of four years had passed between the first and second operation, the patient was well six years later. In a fourth case (6), where a second operation was called for five years after the first, a recurrence took place after a second five years, when chest symptoms appeared. In the fifth case (13), where a second operation was performed five years after the first, and a third small one two years after the second, the patient was active and in good health twelve years after the first operation and five after the last. In the sixth case (16), where a second operation in the scar was performed seven years after the first operation, the patient was well in all ways five years later, or twelve years after the breast was removed.

Of the seven other cases of this section of the group the intervals between the first operation and a recurrence were from ten to thirty years.

In Case 10 of the tables a woman aged fifty-two was operated upon, and had no recurrence for *ten years*, when it appeared in the scar, and as the local disease gave her no pain and was of slow growth it was left alone.

In Case 8, where a woman of fifty was operated upon, a recurrence took place *eleven years* afterwards, when a second operation was performed upon the scar, and she was well two years later.

In Case 3 a woman aged sixty had been operated upon, and a recurrence was suggested *twelve years* later by abdominal symptoms.

In Case 7, a woman aged thirty, a recurrence of disease in the flaps took place *thirteen years* after the primary operation. A second operation and a small third were performed, and one year after the last she was well.

In Case 5 the woman, when thirty-eight, had her breast removed for cancer; twenty-five years later she had a recurrence in the skin over the seat of operation, which spread, but she was alive five years later.

In Case 9, where a woman aged forty-six was operated upon, no return took placed for thirty-one years, when it appeared as a sternal growth, and five years later this patient was eighty-two, and in good health.

In Case 11 the patient had been operated upon when forty-six years of age, and thirty-two years later, when seventy-eight years of age, she had a recurrence upon the sternum, but was otherwise well.

Group III.

This group includes ten cases of recurrent disease after operation in which the second breast was involved; and four cases in which the breast disease was associated with cancer of other parts of the body.

In four of the ten cases (Nos. 22, 24, 26, 29) the second breast was attacked about two years after the first had been removed. In one (No. 27) of the six other cases the

second breast became diseased three years after the primary operation. In two others (25 and 28) ten years elapsed before the recurrence appeared; and in the two other cases (Nos. 21 and 23) the interval between the primary operation and the appearance of the disease in the second breast was respectively twenty-three and twenty-four years, the recurrent disease having in both these cases involved at the same time the scar of the first operation—this fact suggesting to the sceptical mind the truth of the view that the primary disease had been cancerous.

In four of these ten cases the second breast was not removed, the local disease having been extensive and inoperable. In Case 21 the patient was sixty-seven years of age, in Case 23 eighty years of age, in Case 29 forty-eight years of age, and in Case 27 only thirty-eight years of age.

In the remaining six cases the second breast was removed. In one (20) no signs of return were to be traced six years later; in Case 24 no signs of return existed five years later; in Case 26 the patient was well two years later, and in Case 28 three years later. In Case 25 there was no recent history.

The four remaining cases in Group III have been added as cases of interest, but they do not form any part of my tables.

In Case 30 a woman aged sixty had her breast removed for cancer, and came under care fourteen years later, when seventy-four years of age, for cancer of her hand, which was treated by amputation.

In Case 31 a patient who was treated for epithelioma of the nose at the age of sixty-eight with success returned for treatment five years later, when seventy-three years of age, with an acute cancerous affection of her breast, lymphatics, and skin, which was inoperable.

In Case 32 a woman, who came under treatment when seventy-two years of age with atrophic breast cancer of

twenty years' standing, reappeared six years later with an epithelial cancer of her nose.

The last case (33) is one in which an annular cancerous stricture of the rectum co-existed with an extensive cancerous affection of the left breast of four years' growth.

I regard these cases as illustrative of coincidences in the history of cancer, and record them as such.

I propose now, in order to make the questions respecting these Groups II and III of recurrent cases clearer, to analyse them further, and to subdivide them into tables, in order to show—

First, the length of the interval that existed between the first operation and the recurrence of the disease.

Second, as to the seat of the recurrence.

And thirdly, as to the duration of life after operative interference.

Table I of Group II.

Including eight cases of recurrence not requiring operation.

Number of case in Group II.	Length of interval between first operation and recurrence.	Age of patient at time of recurrence.	Seat of recurrence.	Subsequent history.
4	3 years	55	About scar	Spread slowly for nine years, when bladder symptoms appeared.
1	6 years	56	Sternum	In good health.
2	7 years	61	About scar; chest symptoms	—
10	10 years	62	About scar	Very slow growth.
3	12 years	72	Abdominal symptoms	—
5	25 years	63	In scar	Atrophic cancer, alive five years later.
9	31 years	77	Sternum	Alive five years later, aged 82.
11	32 years	78	Sternum	In good health.

In this group of eight cases five had survived the primary operation from 6 to 36 years, one had died from lung disease 9 years after operation, one was evidently suffering from bladder disease 12 years after operation, and a third sinking with chest disease 10 years after operation,—all, it may be assumed, of a cancerous nature, the eight cases having respectively survived the first operation 6, 9, 10, 10, 12, 30, 32, and 36 years, and five of these having apparently some years of life before them.

TABLE II, GROUP II.

Including eleven cases of recurrence with second and third operation.

No.	Interval between first and second operation.	Age.	Seat of return.	History.
12	1 year	51	In scar	10 years later well.
17	1 year	42	In scar	4 years later well.
18	1 year	48	In scar	4 years later well.
19	3 years	60	In axilla and scar	6 years later died of lung disease 9 years after first operation.
14	3 years	55	In scar	6 years later well, or 9 years after first operation.
15	4 years	56	In scar	3rd operation 2 years later; 6 years later was well, or 12 years after first operation.
6	5 years	52	In scar	5 years later another recurrence with chest symptoms, 10 years after first operation.
13	5 years	42	In scar	Also 3rd operation, after which was well 5 years later, or 12 years after first operation.
16	7 years	57	In scar	5 years later well.
8	10 years	61	In scar	2 years later well.
7	13 years	43	Also 3rd operation in scar	1 year later well.

In this group of eleven cases—

Two had lived five years after the primary operation, and were in good health four years after a second operation.

One had lived eleven years after the primary operation, and was well ten years after the second.

One had lived nine years after the first operation, and was well six years after the second.

One had lived twelve years after the first operation, and was in good health five years after the second.

A second had survived the first operation twelve years, and was well two years after a second.

A third had survived the first operation twelve years, a second and a third operation, and six years later had no signs of return.

One survived the first operation fourteen years, and was well one year after the third.

One case had survived the first operation seven years, had endured a second three years after the first, and four years later died from chest symptoms.

One case had no signs of recurrence for twelve years, when, at the age of seventy-two, symptoms appeared suggestive of abdominal disease.

One case had lived twelve years after the first operation, and, five years after the second, had some suspicious chest symptoms.

In three of the eleven cases death had taken place or was near at hand seven, twelve, and twelve years respectively after the primary operation.

And in eight others there was every prospect of continuous health, five, five, nine, eleven, twelve, twelve, twelve, and fourteen years respectively after the breast had been originally removed.

Group III.

Including ten cases in which the second breast became involved in the disease.

Case.	Interval between first operation and recurrence.	Operation.	History.
22	2 years	Yes, aged 55	1 year later recurrence in scar of last operation.
24	2 years	Yes, aged 57	5 years after second operation in good health.
26	2 years	Yes, aged 52	5 years after second operation in good health.
29	2 years	No, aged 50	Open cancer of scar of first operation, with disease of second breast.
27	3 years	No, aged 37	Open cancer of scar of first operation, with disease of second breast.
20	4 years	Yes, aged 54	6 years later no signs of recurrence.
28	10 years	Yes, aged 48	Breast and glands removed; 3 years later well.
25	10 years	Yes, aged 50	Did well after operation, but no recent report.
21	24 years	No, aged 66	Open cancer of second breast.
23	23 years	No, aged 80	Open cancer of original scar and second breast.

In five of these cases no second operation was performed, as in all of them an open cancerous ulcer existed, and it was evident that the sands of life's hourglass had nearly run out. In these cases two, two, three, twenty-three, and twenty-four years respectively had passed before a recurrence of the disease had appeared, and the patients were, when seen with the disease of the second breast, thirty-seven, fifty, fifty-five, sixty-six, and eighty years of age.

In the second five cases the second breast was removed.

In two of these the interval between the removal of the first breast and the recurrence of the disease in the second breast was two years, and both patients, who were at the second operation fifty-two and fifty-seven years of age, were well and free from disease five years later.

In Case No. 20, where the interval between the first

operation and the recurrence in the second breast was four years, and the patient was aged fifty-four, there were no signs of recurrence six years later.

In Case 28, where the interval of recurrence was ten years, and the second operation was performed when the patient was forty-eight, the breast and enlarged axillary glands being cleared away, there were no signs of a return of the disease three years later; and

In Case 25, where the same period of ten years had passed before the second breast was removed, when the patient was fifty, a good recovery followed the operation, but there is no later history.

Taking the ten cases, however, as a whole, it appears that in three instances where recurrence took place in the second breast within three years of the operation upon the first, surgical interference could do but little, as also in two other cases where the subjects were sixty-six and eighty years of age.

It must be recorded that in two others where recurrence took place in less than three years, the patients respectively being fifty-two and fifty-seven years of age, there were no indications of recurrence when last seen five years subsequently, or eight years after the primary operation. In Case 20, where the second breast was removed four years after the first, the patient was well six years later, or ten years after the primary operation; and in Case 28, where the interval between the removal of the first breast and the second was ten years, the patient was known to be well three years later, or thirteen years after the first operation.

Conclusions.

If we look at these tables as a whole it will be evident that the interval which may take place between the primary amputation of a breast for cancer and its recurrence in the scar or second breast, when such occurs, is most uncertain. That whilst in half the cases tabulated recurrence took place in five years or less, in the second half the interval

before recurrence appeared varied from six to thirty-two years; and that in at least two thirds of these cases it occurred after ten years; and also that when second or third operations were undertaken the prospects of life were not bad (Table II, Group II and Group III).

With respect to the seat of the recurrence, it seems that such appeared in or about the scar of the original operation in fourteen cases; in the scar and axilla in only one case; in the sternum in three cases; in the second breast in ten cases, and in five of this ten the scar of the first operation was likewise involved.

I would here ask the surgeons who advocate the clearing out of the axilla of all lymphoid tissue as a rule of practice in every case to consider the fact given above; in only one case was the axilla cleared out in an operation for a recurrent affection, and as already described, it is not my custom to clear it out in all; and yet these results do not suggest an inadequate operation.

I bring this paper before the profession with no little pleasure, for it shows that operations for cancer of the breast, *when undertaken at an early period* of the disease, are not so unsatisfactory in their ultimate results as we have been led to believe. To have been able to tabulate, in Group I, seventeen cases of operation without evidence of recurrence in nine cases from five to ten years, and in eight cases from ten to twenty years after the primary operation, and to add that thirteen out of these seventeen cases are now alive and well, with probably some years of enjoyable life before them, is somewhat startling.

Added to these conclusions is the assurance that should recurrence of disease appear after the primary operation, the prospects of prolonged life without second or third operations, as shown in Table I, Group II, are neither unreasonable nor unsatisfactory; for only two of the eight cases so tabulated had survived the first operation less than ten years, and six had survived from ten to thirty-six years, and five of these had apparently some years of life before them.

To show, moreover, as in Table II, Group II, when recurrence of disease takes place and has been treated by second or third operations much benefit may be conferred, is likewise encouraging, for the study of this table which I ask you to make will suggest that second, and even third operations undertaken as soon as recurrences appear are often followed by fairly long periods of enjoyable life, for in five or six of the cases tabulated the patients were well and in good health five or six years after these operations.

Where the second breast has become involved, a like principle of practice is likewise suggested. In four out of the ten cases tabulated no operation was justifiable, but in six cases the second breast was removed, and in four of these cases there were no indications of recurrent disease five years, six years, two years, and three years respectively; so that it may fairly be said that operations on the second breast are not only justifiable, but conducive to prolonged life. For my own part, I am so much more satisfied with the results of my own practice since I have put together the materials embodied in the paper I have just read, and brought out the results of its analysis, that I do not feel disposed to deviate from it in any great degree unless the advocates of what I must describe as an over-zealous practice can prove to me that I am wrong and that they are right by the publication of material facts better than those I have now recorded.

By way of summary I should like to express my conviction that the results of operations for cancer, whether of the breast or elsewhere, would be much better than they now are if they could always be undertaken during the early development of the disease, as illustrated by the majority of the cases in my tables—Groups I and II; that every breast tumour, neither clearly inflammatory nor encapsuled, which seems to involve gland tissue, and may therefore be cancerous, should be at once explored and removed, if found to be cancerous, with the whole gland; and that recurrent growths when localised should be similarly treated.

In advanced and neglected cases, where the lymphatic glands and covering integument are involved, Moore's, Banks's, Halsted's, or Gould's so-called complete operation may be called for, but its results are not by any means so likely to be as favourable as those I have reported. Lastly, in cases of recurrence not favourable for operation, unless the removal of the ovaries can be shown in the future to be successful, the X rays should be employed, for the benefit which has been derived by this treatment when judiciously applied by men of understanding has in my own experience been so successful as to raise hopes which I hardly like fully to express, and at the same time seems to be free from danger or serious consequences when utilised by those who know the dangers of penetrating rays carelessly employed, and the difficulties with which the practice bristles. I must, however, add that the influence of the rays, to make them effective, must be maintained for several months after it has seemed to be beneficial; a three months' course, with about three applications a week, appears to be the shortest from which any permanent good is to be expected, and this treatment is full of hope.

GROUP I.—*Includes Seventeen Cases of Cancer of the Breast which are now alive or have died without Evidence of Recurrent Disease, five years and upwards after Operation.*

Case.	Primary operation.	Remarks.	Duration of life after primary operation.
1	L. E., a widow, aged 62, was operated upon in May, 1897, for typical cancer	In 1902 she was in good health	Or about 5 years after operation.
2	Mrs. N., aged 74, was operated upon in October, 1894	In 1900 she was in good health	Or 6 years after operation.
3	A widow, aged 52, was operated upon in August, 1893	In 1901 she was in good health	Or 8 years after operation.
4	Mrs. P., aged 45, was operated upon in October, 1893	In 1901 she was quite well	Or 8 years after operation.
5	Mrs. B., aged 44, was operated upon in October, 1893	In 1902 she was quite well	Or 8 years after operation.
6	Mrs. T., aged 52, was operated upon in September, 1892	In 1901 she was quite well	Or 9 years after operation.
7	Mrs. B., aged 57, was operated upon in October, 1891	In 1900 she was quite well	Or 9 years after operation.
8	Mrs. S., aged 46, was operated upon in November, 1892	In 1902 she was quite well	Or 9 years after operation.
9	Mrs. P., aged 43, was operated upon in September, 1891	In 1902 she was quite well	Or 10 years after operation.
10	Mrs. J., aged 51, was operated upon in April, 1884	In 1894 she was quite well	Or 10 years after operation.
11	Mrs. B., aged 58, was operated upon in October, 1885	In 1899 she was quite well	Or 14 years after operation.
12	Mrs. F., aged 56, was operated upon in April, 1862	In 1876 she was quite well	Or 14 years after operation.
13	Mrs. C., a widow, aged 66, was operated upon in January, 1883, for typical cancer	13 years later, in January, 1898, she died from acute intestinal obstruction due to gall-stones, one of which she passed. After death no signs of recurrent disease were found	15 years after the primary operation.

14	Mrs. B., aged 48, was operated upon in March, 1869, for cancer	14 years after, in 1884, she died of acute jaundice, but without evidence of recurrent disease, as proved by a necropsy	14 years after the primary operation.
15	Mrs. L., aged 62, was operated upon in September, 1890	In 1895, when in good health, she was burnt to death	5 years after operation.
16	Mary P., aged 60, was operated upon in 1862	In 1882 she died from old age at 80	20 years after primary operation.
17	Mrs. W., aged 50, operated upon March, 1886, for typical cancer	Has been in good health since	16 years after operation.

GROUP II.—*Includes Nineteen Cases of Cancer of the Breast relieved by Operation, and followed by Recurrence in the Seat of the Primary Operation.*

Case.	Primary operation.	Remarks.	Result.
1	Miss R., aged 50, was operated upon May, 1894, for cancer	In 1900, or 6 years after the operation, she was quite well. In 1901 recurrence appeared on sternum	No signs of recurrence for 6 years and more after primary operation, when a sternal growth appeared.
2	Mrs. M., aged 53, was operated upon in May, 1892	She remained well for 7 years and more; in 1900 chest symptoms appeared suggesting internal growth	No signs of recurrence for more than 7 years after primary operation, when chest symptoms appeared.
3	Mrs. H., aged 60, was operated upon in March, 1888	For 12 years she remained quite well, when abdominal disease appeared	No signs of recurrence for 12 years.
4	Mrs. C., aged 52, was operated upon in February, 1880	In 1883, 3 years later, tubercles appeared in the skin of the flaps, which grew slowly for 9 years, and gave little trouble till 1892, when bladder symptoms appeared, suggesting cancer	Local recurrence 3 years after operation, with very slow growth; 12 years after operation bladder disease.

Case.	Primary operation.	Remarks.	Result.
5	Mrs. S., aged 38, had a breast removed in 1848 for cancer	25 years later, or in 1873, she came into my hands with a small tubercle in the scar, followed in the course of 5 years by many more scattered over the sternum and opposite breast. In 1878, or 30 years after the primary operation, she brought her daughter to me, aged 50, with an acute cancer of her breast, for which nothing could be done, and which proved fatal in 10 months	Local recurrence 25 years after primary operation, and very slow growth of disease. Was alive 30 years after operation.
6 (in book, p. 220)	Mrs. K., aged 47, had a breast removed in 1865 for disease of 2 years' growth	Recurrence 5 years later on flap, when a second operation was made. Five years following this a fresh recurrence appeared, which was not touched, and the patient died a few months later	Recurrence 5 years later and second operation; another recurrence after 5 years, which was not touched. Patient lived 10 years after primary operation.
7	Mrs. F., aged 30, had her breast removed by Mr. Birkett in 1858	She remained well for 13 years, when a recurrence took place in the scar, for which a second operation was performed. 2 years later fresh tubercles appeared, which were removed in 1874, and a year later, in 1875, she was well	Recurrence 13 years after primary operation; second and third operation on scar. In 1875, a year after third operation, she was well—that is 16 years after first operation.
8	A. L., aged 50, in 1865 lost her breast for cancerous tumour of 2 years' growth	She remained well for 11 years, when disease returned in the scar, and was in 1876 removed. Two years later she was well	Recurrence in scar of first operation 11 years after second operation, and 2 years later no return.
9	Mrs. A., aged 46, lost her breast in 1859 for cancer	In 1890, or over 30 years after this operation, she came under my care with a growth fixed to the sternum which was diagnosed as cancerous. In 1895, when she was 82 years of age, this had grown, but not to become a source of much trouble. There was no evidence of disease in any other part. No subsequent history	Recurrence over sternum 30 years or more after primary operation.

10	Sarah C., aged 52, lost her breast in 1874 for what I regarded as cancerous disease	In 1884, or 10 years later, there was a recurrence in the scar, which was of such little trouble that she would not have it removed. No recent history	Recurrence in scar 10 years after primary operation.
11	Mrs. W., aged 78, had her breast removed for cancer by Sir W. Ferguson 32 years before I saw her, when she was 46	In 1891 she consulted me with a tumour over and fixed to the sternum, which was clearly cancerous; it was of recent growth	Recurrence of disease in sternum 32 years after primary operation.
12	Mrs. A., aged 40, had breast removed in 1890	In 1891 recurrence appeared in the sternal end of the scar, which was removed. Ten years after second operation she was well	Well 10 years after second operation for recurrence.
13	Mrs. K., aged 37, lost her breast in 1890 for cancer	In 1895, 5 years later, a tubercle appeared in the scar, which was removed; and in 1896 a second tubercle from near the sternal end of the scar. In February, 1901, or 5 years after this third operation, she had a suppurating gall-bladder, which I opened, and removed from it many calculi. No indications of cancer were then to be felt about the gall-bladder or the breast. At the present time she is well and about, without any evidence of recurrent disease	Recurrence in scar 5 years after the first operation, which was removed. A year later a third small operation. Five years later suppuration of gall-bladder, and without evidence of any recurrent disease, that is 12 years after the first operation.
14	Miss B., aged 52, had her breast removed for cancer in 1893	In 1896 a recurrence took place in the scar, which I removed, and in this year some tubercles appeared in the skin, which entirely disappeared within a year. She is now, in 1902, or 9 years after the first operation and 5 years after the second, quite well	Recurrence in scar 3 years after operation. This removed, and no signs of disease in 1902, or 9 years after first operation.
15	Miss M., aged 52, lost her breast in 1889 for cancer	In 1893, or 4 years later, a recurrence followed in the scar, which was removed, and likewise a second recurrence in 1895. In 1901, or 6 years after the last operation, she was quite well	Recurrence in the 4th year after operation and in the 6th year; both treated by removal. No signs of recurrence 6 years later, or 12 years after first operation.
16	Mrs. F., aged 50, was operated upon for cancer in 1889	In 1896, 7 years later, a recurrence appeared in the scar, which I removed. In 1901, or 5 years after the second operation, she was well	Recurrence 7 years after first operation. Second operation and no signs of disease 5 years later, or 12 years after first operation.

Case.	Primary operation.	Remarks.	Result.
17	Mrs. F., aged 41, was operated upon in December, 1886, for cancer of breast	In 1887, a year later, a second operation was performed for a slight recurrence. In May, 1891, or 4 years later, she was well	Recurrence within a year. Second operation and no recurrence 4 years later.
18	Mrs. M., aged 47, lost her breast in 1888	In 1889 recurrence appeared near the scar, which was removed in 1893. 4 years later she was well	Recurrence within a year treated by operation, and no sign of disease 4 years later.
19	S. W., aged 56, was operated upon in November, 1885	In 1888 a recurrence appeared, which I removed from the axilla. In 1893, or 5 years after second operation, she was well, but died of chest disease in 1895	Recurrence 3 years after first operation; treated by operation. When seen 4 years later was in good health but 2 years later died from chest disease.

GROUP III.—*Including Ten Cases of Recurrent Disease after Operation in which the Second Breast was involved, and Four Cases in which the Breast Disease was associated with Cancer of other parts of the Body.*

Case.	Primary operation.	Remarks.	Result.
20	Mrs. C., aged 50, had one breast removed for cancer in 1891; 4 years later the second breast became diseased, and was removed in 1895	In 1901, or 6 years after the second operation, she was quite well	Both breasts removed at an interval of 4 years. No signs of recurrence 6 years later.
21	Mrs. C., aged 43, in 1832 lost her left breast for cancer at Guy's Hospital, under the hands of Mr. Aston Key	In 1856, or 24 years after the primary operation, when the patient was 66 years of age, she came under my care with an open cancer of the opposite breast	Recurrence in second breast 24 years after primary operation.

22	A widow, aged 53, who had lost her right breast in 1865 for cancer, came into my hands in 1868	With an open cancer attacking the scar of the left breast, which had followed the removal of the gland in 1867	Recurrence in left breast 2 years after removal of the right, with recurrence in the scar of the left.
23	Mrs. H., aged 57, had the left breast removed by Mr. Birkett in 1851. She came under my care in 1874	With an open cancer in the scar of the left breast of some few months' growth, and disease of her right breast	Recurrence of disease at age of 80, 23 years after removal of left breast, and disease of the right.
24	E. T., aged 55, lost her right breast in November, 1865	In February, 1867, she lost her left breast; and in 1872, or 5 years later, she was well	Recurrence in opposite breast 2 years after first operation. Was well 5 years after second operation
25	S. C., aged 40, lost one breast in October, 1861	10 years later, or 1871, the other breast was removed	Recurrence in second breast 10 years after the first
26	A widow, aged 50, lost her right breast in 1865	In 1867 the left became diseased and was removed. In 1869 she was quite well	Recurrence 2 years after first operation. Was well 2 years after the second operation.
27	A childless married woman, aged 35, had her right breast removed in 1861	She came under my care in 1865 with an open cancerous ulcer, of 9 months' growth, in the scar of the operation and in her left breast	Recurrence in scar of operation 3 years after, with disease of second breast.
28	T. B., aged 38, lost her left breast in 1866 for cancer, under Mr. Hilton. The axillary glands were not looked for	10 years later, in 1876, she came under my care with cancer of the second breast and enlarged axillary glands of the same side, all of which I removed. In 1879 she was well	Both breasts removed at an interval of 10 years. 3 years later she was well.
29	M., aged 48, in 1862 had her right breast removed for cancer by Mr. Skey	In 1864 she came under my care with an open cancer of the scar, œdema of the arm, and enlarged axillary glands of the same side; also of disease of the left breast, and chest symptoms indicative of internal disease	2 years after first operation disease of second breast and of the scar of the first.

Four Additional Cases of Cancer in Two Positions.

Case.	Primary operation.	Remarks.	Result.
30	A woman, aged 60, who had lost her breast for cancer by Mr. Birkett in 1863, 14 years before, when 46 years of age	Came under my care in 1877 with a cancerous tumour of her hand, which was treated by amputation	Recurrence in hand 14 years after primary operation upon breast.
31 (Case 4 in book, p. 339)	J. B., aged 68, came under my care in 1861 with an epithelioma of her nose, which was removed and had not recurred	5 years later, in 1866, she came to me again with a typical carcinomatous breast of an acute kind, and cancerous tubercles in skin and disease of lymphatic glands, for which nothing could be done	Cancer of the breast coming on 5 years after the removal of an epithelioma of the nose.
32 (Case 5, book)	F. F., aged 72, came under my care in 1865 with atrophic cancer of her right breast of 20 years' standing, which had been ulcerating for 7 years	In 1871, 6 years later, when 78 years old, she reappeared with an epithelial cancer of her nose, the breast disease having increased but little	Chronic atrophic cancer of breast of 26 years' growth, associated with epithelioma of nose.
33	M. W., aged 44, came under my care in 1864 with extensive cancer of the left breast of 4 years' growth	She had likewise a cancerous stricture of her rectum	—

DISCUSSION.

Sir WILLIAM BANKS desired in the first place to thank Mr. Bryant for the courteous and generous way in which he had referred to such work as he had done in the matter of the operative treatment of cancer of the breast. His first paper on that subject was read to the Lancashire and Cheshire Branch of the British Medical Association in 1877; the next to the Worcester meeting of the Association in 1882, when he narrated the results of forty-two cases; and the next to the Harveian Society in 1887, when he presented eighty-two cases. He finally gave the Lettsomian Lectures at the Medical Society of London in 1900 on the subject of "Cancer of the Breast." He had not overhauled his statistics quite recently, but he felt sure that those which he presented that night were very near the mark.

Table of fifty-eight cases in which patients lived for, or were alive at, periods varying from five to twenty-three years.

(1) *Six cases where death resulted from local return.*
4 patients lived from 5 to 10 years.
2 „ „ to 12 and 14 years respectively.

(2) Twelve cases where there was no local return of the disease, but where it recurred in other parts of the body.
9 patients lived from 5 to 10 years.
3 „ „ to 10, 10, and 14 years respectively.

(3) Twelve cases where there was no return of cancer anywhere, and where the patients died from other diseases.
9 patients lived from 5 to 9 years.
3 „ „ to 11, 16, and 18 years respectively.

(4) Twenty-eight cases now living.
14 alive from 5 to 10 years.
12 „ „ 10 „ 20 „
2 „ at 20 and 23 „ respectively.

He thought the result of the operations which had been done by himself and Mr. Bryant were very satisfactory, as showing that they had evidently prolonged life in many cases, and completely extirpated the disease in not a few. But the great thing now was to encourage the medical profession and the public to look with grave suspicion upon the smallest and most innocent-looking breast tumour, and to have early recourse to operation. The operation he had long ago suggested and for many years carried out reached, he believed, the limits of reasonable surgery. It had been stated by certain surgeons

that the entire credit of the introduction of free operations for breast cancer was due to the researches of Stiles and Heidenhain and the operation of Halsted based thereon. He had the most sincere respect for the works of the two first-named gentlemen, but they were investigations in microscopic anatomy, while all that the surgeon needed to know about the lymphatics of the breast had been common property for long enough. As for Halsted's operation, cancer did not recur in the great pectoral muscle except as progressive from the skin and subcutaneous. The removal of that muscle he considered both unnecessary and unscientific, and he entertained the same view of the removal of the cervical glands as a routine part of the operation. If extra access to the top of the axilla were required, that could be obtained by dividing the great pectoral at its upper end and uniting it again by buried sutures. As for the removal of affected cervical glands, he had tried it many times, and he never knew a case which survived. Finally, it had been maintained that if a patient were alive and well three years after being operated upon she might be put down in a table of statistics as "cured." He agreed with Mr. Bryant that this was a dangerous fallacy, and he trusted that it would in future be banished from the category of reliable statistics.

Mr. Bryant, in reply, said that Sir William Banks's results were entirely confirmatory of his own. From his own expeperience the drastic surgery for cancer of the breast was unnecessary, and the larger operation involved an avoidable risk; the chief point was early operation, when a safer minor operation was sufficient.

ABOUT ALKAPTONURIA

BY

ARCHIBALD E. GARROD, M.A., M.D.

Received October 21st—Read November 26th, 1901

In a paper read before this Society in 1899, the present writer gave the results of the examination of the urine in five cases of alkaptonuria not previously recorded, and a summary of the then state of our knowledge of this rare and interesting urinary abnormality.

The object of the present communication is to call attention to certain facts, and to record some observations, which tend to throw fresh light upon its nature and causation.

1. *The Relation of Alkaptonuria to Consanguinity of Parents.*

That alkaptonuria may be met with in several members of a family was first pointed out by Kirk in 1886, and of the cases since recorded a considerable number have served to emphasise this fact. However, although brothers and sisters share this peculiarity, there is, as yet, no known instance of its transmission from one generation to another, nor is anything known as to the urine of children of alkaptonuric individuals.

On the other hand I am able to bring forward evidence which seems to point, in no uncertain manner, to a very special liability of alkaptonuria to occur in the children of first cousins. The information available relates to four families, including no less than eleven alkaptonuric members, or more than a quarter of the recorded examples of the condition.

I have recently learnt that the parents of my own patient, Thomas P—, and of an infant brother, born in the present year, who also is alkaptonuric, are first cousins, their mothers being sisters.

Again, in the notes which were kindly furnished to me by Dr. Pavy of a family of fourteen, referred to in my previous paper, of whom four were alkaptonuric, it is mentioned that in this instance also the parents were first cousins.

I am also greatly indebted to Dr. Robert Kirk for kindly making inquiries from the father of the three children whose cases were so thoroughly investigated by him some years ago, inquiries which brought to light the fact that their parents also were first cousins, the children of sisters. Dr. Kirk adds that the mother is dead, that the father has married again, and that his only child by his second wife, who is not a blood relation, is not alkaptonuric.

Against this may be set the fact that the parents of the patient studied by Dr. Walter Smith in 1882, and of a younger brother whose urine I examined, were not blood relations.

The children of first cousins form so small a section of the community, and the number of alkaptonuric persons is so very small, that the association in no less than three out of four families can hardly be ascribed to chance, and further evidence bearing upon this point would be of great interest.

In a recent paper by Erich Meyer it is mentioned that the parents of his patient were related, but the exact degree of relationship is not stated. Elsewhere the litera-

ture is silent upon this matter, a silence which counts for little, seeing that the information is not usually forthcoming unless asked for, as Dr. Kirk's experience and my own show.

There are some indications that the younger members of a family are more liable than the elder ones. Thus the alkaptonuric members of the family observed by Dr. Pavy, were the ninth, eleventh, thirteenth, and fourteenth. Thomas P— and his alkaptonuric brother are the fourth and fifth children, and in the family observed by Dr. Kirk, the second, third, and fourth children showed the peculiarity.

The facts here brought forward lend support to the view that alkaptonuria is what may be described as a "freak" of metabolism, a chemical abnormality more or less analogous to structural malformations. They can hardly be reconciled with the theory that it results from a special form of infection of the alimentary canal. There is here no question of the intensification of family tendencies by intermarriage, for in no instance were the parents themselves alkaptonuric, and, as has been already mentioned, there is, up to now, no recorded instance of alkaptonuria in two generations of a family.

2. *The Onset of Alkaptonuria in a New-born Infant.*

That alkaptonuria may persist through life without any apparent detriment to health, and may date from earliest infancy, has long been known, but there have hitherto been wanting observations bearing upon the exact period of its onset in congenital cases. This deficiency I am now able to supply to some extent.

The fifth child (a male) of the parents of Thomas P— and the second alkaptonuric member of the family, was born at 6 a.m. on March 1st, 1901. The mother was tended after her confinement by a district nurse, and both she and the nurse were fully alive to the possibility that the child might show the same peculiarity as its elder brother, and were on the look-out for any indication that

this was the case. The information which follows was given to me by the nurse within a few days of the infant's birth.

During the first day of life the child was put to the breast, and was given a teaspoonful of butter and sugar, according to a practice common among the poorer classes. The napkins were first changed at 9 p.m. on March 1st (when the child was fifteen hours old), and it was specially noted that, although urine had been passed freely, there was no indication whatever of the staining which was so familiar in the case of the elder child.

When the napkins were next changed, at 11 a.m. on March 2nd, the nurse noticed a slight staining, and at 10.30 a.m. on March 3rd (fifty-two hours after birth), and on all subsequent occasions, the napkins were deeply stained in the characteristic manner.

The child had been put to the breast during the previous night, and on the morning of March 3rd the nurse found that the mother's breasts contained milk, but were not full. The mother was not conscious of the "draught" until a later hour on March 3rd.

Some urine collected during the eighth to eleventh days of life reduced Fehling's solution, and had all the ordinary properties of alkapton urine.

The above facts, carefully recorded by one who was wholly without bias in favour of any theory of the nature of alkaptonuria, or knowledge of the questions at issue, nevertheless agree completely with what was to be expected on theoretical grounds.

The evidence available points to tyrosin, formed as a product of pancreatic digestion, as the parent substance of the homogentisic acid which imparts to alkapton urine its peculiar properties, and we should anticipate that the peculiarity of metabolism would first manifest itself after the entry of proteid food into the alimentary canal. As, moreover, the human tissues appear to be able to destroy a certain amount of homogentisic acid, this substance would not be excreted until this destructive power was overtaxed.

The observations on the new-born infant appear to be most readily explained on the assumption that the development of alkaptonuria resulted from feeding, but as the child was suckled, the exact time when food began to enter the alimentary canal cannot be fixed with any degree of certainty.

When the elder child was first seen by me the mother stated that in his case her attention had been first called to the staining of the napkins on the day after his birth, thus in both instances the condition may be fairly described as congenital. In this connection a most interesting case recently recorded by Winternitz may be referred to. He had under observation a family of three alkaptonuric children, a boy aged twelve, a girl aged ten, and another girl aged six. The mother, who stated that the urine of the two elder children had stained the napkins from the first days after their birth, added that this had only been the case with the youngest child during the last year. This recalls Maguire's case in which the condition was said to have dated from the age of twenty-seven, the intermittent case recorded by Stange, and the still more puzzling cases of temporary alkaptonuria.

3. *The Relation in Time of the Output of Homogentisic Acid to a Proteid Meal.*

In a quite recent paper, which embodies many other observations of much interest, Mittelbach gives the results of the estimation of the reducing power of the samples of urine passed by his patient at different periods of a twelve-hour day, which show the maximum excretion of homogentisic acid following within the first two or three hours after the chief meal, and not, as is the case with the ordinary products of metabolism, appearing in the urine in the largest quantities from five to seven hours after a meal.

This result was so unexpected, and seemed so difficult to reconcile with the view that tyrosin is the parent substance of homogentisic acid in these cases, that further observations upon the point appeared desirable. I accord-

ingly estimated the reducing power of the several specimens of urine passed by Thomas P— (aged four) during three periods of twenty-four hours each, and the results are embodied in the following tables. The estimations were made by Baumann's silver method, but, owing to the small bulk of many of the specimens, 5 instead of 10 c.c. of urine were used for each testing, and it was not attempted to secure estimations within 0·5 c.c. of $\frac{N}{10}$ silver nitrate solution.

The urine of the child is always rich in homogentisic acid, and the daily output approaches that of some of the adult patients. At the age of three the average daily excretion during seven days was 2·6 grms. of homogentisic acid, and that of Meyer's patient of about the same age was 3·24. The figures for adults vary between 3 and 6 grms. per twenty-four hours.

Day 1.—On this day the patient was taking the ordinary hospital diet for children of his age. The first meal was at 5 a.m : dinner consisting of minced meat and rice pudding at 12 noon; tea including an egg at 3.45; supper consisting of milk and bread and butter at 6 p.m.

Hour of day.	Quantity of urine passed in c.c.	No. of c.c. $\frac{N}{15}$ silver nitrate solution reduced by 5 c.c. urine.	No. of c.c. $\frac{N}{10}$ silver solution reducible by total urine.	Corresponding to a reducing power per hour of—
A.M. 9.30	60	10	120	—
P.M. 12.30	53	10·5	111·3	37 c.c.
4	46	13	119·6	34·2
5.55	27	16	86·4	45
9.30	55	11	121	33·7
A.M. 12.45	35	9	63	19·3
3.45	28	5·5	30·8	10·2
6	25	5·5	27·5	12·2
Totals	329 c.c.		679·6 c.c. (corresponding to 2·79 grammes of homogentisic acid)	

Here the maximum excretion per hour was between 4 and 5.55 p.m., *i. e.* four to six hours after the chief meal,

but the results are somewhat obscured by the overlapping of the effects of several meals rich in proteid.

Day 2.—On this day the diet was so arranged that the articles richest in proteids were given at the chief meal, which, as before, was at 12 noon, and hourly specimens of urine were fortunately obtained from 4 to 9 p.m. inclusive. It is clearly seen that although there is a conspicuous rise in the specimen passed at 1·30 p.m., the maximum excretion was between 3 and 7 p.m.

Hour of day.	Quantity of urine passed in c.c.	No. of c.c. $\frac{N}{10}$ silver nitrate solution reduced by 5 c.c. urine.	No. of c.c. $\frac{N}{10}$ silver solution reducible by total urine.	Corresponding to a reducing power per hour of—
A.M. 9.55	26	6	31·2	—
11.40	43	5·5	47·3	27 c.c.
P.M. 1.30	25	16	80	43·6
2.50	30	10	60	45
4	30	11·5	87	84·5
5	32	15	96	96
6	20	15	60	60
7	31	14	86·8	86·8
8	25	10	50	50
9	24	8·5	40·8	40·8
10.55	65	3	39	20·3
A.M. 12.55	27	6	32·4	16·2
2	5	6 or 7	8·4?	7·7
4.40	16	7	22·4	8·4
8	41	8	65·6	19·6
Totals	440 c.c.		805·9 c.c. (corresponding to 3·327 grammes of homogentisic acid)	

The total excretion of homogentisic acid was increased, owing to some increase of the proteid food, partly in the form of Plasmon. The effect of the early breakfast at 5 a.m. is still clearly marked.

Day 3.—On this day the meal richest in proteid was given at 9 a.m. instead of at noon, and the maximum output of reducing substance per hour was also three hours earlier, viz. between 12.15 and 4.25 p.m. The rise during the hours immediately following the meal is again very

noticeable. The total reducing power of the twenty-four hours' urine was on this day somewhat larger still.

Hour of day.	Quantity of urine passed in c.c.	No. of c.c. $\frac{N}{10}$ silver nitrate solution reduced by 5 c.c. urine.	No. of c.c. $\frac{N}{10}$ silver solution reducible by total urine.	Corresponding to a reducing power per hour of—
A.M. 6	32	6	38·4	—
8	30	?	?	—
9.25	26	5	26	18·3 c.c.
11.15	46	8	73·6	40·1
P.M. 12.15	29	9	52·2	52·2
4.25	99	14	277·2	66·5
6	46	8·5	78·2	49·3
9.30	95	6·5	123·5	35·3
11.45	31	7·5	46·5	20·6
A.M. 2.50	35	6	42	13·6
4.45	41	4·5	36·9	19·2
Totals	510 c.c.			

It will be at once apparent that these results do not bear out Dr. Mittelbach's observation that the reducing power of the urine reaches its maximum within two or three hours of a proteid meal, but show, on the other hand, that in the case of my patient, although such a meal is quickly followed by a much increased excretion of homogentisic acid, a still larger amount is excreted during the second period of four hours than during the four hours immediately following the meal. In a word, they tend to support the view that the change from tyrosin to homogentisic acid takes place in the tissues after the absorption of the former, rather than the alternative view that the change in question is brought about in the alimentary canal.

Since the publication of the previous paper in 1899, cases of alkaptonuria have been recorded by Winternitz (three children in one family), E. Meyer (one child), and Mittelbach (an adult male); and these with the infant above described raise the total of recorded examples to thirty-seven.

The following additions may also be made to the bibliography there given:

HUPPERT, H.—Ueber die Homogentisinsäure. Deutsches Archiv f. klin. Medicin, 1899, lxiv (Festschrift), p. 129.

WINTERNITZ.—Münchener med. Wochenschr., 1899, xlvi, p. 749.

ORTON, K. J. P., and GARROD, A. E.—The Benzoylation of Alkapton Urine. Journal of Physiology, 1901, xxvii, p. 89.

MEYER, ERICH.—Ueber Alkaptonurie. Deutsches Archiv f. klin. Med., 1901, lxx, p. 443.

MITTELBACH, F.—Ein Beitrag zur Kenntniss der Alkaptonurie. Deutsches Archiv f. klin. Med., 1901, lxxi, p. 50.

DISCUSSION.

The CHAIRMAN (Dr. C. THEODORE WILLIAMS) expressed regret that more papers on chemical pathology were not communicated to the Society. It was along these lines that the greatest advance in medicine had been made. After alluding to the importance of being able to recognise the presence of alkapton in the urine in examination for life insurance, he asked by what test it could be distinguished from sugar in the urine.

Dr. W. A. OSBORNE mentioned the case of a man who was rejected for life assurance because his urine reduced Fehling's solution, which he had found to be due to alkapton. A second and a third brother were similarly affected, and their parents were first cousins. These were the three cases that had been described by Dr. Pavy. Homogentisic acid was present in the urine as a salt. If homogentisic acid was derived, as was suggested, from tyrosin, then a person the subject of alkaptonuria if fed on a tyrosin-free diet should cease to pass alkapton in the urine. Such a diet might consist of sugar, fat, and gelatine. It was very difficult to understand on chemical grounds how tyrosin could become changed into homogentisic acid. He suggested that it might be a good plan to give an alkaptonuric patient some of the intermediate substances between tyrosin and homogentisic acid, and observe the effect on the excretion of alkapton in the urine.

Dr. GARROD, in reply, said that it would be difficult to give a tyrosin-free diet in his case, as the patient was a child of four years. The experiment had been tried abroad by Mittelbach, whose adult patient had consented to take only tea and brandy for three days. Mittelbach found that after such fasting the homogentisic acid excretion fell to about one third of the usual amount, but that the acid did not completely disappear from the urine.

TWO CASES

OF

LIGATURE OF THE LEFT CAROTID

FOR

ANEURYSM OF THE ARCH OF THE AORTA

WITH THE POST-MORTEM SPECIMENS OF FOUR CASES

BY

CHRISTOPHER HEATH, F.R.C.S.

CONSULTING SURGEON TO UNIVERSITY COLLEGE HOSPITAL

Received October 29th, 1901—Read February 11th, 1902

THE following are the notes of the sixth and seventh cases in which I have tied the left carotid for aneurysm of the arch of the aorta. I briefly referred to the sixth case in some "Remarks on the Distal Ligature in the Treatment of Aneurism," published in the 'British Medical Journal' of February 19th, 1898, but the seventh case occurred after that date.

CASE 6.—Martha Fogarty, aged 61, following the occupation of a monthly nurse, came under the observation of Dr. Robinson at the Mile End Infirmary in July, 1890.

Since her husband's death she had supported herself by monthly nursing and the letting of lodgings, and never undertook anything like hard work. Three years before she experienced pain in her right shoulder, which was shortly afterwards followed by the discovery of a pulsating swelling above the right clavicle. She thereupon went to the London Hospital and remained there three weeks. It was then proposed to perform some operation for her relief, but this she declined, and took her discharge.

In December, 1889, when nursing a lying-in case, she noticed that the act of coughing caused her great pain in the supra-clavicular region, and about the same period her voice became cracked. Soon after Christmas of 1889 she could not lie comfortably on her back, and when she did so experienced a feeling of impending suffocation.

In July, 1890, she was admitted to the Mile End Infirmary, when Dr. Robinson noted a marked pulsation in the supra-sternal notch. She was kept closely in bed, and iodide of potassium was administered in full doses for many weeks, but no alteration in the pulsation resulted. She suffered a good deal from cough, and complained of constant pain in the neighbourhood of the pulsation, and this was much intensified during the act of coughing. Over the pulsation a marked bruit was audible, a similarly well-marked systolic bruit being heard at the apex-beat. The pulses in the wrist were equal in volume and regular, 92. There were no signs of arterial degeneration in the superficial vessels anywhere. Her invariable position in bed was a sitting one, with the knees drawn up and her head resting upon them. On the slightest inclination backwards there was an increase of the dyspnœa, and inspiration was accompanied by stridor.

The patient was small and of spare build. Her hair was turning grey, and the arcus senilis was well marked. Her complexion was sallow. There was no local œdema. After the treatment by rest and the iodide had been pursued for some weeks without any improvement in the patient's condition, the advisability of submitting

to an operation was placed before her by Dr. Robinson, and she consented to it. Accordingly, Mr. Heath applied a carbolised silk ligature to the left carotid, above the omo-hyoid, on November 16th, 1890, no anæsthetic being employed. On the evening of the operation the temperature of the left side of the face was 82°, that of the right side being 94°. The pulse in the left radial was noticed to have diminished in volume considerably. The patient at this time complained of a throbbing pain in the neighbourhood of the incision, and also of dysphagia. The pain in her right shoulder she declared to be gone.

On November 19th (third day) it was noted that respiration, which had been distinctly noisy, particularly inspiration, was now unaccompanied by the least noise. The patient was much better, able to recline against her pillows and indulge in sleep, and declared herself quite comfortable. The pupils were noted to be equal and active.

On November 22nd she was able to sleep for seven hours, a thing she had long been a stranger to. The wound healed by first intention, and her progress was uneventful until January 3rd, 1891, when she complained of some return of the pain in the right shoulder and in the interscapular region. She had occasional attacks of epistaxis about this time without obvious cause, and some cough of a laryngeal character persisted. In February, 1891, the pulsating tumour above the sternum, though still visible, was thought to have contracted, and the patient was able to lie and sleep in any position without discomfort. On April 3rd (five months after the operation) she complained of some return of dysphagia. In June, as she complained of some recurrence of pain in the right shoulder, and the pulse was full and hard, Dr. Robinson ordered her tablets of nitro-glycerine, under which the pain subsided. She continued to improve, and was discharged from the infirmary at her own request on August 8th, 1891.

This patient was admitted to the London Hospital

under Dr. Gilbart-Smith on September 3rd, 1891, when a pulsating tumour existed at the inner end of the right clavicle, and could be just felt about it. About the middle of November she began to complain of great pain shooting through the sternum and between the shoulders, and died suddenly on November 29th, 1891, more than a year after the operation.

For the following abstract of a case of aneurysm under the care of Dr. Roberts, I am indebted to Mr. Bucknall, late Surgical Registrar of University College Hospital.

Case 7.—James Smith, aged 36, a labourer, was admitted November 4th, 1898, complaining of "pain in the chest." From boyhood till the age of twenty-six he served as a hand on a fishing smack. Since then he has worked as a rough labourer, doing heavy lifting. For seventeen years he has served his time in the Militia Artillery, "lifting guns." Was in bed with rheumatism for seventeen weeks at the age of twenty-two. Had some swellings in the groins once, but *no syphilis*. Often drunk, and smoked half an ounce of shag daily.

Family history.—Father died of consumption aged twenty-eight.

Present illness.—Began in April, 1898, with pain behind the sternum, which came on when he ceased working, and lasted till he settled to work again, and "warmed to his work."

In September the pain became worse, and spread over the right upper chest to the scapula, and ran down the right arm as far as the internal condyle. He had to give up work and go to bed for four days.

The pain continued to get worse, and was least felt whilst doing manual labour.

During October, 1898, he had a cough.

State on admission (November 5th).—Patient pre-

sented all the signs of an aneurysm projecting forwards in the first and second right intercostal spaces. The first and second right spaces were bulged, and dull on percussion for a distance of one inch from the sternal margin, and pulsation of an expansile character could be seen and felt here, and in the episternal notch and right supra-clavicular fossa.

The inner ends of both clavicles were projected forwards by the swelling, especially the right, and each beat of the pulse threw them further forward, and caused a heaving of the upper part of the chest.

Some dilated veins lay over the front of the chest, and the jugulars were also distended.

Patient had a frequent brassy cough, and the voice was harsh; but the laryngoscope showed that both cords moved equally. There was marked "*tracheal tugging*," the right pupil was larger than the left (slightly), and the right radial pulse might have been a shade earlier than the left; it was certainly much larger in volume. The pulse was regular, 68 to the minute, high tension, large, collapsing rapidly during diastole in a manner typical of aortic regurgitation. Heart apex-beat heaving in fifth space, in the nipple line.

On auscultation a blowing systolic murmur could be heard over the aneurysm; the second sound could be clearly heard in the second right interspace, and along the left border of the sternum a murmur could be heard following the second sound, and running through the whole period of diastole. A blowing systolic murmur could be heard at the apex. The lungs were examined and found healthy.

Notes before operation.—During November and December, 1898, and the first half of January, 1899, patient had severe attacks of pain in the shoulders, back, and side of the neck and face. The aneurysm at first became smaller, but during January it increased in size, and definite swelling and pulsation appeared beneath the pectoral just below the right clavicle. During this

period patient's temperature remained normal. On January 18th patient was transferred for operation.

Operation (January 18th, 1899, by Mr. Heath).—The left common carotid was ligatured with carbolised silk opposite the cricoid, eucaine β being used as a local anæsthetic. There were no succeeding nervous symptoms.

On January 19th and 20th the patient slept badly, owing to pain in the region of the aneurysm. On the 20th the pulsation in the aneurysm was distinctly less marked, and daily improvement was noted until February 1st, when he returned to the Medical ward with the operation wound healed. The pulsation was now much less distinct and forcible, and patient was free from pain and had slept well since January 20th. His cough was less frequent, and less brassy in character.

On February 16th patient complained of pain in the chest and cough, and, on listening to the chest, râles and rhonchi could be heard scattered over both lungs.

On February 17th his temperature shot up to 103°, and from this date till the day of his death (March 21st) he had constant remittent fever varying between 100° and 104°, usually about 102°, with daily remissions of two to three degrees. The lungs showed all the signs of rapid and wide-spread tubercular infiltration and consolidation, and later cavity formation at the apices was evident.

The patient grew thinner and weaker daily, and expectorated copious purulent sputa containing tubercle bacilli. He sank and died on March 21st, having been ill a little over a month. The aneurysm gave rise to no symptoms during this time, and was daily less evident.

The post-mortem specimens from patients on whom I have tied the left carotid for aortic aneurysm are four in number.

1. The patient was a labourer who had had a pulsating swelling in the neck for nearly a year, and was under the

late Dr. Cockle when I tied his left carotid with catgut in February, 1872. "The symptoms due to the pressure of the aneurysm at once abated." When seen in March, 1873, he was in a very satisfactory condition, but in June, 1875, after resuming his laborious occupation of hedging and ditching, a pulsating tumour much larger than before the operation projected above the sternum. The aneurysm burst externally in September, 1876. (See 'Clin. Soc. Trans.,' vol. v, p. 183, and vol. x, p. 96.)

"The arch of the aorta is generally dilated; upon the anterior surface of its ascending portion is an oval opening, about an inch and a half in diameter, which communicates with a large sacculated aneurysm. The aneurysm projects forwards, and ascends in the neck beneath the sterno-hyoid and thyroid muscles as high as the cricoid cartilage, where there is a large opening, at which it had burst through the skin. The transverse portion of the arch is compressed by the sac, and the left brachio-cephalic vein is obliterated. The posterior surface of the sternum is eroded and forms part of the wall of the aneurysm, which had also compressed the left lung. The left carotid artery is obliterated and contracted at a point half an inch below the cricoid cartilage, where a ligature has been applied; it contains a fibrinous coagulum only adherent at the seat of ligature. There is no evidence that the internal coats of the artery were divided by the ligature" (College of Surgeons Museum, 3167).

With regard to this last statement, I may mention that the catgut broke in tying, and that I then doubled it and tied the artery as firmly as I dared. The drawing given by Messrs. Ballance and Edmunds ('Ligation in Continuity,' p. 193) of this preparation is in my opinion incorrect. It will be noticed that the sac contains no clot, for the reason that the examination took place three days after death in very hot weather, and in the country, and the decomposed condition of the clot necessitated

the washing of it away. This was most unfortunate, as it has led to the idea that no coagulum had formed as a result of the operation, whereas a large amount of clot had formed, and had led to the apparent cure of the aneurysm until the patient resumed his labour, when it again grew and burst externally, four and a half years after the ligature was applied.

Specimen 2, from a man aged 38, whose case is reported in the Clinical Society's 'Transactions' for 1891, by Dr. H. E. Harris, under whose care the patient was in the St. George's-in-the-East Infirmary. I tied the left carotid on March 8th, 1890. For a fortnight the aneurysm appeared to decrease in size, but the patient was more distressed with dyspnœa and cough. After that date it again increased in every direction and became more prominent, and the patient died suddenly on May 12th, two months after the operation.

The aneurysm springs from the upper and anterior part of the transverse portion of the arch, with which the sac communicated by a rounded opening of $1\frac{1}{8}$ inches diameter. The opening is entirely to the proximal side of the great vessels, and the sac projects upwards and to the left, its summit being $1\frac{3}{4}$ inches above the sternal notch.

The sac is entirely filled with clot, of which the outer layer, from $\frac{3}{4}$ to 1 inch in thickness, is composed of decolourised fibrin, while the central portion is made up of ordinary red coagulum.

From the orifice of the aneurysm ante-mortem clots extend in a radiate fashion into the aorta, and into the innominate and left subclavian arteries, in which they tail off to threads. This clot, after being subjected to the action of weak spirit, was smooth, well defined, of considerable consistence, and separated like a membrane from the body of the clot. A section of the main clot showed it to be fleshy, and slight pressure caused it to split up into laminæ. Just above the aortic opening

the clot was distinctly adherent over a surface nearly an inch in length to the concavity of the arch, which was extensively calcareous.

A ligature had been applied to the left carotid five eighths of an inch below the bifurcation of the artery, at which point the vessel is interrupted for about half an inch by a mass of fibrous tissue. Above, the artery is completely filled by an organised but still coloured clot. Below, a completely decolourised clot extends along and is firmly adherent to the posterior wall of the vessel; this clot ceases one and a quarter inches above the commencement of the artery, with the exception of an exceedingly fine filament, which is continuous with the clot in the aneurysm. The remains of a ligature may be observed embedded in the fibrous tissue, which has also entangled the pneumogastric nerve (College of Surgeons Museum, 3167A) (Plate I).

Specimen 3 was from Dr. Robinson's patient (Case 6). The first part of the arch of the aorta is uniformly dilated. From the right superior aspect of the transverse arch, in front of the innominate artery, which is dilated and involved, springs an aneurysmal sac of the size of a small orange, with an opening into the aorta of the size of half a crown. The sac was adherent to the trachea, and is almost completely filled with laminated clot. The left carotid is filled with firm adherent clot, and higher up is obliterated by a ligature, which has disappeared. The clot in the left carotid does not extend into the aorta. The aorta was extensively diseased (University College Museum, 1233) (Plate II).

Specimen 4 was from Dr. Roberts's patient (Case 7). There are two aneurysmal sacs, a large one springing from the ascending aorta, and a smaller one arising from the back of the innominate artery. Both contained laminated clot.

The aortic aneurysm forms a tumour as large as a

clenched male fist, lying to the right of the extra-pericardial ascending aorta, and communicating with its lumen by an orifice the size of a florin. This pierced the antero-external wall of the vessel about midway between the pericardium and the origin of the innominate. The fibrous tissue forming the wall of the aneurysm extended around the vena cava and the origin of the innominate, and to the jugular vein. The manubrium sterni and ribs are adherent to the sac.

On opening the aneurysmal sac it was found to be filled with clot, the central part soft and rather fluid, the main mass distinctly laminated. The most peripheral portion was decolourised.

The innominate aneurysm forms a tumour as large as a hen's egg arising from the artery a quarter of an inch from its bifurcation. It lay behind and to the left of the larger aneurysm, to which it adhered, being in close contact with, and adherent to, the trachea on the inner side. It contained laminated clot, and communicated with the larger sac by its lower end.

The left carotid is obliterated an inch below the bifurcation and converted into a fibrous cord half an inch long. Below that there is solid clot filling the vessel to within half an inch of the aorta close to the larger sac. The aorta is extensively diseased. The lungs were universally adherent and solid with tubercles, which had broken down beneath the apex of both upper lobes, leaving a ragged cavity the size of a small hen's egg in each (University College Museum, 1234) (Plate III).

That the application of a ligature to the left carotid has an effect upon an aneurysm of the transverse portion of the arch of the aorta is, I think, sufficiently shown by the cases just read. In the woman it is noted that on the third day after the operation the respiration, which had been distinctly noisy, had become quiet, and the patient was able to recline against her pillows. On the sixth day she was able to sleep for seven hours consecutively.

In the man, on the third day the pulsation of the aneurysm was distinctly less marked. But the relief in my first case (Dr. Cockle's patient) was even more marked, for I brought him before the Clinical Society more than a year after the operation, when it was recorded that "the patient is in perfect health, and feels no inconvenience from his chest. He sleeps well and can lie on either side equally well. The right chest wall in front is quite restored to its natural shape, or if anything is a little flatter now than its fellow. On palpation, the heaving impulse formerly existing over the right anterior chest wall is almost entirely gone. On percussion, the right anterior chest wall, formerly so dull, has, to a considerable extent, recovered its normal condition" ('Clin. Soc. Trans.,' vol. vi, 1873).

The preparation from this patient shows no clot, for the reason I have already given, but the other three preparations show thick laminated clot in each sac, and in the last case (Dr. Roberts') the second or innominate aneurysm was also full of clot.

Various theories have been advanced to account for the formation of laminated clot in these cases. The simplest was that it depended upon the enforced rest in bed following the operation; but the fact is that in every case the effect of prolonged rest in bed had been tried for many weeks without the slightest benefit. Next it was suggested that the clot, beginning at the point of ligature, spread down into the aneurysm, and thus led to the formation of a coagulum in the sac. This is contrary to fact, as shown in the preparations before you, for in no single case was the left carotid involved in the sac, and it is noted that the small thread-like clot, which, in some instances, spread down the carotid, in one case only extended into the aorta, and joined that in the aneurysm. I maintain the view which I have always held about these cases, viz. that the distal ligature affects the current of blood in the aneurysm, probably by

retarding it, and thus causes it to flow around the sac instead of directly through or past it, and in this way leads to the deposit of laminated fibrin on the probably roughened wall of the sac.

When this normal cure of the aneurysm has gone on for some time, there is no doubt a tendency for the small remaining cavity to become blocked with soft coagulum, and this is probably a critical moment for the patient, and may account for the sudden deaths which have occurred at considerable periods after the operation. An aneurysm which is semi- or completely solid must necessarily exercise much greater pressure on its surroundings than one which only contains fluid blood, whilst the sudden arrest of a stream of blood through a sac so near the heart would be likely to interfere with its action and lead to syncope.

For the drawings of three of the preparations illustrating this paper I have to thank Mr. T. W. P. Lawrence, F.R.C.S., Curator of the University College Museum.

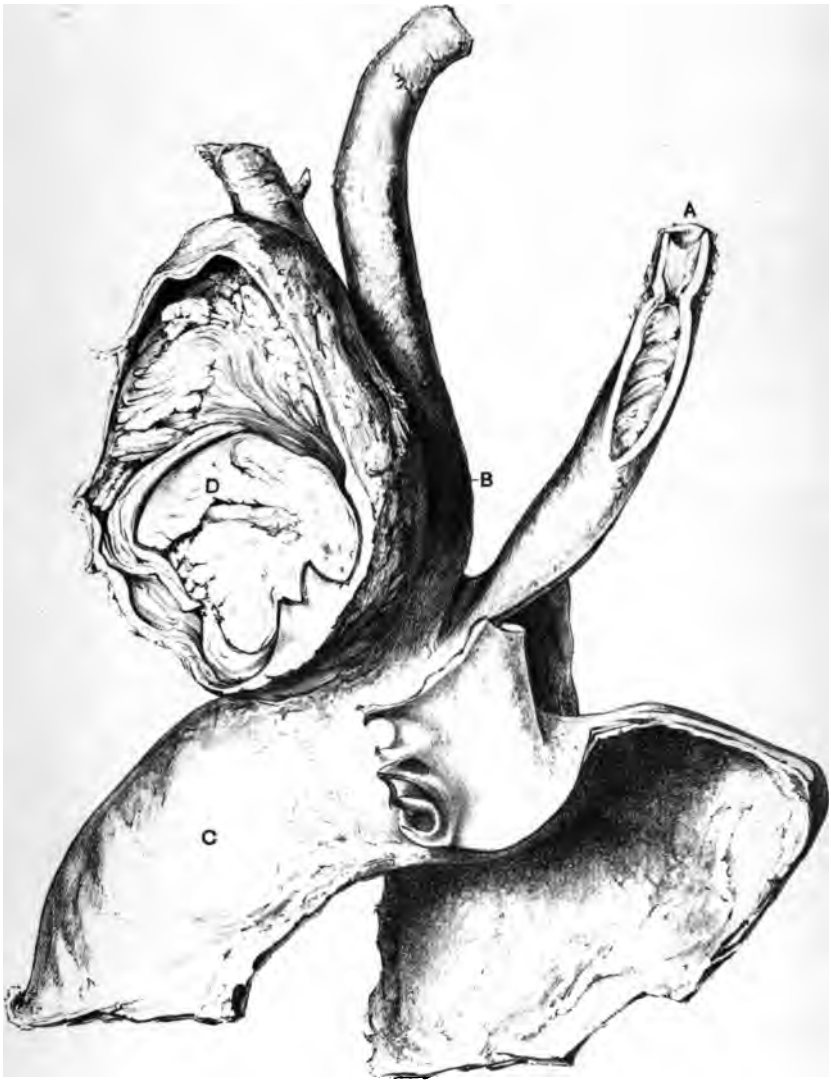
Heath : Ligature of Left Carotid. Plate I.

CASE 5.

A Left carotid at ligature. **C** Loose clot in Aorta.
B Right carotid. **DD** Aortic Aneurysm.

Bale & Danielsson, Ltd., Lith

Heath: Ligature of Left Carotid. Plate II.



CASE 6.

A Left carotid at ligature. **C** Arch of Aorta.
B Innominate artery. **D** Clot filling Aneurysm.

Bale & Danielsson, Ltd., Lith

CASE 7.

A Left carotid at ligature. **C** Arch of Aorta.
B Right carotid. **D** Clot filling Aneurysm.

Bale & Danielsson, Ltd., Lith.

DISCUSSION

Mr. A. Carless referred to the case of a woman aged 40, with aneurysm of the aorta, the specimen from which was shown, which was published in the 'British Medical Journal' for December 3rd, 1898, p. 1685, in whom the left carotid had been ligatured by Mr. Carless, and the left subclavian by Mr. Rose, with improvement in the signs and symptoms in the case. During the five months before coming under observation she had suffered from pain in the right arm, shoulder, and neck, with dysphonia and dysphagia. She had a dry ringing cough. There was no specific history, and she had had two healthy children. On admission to hospital there was bulging of the chest wall at the level of the first and second ribs on each side, with marked pulsation in the intercostal spaces. In addition there was a pulsating swelling below the insertion of the left sterno-mastoid muscle. The left temporal and radial pulses were less than the right. While in hospital several attacks of syncope occurred, and as the tension of the projecting saccule beneath the sterno-mastoid appeared to be increasing, the left carotid was tied. For a few days afterwards there was a slight increase of tension in the sac, with some paresis of the left side of the face and tongue; but these symptoms soon disappeared, the dyspnœa became less, and the pulsation diminished. Twenty-four days after the ligature of the carotid the subclavian was tied. Six months later there was very little pulsation above the clavicles, and she was able to do her work for three years, with some short intervals of rest, when she suddenly died from rupture into the left pleural cavity. The aneurysm was found at the necropsy to be non-sacculated. The left subclavian and carotid were contracted, and the innominate had been absorbed into the general aneurysmal mass.

Dr. Frederick T. Roberts remarked that the chief point for discussion was under what conditions distal ligature of arteries should be performed for thoracic aneurysm. In his case upon which Mr. Heath operated the two main causes of the aneurysm were heavy work and alcoholism; he thought that excessive smoking might also have had some influence. There was no history of syphilis. The indication for the operation was the extreme suffering which the patient endured, and it was performed in spite of the fact that there was free aortic regurgitation and marked arterial degeneration. Much benefit was derived from the operation, both as regards the physical conditions of the aneurysm and the sensations of the patient. Dr. Roberts alluded to another case of aortic aneurysm recently

under his care, in which first the left carotid and afterwards the subclavian artery were ligatured by Mr. Bucknall, with decided benefit.

Mr. R. Barwell said, in reference to the suitability of cases for operation, that clot was not likely to form in symmetrical dilatations of the aorta, even if the vessels were tied; but if the aneurysm were sacculated clot was much more likely to form after distal ligature. He agreed with Mr. Heath that the clotting probably originated in the sac itself, close to the exit of the ligatured vessel from the sac, and not at the seat of ligature. In his opinion there were cases in which it was preferable to tie the carotid and subclavian on the right side rather than the left, the indications as to which side should be tied being derived from the state of the vocal cords and pupils. Among such cases he would have been inclined to have placed those described in the paper.

Mr. T. R. H. Bucknall referred to the case of a man aged 47, who had had syphilis, in whom a swelling below the left sterno-mastoid was present. There were indications of pressure on the trachea and left bronchus, but none of pressure on the recurrent laryngeal nerve. The left carotid and the third part of the subclavian were tied, with the result that the patient was free from symptoms for two months, the pulsation becoming less. Mr. Bucknall then discussed the question of danger from ligature of the carotid, particularly the cerebral symptoms which formerly preceded death not infrequently.

Mr. Heath, in reply, said that his rule had been to tie the artery which came off next beyond the aneurysm, and he quite agreed that in some cases it might be better to tie the right than the left carotid.

THE SURGICAL TREATMENT

OF

OBSTRUCTION IN THE COMMON BILE-DUCT BY CONCRETIONS

WITH ESPECIAL REFERENCE TO THE OPERATION OF CHOLEDOCHOTOMY AS MODIFIED BY THE AUTHOR, ILLUSTRATED BY SIXTY CASES

BY

A. W. MAYO ROBSON, F.R.C.S.,

SENIOR SURGEON TO THE GENERAL INFIRMARY AT LEEDS; EMERITUS PROFESSOR OF SURGERY IN THE YORKSHIRE COLLEGE OF THE VICTORIA UNIVERSITY

Received December 16th, 1901—Read March 25th, 1902.

When once gall-stones have reached the common duct, their attempted dislodgment by purely medical means is with few exceptions disappointing in the extreme, and the unfortunate patients are condemned to a lingering and painful illness usually ending in death, unless the obstruction can be removed by surgical intervention.

Seeing that it is only twelve years since Courvoisier first removed a gall-stone from the common duct by direct incision, the progress in this branch of surgery must be very pronounced when we can safely affirm that there is no portion of the gall-bladder, cystic, common, or primary

division of the hepatic ducts which cannot under ordinary circumstances be reached for the removal of concretions, and that with great probability of success.

No surgeon should attempt the removal of gall-stones unless he is prepared for any of the various operations on the biliary passages, as it is almost impossible to say beforehand what may be required until the ducts have been explored by the fingers and the condition of the parts ascertained, and no operation should as a rule be concluded until it is clearly made out that the ducts, including the hepatic and common, are quite free from concretions, otherwise disappointment and dissatisfaction are certain to follow.

Arguing from some hundreds of cases of cholelithiasis on which I have operated, I find that the common bile-duct has to be attacked in one out of every five or six cases.

In a few cases, concretions may be manipulated backwards into the gall-bladder, and thence extracted by scoop or forceps, but this can only be done when the cystic duct is dilated. I have been able to clear the ducts in this way on ten occasions. Occasionally a small stone may be pressed into the duodenum, but this is exceptional and inadvisable, as it may be pushed into the diverticulum of Vater and so be missed, and the whole operation rendered futile. In patients too old or too ill to bear choledochotomy, a rapid cholecystotomy may be performed, so as to relieve the jaundice and allow solvent injections to be employed; but my experience of this treatment has not been so favourable as to make me very hopeful of accomplishing the solution or the diminution to the passing point of the concretion deliberately left behind, and a subsequent operation is usually necessary.

Crushing concretions by means of pressure by the finger and thumb through the duct walls is a method I formerly employed in over thirty cases with considerable success and without fatality, but it is only available for soft concretions, and fragments are apt to be left and then

to produce further trouble. I have not once adopted this method during the past two years unless I could at the same time remove the fragments.

Cholecystenterostomy or short-circuiting the obstruction should never be performed for gall-stones, as it leaves the cause untouched, and the small opening is apt to contract and lead to speedy recurrence of the symptoms. This has actually occurred in my own practice, and in that of other surgeons. Out of nearly thirty cholecystenterostomies that I have performed, I have only done it on ten occasions for gall-stones, and not once during the past two years.

If the patient be too ill for choledochotomy, the gall-bladder can be very rapidly united to the colon with very little disturbance of adhesions, and this as a means of giving relief answers quite as well as the more difficult operation of uniting the gall-bladder and duodenum: this operation is, however, only applicable when the gall-bladder is dilated, which is unusual in cholelithiasis.

The operation of uniting a dilated duct to the intestine or draining a dilated duct on to the surface may be occasionally called for, and I have twice done the former and once the latter operation, in all the cases followed by recovery.

Reaching the common duct through the opened duodenum, a modification of choledochotomy seemed to me, when it was first suggested by Dr. McBurney, an easy and ideal operation, and at that time it was easier than ordinary choledochotomy; but I feel sure that there is a greater danger of sepsis by this method owing to the necessary enterotomy, and since I have adopted my modification of choledochotomy I have not repeated the operation through the duodenum, since I am quite clear that it is not only more difficult and more dangerous, but that it does not afford so great a facility in clearing the whole of the ducts of concretions. I have performed it eleven times with three fatalities, which compares unfavourably with the ordinary operation of choledochotomy.

Lastly, and most important, we come to the ideal operation for the removal of stones from the common duct, choledochotomy, which, after experience of all other methods in vogue for the removal of gall-stones from the common duct, I have come to the conclusion is the only one to be relied on, and as an operation is therefore worthy of special study.

Moreover, as the result of my experience in sixty cases, I have been able to modify the operation in such a way, that what was formerly a most difficult procedure, involving prolonged manipulation, special appliances, and at least two assistants, and only to be undertaken after all other means had failed, is now a comparatively simple operation in the greater number of cases, only requiring the help of one assistant and not requiring the use of any special apparatus.

By this method the time involved in the operation is reduced considerably, and where adhesions do not give unusual trouble it is easy to complete the work in from thirty to forty minutes, which not only means a saving of time and fatigue to the operator, but a considerable saving of shock to the patient.

I always employ a firm sand-bag under the back opposite the liver, which not only pushes the spine and with it the common duct forward, so that it is several inches nearer the surface, but acts like the Trendelenberg position in pelvic surgery by letting the viscera fall away from the field of operation. I then make a vertical incision over the middle of the right rectus, the fibres of which are separated by the finger, which I find to be the most expeditious and the most effective method of exposing the gall-bladder and bile-ducts; but when it is necessary to open either the common duct or the deeper part of the cystic duct, instead of prolonging the incision downwards as was formerly done, I now carry it upwards in the interval between the ensiform cartilage and the right costal margin as high as possible, thus exposing the upper surface of the liver very freely. It

will now be found that by lifting the lower border of the liver in bulk (if needful, first drawing the organ downwards from under cover of the ribs), the whole of the gall-bladder and the cystic and common ducts are brought quite close to the surface, and as the gall-bladder is usually strong enough to bear traction, the assistant can take hold of it by fingers or forceps, and by gentle traction can keep the parts well exposed, at the same time that, by means of his left hand with a flat sponge under it, he retracts the left side of the wound and the viscera, which would otherwise fall over the common duct and impede the view.

It will now be observed that instead of the gall-bladder and cystic duct making a considerable angle with the common duct, an almost straight passage is found from the opening in the gall-bladder to the entrance of the bile-duct into the duodenum, and if adhesions have been thoroughly separated as they should always be, the surgeon has immediately under his eye the whole length of the ducts with the head of the pancreas and the duodenum. So complete is the exposure, that if needful the peritoneum can be incised and the common duct separated from the structures in the free border of the lesser omentum; but this is not necessary except where a growth has to be excised.

The surgeon, whose hands are both free, can now with his left finger and thumb so manipulate the common duct as to render prominent any concretions, which can be directly cut down on, the edges of the opening in the duct being caught by pressure forceps.

The assistant can now take hold of the forceps with his left hand, as they with the sponge will form sufficient retractor, since the duct is so near the surface.

When the duct is incised there is usually a free flow of bile, which it must be remembered is probably infective; but a sponge in the kidney pouch, and rapidly mopping up the bile as it flows, by means of sterilised gauze pads, avoids any soiling of the surrounding parts, and if

thought necessary the bulk of the infected bile can be drawn off by the aspirator, either from the gall-bladder or from the common duct above the obstruction, before the incision into the duct is made.

After removing all obvious concretions, the fingers are passed behind the duodenum and along the course of the hepatic ducts, to feel if other gall-stones are hidden there, and a gall-stone scoop, the only special instrument I use, is passed quite up into the primary division of the hepatic duct in the liver, and quite down to the duodenal orifice of the common bile-duct; and if thought necessary to insure the opening into the duodenum being patent, a long probe is passed into the bowel.

The incision into the bile-duct is now closed by an ordinary curved round needle held in the fingers without any needle-holder, a continuous catgut suture being used for the margins of the duct proper, and a continuous fine chromic catgut or spun celluloid thread being employed to close the peritoneal edges of the duct.

In some cases, where the pancreas is indurated and swollen from chronic pancreatitis, and likely to exert pressure on the common duct for a time, I insert a drainage-tube directly into the duct and close the opening around it by a purse-string suture, the tube being fixed into the opening by a catgut stitch which will hold for about a week; but where this is not done I usually fix a drainage-tube into the fundus of the gall-bladder in the same way, as this drains away all infected bile and avoids pressure on the newly sutured opening in the duct.

So easy is it to remove impacted stones after this method of exposure that I now never spend much time in manipulating stones impacted even in the cystic duct, but at once incise the duct, remove the concretions, and close the opening, without damaging the duct by much pressure and prolonged manipulation.

Although there is seldom any fear of leakage or of infection, yet, owing to the separation of extensive adhe-

sions, there is usually some tendency to pouring out of fluid in the first twenty-four hours. I therefore generally insert a gauze drain through a split drainage-tube, bringing it out either through a stab wound in the loin or forwards by the side of the gall-bladder drain.

The wound is closed in the usual way by continuous catgut sutures, first to peritoneum and deep rectus sheath, next to the anterior rectus sheath, and lastly to skin.

To those having little experience in this operation, the modifications I have employed may seem trivial, but to those who have experienced the difficulties of the ordinary operation I feel sure the method I have described, which enables the whole of the bile passages to be dealt with as a straight tube close to the surface, will be sufficiently appreciated.

But the technique of the operation is not the only important part of the treatment of these serious cases, which require thought and care not only before and at the time of, but subsequent to operation.

A careful study of the causes of mortality in operations on the common duct shows that hæmorrhage, either immediate, consecutive, or secondary, cannot be ignored as a danger, and that shock, apart from hæmorrhage, has next to claim our attention.

Sepsis is no longer the bugbear that it used to be, thanks to a rigid all-round asepsis, the employment of gauze drainage, and the careful avoidance of soiling the wound by infected bile.

Although there is a greater tendency to bleeding in chronic jaundice from pancreatic disease than when jaundice is due to gall-stone obstruction, I think there can be no doubt that in all cholæmic conditions the blood becomes so altered that the coagulability becomes seriously diminished, and that these factors demand serious attention before any operation is undertaken in cases of common duct cholelithiasis. After reading Professor Wright's researches on the coagulability of

the blood, published in the 'Brit. Med. Journ.' for December 19th, 1891, my mind was prepared to grasp the possibility of turning the experience gained on dogs to practical uses in the human subject, as I had lost two jaundiced patients, one in 1888 and one in 1890, from persistent oozing of blood subsequent to operation. I therefore at once began to employ it in these cases, and with benefit; but it has been only within the last two years, since using chloride of calcium in apparently heroic doses, that I have been able to get at the real value of the drug, which I now always employ in jaundiced patients, both before operation in thirty-grain doses by the mouth, and afterwards in sixty-grain doses by the rectum thrice daily for several days.

The following case, reported by Dr. W. Gough, affords a good example of its utility.

Mrs. M. E. G—, aged 38, was admitted to the Leeds General Infirmary on January 23rd, 1901.

History.—She had had typhoid fever in September, 1899, and had never been quite well since. Shortly afterwards she began to suffer from biliary colic, though she had never been jaundiced till six months before admission, from which time jaundice had never left her. On December 24th, 1900, she became much worse, and had very severe paroxysmal pain, accompanied by shivering and profuse sweats. From that time she lost weight very rapidly and the jaundice deepened. On admission the liver could be felt below the ribs, and there was a distinct fulness on deep palpation in the region of the pancreas. From January 21st to 31st she took calcium chloride in twenty-grain doses thrice daily.

Duodeno-choledochotomy was performed on January 31st. There was very little bleeding. A stone nearly as large as a pigeon's egg was removed from the ampulla of Vater, which was laid open over a director, introduced through the papilla at its opening into the duodenum. The head of the pancreas was felt to be much enlarged and hard. The incision into the ampulla was not

sutured, but through it the common bile-duct, very much dilated, was explored by the finger. The anterior wound in the duodenum was then sutured and the abdominal wound closed. A drainage-tube was inserted through a stab wound in the right loin. The patient, inadvertently, did not have calcium chloride given in the nutrient enemata, as is usual in these cases.

After history.—She did well till the morning of February 2nd, when the nurse noticed at 3 a.m. that the dressings were soaked with bright blood. The drainage wound was exposed, but no hæmorrhage was occurring there. On examining the abdominal incision blood was seen to be slowly oozing from it and the stitch punctures. One drachm of calcium chloride was at once administered by the mouth, and three stitches were removed; the surface of the wound was then seen to be oozing all over. It was packed with gauze soaked in tincture of hamamelis, and a firm dressing applied. One drachm of calcium chloride was given again in two hours, and afterwards repeated in thirty-grain doses every two hours for six times, it being then given thrice daily. There was no recurrence of hæmorrhage, and the patient made an uninterrupted recovery. The drainage-tube was removed on February 4th, and she returned home within the month. An examination of the blood showed a very marked diminution in the blood-plates.

I think it is important to ligature all bleeding points, and not to trust simply to forcipressure; and while in non-jaundiced patients adhesions may be simply separated, in these cases I prefer to divide adhesions between ligatures where practicable.

Where there is persistent oozing of blood from innumerable points, a tampon of sterilised gauze forms a useful means of hæmostasis, and this may be made more efficient by employing at the same time a solution of supra-renal extract to the bleeding surfaces.

The best treatment of shock is preventive, and to that end it is desirable to lose as little blood as possible,

though I do not agree with those who assert that shock in operation is always dependent on loss of blood. The patient is enveloped in a roughly made suit of gamgee tissue, and where he is very feeble, or the operation is likely to be prolonged, it is performed on a heated table. A large enema of normal saline solution, with or without stimulant, given fifteen to twenty minutes before, and the administration of ten minims of Liq. Strychniæ subcutaneously just before commencing anæsthesia, are useful. Expedition in operating is an important factor in lessening shock, especially in abdominal surgery, for it stands to reason that prolonged manipulation and exposure of the viscera in patients so ill as the class of cases we are now considering must generally be, will be badly borne; for it is not only the work of the surgeon but the deep anæsthesia that adds to the shock, since, for choledochotomy to be well and expeditiously performed, the muscles must be well relaxed. Choledochotomy should occupy from half an hour to an hour, and in case of unusual complications a little longer; but it seems to me that the surgeon who spends two, three, or four hours over one operation is either lacking in skill or judgment.

After operation, a pint of saline fluid, with one ounce of brandy, is given by enema, and five minims of Liq. Strychniæ are given subcutaneously every two hours for several hours if called for. Subcutaneous injections of saline fluid or intra-venous infusion are only rarely required.

As I have performed the operation of choledochotomy for the removal of gall-stones from the common duct on sixty occasions, it would be too tedious to read even a short abstract of them all; I shall therefore refer those who wish for more detail to the second edition of my book on 'Diseases of the Gall-bladder and Bile-ducts,' where an account is given of all my cases, twenty-eight in number, operated on up to December 31st, 1899, and to

the list of cases, thirty-two in number, operated on since January 1st, 1900, copies of which I hand round.

It is interesting to note that the mortality of the whole series of sixty choledochotomies is 16·6 per cent., or, excluding the duodeno-choledochotomies, 14·2 per cent.; but, while those operated on before 1900 give a rate of 23·8 per cent., those since January 1st, 1900, show only 7·1 per cent. of deaths, and of the two fatal cases, one was from heart disease and the other from pulmonary congestion and shock, both deaths, I believe, being essentially due to ether anæsthesia, the ether having been given by means of the old apparatus with an india-rubber bag, a method which I have discarded on account of its asphyxial tendency and its want of cleanliness.

I think, therefore, it is quite reasonable to assume that, with due precautions, the mortality of the operation of choledochotomy should be reduced to 5 per cent. or under.

P.S., March 21st, 1902.—Since handing my paper to the Secretary I have had eight additional cases of choledochotomy, all of which have recovered. It may interest the Fellows of the Society to note that the final paragraph of the paper was a forecast. Fortunately the unavoidable postponement of the communication has enabled me to prove that a 5 per cent. mortality was a reasonable estimate, for it will be seen that of the cases—twenty-one in number—operated on since July of last year all have recovered; and that including all my cases of choledochotomy since January, 1900, there has only been a mortality of 5·5 per cent.

CHOLEDOCHOTOMIES.

Cases previously reported, total twenty-one, with five deaths.

No.	Date.	Initials	Sex and age	History.	Nature of case.	Operation.	With whom seen	Result.	After history.
22	1900 Jan. 28	G.	M., 50	Gall-stone colic 2 yrs. and 3 months; loss of flesh and strength	Jaundice and infective cholangitis; stone floating in common duct; fistula between gall-bladder and colon	Stone removed from common duct; fistula between gall-bladder and colon closed	Dr. Windle, Ovenden	R.	Reported quite well Sept., 1901.
23	March 29	M. B.	F., 44	Fourteen years' history of gall-stones; great failure in health	Jaundice intensified after each seizure	Choledochotomy; cholecystotomy	Infirmary	R.	Well 1901.
24	June 7	Mr. B.	M., 52	Some years' history of "spasms;" in later years jaundice and infective cholangitis	Stone floating in common duct; jaundice and infective cholangitis	Choledochotomy; cholecystotomy	Dr. Ellis, Shipley	R.	Well July, 1901.
25	Aug. 22	Mrs. S.	F., 54	Colic 2 years ago, followed by jaundice; several severe attacks since with ague-like seizures and loss of weight	Five stones in common duct	Choledochotomy; anterior drainage	Infirmary	R.	Well Dec., 1901.
26	Aug. 22	M. H.	M, 56	Diarrhœa and indigestion 25 years; first attack of gall-stone, colic, and jaundice six months before	One stone ulcerating through gall-bladder; one in common duct; muco-pus in ducts; suppurative cholangitis	Choledochotomy; cholecystotomy; posterior drainage	Dr. Barrs, Leeds	R.	Recovery tardy. Well since, except for occasional attacks of pain.

27	Oct. 26 1901	M. D.	F., 43	Colic 6 years; jaundice 2 months; two recent colicky attacks followed by fever and rigors	Jaundice; infective cholangitis; one stone in common duct	Choledochotomy; one rough stone removed; anterior drainage	Dr. Davies, Newport	R.	Severe attack July, 1901; another stone passed. Patient very well Nov., 1901.
28	Jan. 31	E. H.	F., 45	Colic 8 years	Jaundice increased after each attack	Choledochotomy; cholecystotomy	Infirmary	R.	—
29	Feb. 15	J. O'C.	M., 51	Twenty years' symptoms, "pains;" infective cholangitis; loss of flesh and strength	Infective cholangitis; fistula between gall-bladder and duodenum; intense adhesions; jaundice	Choledochotomy with closure of fistula and cholecystotomy	Infirmary	D.	Patient took ether badly, became livid, and had much mucus in bronchi. Died from shock and congestion of lungs.
30	June 2	M. C.	F., 50	Colic attacks over long period; jaundice moderate	Fistula between gall-bladder and duodenum; two stones in gall-bladder, one stone in common duct	Choledochotomy, cholecystotomy; fistula repaired	Dr. Mitchell, Cockermouth	R.	Complete and perfect recovery. Well Sept, 1901.
31	June 8	Mrs. W.	F., 57	Many attacks of colic; jaundice 3 weeks; rapid loss of flesh	Jaundice; one stone in common duct, one in gall-bladder. Gall-bladder calcareous and forming hard tumour	Choledochotomy, cholecystectomy	Dr. Mallett, Bolton	R.	Patient very well Nov., 1901.
32	June 17	Mrs. B.	F., 40	Three years colic and jaundice	Jaundice and infective cholangitis; 8 stones in gall-bladder, one in common duct	Choledochotomy, cholecystotomy	Dr. Godfrey, Scarboro'	R.	Very well Nov., 1901.
33	July 1	Mrs. T.	F., 48	Colic one year; jaundice; fistula after previous operation performed elsewhere in October, 1900, when two stones found in cystic duct	Biliary fistula; slight jaundice; rigors if fistula not kept patent	Choledochotomy; excision of fistula; 2 stones in common duct	Dr. Wakefield, London	R.	Very well Nov., 1901.

No.	Date.	Initials.	Sex and age.	History.	Nature of case.	Operation.	With whom seen.	Result.	After history.
34	July 6	M. W.	F., 28	Spasms for years; 25 weeks colic and jaundice. July 24th, 1900. — Duodeno - choledochotomy, 3 stones removed; wound healed but reopened after attack of pain, followed by rigor; returned July 6th, 1901. with jaundice	Biliary fistula; infective cholangitis	Choledochotomy; removal of 1 gall-stone; excision of biliary fistula	Dr. Graham and Dr. Altham, Cockermouth	R.	Well Nov., 1901.
35	July 20	Mrs. S.	F., 30	Previous cholecystotomy for gall-stone colic followed by jaundice was performed February, 1900, by a surgeon in Manchester	Biliary fistula with jaundice if fistula not not kept patent	Choledochotomy, 1 stone in common duct; cholecystotomy	Dr. Sprawson, Blackpool	R.	Well Nov., 1901.
36	July 21	Mrs. H.	F., 38	Some years' history of spasms, infective cholangitis, and jaundice. High temperature and incessant vomiting for a week before operation; patient extremely feeble	Jaundice; infective cholangitis; vomiting; heart disease and deep jaundice	Choledochotomy; several stones removed from common duct	—	D.	Death from shock and heart failure. Patient took ether badly, became very livid and almost pulseless.
37	July 30	Mr. W.	M., 40	Indigestion and pain 1 year; loss of weight and deep jaundice 5 weeks	Five stones in common duct, 35 in gall-bladder	Choledochotomy, cholecystotomy, lumbar drainage	Dr. Taylor, Derby	R.	Patient went home with small tube in sinus. He had lost his jaundice and was otherwise well. In 1902 well.
38	Aug. 5	Miss T.	F., 45	Gastric ulcer 2 years; colic and jaundice;	Pyloric stenosis with dilatation of the sto-	Choledochotomy, chole-	Dr. Williams, Harrogate	R.	Patient well Nov., 1901.

				loss of weight and strength	mach; 20 gall-stones from gall-bladder, 2 from common duct	cystotomy, partial cholecystectomy, pyloroplasty			
39	Aug. 8	Mr. F.	M., 46	4 months colic and jaundice; loss of weight	Jaundice very deep indeed; 1 stone in common duct, 2 stones in gall-bladder; pancreas swollen	Choledochotomy, cholecystotomy, anterior drainage	Dr. Luce, Derby	R.	Patient well Nov., 1901.
40	Aug. 8	Mr. M.	M., 62	2 years colic; 1 year jaundice; loss of weight; 5 stones. Previous cholecystenterostomy by another surgeon with temporary improvement. Relapse; recurrence of jaundice, with great loss of flesh and strength	Jaundice; 3 stones in common duct, many in gall-bladder; cancer of head of pancreas	Choledochotomy, cholecystotomy, anterior drainage	Dr. Lawrence, Darlington	R.	Patient relieved of pain and jaundice, and returned home within the month. Gradual loss of strength due to cancer of pancreas, ending in death in Oct., 1901.
41	Sept. 1	Miss M.	F., 50	Spasms and jaundice for years; ague-like attacks; loss of flesh	Infective cholangitis; jaundice; 1 stone in gall-bladder, 5 stones in common duct	Choledochotomy, cholecystotomy, lumbar drainage	Dr. Wilson, Haworth	R.	Patient well Nov., 1901.
42	Sept. 17	A. F.	F., 47	5 years colic and jaundice with ague-like seizures	Jaundice intensified after each attack	Choledochotomy, cholecystotomy	Infirmary	R.	Well Dec., 1901.
43	Sept. 27	M. L.	F., 38	Colic February to June, 1900, with jaundice and signs of suppuration; cholecystotomy and draining abscess between gall-bladder and intestine; some gall-stones worked back from common duct. After 2 months colic again with persistent jaundice and infective cholangitis	Infective cholangitis and jaundice	Choledochotomy, cholecystotomy, one stone removed	Infirmary	R.	Well Dec., 1901.

No.	Date.	Initials.	Sex and age.	History.	Nature of case.	Operation.	With whom seen.	Result.	After history.
44	Sept. 29	Mr. B.	M., 51	Slight colic, but deep jaundice, a year; great wasting	Infective cholangitis; 5 stones in common duct	Choledochotomy; duct drained, also lumbar drain; chronic pancreatitis	Dr. Hill, Wisbeach	R.	Patient very well; had gained 1 st. 3 lbs. in weight, Nov., 1901.
45	Oct. 10	C. S.	M., 53	Colic 12 years; jaundice 2 years	Deep jaundice	Choledochotomy; stone removed	Infirmary	R.	Well Dec., 1901.
46	Oct. 21	Mr. W.	M., 58	Very deep jaundice; 3 years biliary cirrhosis and ascites	Jaundice; 1 stone in common duct; cirrhosis of liver and ascites	Choledochotomy; omentum fixed to anterior abdominal wall; anterior drainage	Dr. Rigby, Preston	R.	Dec., 1901, letter to say regaining strength and feeling well.
47	Nov. 7	R. K.	F., 46	History of spasms years before; 8 months' history; very little pain, but temperature 103° to 105° every night	Jaundice and infective cholangitis	Choledochotomy	Infirmary	R.	The temperature dropped to normal the day of operation, and remained normal.
48	Nov. 30	Miss S.	F., 52	Spasms for years; jaundice for 4 months; pancreas large	65 calculi, including 18 in common duct	Choledochotomy, cholecystotomy	Dr. C. Brook, Lincoln	R.	Well at present.
49	Dec. 12	M. D.	M., 48	Stout, unhealthy subject, with history of former intemperance; 20 years' history of gall-stones; jaundice with infective cholangitis 8 months	126 gall-stones removed from gall-bladder and 88 from common duct	Choledochotomy	Infirmary	R.	Well at present.

DUODENO-CHOLEDOCHOTOMIES.

Cases previously reported, total seven, with two deaths.

No.	Date.	Initials.	Sex and age.	History.	Nature of case.	Operation.	With whom seen.	Result.	After history.
8	1900 July 23	Mrs. F.	F., 34	Many years colic; intermittent jaundice; cholelithotrity in Feb., 1891; well 1896; recurrence of pain with jaundice and ague-like attacks	Dense adhesions and very difficult operation; *débris* only removed; pancreas large	Duodeno-choledochotomy; gall-bladder drained; no lumbar drain	Dr. Coleman Hemsworth	D.	Patient died on 16th day from exhaustion due to leaking duodenum
9	July 24	Miss W.	F., 28	28 weeks colic; 14 weeks jaundice	Distended gall-bladder; one stone in cystic duct; two more removed from common duct by duodenal route	Duodeno-choledochotomy	Dr. Graham and Dr. Altham, Cockermouth	R.	Wound healed, but reopened after attack of pain followed by jaundice. The biliary fistula which followed was subsequently cured by the removal of another gall-stone from the common duct by choledochotomy.
10	1901 Jan. 31	M. G.	F., 38	Colic and jaundice 6 months; loss of weight and strength	Deep jaundice; gall-stones in common duct	„	Infirmary	R.	Well some months later.
11	Mar. 22	W. S.	F., 49	8 years colic; varying jaundice	8 large and some small stones in common duct	„	„	R.	Well some months later.

DISCUSSION

Sir Dyce Duckworth.—Although Mr. Robson's communication was mainly of surgical interest, there were some points in it of interest to physicians. He would first remark that physicians gladly recognised the importance of surgical treatment for biliary calculi, and especially because no method of medication was really known to be efficient in causing the expulsion of these concretions. The important matter for the physician here was an accurate diagnosis. It was found that many cases of biliary colic occurred where the physical signs were of little aid to the diagnosis. Symptoms of pains, rigors, and vomiting were met with in the absence of signs of obstruction, and such were apt to recur from time to time, all indicating the presence of calculi or grit in the gall-bladder, hepatic or cystic ducts. The liver might, or might not be palpable, and the fundus of the gall-bladder was seldom detectable. Such cases commonly demanded operative interference, and evacuation of the gall-bladder generally proved lastingly curative of all the untoward symptoms. The diagnosis between impacted calculus in the common bile-duct and obstruction by new growth in the duct on the head of the pancreas was often of extreme difficulty, wasting being common to both conditions with chronic jaundice. Simple biliary colic was generally unattended by pyrexia, and so was occlusion of the cystic or common duct for the first few days. Subsequently, febrile symptoms were apt to supervene, and some inflammatory change in the gall-bladder or ducts was then to be suspected. Operation was called for in such cases, and not seldom pus was met with in the gall-bladder or ducts, together with calculi. It would be wrong nowadays to delay interference in such cases. No doubt many persons lived for years with many calculi lodged in the gall-bladder, but there was always a risk of biliary colic, or of the onset of malignant disease in the coats of the gall-bladder, or the ducts, as advancing years progressed, from their prolonged irritation. It was commoner to meet with gall-stones in persons of the arthritic habit with gouty inheritance, and by diet and other measures their formation might be prevented. Physicians now gladly sought the assistance of surgeons in most of these cases, and fully appreciated the skill brought to bear with so much success in the removal of biliary calculi. Each case, however, demanded special attention, and not every case was suitable for, or required, surgical interference.

Mr. Godlee, after congratulating Mr. Mayo Robson on the large number of successes, and remarking on the very large

number of cases that came under his care, expressed his regret that the author of the paper was unable to be present, as he would have liked to put some questions to him. In the first place he would have been glad to know what length of incision was found necessary in order to pull down and forward the liver to such an extent as to bring the cystic duct and the common bile-duct into one straight line. He showed that the incision proposed was a combination of that which was common in the early days of this department of surgery, and that which had been more frequently used of recent years. The plan of putting a large sand-bag under the spine was one the advantages of which he could bear witness to. He desired in the next place to inquire whether Mr. Mayo Robson considered that it was equally good in all cases requiring anastomosis to join the gall-bladder to the colon or to join it to the duodenum; it was difficult to accept this position, as the uses of the bile in intestinal digestion would then be only to a small extent available. He also would have liked to know in what proportion of the cases which were jaundiced at the time of the operation the characteristic hæmorrhage occurred, and also what proportion of them were receiving the heroic doses of chloride of calcium. He had met with four cases of this bleeding, one a case of gall-stones complicated with carcinoma of the hepatic ducts in which bleeding occurred several days after the operation, and continued for the several weeks which intervened before the patient's death. In this case no chloride of calcium had been given. In another case of carcinoma, chloride of calcium had been given before the operation, which consisted only in an exploratory incision, but bleeding began as soon as the patient was put to bed after the operation. A third case will be reported shortly to the Society in which jaundice was due to an obstruction to the common duct, and in which cholecyst-duodenostomy was performed after giving large doses of the drug. Bleeding occurred a few days after the operation, but ceased as the jaundice subsided. A fourth case was one of very chronic jaundice due to a large stone impacted in the ampulla of Vater. Chloride of calcium had been given freely, but the bleeding occurred within a few days of the operation. The drug had not, however, been given by enema after the operation, as Mr. Robson recommends. Finally he would have liked to hear what Mr. Robson would do in a case where numerous small stones occupied the hepatic ducts. In such a case it would be impossible to be certain that all had been extracted by the scoop, and it would, no doubt, be safer to drain the duct for some time. Mr. Godlee emphasised the importance of the assertion that the contents of the common bile-duct in which stones have been long impacted are usually septic, and pointed out that the introduction of the scoop into the duodenum would necessarily

introduce some of the intestinal contents into the wound. He also pointed out how readily the duodenal contents made their way back into the common duct after it was opened, and illustrated this by a case in which a large gall-stone had made its way by ulceration into the duodenum and become impacted in the ileum; about twenty grape-skins occupied the indurated and shrivelled gall-bladder.

Mr. Godlee replied that he had given 40-grain doses four times a day, but he had been rather disappointed with the results. He pointed out that one cause for the subsidence of the bleeding might be the disappearance of the jaundice due to the operation. This occurs in all cases except those of cancer, in which, moreover, another cause of the tendency to bleed might be the presence of the cancer itself.

Dr. J. H. KEAY said that, while highly appreciating the improved methods of surgery by which the mortality from operation for the removal of stones from the common bile-duct had been so greatly lessened, he could not agree with Mayo Robson in almost entirely ignoring the effect of medical treatment. There could be no doubt that, whether owing to medical treatment or the *vis medicatrix naturæ*, there were many who suffered from impacted stones who made an excellent recovery without operation. He spoke from personal experience. After many attacks of severe biliary colic he at length became jaundiced through occlusion of the common duct, and was just about to undergo operation when he passed several stones, and now, for years past, he had not suffered from gall-stones in any form. He referred to those cases, well known to medical men, where the symptoms were obscure, and were often regarded as pointing to malignant disease, and yet the patients, either through the direct passage of the stones through the duct or their indirect passage into the intestines, got rid of all pain and jaundice, and continued to live in robust health. It was quite true that little could be expected from remedies given for the solution of stones unless in those cases where the stone was protruding into the duodenum, but there was no reason to doubt that by medical and hygienic treatment the quality of the bile could be so altered as to relieve spasm of the duct. He would have liked to ask Mayo Robson how long one might suffer from jaundice before resorting to operation. The answer to this question must of course depend on whether there was greater risk in submitting to operation or in waiting for a possibly favourable issue. He noted in the appendix to Mayo Robson's paper that the condition of the patient was given a year or a year and a half after operation. This was not sufficient. He had recently under his care a patient on whom cholecystotomy had been performed in 1895, and cholecystectomy in 1897, and on both occasions she was discharged from the hospital as cured. She

has since suffered almost as much as before operation, and on one or two occasions been almost *in articulo mortis*. Medical men in general practice had greater opportunities of following the life-history of a patient than those attached to hospitals or in consulting practice, and could not help observing how often the power of resisting and overcoming disease was lowered in those who had undergone major operations. In some cases of obstruction of the common duct operation was certainly advisable. It could not, however, be regarded as a radical cure. If there was a tendency to gall-stones, their removal by the knife did not preclude others being formed.

Dr. H. A. Caley remarked, with reference to the use of calcium chloride as a hæmostatic, that, had Mr. Mayo Robson been present, he would have inquired as to the reasons which had led him to prescribe it in such much larger doses than formerly. The author of the paper had referred to Prof. Wright's experiments on the effect of calcium chloride in increasing the coagulability of the blood, but the amount of the salt given by him was considerably larger than that originally suggested by Prof. Wright, who had indicated that to overstep a certain limit of dosage might have the opposite effect of again reducing coagulability. This question as to the amount requisite to produce the maximum degree of coagulability in conditions such as those referred to in the paper had an important bearing on its employment in hæmorrhagic conditions generally.

Mr. Butler-Smythe thought it was a matter for regret that so valuable a contribution to the surgery of gall-stones should have been brought before the Society at a time when so many surgeons who were interested in this subject were absent from town. He, too, wished Mr. Mayo Robson had been present, for there were some questions he would have wished to ask him. He could not imagine anything more difficult or disagreeable than to criticise a paper or to enter into a discussion in the absence of the author. However, there were one or two matters relating to the technique of the operation which seemed to him to call for remarks. As regards the incisions, no mention was made as to the extent of the vertical one, and to his mind an author, bringing before a society a modification of any surgical procedure, could not be too exact in explaining the details. He agreed that the oblique incision, when carried out, would find more room, but he would like to say that he had frequently seen the common duct well exposed by a 5-inch vertical incision outside the rectus muscle. Then with regard to the amount of Liq. Strychninæ administered, he would like to know if Mr. Mayo Robson carried out this method as a general routine, or only in exceptional cases. Knowing how different individuals were more or less susceptible to that drug, and also that it was a cumulative poison, he thought the doses given were, to say the

least, heroic. In his practice he had lately seen a fatal issue, with all the symptoms of strychnia poisoning, following the subcutaneous injection of that drug in ten-minim doses, thrice repeated within three hours. He would like to have the opinion of some of the physicians present as to the quantity of Liq. Strychninæ that might safely be injected in repeated doses.

Mr. HERBERT PATERSON thought that the most remarkable feature in Mr. Mayo Robson's brilliant series of cases was the striking freedom from sepsis and consequent low mortality. From what he had seen, sepsis was by far the commonest cause of death in these operations. With regard to cholecystenterostomy, it was interesting to recollect that the first case in this country was brought before this Society by Mr. Mayo Robson, and he believed that he was right in saying that it was our present President who was the first to suggest the performance of this operation in cases of gall-stones, as an alternative to leaving the patient with the discomfort and inconvenience of a permanent biliary fistula. He was of the opinion that further evidence was required as to the value of calcium chloride in diminishing or arresting hæmorrhage, and it did not seem clear that the case quoted by Mr. Robson was evidence of the value of this treatment. For notwithstanding that the patient had had calcium chloride before operation, there was bleeding from the wound. The wound was packed with gauze soaked in Tr. Hamamelis, and the bleeding ceased. Surely the stoppage of the bleeding was due to the hæmostatic action of the hamamelis, rather than to the *subsequent* administration of calcium chloride. He thought it was scarcely fair to attribute the vague symptoms comprised in the term general debility to an operation performed many years before, as had been suggested by one of the speakers. It was within the experience of all how ready patients were to find some cause, often clearly an erroneous one, for all their ailments, and their morbid minds eagerly grasped at a previous operation, however long ago performed, as furnishing the *fons et origo* of any real or imaginary indisposition. As to the administration of large doses of strychnine, he had given as much as forty minims subcutaneously within four hours with a successful result.

He ventured to disagree with the opinion expressed by Mr. Robson that *deep* anæsthesia added to the shock of the operation, for he was convinced that the shock produced by the operation was inversely proportional to the depth of the anæsthesia; the lighter the anæsthesia the greater the shock; the more profound the narcosis, the less was the patient affected by the manipulations of the operator. He was very glad that Mr. Robson had spoken unfavourably of ether as an anæsthetic in these cases. Personally, he was most strongly against the use of ether in any

abdominal operation, and it was his firm belief that in these severe and prolonged operations the use of ether greatly militated against a favourable result; indeed, from his own experience he believed that in such cases it increased the mortality as much as 5 per cent.

The PRESIDENT desired to associate himself with the expressions of deep regret at Mr. Mayo Robson's absence; obviously it rendered the discussion much less thorough and complete. He would have liked to ask Mr. Mayo Robson one or two questions. It would be instructive to learn in what number or proportion of cases of obstruction in the common duct had the diagnosis of concretions in that duct, previously made, proved to be well founded. Again, was jaundice due to this cause as frequent as jaundice due to new growth or other external cause? Did Mr. Mayo Robson's experience lead him to attach importance to any one or more symptoms as indicative of obstruction by concretions? From his own experience he could confirm the author's observation of the alteration in direction and relation to each other of the cystic and common ducts in these cases of concretion in the common duct producing obstruction.

Author's remarks on paper read in his absence, from abstracts of the discussion kindly furnished by the Secretary of the Society.

Mr. MAYO ROBSON wished first to apologise for his unavoidable absence from the meeting when his paper was read, the reason being that he was detained in the south of Europe by an operation that could not be deferred.

Sir Dyce Duckworth's remarks on diagnosis and treatment entirely coincided with the author's views, and although the paper was essentially surgical, he fully grasped the fact that all these cases were to begin with purely medical, and only surgical after the diagnosis had been reasonably established and general treatment had failed to bring about relief.

Mr. Rickman J. Godlee had asked as to the length of incision; it was a modification of the vertical incision that the author had been employing for several years, the incision being extended upwards over the liver, if necessary, quite up to the angle between the right costal margin and the ensiform cartilage, the length of this extension depending mainly on the size of the liver, the incision being also a little nearer the mid-line, so as to obtain the extension by splitting rather than dividing the fibres of the rectus, except a little obliquely towards the upper end. The average length of the incision would be about five inches

but this might be exceeded, if necessary, without any weakening of the abdominal wall if the anterior and posterior sheath of the rectus were sutured separately. The author had done it through a four-inch incision, but did not hesitate to extend the incision to whatever extent necessary to draw forward the anterior border of the liver.

As to the question of draining the gall-bladder into the bowel, if the patient be in a condition to bear exposure of the duodenum to the extent necessary to effect the anastomosis, the common duct will be efficiently exposed by the operation now described, and can be readily cleared; but if the patient be in a very poor condition the author finds by experience that an anastomosis between the gall-bladder and colon, which can be effected in a few minutes with very little exposure of viscera, is quite efficient in giving relief, and in fact his first case of cholecystenterostomy, in 1888, was a gall-bladder-colic anastomosis, and the patient is now in good health; so much, therefore, for the digestive use of the bile. For the control of hæmorrhage in deeply jaundiced cases by chloride of calcium it must be given by enema in 60-gr. doses subsequent to operation until the blood has clotted in the divided vessels. As to the question of clearing the hepatic duct, by the method he had described the hepatic duct quite up to the liver was fully exposed, and could be freely manipulated. Through the opening in the common duct the author had passed his finger up to the division of the hepatic duct, and had been able to discover and remove concretions from it by means of his gall-stone scoop. The passage of a probe (not the scoop) into the duodenum, in order to prove the duct clear, had not in the author's experience led to any untoward result, though it must be accepted as a fact that the bile in any case requiring choledochotomy is always infective.

Dr. J. H. Keay's views as to the utility of olive oil were so fully answered by Sir Dyce Duckworth's very extensive experience of its uselessness, that it seems needless to further discuss the question.

As to the subsequent history of cases operated on, if Dr. Keay will refer to the second edition of the author's book on 'Diseases of the Gall-bladder and Bile-ducts' he will see that many of the cases are referred to years after as quite well; but the cases here brought forward to illustrate the operation described are of course only comparatively recent, as the complete operation, which is the "*raison d'être*" of the paper, is of quite recent date. A great number of the patients operated on are private cases, and can easily be referred to, and many of them are well known to the author to be now in excellent health, as one would expect when the only cause of their illness has been removed.

Mr. Mayo Robson wished to emphasise the fact that recurrence

of gall-stones after operation in his experience is extremely uncommon, and almost unknown if the ducts have been thoroughly cleared at the time of operation, though subsequent operations may be necessary if the operation be incomplete, as it necessarily was in some of the early cases. It is with this view of making the operation absolutely complete and efficient that the author has devised this radical operation.

Dr. H. A. Caley had asked as to the reason of giving large doses of calcium chloride; that had arisen as the result of experience. In order to get the maximum effect the drug should be given not longer than two or three days before operation, and it was not necessary beyond two or three days subsequently, but during that time it was desirable to have a sufficient amount of the lime salt in the circulation, in order to increase the coagulating power of the blood.

In answer to Mr. A. C. Butler-Smythe, strychnia given subcutaneously is, in the author's experience, much less toxic than when given by the mouth, and the speaker's remarks as to the danger would not lead him to alter his practice in employing it, as he had found the drug undoubtedly very useful.

In answer to Mr. Herbert Paterson, the author thought that ether, if carefully administered, was a safer anæsthetic than chloroform at the time of operating, but he entirely agreed with the speaker that chloroform was a very comfortable anæsthetic both for the patient and operator in abdominal operations if skilfully given, and probably, looking to the after progress, in many long and serious cases a safer all-round anæsthetic. He felt the difficulty of proving the value of calcium chloride, and its use in the case quoted is open to objection in that hamamelis was used as well. Nevertheless the author's experience in a large number of cases left no doubt on his mind that the drug was of great value. He was glad that Mr. Paterson agreed that it was scarcely fair to attribute the vagus symptoms comprised in the term general debility to an operation performed many years before, as had been suggested by one of the speakers.

In answer to the remarks of the President, Mr. Mayo Robson had seldom operated expressly for gall-stones and found malignant disease to be the sole cause of the jaundice; but in a number of cases where the operation had been undertaken as an exploratory procedure, and where cancer was suspected, gall-stones or chronic pancreatitis had been discovered, and the patient had been cured by the operation.

In a paper recently given before the Medical Society of London, "Observations on the Surgical Treatment of Obstructive Jaundice from an Experience of over 200 Cases," and published in the 'British Medical Journal' for January 18th, 1902, the author had dealt at length on the diagnosis of the

causes of obstructive jaundice, and had given his experience of operations on 212 cases, out of which 60 were the subjects of malignant disease, and 152 had suffered from gall-stones or other non-malignant causes of obstruction.

A CONTRIBUTION

TO THE

STUDY OF TROPICAL ABSCESS OF THE LIVER

BY

RICKMAN J. GODLEE, M.S.

Received March 6th—Read May 13th, 1902

In the course of an experience of tropical abscess of the liver, somewhat large for an English surgeon, though ridiculously small as compared with that of those practising in the tropics, I have been led to think about the disease and its complications, and trust that the following contribution to its study may be, if disjointed, not therefore without interest. Possibly it may elicit some useful comment and criticism, and it is on this account that the statements contained in it are somewhat dogmatic. I will first deal with some of the complications.

Perihepatitis, usually perihepatic peritonitis, is an almost invariable accompaniment of tropical abscess, and must always exist to a greater or less extent when the

abscess reaches the surface of the liver; but it is astonishing how little there is in some cases even when the matter has approached within perhaps half an inch of the surface. If there be no adhesions the introduction of an exploring trocar or an aspirator needle into the liver will probably be immediately followed by the escape of pus into the peritoneal cavity. Supposing the needle has been introduced for the sake of discovering the existence of an abscess, and the operation for its evacuation be immediately proceeded with, there may yet have been time for a quite considerable quantity of pus to have escaped in the few minutes which have elapsed before the peritoneum is reached. It is even possible to mistake this free pus for the abscess itself. The further exploration of the liver under these circumstances is essential. But first the peritoneal cavity in the neighbourhood of the puncture should be carefully sponged, for although the pus may contain no other organism than the *Amœba coli*, and need not necessarily give rise to septic peritonitis, there is no security that streptococci or staphylococci or *Bacterium coli commune* may not be present. It is scarcely possible under these circumstances to sew the liver up to the abdominal wall, as each stitch will most likely enter the abscess cavity, and the stitch holes will leak. It is safer, therefore, to pack round the part where the incision is to be made before the abscess is actually opened, and after the opening has been made to manipulate the parts as gently as possible.

Perihepatic peritonitis is the cause of the acute pain that is often met with as distinguished from the typical liver pains. This pain is of course superficial, and is accompanied by acute tenderness easily elicited by even light percussion. Peritonitis occurs often in definite attacks, accompanied by fever and vomiting, and often yields a useful physical sign, *i. e. friction*, which may be felt with the hand and heard with the stethoscope over the liver. It exercises a protective effect by shutting off the part

into which an abscess is pointing, so that when rupture occurs a subdiaphragmatic abscess results, and not a general peritonitis.

As might be expected, peritonitis most commonly affects the convex surface of the liver, which thus often leads to the adhesion in whole or in part of this surface of the liver to the diaphragm. When this has occurred an enormous amount of pus may accumulate in the liver without giving rise to any enlargement downwards, and the enlargement upwards may only be indicated by partial dulness in the lower part of the chest; the dulness is least marked when the lung is adherent to the pleura, because the lung cannot then be displaced upwards by the approximation of the diaphragm to the chest wall.

Perihepatic peritonitis may also occur on the under surface of the liver, and may then give rise to very serious results. The stomach, duodenum, and colon may become firmly adherent to the liver, and after the abscess has been evacuated the consequent contraction may lead to considerable displacement of these viscera, giving rise to such troubles as dilated stomach from kinking of the duodenum. But it is a much more serious thing if the transverse fissure and the small omentum have been the seat of the peritonitis, for any one or all of the hepatic ducts may be partially or completely obstructed.

I have recently seen with Dr. Manson a case which bears out this statement.

A man aged 35, in the Civil Service, who had been in India almost continuously since 1887, and had had some pretty severe attacks of fever, began to have hepatic troubles following dysentery in May, 1900. The special points in his illness were that he had had several attacks of acute pain and indigestion, accompanied with high fever; and that an unsuccessful attempt to open the abscess through the chest wall in the lower axilla had been made in Madras in May, 1901. It will be observed that the history suggests a good deal of peritonitis. I opened the abscess without difficulty through the old scar

in July, 1901, and during convalescence, which was rather tedious, there was from the first a rather large quantity of bile in the discharge. This escape of bile is not uncommon; it indicates that a bile-duct of some size communicates with the abscess, and though it delays healing seldom gives rise to trouble. In this case, however, after the abscess had contracted and pus had ceased to flow, bile still came from a small opening in the scar. After a while it nearly stopped; but at the end of September, after some abdominal pain, all the bile came through the wound, and the stools became white. On November 7th the flow of bile ceased, and the patient became intensely jaundiced.

Knowing the danger from hæmorrhage of operating on a jaundiced patient, I passed probes into the wound and fortunately re-established the flow of bile by this channel; and when the jaundice had diminished, and after the administration of large doses of chloride of calcium, I opened the abdomen on November 28th, and found the matting together of the viscera I have described above. It was quite hopeless to free the common bile-duct from the dense surrounding adhesions, as I had hoped to do, and so, as the gall-bladder, though not distended (because the bile was escaping through the wound), contained bile, I anastomosed it with the first part of the duodenum, using a Robson's bobbin. It was a difficult task. I ought to have waited still longer, *i. e.* until the jaundice had completely disappeared, for after a few days hæmorrhage occurred in the wound, and to the consequent stretching of the parts I attribute the partial giving way of the junction, and the escape of bile and duodenal contents through the abdominal wound for a while. At last, however, the leak stopped and the wounds healed, the stools becoming of normal colour, and the patient's health soon re-establishing itself. I must add, however, that he had an attack apparently of indigestion in January, 1902, followed by the escape of bile for a few days from the abdominal wound. But this has again closed, and at

present he is quite well. His condition for the next six months will be watched with interest.

The *escape of the whole of the bile through the wound* is fortunately not of common occurrence. I have never before met with it in cases of tropical abscess, though I have seen it in hydatid of the liver. It does not necessarily lead to loss of appetite or difficulties in digestion, but it sooner or later causes emaciation, which may become extreme. I have seen bedsores form over the projecting angles of the ribs and angles of the scapulæ.

I am permitted by the kindness of my friends Dr. Manson and Sir W. H. Bennett to mention the only other two instances I have heard of in connection with tropical abscess, and I would suggest that in fatal cases the same condition as that which occurred in the one I have just described will be met with. In Dr. Manson's case (Case 1) the patient died after four months in a state of extreme emaciation, though the fistula was apparently beginning to close; and in Sir William Bennett's case the sinus gradually closed after seven months, and the patient made a complete recovery (Case 2).

In connection with this subject reference may be made to cases, with which all will be familiar, where there is complete obstruction to the common bile-duct, either from calculus, tumour, or inflammatory adhesions. These patients do not, as far as I have seen, emaciate; on the contrary, their nutrition may remain fair for years, and it is quite remarkable how long the fatal event may be delayed.

It has been shown by the beautiful observations of Klein that the direction of the flow in the lymphatics of the diaphragm is upwards. As might, therefore, have been expected, *pleurisy* is a very common accompaniment of tropical abscess. It is a useful diagnostic sign. It is also a note of warning. Rupture into the pleura is in my experience one of the most dangerous outcomes of this disease. I could, if it were advisable, give more

than one example of this catastrophe being followed by the most extraordinarily rapid pleural effusion. In one, indeed (Case 3), though I saw the patient within twenty-four hours of the rupture of an abscess which had for so many months remained dormant that its very existence had been doubted, the patient appeared to be moribund, and the pleura contained an enormous amount of fluid.

But even when the pleura is opened at once, if the lung have not previously contracted adhesions to the chest wall, it is not unlikely that it may collapse against the spine and never expand again, and the most extensive Estlander's operations may fail to effect a cure.

The patient, therefore, who has extensive pleural adhesions is saved from very grave dangers.

It would often save trouble and anxiety if the presence or absence of such adhesions could be determined. This is, however, notoriously difficult and often impossible, which is my excuse for mentioning two aids to diagnosis which have not, I think, received sufficient attention.

1. The upper level of the dulness caused by a liver which is enlarged upwards is, speaking generally, considerably lowered when the patient is placed on his left side. If, however, the lower part of the pleural sac is obliterated by adhesions this does not occur, because the lung cannot be sucked down into the pleural sinus by the falling away of the liver—that falling away which so frequently causes pain when the patient attempts to lie on the left side.

2. In very thin people, even when the parts are normal, I have pointed out ('British Medical Journal,' October 6th, 1900, p. 997) that it is often possible on careful inspection to see the lower margin of the lung on the right side move upward and downward on respiration. It may sometimes be observed on the left side, especially if the spleen be enlarged, or there be a great enlargement of the left lobe of the liver, or, indeed, any tumour in this situation; and on the right side it is more obvious when

the liver is enlarged than when it is of normal size. This sign requires somewhat careful inspection for its discovery, and is only of use in emaciated or very thin subjects. If it be present it is a certain proof that there are no adhesions in this situation, and that there is little or no pleural effusion.

Whilst writing this paper I had under my care a young woman in the last stage of emaciation from advanced actinomycosis, in whom I was able to demonstrate this phenomenon to spectators standing at some little distance from the bed. It was in her visible on both sides. It has been doubted by physiologists whether on deep inspiration the lung ever reaches the bottom of the pleural sinus. Careful observation of this patient showed that on very deep inspiration the lower border moved quite as far as the anatomical limit of the pleural cavity.

Though not quite germane to the subject, it may not be out of place to throw out the suggestion that when the liver is much displaced downwards by a pleural effusion, and there is no corresponding displacement of the heart outwards, it is almost certain that the base of the lung is not adherent to the diaphragm. Under these circumstances it may be almost impossible to say whether the fluid is above or below the diaphragm.

In connection with the question of adhesions I desire to make a practical suggestion. If the incision for opening the abscess has necessarily to pass through the pleura below the lower border of the lung, it is often difficult or impossible to tell whether the cavity is obliterated by lymph or not. I have more than once cut down through both layers of the pleura, and as no air entered, have gone on to incise the diaphragm and liver. On withdrawing the finger, however, the ominous sound made by air rushing into the chest has shown that the two layers of the pleura were at first only held together by the attraction of cohesion or by very feeble adhesions, and a laborious process of stitching up the hole had to be

undertaken. The right way to proceed, if there be any doubt, is to cut through the diaphragm across its fibres at the part nearest its costal attachment, and to sew up a flap of the muscle to the intercostal muscles at the opposite end of the wound. The incision into the abscess can then be undertaken without fear of producing pneumothorax. By adopting this precaution I do not expect to meet with the above-mentioned accident again.

Pleurisy may lead to serous effusion, or to empyema, without any rupture of the liver abscess through the diaphragm, and such an effusion may keep up the temperature after the successful evacuation of the hepatic abscess.

I once opened a large tropical abscess on an Indian army surgeon who was in an extremely exhausted condition. There remained some dulness in the back, and the temperature did not fall to normal. The removal by aspiration, several days later, of a few ounces of clear fluid was immediately followed by disappearance of the pyrexia, and from this time the patient made an uninterrupted recovery.

On another occasion, when the physical signs and symptoms were almost the same, the fluid was pus, and an equally good result followed the insertion of a drainage-tube into the pleura.

A less successful result followed in the case of a young officer whom I saw with Dr. Ringer, and whose abscess I had opened in the lower axilla. Repeated attempts at aspiration of the chest were unsuccessful, because there was much recent lymph as well as fluid in the pleura. In the hope of securing better drainage, I made a second incision into the abscess behind, and in doing so found that I had opened the pleura, and a considerable quantity of clear fluid and large masses of lymph escaped. Thinking that some of the pus from the abscess had probably entered the pleura, I therefore placed through this second opening one tube into the abscess and one into the

pleura, but I am sure it would have been better to have sewn up the opening in the pleura, and to have left this cavity for treatment later on; for the recent pleural adhesions gave way owing to the violent coughing of the patient, the lung collapsed, and a general pneumothorax was produced. The case was a very septic one, and a fatal result was probably inevitable; but I think it was hastened by the pleural complication.

I said that the existence of friction was an *ominous* sign, for although rupture into the lung, as every one knows, often leads to a rapid cure of the disease, no one who has seen the disastrous results that may ensue would dream of waiting for it to occur. *When there is marked pleural friction, therefore, exploration of the liver should be made without delay.*

I am thus led to the next part of my subject.

Changes that take place in the lungs.—It is obvious that in every case in which an abscess bursts into the lung a certain amount of lung tissue must be destroyed, and an abscess of greater or smaller extent must be formed. In those which recover in a short time this is a negligible quantity. But it is often far otherwise. The next most favourable cases are those in which a small abscess forms in the lower part of the lung either in front or behind. Most commonly the signs of cavity will be met with below and to the inner side of the right nipple; but the cavity not infrequently occurs behind, and in rarer cases a liver abscess bursts into the left lung and forms a cavity there. At the end of the paper will be found an account of cases illustrating each of these conditions (Cases 4, 5, and 6).

If such abscesses are opened as soon as they are discovered it is probable that they will heal readily. But, if operation is delayed, a series of phenomena may occur that are not unlikely to give rise to a very chronic condition which may end fatally. These abscesses do not behave like those resulting from pneumonia, injury, or tubercle, but have a peculiar tendency to burrow by means of long

and intricate tubular processes which are very difficult to follow up and drain, and may lead to the gradual destruction of large areas of lung tissue. I have seen one lobe almost completely disorganised in the course of an illness extending over many months (Case 7). The suppurating tracks are sometimes in the lung itself, sometimes partly in the lung and partly in the pleura. The pus discharged from them has the characters which we are accustomed to associate with liver pus,—that is, it is inodorous, thick, slimy, and chocolate-coloured, and may contain the *Amœba coli*, so that I am inclined to believe that the peculiarity of the process depends upon the presence of this parasite. In these cases hæmorrhage is a common symptom; it is often frequent and severe, and occasionally fatal (Case 8). The continued discharge of what has been recognised as liver pus has led to the assumption that the abscess of the liver is still unhealed; but this is certainly not always the case, for I have met with an instance in which the patient, a young man from an unhealthy Indian tea-garden, died of hæmorrhage, and at the post-mortem examination so little trace of the liver abscess remained that the medical man who made the necropsy stated that there never had been an abscess of the liver at all. This, however, I knew to be incorrect, for I had had my finger in the patient's liver, and the observation did not surprise me, for I have been struck with the complete way in which all traces of a liver abscess even of large size may disappear (Case 8). This is surely a not unimportant fact in making a prognosis, and accounts for the completeness of the cure if recovery takes place. It is also interesting to note that if a secondary abscess forms in the left lung, as a result of the inspiration of matter from the opposite side, the pus formed in it may be slimy and chocolate-coloured like that which came from the original abscess (Case 9). I do not mean to say that chocolate-coloured pus may not come from a primary abscess of the lung; it may do so, but physicians will agree that this is a most exceptional occurrence. *It may be*

safely stated that the persistent discharge of chocolate-coloured pus does not prove that the liver abscess is not healed.

Another danger to which a patient with an imperfectly drained abscess of the lung is exposed is abscess of the brain. I have met with two such cases following tropical abscess (Cases 9 and 10). In one I opened the abscess myself, and in the other I directed the operation; both ended fatally.

The practical deduction from what has been stated is that *pulmonary abscess consequent on hepatic abscess should be opened without delay*, and that a careful search should be made for outlying suppurating tracks; that these should be drained by the insertion of full-sized tubes, and, as far as possible, laid freely open, even if this should necessitate extensive removal of ribs; and, finally, that the tubes should not be removed or shortened until the surgeon is satisfied that closure of the abscess is almost complete.

Dr. Manson, with whom I have been associated in several of the cases that have come under my care during recent years, has shown that, in the majority, the *Amœba coli* is present in the pus. In some its presence is more easily demonstrated a few days after the abscess is opened than on the day of operation. In some it is found in abundance many weeks later, when the case is, perhaps, pursuing a normal course towards recovery. There is, therefore, strong suspicion that the amœba has something to do with the causation of tropical abscess. This persistence of the organism, and the very slight effect it may, under some circumstances, produce, possibly account for one of the most striking features of this disease—its *occasional extreme chronicity*. One cannot fail to be struck with the long periods of time during which an abscess may be latent, with the way in which symptoms that point strongly to the existence of an

abscess may disappear, and with the comparatively slight disturbance of health that is not inconsistent with the presence of an abscess. I have known a young officer, who was suspected of having an abscess, go through a winter's hunting and return to show himself, as he thought, well, though the abscess was almost pointing at the epigastrium; and it is well known that an abscess may show itself years after a patient has returned from the tropics.

Some abscesses, on the other hand, after a long period of latency suddenly become acutely septic. These cases most likely become infected from some part of the intestinal tract with the *Bacterium coli commune* or some other septic organisms.

I had under my care last year a young officer invalided home from India on account of liver abscess. But his symptoms so completely disappeared that he was supposed to be well, and was sent abroad to recruit. He had, however, occasional attacks of acute epigastric pain, in one of which a swelling formed at the epigastrium, which was caused by the rupture of the abscess in this situation. This was accompanied by great collapse, and was quickly followed by double parotid bubo. The abscess and the two parotid buboes were opened, but the patient died in a few days with all the symptoms of acute septicæmia.

Some liver abscesses contracted in the tropics are, on the other hand, acute and septic from the first. These should, I think, be placed in a class by themselves, and are not, properly speaking, examples of tropical abscess, but are part of a general pyæmic process. They may arise in this country. They are often multiple, and, as far as my experience goes, are quite hopeless cases to treat.

But some genuine tropical abscesses are very acute. I saw lately a young man from Central Africa who had had many attacks of fever, but no hepatic symptoms till he reached Europe on leave. In the course of a few weeks he developed a small abscess in the right lobe. It

was easily reached, and healed in a shorter space of time than any other case I have had to treat, a very few weeks sufficing for the cure.

The majority of tropical abscesses that I have seen have been single, and I believe that many of the cases which have been reported of a second abscess following the first are those in which the drainage-tube has been removed too soon, or in which a diverticulum of the original abscess has never been properly drained, and has been shut off from the main cavity. I think it is of great importance at the time of the operation to explore the cavity very carefully with the finger, and to open up all branches of it, as far as possible, before the drainage-tube is inserted.

I am, of course, not likely to forget that the cases we see in England have been sent home from distant parts of the world, and am prepared to hear that others of a totally different type may be met with by surgeons who are on the spot where the disease originates. I have, indeed, been frequently told that we in England do not see the really bad cases, which may be true, though it must be owned that some of them are bad enough in all conscience.

The last point I would deal with is the question of the *best place to make the incision*, and will begin by stating that I consider, if possible, the seat of election for the operation is in the lower axilla. If two lines be drawn vertically downwards, prolonging the anterior and posterior folds of the axilla as far as the margin of the ribs, they will, at the lower part, enclose the space where the widest interval intervenes between the lowest part of the pleura and the costal margin, an interval generally of two inches, and often even greater. The incision may conveniently be made transversely,—that is, parallel with the lower margin of the pleura; and the portion of one rib

and cartilage that crosses the wound obliquely should be removed, great care being taken to separate the structures on the deep surface of it without injuring the pleura, in case, by chance, it should extend lower than usual. Generally the structure thus exposed consists only of the origin of the diaphragm, but if the pleura should be low, it is easily recognised, and may, without any difficulty, be dissected up without injuring it, and fastened, out of the way, to the upper part of the wound. If it should accidentally have been opened, the suture of the opening is a simple matter, and it is essential to make it perfectly air-tight before proceeding. The rest of the operation consists in incising the diaphragm, either in the direction of its fibres or across them (I prefer the former method), and then cutting through the diaphragmatic peritoneum. If there be no adhesions the liver may either be sutured to the diaphragm and chest walls, or the parts around the opening may be carefully plugged with some antiseptic material. If the latter course be adopted, it must not be forgotten that if the abscess be large the liver will at once shift its position, and that this shifting will take place in the upward direction.

It may be asked, "Why lay down this dogmatic rule when it is well known that the abscess may occur in any part of the liver?" But to this it may be answered that, in my experience at all events, by far the majority of abscesses occur in the right lobe; and that, if they be of large size, they can generally be opened in this situation, even if they form a projection in the epigastrium or most nearly approach the surface behind. I have already referred to the difficulties and dangers of incising the liver behind, depending upon the fact that it is necessarily a transpleural operation. The epigastric incision does not drain well, and I am therefore in the habit, even if the abscess appears to be pointing in this situation, of ascertaining to begin with whether or no the matter can be reached at a moderate distance from the side. It is a question whether, if this be found to be the

case, and after making the lateral incision the epigastric tumour disappears, the surgeon should be content, or whether he should make an epigastric incision as well. A case recently under my care, and referred to in an earlier part of this paper (page 130), supplies an argument in favour of the latter course. I had been content with the single incision in the side, but it turned out that the epigastric swelling had been caused by a localised peritoneal collection of matter due to the bursting of the abscess in front. The communication was not, however, sufficiently free to allow of satisfactory drainage, and it was necessary to make the anterior opening in the course of a few days.

I do not deny that the epigastric incision is the only possible one in certain abscesses in the right lobe, and in all of those that occur in the left lobe.

I would strongly deprecate a lateral incision below the costal margin, because the opening is certain to become troublesomely oblique in the course of a few days as the liver shrinks up under the ribs.

The opening into the liver itself may conveniently be made with a long pair of dressing forceps followed by the finger. In this way abscesses at a great distance from the surface may be safely reached and effectually drained.

Hæmorrhage is often free, but it usually stops spontaneously if the finger be retained for a minute or two in the wound. Should it not do so, careful plugging round the tube must be practised. Some cases bleed extensively after the operation, and the bleeding occurs on subsequent occasions. I am not referring to the slight hæmorrhages which often occur for many days in cases which are pursuing a normal course, but to those in which the loss is considerable. I look upon this as a grave sign, and think it occurs most in the septic as opposed to what I have called the amœbic abscesses.

A certain amount of blood often escapes into the peritoneum as the result of the preliminary puncture. It

seldom is of any moment, but dangerous and even fatal cases of exploratory puncture have been recorded. It is not unlikely that these patients were either jaundiced or suffering from leucocythæmia.

I cannot conclude without referring to Dr. Manson's ingenious device of introducing a drainage-tube through a large trocar plunged boldly through all the superficial tissues into the abscess. It has yielded excellent results in the hands of many, and I would not say a word against it. But for my own part I prefer to know in what condition I have left the pleura and peritoneum, and to have ascertained what the size and shape and possible ramifications of the abscess may be; to have opened these up if it appears to be necessary to do so, and to have placed the tube or tubes in what seems to be the best position for the future draining of the cavity.

Illustrative Cases.

Case 1 (*escape of all the bile through the incision made into the abscess; death*).—This case was under the care of Dr. Manson, and has been published by him in 'Medical Reports,' Imperial Chinese Maritime Customs, *circa* 1884.

The patient was a man aged about 34, a tea-taster, resident fifteen years in China, chiefly at Amoy. Ten years previously he had had pleurisy, and suffered from stricture and chronic sores on the legs. For the previous four or five years he had suffered from chronic dysentery, and had been of habitually intemperate habits.

Liver symptoms began in February, 1883; there were pain, fever, and other symptoms of hepatitis. By August the liver had considerably enlarged and some friction was heard.

On September 14th he reluctantly consented to an exploration, and pus, which was found at a depth of two or three inches in large quantity of dark chocolate-brown

colour, escaped freely through the cannula which had been introduced. A drainage-tube was passed through the cannula, eight inches long, and through this the residual pus was from time to time removed by aspiration. Bile in small quantities appeared in the pus, and by the beginning of October it was large in amount.

On October 13th there was an extensive hæmorrhage.

On October 16th the abscess was irrigated with a solution of salicylic acid, and some sloughy material escaped.

On October 19th the bile was discharged in very great quantity, and on the 30th it all came through the wound, and the stools became white. About the same time some thick sloughs came away. After this the bile was collected in a bottle, and the daily amount varied from 28 oz. to 35 oz. His weight diminished, but the appetite remained good.

On December 22nd the tube was plugged, but this was followed by a rise of temperature, so the plug was removed.

On December 24th, 25th, and 26th there was bile in the motions.

He was then sent on a voyage to America, but on the way he died, on January 29th, 1884, apparently from exhaustion, owing to the very rough weather; but it was noted that the amount of bile discharged was reduced to 14 oz. *per diem*, and that the stools were coloured, so it is probable that he would, like the following case, have recovered if he had not experienced such a tossing upon his voyage.

Case 2 (*escape of all the bile through the incision made into the abscess; recovery*).—The patient was a man aged 46, a railway engineer, under the care of Sir W. H. Bennett, who has kindly furnished these notes. He had lived in India since the age of three years. He was well until October, 1898, when he began to have occasional attacks of acute diarrhœa and severe malarial fever.

In May, 1899, he came under observation suffering

from an abscess in the right lobe of the liver, which was opened below the end of the eleventh rib, at which spot the abscess was bulging, on May 22nd.

Nothing peculiar was noticed at the operation, but seven hours afterwards the dressings were thoroughly soaked through with pure bile, which continued to flow intermittently.

The stools were generally white but sometimes piebald, and partially regained normal colour during the occasional stoppage of the leakage. Dyspepsia was troublesome. There was often great constipation, sometimes diarrhœa. Emaciation was extreme.

The wound was open from May 22nd to December 26th, when permanent closure occurred.

Leakage stopped suddenly twice after gradually diminishing to a certain point. The first stoppage of leakage occurred on September 28th, and no bile was seen for a week. Then a profuse flow began suddenly. During the cessation of leakage the stools became more normal in colour, but whitened again with the recurrence of the flow of bile.

The patient was more or less jaundiced until about a fortnight before the final healing of the wound.

Case 3 (*rupture into pleura; very rapid effusion*).—The patient was a man aged about 25, a Ceylon tea planter, under the care of Dr. Bramwell of Cheltenham. He had had hepatic symptoms dating from August, 1895. They subsided, and it was thought that the abscess had disappeared.

Symptoms reappeared in the spring of 1896. The only physical sign observed was a slight elevation of dulness in front. It was intended that I should see him on April 26th, but on April 23rd the abscess burst into the pleura during the night.

On the morning of the 24th the right side was dull all over, and the patient was very ill indeed.

In the afternoon I saw him and first aspirated two

pints of sticky material resembling pea soup, and then, without removing any rib, let out a further large quantity by an incision on the seventh interspace in the axilla. I did not think he could stand a more severe operation.

He died in a day or two, and a second abscess was found which was apparently about to burst into the peritoneum. The position of it was not stated.

Case 4 (*pulmonary abscess in anterior part of lung*).—The patient was a lady aged about 30, who had been in India, and who was seen with Sir Richard Douglas Powell. This is one of the very few cases of tropical abscess I have met with in women. The patient developed an abscess in the right lobe of the liver, which burst into the lung, and which I opened by means of a lateral incision in July, 1896.

The fever did not completely subside, and there were signs of right pleurisy, but exploration with the aspirator revealed nothing. The expectoration continued.

She left for the country with the wound unhealed, and by November, 1896, it was obvious that there was an abscess in the lung to the right of the sternum, about opposite the fifth rib. This was opened and drained. The expectoration stopped, and both wounds finally healed soundly.

The patient has remained well since.

Case 5 (*pulmonary abscess in base of lung behind*).—Dr. S—, aged 35, I.M.S., China and India. He had had dysentery and hepatitis in 1894, and was invalided home September, 1894. He had one rigor in October, 1894, and right pleurisy, lasting one month, followed by normal temperature for a fortnight. The abscess burst into the lung in December, 1894.

I saw him in January, 1895. The physical signs indicated a liver of normal size and a pulmonary abscess behind. The expectoration was about six ounces of blood-stained muco-pus *per diem*. The pulmonary abscess was opened January 31st, 1895. The expectoration stopped

thirty-six hours after operation. The abscess was completely healed in three weeks.

CASE 6 (*abscess in left lung*).—Lieut. ——, aged 28. Sent to me by Dr. John Anderson. He had been in India from 1892 to 1899. He had had no fever or dysentery. Hepatic symptoms began in May, 1899. An operation was performed, but no pus was found. Expectoration of blood and pus began July, 1899, after a fall. He was invalided home in November, 1899.

Left empyema was diagnosed. An operation was performed, resulting in the discovery of some clear fluid in the left pleura and the absence of an abscess beneath the left side of diaphragm.

On a subsequent occasion the right pleura and subdiaphragmatic region were explored and found to be healthy.

I first saw the patient March 17th, 1900. The physical signs pointed to an abscess in the base of the left lung, behind and inside, and below the angle of the scapula. There was no marked enlargement of the liver. There was copious expectoration of reddish-brown pus. The expectoration occasionally stopped, which always caused a rise of temperature.

I opened the abscess in the situation indicated by the physical signs, namely, higher up than the previous incision into the left pleura, removing a piece of the eighth rib. The abscess was in the lung ; it had thick walls and many prolongations. No communication was found with the liver.

His general health at once became quite good, but healing was, as might have been expected, slow, the wound not being completely closed till November, 1900.

CASE 7 (*extensive and fatal destruction of lung*).—The patient was a man aged 46. When first seen in June, 1897, by Dr. Hector Mackenzie he had been twenty-two years in India.

In 1890 he had malarial fever.

In 1892 he had typhoid fever and was afterwards invalided home. There was no history of dysentery.

In February, 1897, he was quite suddenly attacked with diarrhœa, and temporarily lost power over the sphincter ani. He had pain over the ribs on the right side at the same time. He went up to the hills for a time, and while there was very ill with fever every night and rigors. Then a purulent discharge from the rectum came on. He returned to Calcutta, and an abscess of the rectum was diagnosed; he was relieved by hot hip-baths, but was very ill, and lost flesh and strength.

The pain in the side continued, and in March, 1897, a severe dry cough came on. In April he began to expectorate pus stained with blood, and continued to do so more or less till the end of the case. He was sent to England. Cough, sweating, pyrexia, and depression of spirits continued on his voyage home.

When examined by us there were signs of enlargement of the liver upwards in front (level of third rib), but no increase of liver dulness downwards. The rectum appeared to be healthy.

On July 8th, 1897, a large abscess was opened anteriorly and laterally, and this was followed by a gradual improvement in health; but drainage was never satisfactory, and there were occasional attacks of increased cough and expectoration owing to retention of discharge.

On December 6th, 1897, the ramifications of the abscess, which were now found distinctly to involve the pleura, were very thoroughly opened up.

During the year 1898 there were periods of improvement and relapse. At one time it looked as if he would make a good recovery; but at the end of the year the tendency was gradually downhill, and his condition became more obviously septic.

On March 27th, 1899, though he was then very ill indeed, a further attempt at opening up the suppurating tracks was made. They were found to be very extensive, reaching up almost to the apex of the lung. The cavities

were bounded in part by the chest walls, in part by broken-down lung tissue. A great portion of the anterior aspect of the right lung had been destroyed by the suppurating process. He only survived this operation a few days. There was no post-mortem examination.

CASE 8 (*destruction of lung; fatal hæmoptysis*).—The patient was a man aged 26, a tea planter, born in India, educated in England, and who returned to India in 1886 and stayed there till 1893, and had had some attacks of fever.

He had dysentery in March, 1893, and was in hospital in Calcutta. The dysentery was not quite cured, and he was invalided home in November, 1893, in a very bad state. The dysentery stopped on the voyage, and on arrival home he was well except for a dry cough.

On December 13th he began to spit blood and matter.

On February 20th, 1894, he came under my care. He had lost three stones in weight, and had constant cough; copious chocolate-coloured expectoration; hectic temperature; diarrhœa with blood and mucus; pain in defæcation and difficulty in micturition. His appetite was good; he was given a milk diet. The physical signs indicated great enlargement of the liver and an abscess at base of right lung.

An operation was performed on February 21st. An abscess in the lung was opened behind and a finger passed through the diaphragm into the liver. Considerable improvement followed, but there were frequent hæmoptyses.

Fatal hæmoptysis occurred on April 24th, 1894.

Post-mortem.—The liver was found to be firmly adherent to diaphragm at the upper part, but there was no sign of an abscess. The right lung was firmly adherent to the diaphragm and to the chest wall up to the level of the eighth rib. A large irregular cavity occupied the lower and middle lobes, and there was a cavity as large as a Tangerine orange at the right apex. The liver and kidneys were amyloid.

CASE 9 (*abscesses in both lungs and in brain*).—The patient, a man aged 43, had lived in India for ten years. He had dysentery soon after his arrival, from which he completely recovered. Sixteen months before his admission to hospital he had had symptoms of liver abscess. Five months later the abscess burst into the lung, and four months after that he was operated upon, portions of the seventh rib in the axilla and of the eighth and ninth ribs behind having been excised. This had not relieved the patient; it is uncertain if pus was found.

On admission there were no signs of enlargement of liver. The physical signs pointed to the existence of an abscess at the base of the right lung. Exploration, however, failed to detect the presence of pus.

Three days after operation symptoms of cerebral abscess commenced. Eight days later an abscess was opened in the right occipital lobe, the only localising sign being tenderness on percussion. On the same occasion an abscess in the base of the right lung was opened through the old scar in the axilla, more space being gained by removal of more portions of ribs.

No improvement followed, and another trephine opening was made in the hope of finding a second abscess in the brain, but none was discovered, and the patient died twenty days after the first operation.

At the post-mortem examination the lower lobe of the right lung was fibrotic and riddled with cavities. The right pleural cavity was obliterated at the base. The left lung contained a cavity as large as an orange opposite the fifth, sixth, and seventh ribs, and the pleura in this situation was obliterated. The brain contained a large abscess in the right occipital lobe, but there were no other collections of pus. There was little or nothing to indicate the old abscess in the liver.

CASE 10 (*abscesses in the lung and the brain*).—The patient was a man aged 33, a merchant, and was seen with Dr. Crombie January 9th, 1901. He had been in Calcutta seven years. He had had very little fever.

In May, 1897, he had dysentery and congestion of liver, which lasted till end of 1897. He was invalided home on account of sprue in January, 1898, and remained till September, 1898. Liver symptoms began in September, 1899. Pus was removed by aspiration in October, 1899. Aspiration was again performed in November, 1899, but nothing was found.

In January, 1900, cough began, and had continued ever since. He had had intervals from fever and cough lasting a week, but not longer. These were followed by a rise of temperature and expectoration. He had lost some flesh, but not much.

In June, 1901, he had signs of a pulmonary abscess at the right base, which was opened by a posterior incision opposite the eighth rib. There was a gradual improvement, though drainage was never perfect, because the cavity had many branches, and a considerable amount of bright blood and pus was discharged from the wound and expectorated as well. He improved so much, however, that he was sent to Christchurch, Hants, under the care of Dr. Leslie Burnett.

In September symptoms of cerebral abscess manifested themselves, and before long symptoms pointing to affection of the left motor area appeared.

Dr. Burnett explored this region, but found no pus. A few days later, at my suggestion, he explored further back and found a considerable abscess in the left occipital lobe. There was some improvement, but the patient died on August 3rd.

Post-mortem.—The right lobe of the liver was found to be adherent to the diaphragm, but contained no pus (cf. Case 8); the abscess in the lung was extensive, and branched out into numerous pockets; part of its wall was formed by the ribs (cf. Case 7). A considerable cavity existed just beyond the end of the drainage-tube. The abscess in the occipital lobe was not empty, an accumulation existing beyond the end of the drainage-tube.

DISCUSSION.

Dr. PATRICK MANSON, referring to the operation he had devised, said it was meant for an ordinary surgeon away from all assistance, as in tropical countries. He asked why it was that abscess of the liver extended upwards and did not lead to depression of the liver, as a pleural effusion did. Had Mr. Godlee ever seen the escape of hepatic pus into the peritoneal cavity produce serious consequences? for he himself had not.

Dr. A. CROMBIE alluded to the time when it was the invariable custom to empty liver abscesses by the aspirator, and said that small and recent abscesses were still successfully treated by this method. Even in the case of larger abscesses, if acute and recent, recovery had followed repeated aspiration, in one case after so often as fourteen times. The operation itself produced no constitutional disturbance, and Dr. Lawrie had described cases in which a single aspiration had effected a cure, and other similar cases he had himself met with. During a thirty years' experience of the treatment of liver abscess, in only one case had the liver been stitched to the parietes, and never once had he seen any ill result from the escape of pus into the abdominal cavity after direct incision. In the tropics, at any rate, such pus was aseptic, and it often probably escaped into the peritoneal cavity. Reference was made to the occurrence of severe hæmorrhage during operation, which, however, was always controlled easily by pressure.

Mr. CANTLIE was a thorough upholder of Dr. Manson's method of aspiration for the treatment of liver abscess. It was a very appropriate operation for surgeons undertaking the operation single-handed, and had the advantage in being, as it were, a natural sequence of the exploratory puncture. In the absence of suitable nursing and other assistance it was a great boon. Hæmorrhage with Dr. Manson's operation could hardly occur; cutting the liver with the knife was the chief cause of hæmorrhage. A metal drainage-tube was not advisable in liver abscess; an india-rubber tube which would be compressed by inspiration and expand again during expiration was good in preventing escape of pus by the side of the tube. He had only had two fatal cases among the many in which he had employed this method, and these were the first he had operated on.

Dr. WILLIAM GABRIEL ROCKWOOD, during twenty-five years, had had over a hundred cases of operation for abscess of the liver. In the earlier he had aspirated; subsequently he had only operated by incisions between ribs. The latter method in acute cases proved of no avail; now he was accustomed to

excise a portion of the rib. In chronic afebrile cases aspiration might suffice; in acute cases, with thick pus and much *débris*, nothing short of excision of a portion of rib would do good. Lateral incision even might not be sufficient, and incision in the middle line might be required.

Mr. GODLEE, in reply, thought that the liver sometimes enlarged upwards because adhesions had been formed between the lower part of it and the abdominal wall. He could not say that he had ever seen any serious harm from the escape of pus into the peritoneal cavity, but he referred to one case which was followed by severe pain apparently indicating general peritonitis, from which, however, the patient recovered. Bursting of the abscess into the peritoneum was, of course, disastrous. Aspiration in liver abscess was much on a level with that for empyema; a certain number recovered, but probably the majority came to operation sooner or later, and aspiration merely meant delay. At the time of operation bleeding was seldom serious, and was almost certainly stopped by pressure, but the later hæmorrhage could not thus be arrested.

SOME GENERAL AND ETIOLOGICAL DETAILS

CONCERNING

LEPROSY IN THE SUDAN

BY

T. J. TONKIN

LATE MEDICAL OFFICER TO THE HAUSA ASSOCIATION'S CENTRAL SUDAN EXPEDITION, 1893-4-5.

Received October 29th, 1901—Read May 27th, 1902.

THE object of the paper which I have the honour to read to you this evening is to bring before your notice an account of some of the ætiological factors probably concerned in the maintenance of leprosy in the Sudan. When the opportunity, which I am now enjoying, first presented itself to me, my idea was to make the scope of the paper wider, but considerations of time rendered that impossible. Before I enter on the subject proper, however, it is perhaps desirable that I should tell you something about the leper field from which I have drawn my results. I will begin by defining the term Sudan. Our Imperial losses and gains in the country immediately around Khartoum have tended to concentrate our national attention on that particular scrap of country to such an extent, that it is probable that many people are ignorant that any other Sudan than that to the south of Egypt exists. The Egyptian Sudan, however, is only a small part of a great whole. The Sudan proper is an immense reach of country stretching across the continent of Africa

at its widest part. The seaboard of the Atlantic from Cape Verd to the mouth of the River Roquelle is its western boundary; its boundary on the east is the valley of the Nile. Its northern edge from Egypt to the mountains of Senegambia coincides with the southern fringe of the Sahara, while to the south its limit may be placed at a line drawn from the mouth of the Roquelle,—that is of course Freetown, Sierra Leone,—to the outfall into the Nile of the Bahar Eg-Gazal.

The Sudanese leper field is on a scale proportionate to the region that contains it. It lies in the centre and to the west of the centre of the Sudan. I would describe it as a belt, say five hundred miles wide, coming out of the eastward probably from beyond Darfur, embracing Lake Chad, stretching across our Northern Nigeria, holding its own over and beyond the waters of the Middle Niger, and finally losing itself as it approaches the upper waters of that river away to the south-west of Timbuctoo. The whole of this area is very strongly affected by the disease. The Northern Nigerian regions in which I travelled are especially unfortunate in this particular. They are occupied by the sufferers from leprosy as by a vast standing army. Everywhere and on all sides the familiar uniform is met. Large towns are heavily garrisoned; the smaller have detachments and companies proportionate to their size. During parts of my journey I do not remember touching at any village so small that it had not some lepers. In places I found settlements of considerable size, apparently specially designed for them, at any rate almost entirely populated by them. In the large towns lepers may be seen in almost every street and square. In some of the streets they sit in rows and companies, in others, and near the borders of the market places and on the open spaces by the gates they collect in gangs and troops.

Kano, the principal commercial city of Northern Nigeria, is a veritable hive of lepers. In that city (of the size and importance of which something may be inferred

from the fact that it is protected by fifteen miles of earthworks, has fifteen gates, and a daily market on which from twenty to thirty thousand people may often be seen at once) hundreds of lepers live together in various houses or collections of houses. There are many such colonies in Kano. In them young and old, male and female, the well-nigh healthy and the fearfully diseased, the vigorous and the dying, promiscuously herd. With regard to the surroundings of these communities, insanitary as a descriptive term would be feebleness itself. The apathy that gradually creeps over the leper as the disease closes its grip upon him, makes the inhabitants of such places less careful about personal cleanliness and the cleanliness of their dwellings than the average native is. The result is easily evident. In the dark tomb-like huts which the heat and glare of the sun, and the persistent attentions of the fly tribe, render necessary in these parts of the Sudan, the smell emanating from the neglected ulcers of scores of leprous occupants hangs like an oily fœtid fog upon the air. Inside and outside, foodstuffs and other matters in decaying conditions are allowed to accumulate. The usual etiquette of the Hausa household is suspended, and it is among such surroundings as these that the lepers, representing among themselves every age and every degree and variety of their disorder, live and die.

In Northern Nigeria familiarity with leprosy is a social characteristic. The disease is so common that in spite of the repulsive appearance of the sufferers, the general public have, as far as I could make out, no active objection to it. They are accustomed to it, and regard it as one of the stable things of the world, and the chance of catching it as one of the ills to which human flesh is inevitably heir. They do nothing so far as I know to limit the chance of contagion. Lepers are permitted to mingle freely with the healthy population, engage in business, and marry whom they will. When they live in communities it is not because they are forced to do so, but

rather because community of interest acting through long years has drawn them together. Lepers are not subject to any municipal or social disabilities on account of their disease. I have frequently seen them tailoring, selling second-hand clothes, and presiding at provision stalls. Nor did I notice any repugnance on the part of the people to the idea of having their national food (which is thick and porridge-like) served out by a pair of scaly, mutilated, and often ulcerated hands; time and old custom have hardened them to it. The native of Northern Nigeria regards a man whose limbs have been reduced to a mere fraction of their normal proportions, and whose skin is broken, seamed, and puckered by leprosy, in much the same light as we should regard a person with a club foot or a wooden leg, and the idea of walking twenty yards further for the privilege of buying a meal from a healthy salesman or woman, would, if it were ever suggested to the native mind, be derided as unnecessary and foolish. This is the state of things in Northern Nigeria, a region extending over some five hundred thousand square miles, and I have every reason to believe that it is only a slightly accentuated example of the similar conditions obtaining elsewhere in the Sudan.

Having, then, given you some sort of impressionist idea of the locality and extent of the Sudanese leper field, I will pass on to the ætiological portion of my paper.

There is, I take it, at this time of day, little need to insist on the improbability of the transmission from parent to offspring of a disease which depends for its causation upon the action of a specific poison. In a paper of this kind it is quite unnecessary for me to bring forward evidence either from the Sudan or elsewhere bearing on this point. It is generally admitted that in no sense of the term can leprosy be regarded as a heritable disease, and I am not taking any liberty, therefore, in starting with the assumption that in every case the disorder is the result of a fresh individual infection by the specific bacillus.

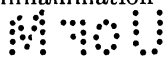
The first question that naturally arises in connection with the spread of leprosy relates to the working sources of the bacillus. There is a peculiar appropriateness in dealing with this question in relation to West Africa. Evidence that bears strongly on this point is closely interwoven with West African history, and with the great enforced migration of West African natives that was determined by the now extinct trans-Atlantic slave trade from the Guinea Coast to certain parts of America. At the time of the discovery of America that continent was free from leprosy, and it continued to be so until the middle of the sixteenth century. Then, with the ever-increasing demand for. labour set up by the cotton and sugar plantations, came the slave traffic, and, by means of that traffic, wide areas of the Western World were flooded with Africans, drawn from the very infected region that I have just been describing to you.

From the Sudanese leper field these people took the disease with them across the ocean, with the result that America was infected, an infection that is responsible for the existence of the disease in that continent to-day. The American leper field is the daughter of the Sudanese, and the link between them was evidently individual, man-to-man infection. Such facts as these, even if they were unsupported, which they are not, would appear to be inconsistent with the supposition of any other regular source of the bacillus than the diseased tissues of previously infected individuals.

If it be, then, granted that in every case leprosy is the result of a fresh infection of the individual by the bacillus, and admitted that the immediate source of the bacillus is usually the damaged tissues of previously affected individuals, the next problem that presents itself for solution is the determination of the general mode by which transference of the bacillus from previously affected to fresh subjects is accomplished. I do not use the word infection in this instance, because, in the case of leprosy, it is probable that more than mere transference of the bacillus

is necessary to the initiation of the disease. As far as the actual first transference, however, is concerned, it seems likely that it is achieved by a process of mediate contagion. The bacilli are transferred from their source, disintegrating leprous surfaces, to surfaces previously healthy by the agency of various things, among which personal clothing and bedding occupy the chief place. Penetration of these fresh surfaces is, however, still necessary even to the possibility of infection, and this is probably effected by the help of other influences, which act as introducing media by making breaches in the protecting epidermal layers and allowing the bacillus to reach the deeper and more readily damageable structures of the skin. These introducing agencies are of various natures, atmospherical, frictional, due to the attacks of insects, and variation in the particular influence at work, and, more or less, the particular surface affected, occur with fair constancy among the various leper fields of the world.

It can hardly have escaped the notice of people interested in leprosy that the feature known as leontiasis is more marked and much more common in extreme northern and southern leper fields than in those situated in warmer regions. The feature referred to is of frequent occurrence among the Icelandic and Norse lepers. In the Barbary States I can say from personal experience that it is very much rarer, while in the Sudanese area, which I have had particular opportunities for observing, it is most infrequent. The explanation of this relative variation in the occurrence of what has come to be regarded as a classical feature of the disease is probably to be found in a sort of general rule, in response to which the first advance of certain microbic skin affections, and among them the initial lesions of leprosy, tend to fall with major severity upon surfaces of the body most exposed to wear and tear. The frequent appearance of the common boil at the collar line on the neck, of erysipelatous inflammation at the junction of mucous and cutaneous,



ulcerated and sound surfaces, of seborrhœa under the irritative pressure of the hat-band, and of a whole collection of parasitic disorders in the much-scratched region of the hairy scalp, are instances of this. The leontiasis of leprosy is a further case in point. The face of the Scandinavian, exposed as it often is to violent alternations of temperature, from the warmth of the house to the biting frost and scathing wind of the outer air, is prone to chap and crack, and to have thereby its more sensitive layers exposed by the damage to the epidermis, and their resistance to morbid influences lowered by the consequent congestion of the blood-vessels. The habits of the people in Norway with regard to bedding are, I understand, gregarious, and have not, I believe, until recently been affected by the condition of any member of the family that may have happened to be suffering from the endemic disease. Bacilli, freed from the surfaces of a suppurating leper and lying ready for mischief in the bed, would, at any rate, find some difficulty in making an impression on the smooth oily surfaces of the parts of the body that are, in those countries, constantly protected from the air. With the skin of the face, however, matters would be different. There would be little hindrance to their effecting an entrance through its cracked weather-damaged surface, and, other things being favourable, little difficulty in increasing the already irritated and thickened condition of the cutis and of the subjacent structures up to the intense visible specific disturbance referred to.

The native of warmer climates is not exposed to this particular localising influence, hence the rarity of the resulting feature among them. But they are subject to others from which the Northerner is exempt. One of these, quite as characteristic in its way as leontiasis, though not so readily apparent, is the thickening of the outer borders of the feet in barefooted races. The feet of the tropical native who pads unprotected over sand and rock, through mud and water, are especially prone to fall early under the

influence of the bacillus. The inner borders of the feet are held safe by the protecting influence of the plantar arch, but the outer are in contact with the ground, and the skin over them is thickened and cracked by constant exposure to alternating conditions of wet and dryness, and by frequent small violences, and becomes thereby reduced to a condition parallel to that described as affecting the face of the Norseman. Long nights, during which the injured outer surfaces are scuffled up and down over harsh sleeping mats, supply, should the mats have been previously infected by the discharges of a suppurating leper, an opportunity by which the bacilli may gain access not only to the most intimate structures of the skin itself, but also to the areolar tissue that lies below. The dense infiltration of these outer borders of the feet, the consequent interference with adjacent plantar nerve supplies, and the resulting injury to or loss of the lesser toes which so frequently follows among barefooted races, at least in Africa, I regard as the analogue of the leontiasis of the boot-wearing Northerner; and both I look upon as instances in which free germs from infected garments or bedding obtain direct entrance into and through a damaged and chronically irritated skin surface, the pre-existent damage and irritation being answerable for the marked neoplastic changes that equally in both places ensue. That in the Sudan these and similar processes are the ordinary modes of leprous infection is highly probable. The situations in which the other early lesions of the disease first show themselves lend colour to this view. The early lesions first show themselves on the prominences of the body, on the cheek-bones, the temporal ridges, the outer surfaces of the extremities, the scapular region, the buttocks. In all the situations mentioned the skin is at a disadvantage with regard to wear and tear. Lying about, as the average Sudanese native does, on the hard ground, or on a mat almost as hard, these parts are in constant, and to a certain extent violent contact with his garments. To those who know the Sudanese native well this is a circumstance full of

significance, and one which it is difficult not to associate with the preference evinced by early leprous lesions for these localities. The average Sudani is not a cleanly person. The clothes he wears, the mats, whether of skin or grass, on which he lies, the loose covering with which he keeps off the chill of the early hours of the morning, are never washed, and are used indiscriminately by himself and friends. It is uncustomary for a Hausa to wash anything that belongs to him, or to have it washed. Wealthy men buy their robes, which are made of cotton textiles, new, and when they are dirty they sell them to less fortunate people than themselves, people who cannot afford to be so nice in these particulars; or they give them away. It is considered an honour to be the recipient of a king's raiment. But, however they may obtain them, the people who get these second-hand garments wear them as long as they think proper; and then, when they feel they can afford it, or when the clothes become too dirty for a person in their particular class of life to wear any longer, they pass them on to some one lower in the social scale. In this way a regular circulation in clothes is established, the rich selling or giving to the middle classes, the middle classes to the poorer, and the poor borrowing, begging, stealing, selling, or lending among themselves. A single robe, during its life as a robe, may have in this way from five to fifty different owners. As long as half a dozen shreds of it continue to cling to the neck-band, so long does it continue to do its duty. It responds during the course of its existence to the influence of a kind of gravitation, falling layer by layer through the various strata of society, till, from gracing, it may be, in its crisp new early days, the shoulders of a prince, it may come at last in its thread-bareness to be the only covering of the poor man's slave, or later still its tattered remnants may be found to be conveniently lending themselves to the exhibition of the alms-earning ulcers of the wayside beggar. But with all its varying fortunes it will probably never—and this is where the peculiar danger comes in—have been washed.

In accordance with this unfortunate custom, robes stiff with leprous exudation often pass warm from the bodies of lepers to those of previously unaffected persons. Let us take an example. A really well-to-do man buys his robes new. If he wears white he will on an average become the possessor of two or three new garments every month. When he buys a robe it is fresh from the hands of the maker. He puts it on, wears it till it is dirty, then sells it. He thus puts into circulation from twenty to forty robes a year. Putting aside all other sources of contamination, just imagine the possibilities of the case if that man happens to be a suppurating leper. Another man buys one of those robes, wears it by day and rolls about by night with nothing between him and the hardness of the ground but that leprous garment. The patches of skin covering the prominences of his body bear the brunt of contact with the garment. Their superficial irregularities, the follicles and the like, become stocked with bacilli which are rubbed into them from the surfaces of the infected garment. Then an abrasion of the epidermis, which may be due to accidental violence, to coincident disease, or, as is probably more often the case, to the attacks of mosquitoes, fleas, or body lice, and consequent scratching, occurs, and the horny protecting layers of the skin are injured. Through the trifling wounds of the nature suggested, the bacilli make their way from the outer works of the skin to the innermost recesses of its structure, and an invasion is complete. Whether the invaders will make good their local foothold or be destroyed, or whether, should they succeed in making their local footing good, they will then be held powerless for evil or succeed in making further advances against the defences of the system they are invading, will depend upon the powers of resistance to which they find themselves opposed, and probably to some extent upon the numbers and virulence of the invading bands of bacilli. If the system be vigorous it is probable that no evil result will accrue; but if the reverse be the case, if the system attacked be under the ban of

the influences that determine the occurrence of the predisposition to the disease, if its powers be, moreover, depressed by ill-health, privation, over-strain, or general reverses, the bacilli may make good their foothold, and an attack of the disease, more or less severe according to individual circumstances, may result.

But it is with the predisposition to the disease that we get into the actual working habits of the leprosy bacillus. It is well known that individuals of every race may remain exposed during long periods of time to all the possibilities just sketched, and may even certainly incur the risk of constant and close association with lepers without affording any evidence of having contracted the disease. This state of affairs is explained on the supposition that a certain condition of contributory lowering of vitality is necessary on the part of tissues attacked before the bacillus can effect its characteristic results. This condition of lowered vitality is called a state of predisposition, but what that condition may actually be, and how exactly it is acquired, are still questions that are being debated.

It has been suggested that the tendency to leprosy is natural to certain races. This can hardly be the case, for if it were so each member of such races would possess the innate characteristic in common with the rest. It does not appear, however, to be of such general occurrence. Healthy persons not only may, but often do live for indefinite periods of time on terms of the closest intimacy with lepers of their own race, tribe, and even family, without developing the disorder. This state of things would appear to be incompatible with the existence of a natural susceptibility, and we are probably justified, when considering the factors that have to do with the causation of leprosy in leaving it out of the question. As susceptibilities must be either natural or acquired, this leaves us with the acquired group on our hands. Now, it being admitted that a parent is capable of transmitting to his offspring a disposition obtained by himself, it follows that susceptibilities falling within this class must be either acquired by an

individual personally, or received by him as an inheritance from the parent or more remote ancestor with whom they originated. In other words, they may be either acquired personally or inherited. But this classification is not the one best suited to present requirements, the question of first importance with regard to leprosy being not when, but how the predisposition is acquired. It will be, therefore, more to the point to take acquired susceptibilities in bulk, and divide them with reference to their probable causes into two classes: (1) those that can reasonably be ascribed to the leprosy of a parent or more remote ancestor, namely, specific tendencies; and (2) those that cannot.

When this is done we shall find that we have to some extent affected the question of time also, for it is evident that all susceptibilities due to leprosy must be inherited, while those due to other causes may be either inherited or personally acquired, or both. This classification is also one that lends itself readily to further simplification, for the first class may be shown to be of doubtful occurrence. The same evidence that is advanced against the theory of heredity as applied to the disease itself may with equal relevance be urged against the theory of a specific origin of the tendency. Of my own results, I found among the lepers I examined in the Sudan that only about one out of every ten was born of tainted ancestry,—that is, in only about one case out of every ten did leprosy occur among the more immediate forebears of the patient. Moreover, of the children of lepers it appeared that less than ten per cent. ultimately developed the disorder. It is manifestly impossible that the condition that predisposed the leper to his disease should have had its source in ancestral leprosy when the individual's progenitors as far back as could be ascertained have been free from the disease; so as only one leper in every ten is born of tainted parentage, such a source of the tendency could only be possible in a similar proportion of cases. Moreover, if the leprosy of a parent or ancestor were a regular source of the tendency in a descendant, the resulting tendency should be at its strong-

est in the immediate descendants, namely, the children of lepers; yet even of such children as are born after the inception of the disease in their parents, and exposed to contagion during their infancy, childhood, and often adolescent and adult years as well, less than 10 per cent. ultimately develop the disorder. If these data are correct, a specific source of the tendency is only possible in one case out of every ten; and out of every ten cases in which it might occur, and if it did occur might reasonably be expected to be at its strongest, in less than one is there evidence of the existence of any predisposition at all. Out of 220 of my own cases in which I went closely into family history, in only eighteen could the tendency which we suppose to be necessary to the development of the disorder have possibly had its source in the disease of a forebear, since the immediate ancestors of the rest for two generations at least had been free from taint. If the diffusion of leprosy depended to any extent upon the transmission of a tendency of specific origin it would not be unreasonable to expect a larger proportion than 18 possibilities out of 220.

It is fairly clear that the bulk of the persons who suffer from leprosy do not owe their liability to the pre-existent disease of an ancestor. What, then, is the source of the tendency? In reply to this question, I should say that it is probable it may be more accurately traced to adverse circumstances of a general nature; but, among these, I think a leading place should be accorded to defective diet. I do not refer to any particular improper foodstuff, but to a definite dietetic defect.

It appears to me that there is one great common factor pervading the leper fields of the world. That common factor is a diet which, when considered chemically, will be found deficient in one specific direction. In all the countries of the world without exception, in which leprosy has acquired anything like a footing, the national diet is wanting in nitrogenous elements. In India and China rice is the staple foodstuff of the masses. In

Scandinavia and in Iceland the exigencies of climate call for the consumption of large quantities of fat, to the displacement of other necessary aliments. In the West Indian and Pacific islands the bulk of the population live largely on vegetable food, and in the Sudan the existence of millions rests on a porridge-like preparation of dhurra or a solid substratum of yam.

It is probably not too much to say that eighty per cent. of the inhabitants of the endemic area of the Western Sudan subsist on a vegetable diet of the straitest sort, and I believe that this circumstance definitely affects their resistance to leprosy.

It is a matter of common knowledge how excessively prone to ulcerative changes are the peripheral tissues of the tropical native of the poorer classes. That the rest of his tissues are in the same tumble-down condition is doubtless the case, but we can see his skin, and the readiness with which ulcers follow the slightest scratch, or appear on the cornea without any apparent encouragement at all, is so well marked as to be immediately evident even to the most casual observer.

This state of things is probably due to the specific defect in his diet. It is not always that the native does not get enough food, but that he does not get the right kind of food. He needs a certain definite amount of nitrogenous nourishment for the effective discharge of the functions of his body, and for the maintenance, at a normal standard, of the vitality of that body's tissues, and the getting of that certain definite amount he fails to achieve. In the effort to get it, moreover, out of a national array of foodstuffs that contain an overwhelming percentage of carbohydrate or hydrocarbon material, as the case may be, and very little more than a mere trace of the desired element, he still further adds to his embarrassments. In the vain attempt to get enough nitrogen for his needs he charges himself with very bulky meals, taxing his digestive organs to their utmost limit. And, in the end, he probably does not succeed in getting the necessary amount,

because the percentage of nitrogenous material in his foodstuffs is so low that he has eaten all he can hold long before he has got the quantity commensurate to his needs. All he does by his efforts is to further increase his difficulties by encumbering his economy with a large amount of superfluous, and therefore deleterious carbon. Enfeebled as the resistive powers of the tissues are already by lack of nitrogen, it is not difficult to imagine that this overburden of carbon, littering up blood, lymph, and tissue elements, may have the effect of still further reducing their power of resisting morbid changes, and, as a consequence, rendering the individual yet more liable to the endemic disease.

But I do not claim that this dietetic factor is by itself sufficiently potent to lay a man of ordinary powers open to the attack of the leprosy bacillus. I only suggest that it is a factor common to all the leper fields of the world, and that it diminishes to such an extent the resistance naturally offered by the tissues of the normal body to disease, that that resistance is, on the supervention of (in some cases even slight) further adversity, readily disposed of altogether. The additional adverse influence often shows up very clearly. In the Sudan, among the bulk of the people time is measured and dates are defined by events, and I have been struck by the frequency with which the year or so immediately succeeding some untoward event—a war, a famine, or a pestilence—have been named by patients as the time of the onset of their disease. In many cases business reverses or domestic losses involving sudden poverty or grief have appeared to pave the way for the malady. In women the first signs of its invasion not infrequently appear during lactation. The bodily prostration consequent upon the dangers and privations attending pilgrimages, and other long journeys over wild and savage countries, is frequently taken advantage of by the disease, and prisoners of war often develop it within a reasonably short space of time from their introduction to a life of slavery.

I look upon these last-named adverse circumstances, however, only in the light of last straws, and they may take a hundred different forms according to the nationality, habits, age, or sex of the persons affected; they are, in fact, mere accidents. The rank of common and constant factor predisposing to the disease can in my opinion only be accorded to one thing, and that is an absence from the dietaries of the affected races of the amount of nitrogen necessary for their needs.

In the near future I hope to have the opportunity of working up this defective diet idea on fuller lines, and in the light of more exact information. For the moment I must content myself by hoping that the a-nitrogenous theory which I have advanced this evening may prove a possible basis on which to account for the occurrence of what is emphatically the most important factor that has to do with the causation of leprosy.

For discussion see end of Mr. Hutchinson's paper.

LEPROSY IN NATAL AND CAPE COLONY

BY

JONATHAN HUTCHINSON

Received 4th April, 1902—Read 27th May, 1902

HAVING recently returned from a short tour in South Africa, undertaken with the object of inquiring as to the causes of the prevalence of leprosy there, I am desirous to submit to the criticism of the Royal Medical and Chirurgical Society the conclusions which have been arrived at. At the outset I may admit that although I went, I trust, with an open mind as regards the reception of evidence, it was not without strong prepossessions. For now nearly half a century I have felt convinced that the origin of leprosy must be in some way connected with the use of fish as food. To this conclusion the general facts as regards the distribution and prevalence of the disease, its decline in some regions and its persistence or even increase in others, seemed conclusively to point. As years have gone on and evidence has accumulated, this conviction gained strength, and also assumed better definition. At the same time I have, in common with many other observers, been inclined to discredit the opinions of those who hold that contagion is the principal, if not the sole cause of the spread of the malady.

Such being my convictions, a study of the facts offered by South African observers as to the conditions under which the disease had developed and was spreading in their regions, led me to believe that a quite exceptional

opportunity was afforded for an attempt to solve problems of great importance. The disease in Cape Colony had been only recently introduced, and was as yet only very sparingly prevalent; whilst in Natal and some other parts its first occurrence was of yet more recent date,[1] and its dissemination yet more scanty. In countries where it has long been endemic and prevails extensively, the possibilities as regards hereditary transmission and contagion become so inextricably mixed up with those as to fish-food, that it is almost impossible to feel confidence in any conclusions which may be suggested. It occurred to me that in South Africa, with a quite recent development of the disease in virgin populations, representing very different races, and scattered sparingly over immense tracts of country, the facts might be more easy to deal with. I was further encouraged by the knowledge that these facts had already received the attention of the Colonial Governments, at whose request the district medical officers—a body of men second to none in intelligence and capacity for such observation—had made local inquiries, the results of which would be available. A further stimulus was added by statements which came from the Natal colony, to the effect that there the disease occurred to those who never, under any circumstances, eat any kind of fish. In addition to examining the facts as to leprosy itself, it seemed desirable to obtain detailed knowledge as to the extent to which fish is employed as food in the different regions of South Africa, and the conditions under which it is supplied. On these and other subjects I had previously sought information by correspondence, and with only very partial results.

It may be convenient at the outset to say a few words as to whether or not leprosy is a new disease in the districts in question. In the more northerly parts of Africa;

[1] In Captain Lucas's report of evidence before the Commission in Natal, 1886, he states that he made inquiries of Mr. Osborne (residing in the Zululand Reserve), who made investigations and could not find that leprosy was known or had ever been heard of in the Zulu country.

about the Zambesi, the great Lakes, Lake Chad, and on both the west and east coasts there is no doubt that it has long prevailed as an indigenous disease. As regards South Africa the facts are, however, in dispute. The dispute concerns the Hottentot (or Gariepine) races only ; for all admit that in the eastern districts amongst the Bantu tribes (Kaffirs and allied races) it was unknown. The evidence as to the Hottentots is almost none, and it is quite certain that if they knew the disease before the Dutch occupation it was to an exceedingly small extent. No Dutch record of such disease occurs, and the Dutch settlers were well familiar with it in other colonies. The first record of leprosy in South Africa was in 1756, when three Dutch persons living on a farm at Stellenbosch, near to Cape Town, were found to be its subjects. A Government inquiry was made, the records of which are extant, and not a hint is given that the disease was known amongst the Hottentots, who at that date were engaged in large numbers as slaves on the farms. During the next fifty years the Government records are silent as to the disease, but at the end of that time disquietude was manifested in the Cape Town district on account of its gradual increase. Two or more different contentions may be sustained as to the mode of its introduction into, or of its origin in, the Colony. It is undoubted that the Dutch had brought over detachments of Malays who were to catch and cure fish in Table Bay and at other places on the coast, and it is certain that the farmers were at that period feeding their slave-labourers on rice and salt fish. The Dutch are, as a race, fond of salt fish, and it may be plausibly suggested that the first victims had developed their malady *de novo* from using this food, and that they were but the first drops of a shower which was about to fall over the whole district. On the other hand, it may be suggested that they obtained the disease by direct personal contagion either from their slaves or from some Malay or other immigrant, who brought it from the East. It may be remarked in passing

that not the slightest suggestion of either of these modes of introduction occurs in the Government records. If it were granted that the Hottentots had the disease, the question—unanswerable, I submit, by any contagionist—remains, "Why had it not spread among them?" Their conditions of life were such as to pre-eminently favour the spreading of a contagious malady, yet it is admitted on all hands that it did not become common among them until they came under the influence of Dutch masters. Excepting in the introduction of some new article of food, no change in their habits can be mentioned which was likely to conduce to the spread of any specific disease. It appears to my mind, therefore, that the contagionist would be wise to abandon the suggestion that the Hottentots had the disease at all, for it would prove too much. There is no doubt, however, that eventually the Hottentots and their bastard descendants were the chief sufferers from it. They are so to the present day. Nor is there any doubt that a certain number of Kaffirs who have acquired leprosy attribute their disease to association with Hottentots. This suggestion is, however, as we shall see presently, capable of a quite different explanation, and may be held to prove nothing more than that the person making it has been into Cape Colony, where Hottentots abound.

It may perhaps not be considered inappropriate to interpolate here a few words as to the use of the words Hottentot and Kaffir, and as to the present distribution of races in South Africa. It was of course with Hottentots only that the early settlers came into contact. They inhabited all the western and south-western part of South Africa, and it was only at a later period that white men came into collision and intercourse with a totally different race consisting of many various tribes now known to be of Bantu stock. It may be convenient in this paper to speak of these Bantu tribes as "Kaffirs." As regards the distribution of the two races, it may be understood that Hottentot tribes occupied the western half of South Africa, and Bantus or Kaffirs the eastern. Both were pastoral and

relied chiefly upon their flocks and herds for food, but the Kaffirs were also to some extent cultivators of the soil. Neither the one nor the other were fishermen, nor did they specially frequent the sea-coast, but it is possible that, on occasion, those who did so ate molluscs and other easily obtainable products of the water. It is certain, however, that they were not addicted to fishing, and that they did not attempt to salt or cure fish. The Hottentots had no prejudice against fish, and appear to have taken to it freely when their Dutch masters placed it within their reach. The Kaffirs, on the other hand, had, almost universally, a strong prejudice against fish, so strong that many authorities state that they would on no account touch it. I shall have to deal with the Kaffirs when I come to consider the introduction of leprosy into Natal, and its prevalence there and in the districts which used to be known as Caffraria. For the present we are concerned only with the Hottentots, for it was amongst them that leprosy first spread, and to whom for nearly a century it was probably almost wholly confined. In former times there was constant feud between Hottentot and Kaffir. The races did not mix nor come into any sort of social contact. The advent of Europeans has largely modified the state of society as we find it at present. The pure Hottentot has been supplanted by a hybrid race of mixed Dutch extraction now known as "Cape boys," and race-antipathies have to a considerable extent disappeared. It is even said that in some parts Hottentots and Kaffirs have intermarried and become the parents of a mixed race. As a natural result of this, aided by the introduction of Christian teaching, the Kaffir prejudice to fish as food has become modified, and many Kaffirs will now catch and eat fresh fish, and a still larger number will eat it freely in the altered condition in which it is presented after being salted or dried. A general observation of much importance to our present inquiry is that both Hottentots and Kaffirs are very prone to wander about the country. The labour market over the whole of Cape Colony is to a large extent

supplied by Kaffirs who have left their native hills in the hope of earning money to buy wives and cattle. They do not migrate with intent to settle, but purpose to return to their kraals as soon as their object is attained. In Tembuland I was assured that there was scarcely an adult native who had not done his wanderjahre, visiting Cape Town, Grahamstown, Kimberley, or Johannesburg. Nor was this willingness to wander confined, my informants stated, to men. Many young women had, I was assured, lived for a time as servants in the large towns, and afterwards returned to their homes to marry and settle.[1]

The first cases of leprosy observed in Cape Colony were, as already stated, in Dutch farmers. This was in 1756. The place was Stellenbosch, a small town not twenty miles from Cape Town itself, now the Nuremberg of South Africa, as containing the oldest and best preserved relics of the original settlers. No further reference to leprosy occurs until 1817, when the disease had so much increased that a leper home, under the care of Moravian missionaries, was established. Its site was a valley in the mountains near to the now fashionable watering-place of Caledon, and not far from Stellenbosch. Hemel en Aarde received lepers for twenty-eight years, and during that period had a total of 400 inmates. It was visited by a very intelligent traveller, Mr. James Backhouse, of York, in 1835, who records that he found it with

[1] A source of many errors in our inferences as to the incidence of leprosy in different places, is forgetfulness of the fact that the leper may have acquired the disease in some place at a distance from where he is found. The incubation period may be long, as long as a dozen years in some instances, and thus there is opportunity for repeated changes of domicile. In every instance in which leprosy occurs in a region supposed to be exempt, the leper should be asked as to where he has lived in bygone years.

In England, at the present time, there are probably not fewer than from 50 to 100 lepers, but they are all imported cases.

It may be the fact that in Persia, Palestine, and many inland places where only a few lepers are found, and but little fish is eaten, some of the cases are imported ones. The Arabs in the north of Africa and the Kaffirs in the south are notably migratory.

eighty inmates, chiefly Hottentots, and that the pastor who superintended it told him that they did not consider the disease contagious. After this, smaller leper homes were formed in different parts, Graaf Reinet, Lovedale, etc., affording evidence that the disease was making its way from west to east. In 1845 the Hemel en Aarde leper home was transferred to Robben Island, and in 1894 the Cape Government, in view of the difficulty of transporting patients, and the expense of their maintenance on the island, formed an eastern establishment in Tembuland, which is now known as Emjanyana. At this latter, natives only are received (with the fewest exceptions).

During quite recent years a leper home was constituted at Pretoria, and just before the outbreak of the war the Transvaal Government had built a larger establishment a few miles from the town. With the exception of a little home with six patients at the foot of the Bluff at Durban, Robben Island, Emjanyana, and Pretoria are, I believe, at the present time the only places in South Africa where lepers are received with the object of segregation.

Robben Island has 560; Emjanyana, 400; and there are at large, *i. e.* not in confinement, in Cape Colony, an uncertain number; in the native territories of Caffraria, 500; in Natal, 200; and in Zululand, 8.[1]

Having thus briefly referred to the chief facts as to races of South Africa and the early history of the spread of leprosy amongst them, it is now needful to give some facts as to the fish industry.

Although the bays and mouths of rivers on the coast everywhere abound in excellent fish, nothing worthy of the name of a fishing industry has ever existed on the eastern or south-eastern shores, and until a very recent period the adjacent districts were quite free from

[1] Dr. Impey, in 1896, estimated the number of lepers in South Africa as being 600 in Cape Colony itself, 250 in Griqualand East, the same number in Basutoland, nearly as many in Natal, whilst the Orange Free State had only 150, and the Transvaal only 30. The location of these numbers confirms the conclusion that the disease had spread from the Cape Town district east and north.

leprosy. On the western and south-western, on the contrary, at numerous places there have been colonies of fishermen who, after primitive fashions, prepared fish, by drying and salting, for use inland.[1] Until recently these have been chiefly in the hands of Malays. The first was in Cape Town itself; but Kalk Bay, Mossel Bay, Saldanha Bay, and other places soon followed. The fish was sent inland in carts, and as roads were bad, it is probable that it did not at first go very far. It is on record that it was in great demand, and Damberger, who in 1797 travelled on foot through the Colony, and was repeatedly beholden to Hottentot slaves for a meal, states that they shared with him their rations, consisting of "*salt-fish and rice.*" As roads were made, and more especially when railways were constructed, we may assume that the salt-fish was carried further and further inland. At the present day large quantities are consumed in Johannesburg and the other mining centres.

It may not be without its object to state that the first South African leper who came under my own notice was a Welshman who had been engaged in laying down the railway to Kimberley. He told me that Cape salt-fish, brought on by the rails, had been the principal article of food for himself and his men. Our best means of estimating the dietetic habits of the Dutch farmers of that day is probably afforded by ascertaining those of the present, and for this a single instance will serve. Malmesbury is an old Dutch town in an agricultural district about forty miles from Cape Town in the direction of Saldanha Bay. Here I visited a fish-warehouse, and saw the salted fish. I was told that the farmers bought it regularly for their labourers, and that, often tempted by the wholesale price, they bought much more at a time

[1] Thus it will be seen that a fisher community may be exempt from leprosy if the art of curing be not practised, and all the fish caught be eaten fresh. A community which has long been accustomed to live on fresh fish with impunity, or with but little leprosy, may experience an outbreak of it if the art of curing be introduced. This occurred in the case of the Sandwich Islands, and possibly in New Caledonia.

than they could consume whilst in good condition. It was not of a quality which would keep good more than a fortnight.

Several Dutch farmers, now themselves inmates of Robben Island, and from various districts, confirmed what I had learned at Malmesbury, and said that it was usual for the labourers to eat salt-fish for breakfast and supper. The kind of fish here referred to is what is known as "sack-fish," because it is sold in sacks, and is an article of which our English market knows nothing. It is usually prepared by steeping large fragments of coarse fish in a very strong brine for about a fortnight, after which it is allowed to dry, is packed in sacks, and will keep without obvious decomposition for about three weeks. There are several better kinds of dried and salted fish in the market, but these fall to the share of the more wealthy. It is the "sack-fish" which is supplied to labourers, and which is almost exclusively under suspicion as the cause of leprosy.

Enough has perhaps been said to prove that this kind of fish has been in the past, and still is, accessible to very large sections of the inhabitants of Cape Colony. It would be by no means difficult to show that, in the main, the districts to which it is chiefly supplied are precisely those in which leprosy is most common. It would, however, be tedious to attempt to do this on the present occasion, and I may freely admit that the data do not at present exist which would justify more than general statements. A leprosy map for Cape Colony has been constructed by Dr. Impey, and statistical tables showing local prevalence have been compiled by Dr. Gregory, the zealous and able Medical Officer of Health for Cape Colony. Both these observers have, however, in discrediting the fish-hypothesis, contented themselves by observing that there is no proof of excessive prevalence on the sea-coast. To this objection the reply is obvious, that it is not where salt-fish is prepared but where it is eaten that we must expect its ill results, and that the chief object of salting is to allow of its being sent inland.

Under certain special local conditions, absence of roads, or periodically recurring inclemency of climate, the inhabitants of fishing villages may be induced to eat the fish which they have salted, but under other conditions they may find it to their interest to send almost the whole of it away. In the early days of Cape history we may believe that the fish caught was eaten chiefly near to the places where it was taken, and in those times, as I have already said, the centres for leprosy were in the neighbourhood of the bays on the coast. The conditions have, however, changed, roads and railways have been made, and the factors which now appear to influence the distribution of leprosy appear to be:—agricultural as opposed to pastoral pursuits,—a fairly dense population, in which the native element (bastard Hottentot) largely predominates:—and Dutch proprietorship. Exceptions to these statements occur in the case of the great mining centres, but in them the population is a migratory one, and, although many may receive the germs of the disease, but few remain there to develop it. As a rule leprosy is not encountered in the large towns of Cape Colony, but in the agricultural districts adjacent to them. In the latter it is scattered sparingly, large districts are free, but here and there a farm has its one, two, or three, and it may be known to have existed for several generations. Nowhere are there many cases, and rarely indeed does it affect more than a few members of the same family. Many examples occur of quite isolated lepers,—that is, of those who have lived at their homes through the whole course of their disease without communicating it. When it shows itself in early life, very usually more than one member of the family is its subject. Instances of the disease in two brothers are far more common than those in which husband and wife suffer together. My inference from this is that children not infrequently acquire the disease from contaminated food which an adult would avoid.

I may perhaps be permitted here to advert briefly to the facts which, in other regions than South Africa, and

not only in our own time but in ages long past, appear to connect leprosy with the use of fish as food.

The disease is one which has prevailed in all ages, and which, whilst by no means ubiquitous, has occurred to almost all races and in the most varied climates. Its sameness under all conditions wholly precludes the idea that it can be produced by any accidental combination of conditions, or that it has anything to do with mere poverty. It has appeared to be incident to a certain stage of civilisation, not the highest and not the lowest, and it has prevailed in some populations coincidently with religious maxims which necessitated a large consumption of salt fish. It has wholly disappeared from certain large territories where those maxims have lost their force, and it still persists in others where they still obtain (Spain and Italy). Roughly speaking, it is now prevalent all over the world in ratio with the salt-fish-consuming habits of the population. In almost all places where it has prevailed a popular suspicion has been entertained, and sometimes a strong one, that it was caused by fish. Now there is no other article of food which can be named, the use of which is common to all leprosy districts.

It is the chief object of the present paper to maintain two principal propositions, and to these I may now address myself.

The first is that leprosy is undoubtedly communicable from person to person, but that the mode of its communication is peculiar and does not come under the head of contagion properly so called.

The second is that, whilst personal communication obviously cannot explain the *origin* of any disease, the facts as regards the origin and distribution of leprosy in South Africa strongly favour the belief that it can arise *de novo* as a specialised form of disease—possibly of tuberculosis—from the use of imperfectly cured fish.

As regards the first half of my first proposition, I well know that my contagionist friends will tell me that I

need not have gone to South Africa to learn that; they had long known that leprosy was communicable. But I may perhaps be allowed to suggest that their belief was in the main an inference from bacteriological theory, and that it was supported by exceedingly little of clinical evidence. The cases with which their writings teem as instances of contagion are all of them open to the objection that the disease might have originated *de novo* from food, since they all occurred in communities where the disease was prevalent and where fish was eaten. The constantly-quoted case recorded by Dr. Hawtrey Benson in Ireland was the only one in which this explanation could not be given. In all the others the supposed exposure to contagion might have been the merest coincidence. I do not think, therefore, that the evidence which I am now about to offer, and which will, I trust, set at rest for ever the discussion as to communicability, ought to be received by the contagionist school with feelings other than those of simple gratitude.

The facts which convinced me on this point were the following:—First, near to the village of Howick, in Natal, on the open veldt, I saw young lads unquestionably the subjects of leprosy, who had never left their native kraals, and concerning whom it was morally certain that they had never eaten salt-fish. They were living in the same kraal with adults who were the subjects of leprosy and who had probably obtained it in Cape Colony. Subsequently in other parts of Natal I met with precisely similar facts. Now leprosy is not endemic in Natal; it cannot be suggested that there are any conditions as regards food or mode of life which can conduce to it. The kraals are widely separated from each other on the open hillside, and their inhabitants have usually enjoyed good health. Leprosy is of recent introduction, and is met with only very sparingly. In all instances in which young persons were its subjects there was the history of its introduction into the affected kraal by an adult who had previously sojourned in a fish-eating district. That

in these instances the young persons derived their disease either by inheritance or personal communication seemed indisputable, and the idea of inheritance appeared to be negatived by the fact that often the young sufferers were the nephews or nieces and not the children of the individual who had originated the disease. Thus, then, the inference seemed inevitable that the disease had been communicated from one person to another. Nowhere, however, had it spread to many. Its incidence appeared to have been most erratic. A few had been taken and a great many, who had apparently been equally exposed, had been spared. Amongst the district medical officers with whom I conversed exactly the same difficulties had been recognised. With very few exceptions all thought that the disease was in some way communicable, but all admitted that it was most difficult to conjecture by what means the communication took place. I am speaking now of observations made in Natal, where little or no fish is eaten, for over the whole of Cape Colony proper the use of salted fish is such that no cases which may appear to imply personal communication can be accepted as conclusive. Reflecting upon the difficulties which the proved instances of personal communication presented, it occurred to me that it might possibly be by the discharges from sores on the hands of lepers finding access to the stomach on articles of food. The more I thought over this hypothesis the better it seemed to fit with the ascertained facts. Suppurating sores on the hands of lepers are in certain stages very common, and they remain for a long time. The Hottentots and Kaffirs are exceedingly careless feeders, and there is nothing in the least difficult of belief that food, fruit, or other dainties might be taken directly from a hand so affected. This would be especially likely to occur in the case of children. Inasmuch as it would make communication a sort of accident, it would well explain both the rarity and the irregularity of its occurrence. The subject is, however, of such importance that I will venture in some detail to state the principal reasons

which induce me to believe that this is the true explanation.

The facts which seem to support the hypothesis that in all cases in which the disease spreads from person to person the bacillus is received by the stomach are the following.

The first symptoms of leprosy are almost always those of a blood disease. There is never any primary sore or other indication of local infection. The earliest phenomena, whether affecting the skin or the nervous system, are as a rule bilateral, and imply blood contamination.

There is not the slightest reason for believing that any recognisable peculiarity in individuals, either as regards temperament or health, in any way predisposes to leprosy.

It is impossible to believe in communication by the breath, for attendants in leper houses, and others who may be in constant and close communication with lepers, never take it. It is also for the same reason impossible to believe in contagion through the skin. To these arguments may be added that it is very rare for both husband and wife to suffer, and that many experiments in inoculation have been tried without result.

The arguments just advanced have been felt to be so strong, by a large majority of medical observers, that prior to the discovery of the bacillus there was a very general disbelief in the contagiousness of leprosy. This was the verdict given by the College of Physicians after a detailed inquiry, and it was that of the best Indian authorities. We are now confronted with the proved existence of a parasite, and with evidence beyond dispute that in some way the disease can be communicated. We are driven, therefore, to seek the explanation of its communication in some direction which has hitherto either wholly or in part escaped notice, and which can be made to fit with the very erratic incidence with which such communication is observed to occur. Now the suggestion that the bacillus is received into the stomach by the direct contamination

of food by leprous discharges does meet these conditions; such contamination of food is not likely to occur, excepting under conditions of extreme carelessness as to feeding, and it is only amongst those who feed very carelessly that proofs of the communication of leprosy are met with. Amongst the cleanly communities of Europe and America, although there are plenty of leper-patients who might serve as sources of infection, no such communication appears to occur.[1]

The question of *de novo* origin is so important that a little further detail respecting it may be allowed.

In South Africa leprosy is rare, and is sparingly scattered over very wide and thinly populated districts. The disease has now been present for several generations, and hereditary transmission is therefore possible. There is no sort of doubt that family as well as regional prevalence is not very infrequently noted. Yet it remains the fact that as far as the evidence can be obtained, a great majority of the cases which occur in adults are *de novo* cases.

One after another, both whites and coloured persons tell us that no relative has suffered, and that they themselves never saw leprosy until it occurred in their own persons. This latter statement is the more credible because the disease is rare, and it would have been difficult for them to find its subjects. In many instances it might have been well-nigh impossible. I will quote directly some facts

[1] The following is a most instructive statement of fact. I quote it from Dr. Thin's pages, where it is cited as if proving the value of isolation:

Dr. T. H. Hall has recorded a case in which "leprous families of negro slaves in Bahia were exiled deep into the fertile woods of Northern Brazil. In their exile they were furnished with means of rearing poultry, pigs, goats, of fishing and trapping game, of cultivating cassava, yams, plantain, maize, etc., and then they were left entirely to themselves. Among these exiles, when visited after the lapse of many years, leprosy was found extinguished; a sound negro colony occupied the place of the old leprous one." It would appear clear from this narrative that in the migration inland the community left behind it the real cause of the malady. It is clear also that personal communication, unaided, was unable to maintain the disease. It died out.

collected by myself, but before doing so I may adduce some, offered without any preconceived object by an observer who, if he had prejudged the question at all, had done so in a direction opposite to my conclusions. Dr. Impey, in his little manual on lepra, has published the portraits of many who were under his care on Robben Island, and he has given with each a brief account of the patient's case. In all he mentions particularly the family history; and presumably when he omits to specify supposed exposure to contagion no facts on this head were to be obtained.

He states, respecting one case, that the man had cohabited with a leper; and in another, that its subject had worn a leper's clothes, and had he been aware of any similar risks run in any other cases, no doubt he would have named them. He does not do so, and the omission extends to thirty out of his thirty-two cases.

As regards family history, it is specially stated to be absent in twenty-three out of the thirty-two cases. Thus we find that a proportion of seventy-two per cent. of the adults in the Robben Island establishment were,—so far as Dr. Impey, who as resident medical officer had every opportunity for investigating the facts, could sift them—*de novo* cases. Most of the patients were men, and of the dark races. Is it unreasonable to believe that such a preponderance of negative evidence does really imply that the disease was, in many instances at least, the result neither of inheritance nor of personal contagion?[1]

Statements precisely similar to those made to Dr. Impey, were made to myself over and over again during my investigations in Natal and Tembuland.

In entering upon my second proposition, that a diet of salt fish is capable of originating leprosy *de novo*, and that it has in South Africa been the one sole cause of its origin, and by far the principal influence in its diffusion,

[1] In the course of a Report on Leprosy in Kashmir, Dr. Neve tells us that out of 143 patients 6 only had leprous relations, 47 knew of other lepers in their villages, and 96 knew of no others in their vicinity.

I am aware that many will think that I have a very difficult task. To some it may appear almost absurd to suggest that a malady, the phenomena of which are certainly in connection with the presence of a specific bacillus, can ever be of *de novo* origin. To such let me explain that by *de novo* origin is meant origin independently either of contagion or inheritance, that is, without personal transference of germ-material. It is not meant that the bacillus can arise *de novo*, but that the disease leprosy can do so. If leprosy can begin in a community in which no lepers have previously existed, then it is convenient to speak of such beginning as *de novo* without for one moment suggesting that it comes without the precedent occurrence of very definite causative influences. It may be that, after all, leprosy is only a modified form of tuberculosis, and that the same bacillus has undergone modification in connection with peculiarities in food supply. Without, however, allowing too much weight to this or any other hypothesis, it behoves us to look the facts fairly in the face. The discovery of the bacillus and the admission which must now be fully and freely made that it may be transferred from person to person, and that when so transferred it produces the full phenomena of the disease, although invaluable evidence of the *quasi*-specificity of the malady, are not facts which cover the whole ground. It still remains for us to examine whether there are other modes apart from personal communication by which the bacillus may gain access to the human body, or by which it may, so to speak, be bred up into specificity from a closely related organism.[1]

[1] An exceedingly important question in connection with fish food and leprosy is whether the fish simply serves as a vehicle for the introduction of the bacillus into the system, or whether it only stimulates the bacillus to activity. On the one hypothesis the continued use of salt-fish diet might be of no moment when once the disease was contracted; on the other it may be very prejudicial. It is possible that the reason why leprosy was regarded as incurable in the past was because fish food was still supplied, and much of the credit which is from time to time accorded to various modes of treatment in asylums and elsewhere may be simply

Taking syphilis as our best example of a somewhat chronic disease of specific character which spreads by contagion, and by contagion only, an instructive contrast may be drawn between it and leprosy. Neither of them show any regard to the race, the age, or the state of health of those whom they affect. Syphilis is, however, met with chiefly in towns, leprosy in country districts. If either one of a married couple contracts syphilis and cohabitation be continued, the other is certain to become infected; this is very exceptional in leprosy. In syphilis there is a primary sore denoting the site of inoculation; none such is ever observed in leprosy. Experimental inoculation always succeeds in syphilis; never in leprosy. Syphilis may be communicated freely in vaccination; it is very improbable that leprosy can be so conveyed. If syphilis has once got foothold in any town or community it will maintain itself from generation to generation; leprosy, on the other hand, although well established, often shows a most definite tendency to die out, without any other assignable cause than gradual changes in the social habits of the community.

The consumption, on a large scale, of the kind of fish under suspicion, has been proved as regards many of the districts in Cape Colony most affected with leprosy, and shown to be at least possible in all.

We are now in a position to state definitely the questions which are at issue in reference to the spread of leprosy in South Africa. Respecting the principal facts there will, I think, be no dispute. It has advanced steadily from west to east during the last century and a half, and is now invading regions to the east of the Drakensberg range, which were until recently free. Not for a moment can it be contended that there has been any change in the general well-being of the communities involved, which would explain it. The malady has proved no respecter of race, and those who have

due to the change in food and the abstinence from fish. No fish is now supplied as a rule either on Robben Island or at Emjanyana.

become its victims were still living under a sunny sky on the slopes of breezy hills, and exempt from hardship. Two conjectures only seem possible. Either the disease has spread by contagion or through the influence of some article of diet which has been introduced during the period under consideration. Now there is no doubt that salted fish has been so introduced, and there is no other article of diet which can be suspected. The issue, therefore, lies between salt fish as a cause of the *de novo* origin of the malady, aided occasionally by personal communication as a cause of increased local prevalence, and contagion pure and simple. There will probably be no dispute as to the mode by which the disease has been recently introduced into Caffraria and Natal. Contagionists, as well as others, will accept the suggestion that it has been carried there by men who have been into Cape Colony. No one will wish to suggest that in these regions it has originated in any sense spontaneously. The question is, then, under what special influences were these wanderers brought in the Cape district which occasioned them to become lepers? Were they the victims of unsuspected contagion, or did they acquire it by eating salt-fish? In slightly varied terms the same question is to be asked respecting all instances of the advance of the disease in Cape Colony itself. I have instanced Natal and Caffraria simply because in them the problem is offered in its simplest and most definite form.

I have made throughout this paper no concealment of my own opinion that the chief cause of the spread of leprosy in South Africa has been the use of salt fish and not contagion, and it is not without some risk of repetition that I now recapitulate the principal grounds for that belief.

Putting aside the cases in which the disease has begun in childhood, and in which personal communication may be suspected, a large proportion of the adult lepers in South Africa assert strongly that they have not had

leprous parents, nor been in any way exposed to risk of contagion. Many assert that they had never in their lives seen a leper until the disease was disclosed in their own persons. These assertions are made not only by coloured persons but by intelligent Dutch farmers and others. Of the latter class several were in confinement on Robben Island when I visited it, and they all made this statement, whilst they all admitted that they had habitually eaten salt-fish. Negative statements of this kind must always be received with caution, but when they are repeated by one person after another it is impossible to put them wholly aside. It must also be remembered that leprosy is a very chronic and very conspicuous disease. It cannot be concealed from relatives and neighbours, and it often leaves indelible and well-recognised traces behind it. If a man has leprous relatives, his neighbours will know of it, and the dread of contagion is such in the minds of most that any association with strangers suffering from the disease would be most carefully shunned. When we remember also that the communication of leprosy would appear to be possible only under conditions of exceptional and most intimate intercourse, I feel convinced that we may accept the statements of patients when they say that they have never been exposed to such risk. A large majority of the South African cases are then, as regards both contagion and inheritance, of *de novo* origin.

Another argument against the suggestion of contagion is the scattered distribution of the disease and the absence of any foci of great prevalence. Unlike syphilis, it is not met with in large towns, but dotted, as it were, very sparingly over very large agricultural districts. Many cases are solitary ones, and although undoubtedly it sometimes affects several members of a family, there is no record of its having spread as an epidemic in any village or district.

Although scattered over the whole of British South Africa, from the north of the Transvaal to the southern

coast, it is by no means evenly distributed, and there are large districts which are yet wholly free. On the theory of contagion no explanation could be offered of its local distribution, whilst the varying facilities in obtaining salt-fish do to a large extent fit with the observed facts.

Excepting in cases of family prevalence, where personal communication may be suspected, there is in South Africa, as in all other regions where leprosy prevails, a marked disproportion in the sexes. Three men to two women is the usual result of statistical calculations, and were the communication cases omitted it would probably make the disproportion two to one. On the theory of contagion I can suggest no explanation of this world-wide fact, whilst in support of that of fish causation it may be plausibly suggested that men engaged in labour eat more than women, that men secure for themselves the larger share of animal food, and that men are less prone than women to object to articles that have been over-kept.

Thus then, in conclusion, I venture to say that the hypothesis of bad-fish causation, taken together with the admitted possibility of food-communication under certain conditions, is one which satisfactorily meets the facts as to leprosy not only in South Africa, but over the whole world. To those who discredit it I throw the challenge to produce any other which makes any approach to doing so.

It is impossible to maintain that personal communication will explain it when in South Africa we find that of those who, as physicians, nurses, etc., associate daily with lepers no one ever takes it, whilst of those who become its victims four out of five believe that they have never even seen a leper.

DISCUSSION.

Dr. G. A. HANSEN (Bergen), in opening the discussion on the two papers read at the last meeting of the Society, expressed the opinion that leprosy was solely contagious. From his first pathological researches he had come to the conclusion that leprosy was a specific disease, which should have a specific course. Illustrations were given of foci of the disease, some of which, at first sight, seemed to favour the old theory of inheritance; but it was pointed out that although the disease occurred in families living together, yet it occurred as frequently in others living together but not related. As to the supposed ætiology of leprosy from fish eating, he thought that leprosy did not occur in many communities where cured fish was an ordinary article of diet. If it were so the leprosy bacillus should have been found in the fish, but that had not been done. The leprosy bacillus was very difficult to cultivate, therefore it was difficult to believe that it could survive for any length of time in salt fish. In regard to the supposed *de novo* origin of the disease, its incubation period was not so long as had been supposed; it was probable that the disease might be existent for several years in a patient but concealed, and hence wide-spread infection might arise. The disease always spreads along the channels of communication between peoples, even among people who ate but little fish; such a channel of communication, for example, as that over the Alps. Although as much fish as ever was being eaten in Norway, yet leprosy was disappearing. From the time that isolation of lepers was enforced—in 1856—the diminution might be traced; this diminution in Norway had been from 2870 cases to, approximately, 1500 cases. Their way of living was just as it was some fifty years ago. The cause of the spread of the cases there had been uncleanliness, leading by a certain prevalent intimacy of intercourse—as that which required hospitality to share the same bed with a guest—to contagion. Many lepers from Norway emigrated to America, but there had been no spread of the disease to the children or grandchildren of these, negativing the hereditary theory. In Bergen very much fish was eaten, both fresh and cured, almost at every meal, yet there was no leprosy.

Dr. GEORGE THIN referred to the admission of Mr. Hutchinson of the contagiousness of leprosy. The case shown by Dr. J. Hawtrey Benson was, in his opinion, a crucial case proving its contagiousness. This observer showed to the Medical Society of Dublin a case of developed leprosy that had come from the West Indies. Several years after this patient's death his brother was affected; the two brothers having occupied the same bed, worn

the same clothes, and used the same eating utensils. But this case was not an isolated one: several others were alluded to proving its contagiousness on intimate association. In relation to the food theory, he had many years ago in China fed a leper on a diet rich in animal food without any amelioration of the disease. In one of the papers the predisposing cause was held to be lack of animal food, in the other it was considered to be due to eating a nitrogenous food. He remarked on the occurrence of leprosy without any obvious primary lesion. The leprosy bacillus produced so little local irritation that it was possible the actual entry of the bacillus might not attract attention; microscopically, too, there were very few inflammatory signs. It was possible that the entry of the bacilli might be by inhalation, which was supported by the symmetrical distribution of the lesions in some cases. The bacillus was shown by Campana at a congress in Rome as growing in sugar agar, but it was said that the bacilli, to give cultivations, must be taken during the so-called leprosy fever. As to the *de novo* theory, there were no recorded facts to show that leprosy had developed anywhere without the possibility of contagion. Lepers had been known longer in Egypt than anywhere else, therefore Africa was the last place to go to for *de novo* cases at the present time. The movements of African tribes being so wide, it was quite likely that cases might have reached South Africa from the northern or interior parts. The fact that leprosy was not recorded among the natives did not negative its possible existence. How could the disease develop *de novo* without the bacilli developing *de novo?* The fish theory had been investigated by a commission in India, who concluded that fish was not the cause of leprosy, and that no form of diet had any specific influence. It was to be hoped that even now some measures might be commenced to mitigate the dissemination of leprosy in India—in the way of segregation especially. Leprosy was introduced from China into Australia, but by careful isolation its spread was prevented. Wherever lepers went leprosy was apt to crop up.

Dr. HANSEN considered that the disease was probably introduced through the skin. There was slow development, but little irritation, and often neglect of his condition on the part of the patient. The antiseptic action of the gastric juice would probably prevent infection through the stomach.

Dr. PATRICK MANSON acknowledged himself a contagionist. The spread of the disease was comparable to that of tuberculosis, only it was a more difficult process. The staining characters of the bacilli in each disease were similar; the difficulty in cultivation was great in the case of tubercle, and almost insurmountable in the case of leprosy; the channel of introduction of either was obscure. The difficulty of accepting the fish theory was very great. It was not definitely stated, however, whether the fish

eaten contained the bacilli, or that it merely lowered the bodily resistance. It was allowed that much fish eating might not be associated with leprosy. It was an acute observation of Mr. Hutchinson that leprosy was a disease of an intermediate stage of civilisation, between the savage and the fully civilised man; the same applied to tuberculosis. No reference was made in the first paper as to whether the Soudanese ate fish, fresh or dried; the negroes probably did not use salt fish. The dietetic theories were neither of them conclusive. Beri-beri had itself been attributed to a defect in the nitrogeneous element of food. Defective diet was a favourite cloak for ignorance of ætiology. Two concurrences were not necessarily related as cause and effect. The occurrence of ainhum had been held to be a manifestation of leprosy, but in his experience this was not so. Was the elimination of a fish diet in Robin Island followed by a greater curability of the disease? In many cases of leprosy which he had seen the first lesion was asymmetrical, and there was no evidence of a constitutional disease. The principal medical officer of Ceylon had recently told him that the Dutch prisoners in Ceylon had succeeded in cultivating the leprosy bacillus in a fish-broth; if this were true it was a strong argument in favour of the fish theory. The origin of the germs of disease was probably in the remote geological past, and had been evolved from those times in remote ancestors. The obstacles in the way of ameliorating leprosy in India were almost insuperable from the ignorance of a lay public. The mode of entrance of the leprosy germ, as was suggested in Dr. Tonkin's paper, was very likely by the use of infected clothing and bed linen. But the spread of leprosy probably depended on a multiplication of opportunities, during some of which, by the fortuitous concurrence of certain necessary but rarely recurring conditions, the disease became implanted; hence the difficulty of the propagation of the disease. It was well known that malarial infection was through the skin; it was probable that the relatively large parasite of ankylostomiasis similarly entered through the skin; and it could easily be understood how that the minute bacillus of leprosy might likewise gain entry through the skin.

Sir WILLIAM R. KYNSEY said the use of dried fish in Ceylon was almost universal, both as a staple food and as a condiment. The importation of dried fish into Ceylon was enormous, the greater part going up country. Leprosy was almost unknown inland, but was prevalent in certain foci along the coast where fresh fish was obtainable. In several instances of single cases of leprosy in a family the patients had been wet-nursed, but he had not been able to ascertain whether the nurses were leprous or not. It was suggested that the bacilli might be in the milk. In several instances it seemed that leprosy was associated with vaccination.

Dr. HANSEN said that in Norway there was no evidence either of the association of leprosy with vaccination or of the occurrence of the leprosy bacilli in human milk.

Sir LAUDER BRUNTON had seen many cases of leprosy at Jerusalem which resembled very much syphilitic cases, and especially were certain late cases of syphilis of the larynx similar in their appearances to the lesions of leprosy. It might be possible that in cases of leprosy there might be a mixed infection. It was probable, indeed, that infection depended both on the bacillus and on a special susceptibility. Such susceptibility might depend on many factors, of which one was not unlikely imperfect feeding, as in the case of tuberculosis. Professor Unna had found that if any oxidising substance such as pyrogallic acid were applied to a leprous sore it was made much worse, and he also found that this could be counteracted by making the blood less alkaline, as by the administration of hydrochloric acid; it therefore might be that by endeavouring to render the blood acid, not necessarily by giving hydrochloric acid, but by giving nitrogenous food even, if it were only leguminous, the disease might be stayed. The ground-nut in the Soudan, one of the Leguminosæ, might supply the necessary nitrogenous element for the Soudanese. If people ate a great amount of fish they would probably eat less of other food; thus fish might act either by replacing flesh food or by itself adding a substance to the blood such as trimethylamine, present in herring brine, which would favour the growth of the organism.

Dr. T. M. YOUNG had seen much leprosy in Siberia, China, India, and the west coast of Africa, and had been struck by the active motility of the leprosy bacillus, suggesting a life history outside the human body, possibly in salted fish. He had not found the leprosy bacillus so numerous in the leprous sores of fishermen as in the deeper connective tissues. It was probable that the bacillus lived in the living tissues, and not in the tissues being cast off, and that the disease was not spread by discharges from sores. The eating of the different forms of fish did not, in his experience, correspond with the distribution of leprosy.

Dr. HERON had served on a committee ten years ago with Mr. Hutchinson to consider the findings of the Leprosy Commission. As Mr. Hutchinson had adopted the theory that leprosy was a communicable disease it was surely not necessary for him to adhere to the fish-eating theory, every part of which was indeed pure theory. A valuable test would be the finding of leprosy bacilli in dried fish in the laboratory. Alluding to the report of the committee on the Leprosy Commission, out of 464 lepers 99 had never tasted fish; 162 lepers in asylums scattered all over India had not eaten fish; some of these came from high up in the Himalayas, and several had never seen fish. Of 200 lepers examined by the Commission, 39 habitually, 57 occa-

sionally, 58 seldom, and 46 never ate fish. All the arguments in the paper more strongly supported the contagion theory than the salt-fish theory.

Dr. ALFRED HILLIER had seen cases of leprosy in Africa, both in the south and north. The exclusion of fresh fish seemed in his opinion rather against the fish theory. The fact that a leprous patient had never, as he believed, seen a case of the disease before had not much significance, as the malady was easily overlooked and often disregarded. The communicability of leprosy by contaminated food was quite understandable. Eating of salt fish was prevalent in certain parts of the home country, and yet there was no leprosy. Leprosy had all over the world vanished with the spread of sanitary conditions.

Mr. TONKIN, in reply, said that with regard to Mr. Hutchinson's paper, he was in agreement with the opinions expressed in it so far as this—that a dietetic factor determined the occurrence of leprosy. That the dietetic factor at fault, however, was an article of diet, and that article fish, he thought hardly so probable. The fish hypothesis did not appear to him to be capable of sufficiently wide application to account for all the facts connected with the spread of the disease. Among the circumstances surrounding leprosy in the Sudan, fish played but an insignificant part. Fish was rarely used by the people as an article of diet. It was certainly consumed in the parts of the country affected by leprosy, but to a small extent. In Upper Hausaland fish-containing water was scarce. The river element was mainly represented by beds that contained torrents during the rains, and were waterless, or only occupied by chains of more or less widely separated pools, during the dry season. Owing to the difficult and dangerous nature of the country, transport from more freely watered districts was expensive, and was therefore generally occupied with more valuable freight. He would grant that what fish did come into this part of the country was of an extremely doubtful nature, but, owing to the circumstances he had stated, its amount was small—so small indeed as to be negligible.

During the whole course of the stay of the Hausa Association's Expedition in Kano town and province, the district generally looked upon in the Sudan as the most leper-stricken, fish was only offered them for sale on one occasion. In the country shops and markets one rarely saw it; even in the biggest towns it could only be procured after a more or less prolonged search for it. Kano market was the greatest market in all central Negroland. During the dry season from twenty to thirty thousand people might often be seen on it at one time; a thing that could be bought anywhere in this part of the Sudan could be bought there. Yet, to illustrate the inconspicuousness of the fish element, he would call attention to the significant facts that

Dr. Henry Barth's minute description of this market, written in the early fifties, contained no mention of fish; that neither of the descriptions that had appeared in his late companion Canon Robinson's two books, 'Hausaland' and 'Nigeria,' contained any mention of it; and that his own lists, carefully compiled from daily notes, and reaching a total of nearly ninety articles and classes of articles, did not include it among the number. Fish in any form was rare in the central parts of Northern Nigeria, and it was in those parts that leprosy was most prevalent. The conditions obtaining in the neighbourhood of Lake Chad were roughly parallel. There was, he believed, when and where the water was accessible, a certain amount of fish caught in the lake on all its sides, but it was only at the north-western angle that the business was sufficiently highly organised to be called an industry. Without arrangements for catching on a large scale, and for transport of the catches that were made, the influence of a sheet of fish-bearing water, no matter how large it might be, could extend little further than the immediate dwellers on its shores. So far as he knew, no such arrangements existed on Lake Chad except at its north-western end. In that locality there was a town that might be called the Grimsby of the Chad. By the inhabitants of that town fish in considerable quantities was caught, prepared, and exported, but the stream of exportation was not into the leper-stricken Sudan, but entirely northward into the Tebu country, a country in which no leprosy was reported to exist. He said that this state of things pointed to a small consumption of fish in the leper area in the Sudan, and he did not think, therefore, that the supposition that fish was intimately connected with the dissemination of leprosy would, even on further examination, be found to receive much support from the facts pertaining to the spread of the disease in that region.

Mr. HUTCHINSON, in reply, stated that he regretted the shortness of time which was at his disposal. He did so the less, however, because, for the most part, those who had spoken had not dealt with the facts stated in his paper, but had been content to enunciate their own opinions. Dr. Manson was the only one who had brought any new facts into the discussion, and his statements as to the hope of cultivation of the bacillus on fish were of great interest. He (Mr. Hutchinson) lived in the daily hope that some one would announce the discovery of the bacillus in decomposing fish. It was, however, an inquiry needing great patience, for if present it is in all probability rare. If it were common, leprosy would be much more general than it is. All who had spoken had slurred over the difficulties which surround the theory of contagion in the ordinary sense. The failure of attempts to inoculate leprosy; the facts that husband and wife very rarely suffer together and that the healthy inmates of leper asylums never contract the disease, were, to his mind, conclusive

against it. It was supported in South Africa by the almost universal assertion of those who suffered that they had never consorted with lepers or even seen them. In reply to Dr. Thin he said that he held it to be a pure delusion that in the Middle Ages segregation measures were the cause of the disappearance of leprosy. There never was any real or efficient segregation, and the leper homes were for the most merely retreats for those who wished to resort to them. The same statement applied, he felt sure, to what was now taking place in Norway. There also there was no segregation which would be efficient on the theory that the disease spread easily by contagion. One third of the Norwegian lepers were still at home with their friends. The disease was, moreover, disappearing just as rapidly in Madeira, where no attempts whatever were made at compulsory segregation. As regards practical measures, he added that he would be quite prepared in South Africa to do away with all compulsory segregation, and to allow the lepers to return to their homes. They should be under supervision, and should be well warned as to the risk of commensal communication. It was probably an exceedingly small one. Above all, the fish-curing trade should be controlled. If no badly cured fish were allowed to get into the market, it was his opinion that leprosy would soon cease to exist.

THE POSSIBILITY OF RECOVERY

FROM THE

ACTIVE STAGE OF MALIGNANT ENDOCARDITIS

ILLUSTRATED BY CASES AND SPECIMENS

BY

WILLIAM EWART, M.D., F.R.C.P.

AND

A. S. MORLEY, L.R.C.P., M.R.C.S.

Received February 11th—Read April 22nd, 1902

CLINICAL and post-mortem observations in the cases to be narrated seem to warrant the conclusion that in its infective stage malignant endocarditis may be amenable to treatment, and the purpose of this paper is to urge the necessity for early diagnosis and for adequate treatment before irremediable structural damage has occurred. Its fatality is great because those who do not succumb early to the infection almost inevitably die of some of its late results. Instances of both these modes of termination are

afforded by the following three fatal cases which were simultaneously under observation.

Cases of recovery from ulcerative endocarditis are not unknown in the modern annals of medicine. As in most of the published cases ocular evidence of the cardiac lesions was not obtained, there must remain some doubt as to the severity of the attack and as to the existence of the disease in its worst form. Fatal cases afford evidence as to the nature and extent of the lesions, and as to their progressive or regressive character; and cases such as two of the present ones, where death interrupted the beginnings of reparative changes whilst revealing the presence of the destructive lesions of malignant endocarditis, are capable of supplying collateral evidence on the question of the curability of the disease, particularly when, as in them, some clinical improvement had occurred in association with the arrest of the local morbid process.

A simultaneous study of the clinical aspects of the three cases which ran a protracted course illustrates various points in the natural history, in the pathology, in the diagnosis, and in the prognosis and treatment of the disease. But the chief conclusions to be drawn from them are based upon the contrast in their pathological appearances. A careful inspection of the three hearts shows that the primary endocardial lesions may, as in one of them, continue to the end to be progressively destructive; or that, as in the other two, they may make room for changes of repair.

This pathological contrast agrees with the clinical differences. In one of our cases death resulted from the local disease, in the other two from its more remote consequences, whilst in the latter the slight improvement noted towards the end suggested the view that the infective virulence of the disease was on the decrease.

Case 1.—C. B—, aged 17, a pale, emaciated, nervous girl, was admitted on April 1st, 1901 (Med. Reg., No. 565), complaining of severe cough, great debility, faintness,

and palpitation. The mother had formerly suffered from rheumatic fever. The patient had enjoyed previously good health, spending most of her time out of doors. In December, 1900, she was laid up in bed for eight days with severe pains in the left leg and hip, and soon after she was laid up with pain in the left foot for three weeks. For the last two months she has had dyspnœa and palpitation, and progressive wasting. On admission she was so pale and thin that the diagnosis of severe phthisis was that which occurred at first sight, and this seemed to be borne out by the aspect and complexion, the wasting of the muscles of the thorax, and the complaint of cough. The pulse was compressible and 140 per minute; the respirations 48; and the temperature 102·2° F. The breath was extremely foul owing to the neglected state of the mouth. The throat was dry and injected. The tongue was dry, cracked, and furred.

On examination the *thorax* does not move freely, and the left ribs are more prominent than the right, though there is no scoliosis. The pulmonary resonance is defective at the left base.

Heart.—The apex beats in the nipple line in the fifth space. There is a loud systolic murmur and a doubtful presystolic murmur and thrill, with an accentuated second sound at the apex. The pulmonary second sound is accentuated with a loud pulmonary systolic murmur.

On April 3rd she complained of pain in the feet, which were a little red and swollen.

On April 15th a cough, accompanied by some blood-streaked expectoration, appeared, and a few crackles with diminished resonance were detected at the right supra-scapular fossa. The temperature was very irregular throughout this period, ranging from 103° to 99° F. There were occasional night sweats. These symptoms and signs, together with the aspect of the patient, led to the adoption of the original diagnosis of tubercular phthisis with rheumatism and mitral stenosis. She had been treated throughout this time with salicylates and

potassium iodide and quinine pills. Her mouth had been carefully disinfected and her bowels regulated. From April 10th to April 23rd she was taking gr. j of protargol in ℥ss of distilled water three times a day, and ʒiss of Easton's Syrup. The diet had been restricted for a few days to milk, and was then gradually increased till a very liberal diet with minced meat and vegetables was allowed.

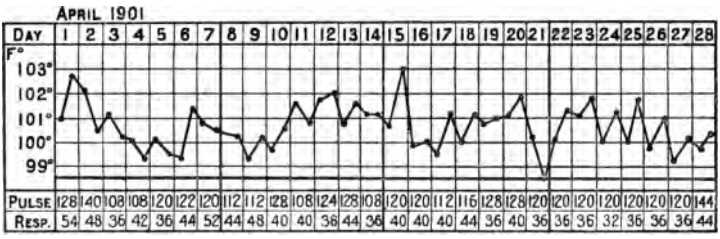
On April 18th she had somewhat improved in colour, and the night sweats had been controlled by atropine.

On the 21st she became drowsy and complained of intense headache. The murmurs varied from day to day, and on the 24th the condition of the heart was reported as follows :—"Apex-beat in fifth space just outside the nipple line. The beat is diffuse. The right ventricle is not much dilated. At the apex there are a systolic and a diastolic murmur; the first sound is loud and flapping. Both sounds at the base are suggestive of murmurs. The action is somewhat cantering and irregular."

On the 26th the headache and drowsiness persisted, and tuberculous meningitis was suspected.

On the 28th she was somewhat better and the drowsiness and headache had quite disappeared, but in the early morning of the 29th she suddenly died of syncope.

TEMPERATURE CHART OF CASE 1.



The post-mortem examination revealed a large patch of heavy, grey, necrotic vegetations at the mitral valve, extending into the left auricle. Some of the chordæ tendineæ of the mitral valve had ulcerated and ruptured.

The heart was slightly dilated and hypertrophied. Pericardial adhesions of some standing occurred over the left ventricle and over the anterior surface of the right ventricle.

The *lungs showed no signs of phthisis.* There were no pulmonary infarcts; but the spleen, which weighed 19 oz., presented small white infarcts and a rather larger hæmorrhagic infarct. The kidneys also contained three or four white infarcts and several recent ones. The brain weighed 3 lbs., and was apparently normal.

Case 2.—E. I—, aged 14, a dark intelligent girl with sharp features, was admitted on April 9th, 1901 (Med. Reg., No. 614), complaining of palpitation, dyspnœa, and cough. There was a family history of rheumatism. She herself had had measles as an infant, and *rheumatic fever* two years previously, for which she was treated in a children's hospital.

On admission she was remarkably pale and emaciated. "*Her teeth and gums in a very bad state.* Tongue clean. *Fingers* clubbed and bluish. Thoracic movement deficient, and myoidema very marked. *Lungs:* tympanitic all over, except at the apex of the right lower lobe, which is dull. *Heart:* apex in fifth space, almost in the left mid-axillary line. At the apex there is a marked thrill with a presystolic and diastolic murmur; and at the aortic site a diastolic thrill, with a systolic and diastolic harsh murmur conducted down the sternum. The diastolic murmur is occasionally distinctly musical. The *liver* is enlarged to two inches below the right costal margin. The *urine* contains much albumen and a deposit of phosphates. *Blood examination:* the red cells number 3,000,000, and the white 15,000 per c.mm."

A provisional diagnosis of aortic and mitral disease and of phthisis was made at first, but in a few days the remittent temperature and the changeable murmurs, together with the leucocytosis, led to the diagnosis of malignant endocarditis, which was subsequently verified.

On April 10th some pus appeared in the urine. The physical signs remained unaltered. The sputum was examined, and *no tubercle bacilli* were discovered.

On April 20th the presystolic murmur was much less marked, but the aortic diastolic was intensely loud and musical, with a very distinct shock perceptible on palpation. The lungs were drier, and no adventitious sounds were audible. The temperature was still markedly hectic, and the pallor was increasing. The pyrexia persisted.

The treatment had consisted of tonics, urotropin, cod-liver oil and malt, and ichthyol administered internally, and of disinfecting lotions for the mouth.

On May 2nd a course of daily injections of anti-streptococcus serum (5 c.c.) was commenced. The effect of these was to produce great mental depression, the patient becoming lachrymose. The following day some red blotches were observed scattered over the face and arms; these, however, rapidly disappeared. There were no rigors.

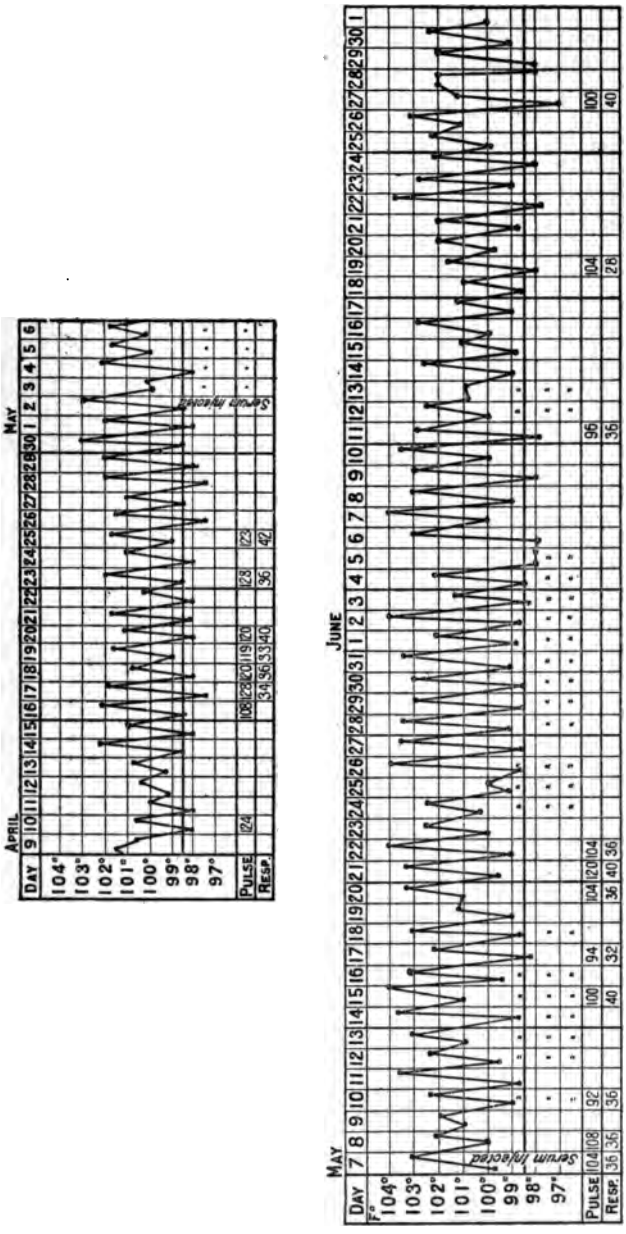
On May 18th the musical aortic murmur was replaced by a soft blowing murmur. The emaciation had increased although the face appeared full in the parotid region. There was slight bronchitis at the time.

At the end of May she was occasionally delirious at night. She ate her food greedily, and, except for fits of depression after the injections of the serum, was fairly cheerful. The pyrexia persisted, and there were a few attacks of pain over the liver and spleen. On one occasion distinct friction was felt and heard over the liver, and suggested the possibility of hepatic infarct with perihepatitis. On another occasion after an attack of pain a little blood was found in the urine, possibly due to a renal infarct.

On May 24th a 20 per cent. ointment of protargol was ordered to be rubbed into the skin twice daily as in the third case, and this was continued throughout.

On May 27th a tonic containing T. Digitalis ℥iv, Liq. Strych. ℥iij, Liq. Hydr. Perchl. ℥viij, and T. Ferri Perchl.

TEMPERATURE CHART OF CASE 2.



mviij was prescribed on account of the "canter rhythm" observed. This had the effect of quieting the heart and of improving its force. On June 10th a pill of nitrate of silver, gr. $\frac{1}{10}$, was ordered to be taken three times a day.

Since that date the case pursued much the same course.

The hectic temperature with high evening rises continued to the end, and she never left her bed. During the last three weeks, however, there was decided improvement in the appetite, in the aspect and manner, and in the strength, and she sat up in bed unsupported at frequent intervals during the day. A definite hope was, therefore, entertained of her ultimate recovery. Death occurred rather unexpectedly on July 8th, after a short period of dyspnœa.

Post-mortem.—Both pleural cavities contained moderate effusions. The pericardium contained about 4 oz. of fluid. The spleen was firm, and weighed 11 oz. The liver presented a nutmegged appearance. The left kidney was the seat of a congenital hydronephrosis. There were no infarcts in these organs. The presence of malignant endocarditis was verified. The affection was limited to the base of the aorta and the semilunar valves, which were thickened by granular and beady deposits. To one of them, that nearest the mitral orifice, a delicate flat ribbon-like pedunculated vegetation, about half an inch long, was attached. This was slightly rough and yellowish, as if atheromatous, with some pink staining. Where this appendage came into contact with the arterial surface above, an uneven but perfectly clean ulceration was found, nearly one third inch in diameter, which perforated the vessel wall as far as the adventitia.

Search was made for necrotic grey vegetations, but none were found, either at the surface of the ulcer, which was perfectly clear of loose deposits and apparently in process of healing, or at the damaged valve, the beady deposit of which was mostly of glistening aspect, not

rough or granular. There was no parietal endocarditis. The tricuspid and the mitral membranes were also perfect, and the auriculo-ventricular orifices and the auricles normal.

The right bronchial artery, which was of large size in connection with the cardiac malformation, was thickened and roughened by yellow atheroma, almost from the point of its origin. A loose atheromatous yellowish plug, about half an inch in length, was removed from the vessel.

The cardiac conditions described were such as would produce very free aortic reflux as well as a systolic bruit, and the regurgitation was probably sufficient to have interfered with the diastolic rise of the anterior mitral flap; but the mitral valve presented no structural change whatever in the direction of stenosis. The heart was large, and the two ventricles presented almost equal thickness of wall and equal dilatation. This was connected with a remarkable anomaly, viz. *complete absence of the pulmonary artery* with considerable deficiency of the septum, which need not be insisted upon, as the specimen will be exhibited and the condition fully described before another Society.

Remarks.—As regards the cardiac anomaly, there was nothing in the history prior to the rheumatic fever to lead to congenital disease being suspected. Although rather small for her age the child was not cyanotic, and her breathlessness on exertion had been chiefly noticed since the rheumatic fever. The diagnosis was correct as regards the causation of the double aortic murmur, and as regards the presence of malignant endocarditis. But mitral stenosis was, as so often happens, diagnosed where it did not exist. At most there may have been pressure upon the anterior mitral flap from aortic regurgitation.

It is noteworthy that in this case few traces of embolism were found except a yellow deposit in the right lung, which proved, on examination, to be beset with tubercle bacilli. At the necropsy there was no evidence of any recrudescence of the virulent affection. The heart

lesions, with the exception of the brittle appendage, were apparently in process of healing.

In this case, as in Case 3, the rheumatic diathesis and the oral sepsis were prominent ætiological factors. This leaves us in doubt as to which of these infecting agents may bear the responsibility for the cardiac changes.

The diagnosis of ulcerative endocarditis had been obvious a few days after her admission, owing to the hectic temperature, the pallor, the night sweats, the emaciation, and the changing cardiac murmurs. But the measures of treatment did not prove adequate. In particular, the injections of antistreptococcus serum were disappointing both in their failure to benefit the condition, and in the depression which they undoubtedly occasioned. Some improvement was apparent after the inunctions of protargol were commenced, and after silver nitrate was administered. Meanwhile the internal treatment by heart tonics and perchloride of mercury was continued, and to the action of these remedies some share in the slight improvement may be ascribed.

Case 3.—H. B—, traveller, aged 32, married, was admitted into St. George's Hospital on April 28th, 1901 (Med. Reg., No. 741), and died on June 9th, greatly emaciated and exhausted, after a long illness, beginning in November, 1900. For the early notes of the case our thanks are due to Dr. A. H. Newth, of Haywards Heath. The patient's previous health had been good, excepting gonorrhœa at the age of sixteen, scarlet fever at the age of nineteen, rheumatic fever at the age of twenty—from which he made a good recovery,—and two or three years ago some acute gastritis with anæmia. His habits formerly had been rather alcoholic. The family history mentions the death of a sister of "phthisis," and temporary hysterical insanity in another sister. The present illness began in November, 1900, with severe rigors, but he went on with his duties for a month afterwards. He was then seen by Dr. George L. Johnson, of Woolwich,

who reports he was suffering from acute gastric catarrh, with congestion and evidence of early cirrhosis of the liver, and from head and back pain, probably due to influenza with abdominal complications. No albumen or sugar had been found in the urine.

On January 26th, 1901, he came under Dr. Newth's care, whose report is as follows:—"Patient very much emaciated; no cough, nor dulness in lungs; liver somewhat contracted; spleen enlarged. No rigors, but night sweats, saturating clothes and blankets. Obstinate constipation. No albumen nor sugar. The blood did not show any remarkable excess of leucocytes, but the globules were crenulated, and there seemed to be a large quantity of free nuclei, and also some blood plaques. The night sweats improved under quinine, nux vomica, nitro-hydrochloric acid, purgatives, etc.; the urine became normal and the sweating lessened considerably, so as to be almost insignificant. He gained strength and was able to walk out. Subsequently he had some rheumatic inflammation in the right foot, which yielded to salicylate; and after this he gradually got weaker."

A consultation was held on March 15th with Dr. John J. Uhthoff, who thought it probable that deep-seated suppuration, perhaps in the liver, existed; but no definite conclusion was arrived at except the desirability of his removal to the hospital.

Dr. Newth's last note (April 21st) is to the following effect:—"There have been no rigors; the night sweating has subsided, but the pain and tenderness in the left side continue. The temperature oscillates between 100° and 102°; pulse about 100. There has been practically no cough, no expectoration, no irritation of the fauces, and vomiting only once or twice; but the emaciation is extreme in spite of plenty of nourishment and of fairly good appetite and digestion. For weeks he has been too weak to get out of bed, except for short periods. The treatment has consisted in quinine and arsenic, and latterly a simple effervescing mixture."

His appearance when admitted on April 28th was, but for the remaining energy in the gaze, that of a man at the extremity of chronic phthisis; he was pale and emaciated, with extreme muscular wasting; his weight reduced to 7 st. 2 lbs. There was no anasarca. On examination a few rhonchi only were found in the lungs, which were clearly not seriously affected; but a double murmur was heard at the aortic area. A murmur was also attached to the first sound at the apex of the heart. The diagnosis of malignant vegetative endocarditis was arrived at on this evidence.

The liver and spleen were apparently normal. An examination of the blood showed a diminution of the red cells to 1,500,000, and an increase of the white to 25,000 per c.mm. The blood proved to be sterile.

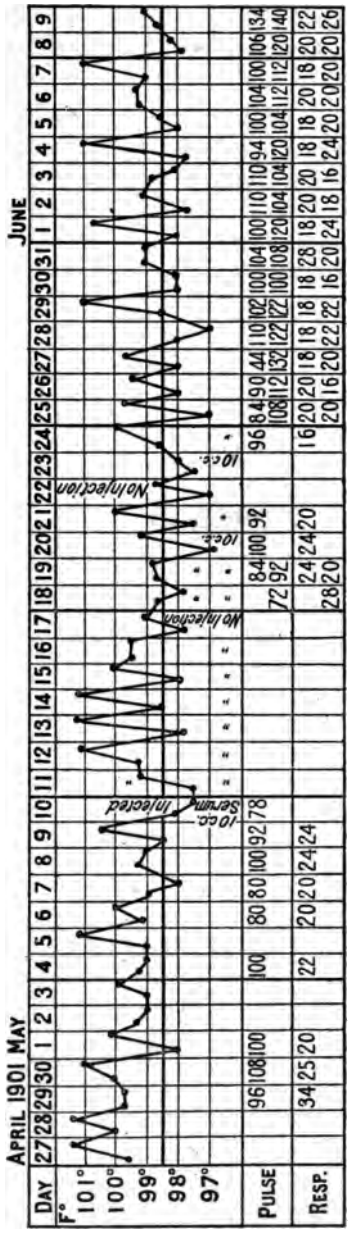
The pulse (100) was of the "water-hammer type," and there was marked pulsation of the carotids and other arteries. The temperature oscillated from 99° to 101·5°. The night sweats were profuse. The urine was high-coloured (sp. gr. 1019), and presented only a cloud of albumen.

The mouth was in a very bad condition, with decaying stumps and fetid stomatitis. This was at once treated.

On May 10th a course of daily injections into the abdominal walls of 10 c.c. of antistreptococcus serum from the Jenner Institute was commenced. They remained without any marked result. Meanwhile the general condition had improved slightly under the influence of food and nursing, but the emaciation continued to increase. A few lardaceous casts were reported to be present in the urine on May 10th.

On May 21st an ointment consisting of 20 per cent. protargol in lanoline and lard was ordered to be rubbed into the skin daily, about ʒj being used at each inunction, and the injections of antistreptococcus serum were continued for a few days longer. From this date a slight improvement was noted. The patient had better nights, and seemed to gain a little strength, although the tempe-

TEMPERATURE CHART OF CASE 3.



rature remained irregular. There were occasional attacks of severe pain in the hepatic and splenic regions, which suggested infarction; but at no time was there any subcutaneous hæmorrhage or hæmaturia.

On June 8th he appeared so much better that his earnest appeal to be allowed to be carried into the quadrangle was granted. Unfortunately even this seems to have been too great an exertion, for an attack of acute cardiac dilatation supervened, from which he died on the following day.

The post-mortem revealed considerable cardiac disease, but no tubercular affection of the lungs or other organs. There was a slight excess of fluid in the pericardium. The vegetations of malignant ulcerative endocarditis occupied the aortic and the mitral valves and part of the adjoining ventricular surfaces. We shall presently revert to the cardiac changes. There were old and recent infarctions of the spleen and kidneys, but no lardaceous disease was present. In the kidneys there was a slight diffuse overgrowth of fibrous tissue. The spleen was firm, and weighed 11 oz. The liver was fatty.

The Appearances presented by the Heart in the three cases, and the Conclusions suggested.

On closer inspection the individual specimens present the following appearances:—The heart in the first case shows malignant endocarditis of the mitral valve in its worst form and stage; the vegetative and the ulcerative processes are alike unchecked. Heavy vegetations, of greyish necrotic aspect, cover the mitral flap, inducing slight infection of the infra-aortic surface opposite, but not of the aortic orifice; and several of the chordæ are ulcerated through. No healthy fibrin is anywhere to be seen, and there are no changes of repair perceptible to the naked eye. These lesions are entirely adequate to explain the symptoms and the fatal termination. The

patient died of the direct effects of the disease, partly destructive and partly toxic.

In the second case, in which it was difficult to determine the precise duration of the acute affection, the changes were limited to the aortic orifice. The vegetative process is here identified by the presence of a long narrow strip of altered fibrin growing from one of the valves, and also inferentially by the embolic mass of similar material occupying the first portion of one of the large bronchial arteries. The malignancy of the ulcerative process is likewise attested by the ulceration through the coats of the aorta near the orifice of one of the coronaries. On the other hand, there is no evidence of recent disease—no grey necrotic aspect of the vegetations. The long pedunculated vegetation was brittle, and presented the same yellowish atheromatous mottling as the embolic plug, apparently indicative of staleness of the fibrin rather than of its recent deposition. The ulceration of the aorta, which had probably been set up by the whipping action of the long vegetation, did not seem to have been progressing, but rather healing, as some of its edges were smooth and its surface not unhealthy; and, with the exception of the long pedunculated appendage, the aortic vegetations were reduced to clear glistening warty deposits. In this case the cardiac lesions found at the necropsy were hardly adequate to explain death; they rather suggested the inference that the endocarditis had been worse at some previous time than at the final stage. But the state of the lung and the plugged bronchial artery established a complication which the patient, in her exhausted state, had been unable to survive.

In the third case it is possible to fix a date for the beginning of the disease, and the continued and progressive symptoms warrant us in regarding it as having been from the first infective. After a duration of six months the disease, if it had remained unchecked, should have culminated in its worst developments. As a fact, the heart suggests an opposite conclusion. In this respect

there is a striking contrast between the heart in Case 1 and this heart, where a deep ulceration of the aorta and a slight ulceration of the aortic cusps are associated with a vegetative affection of the mitral flap and of the infra-aortic surface opposed to it. The lesions are of greater superficial extent, but they present the aspect of healing rather than of degeneration. Though none of the chordæ are ruptured several of them present nodes, which, however, are not granular, but smooth, as though there had been for some time no further deposit of fibrin or micro-organisms upon them. By the side of the few vegetations which remain on the mitral flap there are many smooth knobs of the same character, free from granular fibrin, and glistening as though clothed with endocardium. The fibrin of the vegetations is nowhere grey, but of pink or opaque white aspect. In short, most of the appearances suggest that the destructive stage of the disease had been got over and the necrotic surfaces cleared away, although the healthier fibrin had not been removed nor converted into fibrous tissue. The aortic valves present no rough fibrin, but nodules only. The aortic ulceration is situated just in front of the mouth of the coronary orifice, and the fine nodular deposits close to the ulceration suggest that fibrin might have grown there which had since disappeared. Some fibrin remained, and this helped to plug the channel of the coronary artery. The fatty degeneration visible in the left ventricle was apparently the secondary result which brought about death, and was probably due to coronary obstruction.

An inspection of the three hearts side by side shows beyond any doubt—(1) that in all three the disease was malignant endocarditis; (2) that in Case 1 the lesions were considerably more severe and more active. In this heart are seen—(*a*) several ruptured chordæ; (*b*) abundant shaggy vegetations of greyish, granular, necrotic fibrin. But in the other two hearts, although the vegetations are numerous, none of them present the same grey necrotic aspect or the same granular crumbling surface. None

of them are broken at their extremity, but rounded, as though any ulceration or rupture had been smoothed over. Many of them, too, are beady or knobby, as though they had cast off their appendages. The general impression is conveyed, by the firm look, the clear pinkish colour, and the glistening surface of these vegetations, that they are in a stage of regression of the fibrinous or bacterial deposit.

These differences might be regarded as due to an original difference in the virulence of the affection. It is impossible to form any definite opinion as to what appearances might have been presented by these hearts at their worst stage. The partial destruction of the aorta in both of them is unmistakable evidence of the extreme activity of the disease at some previous time, but it does not appear from the specimens that the worst stage was the final one. On the other hand, the clinical records of the cases enable us to follow the history of the lesions.

In one of them, Case 3, the onset of the acute stage had occurred six months previously, and acute symptoms lasted throughout almost to the end. Yet the terminal condition of the endocardium was not of the worst type. And the progress noticed in the symptoms bears out the view that the endocarditis itself was not in the stage of progression. The same remarks apply in a modified degree to Case 2.

The pathological conclusion which we feel justified in drawing is that the changes in both these cases had previously been of a more active type than they were shortly before death, whereas in the first case the endocarditis was at its acme.

Remarks on the Cases.

In addition to a few remarks as to the source of infection, special attention will be given to the following points :—(1) The insidious onset, and (2) the often pro-

longed duration of the disease; (3) the extreme emaciation and other symptoms simulating phthisis; (4) the difficulty of a correct diagnosis even at a late date. But the chief object of this communication is to illustrate further points of practical importance:—(5) The fatal tendency of the disease if not treated; (6) the possibility of arrest, and of partial recovery even at an advanced stage; and lastly, (7) the share taken by treatment in aiding recovery, and suggestions as to the probable mode of action of the remedies.

In two of the cases an arthritic and probably rheumatic element was present. Although we are scarcely warranted in regarding common rheumatic infection as capable of development into the malignant forms of endocarditis, it is possible that the micro-organisms of rheumatism may have acquired a higher degree of virulence, either alone or in association with other septic organisms, and may have led to the ulceration. Perhaps, however, the cases were due to simple septic infection, and if so, a sufficient supply may have existed in the oral sepsis at least in two of the cases. The blood was found free in one case from staphylococci and streptococci, and in two of the cases the injection of antistreptococcus serum failed to check the disease. In Case 2 there was no recent rheumatic element, nor any other known source of infection beyond that which, from its extent and degree, may be provisionally regarded as an adequate cause, viz. the oral sepsis from decaying teeth.

(1) On the subject of the insidious mode of onset the clinical histories supply their own comment. None of the cases presented initial symptoms identifying the acute attack as one of cardiac disease, nor any subsequent clinical events identifying a late onset, whilst the post-mortem appearances were those of lesions of old standing. Sometimes cerebral embolism occurs whilst the patient is engaged in laborious work, and death follows in a few days. Instances of this kind, such as that diagnosed and treated by one of us as malignant endocarditis probably

originating in dental caries,[1] supply a complete proof that the lesion need not at first give rise to any marked symptoms. The fact that a period of latency may exist in some cases is of clinical importance.

(2) Where an abrupt termination is not occasioned by any major complication the disease under ordinary treatment and nursing may run a protracted course, which in our cases, without allowing for any preliminary period of latency, reached a duration of probably not less than 13, 14, and 26 weeks respectively.

(3) Most striking is the intense emaciation often observed. In our three cases it was the more remarkable owing to its contrast with the liberal amount of food taken, and to the absence of any wearing influence except that of the fever. It was considerable in all three cases, but in Case 3 it was extreme, and such as is seen only in starvation or in the ultimate stage of phthisis. The general aspect of the patients was also that of advanced pulmonary tuberculosis. The hectic temperature without rigors was a point of resemblance between the cases.

(4) Diagnosis is probably impossible at the earliest stage of most cases. Even should a murmur be detected it seems doubtful, having regard to the latency of symptoms referred to above, whether its due significance would be put upon it. Increasing responsibility attaches to diagnosis in the later stages, and the history of each case shows that even then the symptoms may not reveal their real cause.

In all three cases the provisional diagnosis of pulmonary tuberculosis was made at first sight prior to searching examination, and in one of them phthisis or latent suppuration had also been diagnosed, prior to the patient's admission, at a consultation held among physicians in the country.

[1] "A Case of Fatal Malignant Endocarditis apparently due to Infection from Dental Caries and Stomatitis treated by Antistreptococcus Serum and by Saline Injections," read before the Ipswich Meeting of the British Medical Association, 1900, by Wm. Ewart, M.D.; cf. 'Brit. Med. Journ.,' 1900, vol. ii, pp. 906 and 1057.

(5) The cases illustrate the slowly destructive tendency of the disease apart from the fatal complications which so often shorten its course.

(6) The most important teaching conveyed by the cases is that the cardiac lesions of malignant endocarditis are not incompatible with recovery under treatment. Whilst in the case which was under treatment for rheumatism there was no sign of improvement, nor any evidence of repair in the lesions traceable after death, in the other cases in which systematic antiseptic treatment was applied perceptible progress was made clinically, and the improvement was such as to warrant a hope of ultimate recovery, death supervening, particularly in the male case, almost as an accident. The reality of the improvement observed in the patients was borne out by the post-mortem appearances in the heart, where some of the destructive changes had made way for the changes of repair.

(7) Therapeutical conclusions are always difficult, but in this set of cases they are facilitated by the clinical record. In the first case a purely antirheumatic and restorative treatment failed to stay the downward progress. In the other two, antistreptococcus serum from the Jenner Institute was obtained and given a fair trial. But here an important question must remain unanswered. The serum may have made some impression upon the vegetations, but during its administration it had no influence upon the clinical progress of the patients. In one of the latter definite depression was induced by the injections, and the amount had to be reduced. It may be said, then, that the curative value of antistreptococcus serum injections has received better support from some other published cases than from these.

In both cases improvement occurred very soon after protargol inunctions were adopted as an adjunct to previous treatment, and as the improvement continued when the serum injections were left off, it seems warrantable to think that the inunctions had a definite share in

the result. If correct, this view would corroborate similar observations which have been reported by others, and it would warrant our recommending for further trial a method which is entirely free from inconvenience or complications. Questions as to the selection of the ointment, whether unguentum Credé, or protargol ointment, or any other, as to its strength, and as to the frequency of its use, may be left for further trial.

Addendum by Dr. Ewart.

The treatment which I had intended to apply in these cases, but which was postponed in view of the partial improvement otherwise obtained, does not hitherto rest upon any direct observations made in this disease, but upon the good results, hitherto unpublished, which I have obtained in acute pneumonia from intra-venous injections. I believe that the rational, and it is to be hoped the successful treatment of endocarditis will be found in the intra-venous injection method.

Intra-venous injections of perchloride of mercury have been used with marked success in the human subject for the cure of Werlhof's disease by A. Lusignoli.[1] Fischer[2] reports the cure of a malignant carbuncle due to anthrax infection, without any incision, by intra-venous injections of colloidal silver. These facts add some support to the view which I have ventured to formulate; and being satisfied from a personal acquaintance with the method that intra-venous injections may be administered by the expert without too serious a risk, I should be disposed to adopt them in any obvious instance of a disease which, as these cases show, almost inevitably tends to a fatal termination.

[1] Cf. 'Brazil-Medico,' Oct. 15th, 1901, and 'Journal of the American Medical Association,' Dec. 21st, 1901, p. 1713.

[2] Cf. 'Münch. med. Wochenschrift,' Nov. 19th, 1901.

For Discussion see page 239.

A CONTRIBUTION

TO THE

STUDY OF MALIGNANT ENDOCARDITIS

BY

F. J. POYNTON, M.D., M.R.C.P.

AND

ALEXANDER PAINE, M.D.

Received December 4th, 1901—Read April 8th, 1902

I. Introductory Outline.

(A) *A Group of Cases of Malignant Endocarditis closely associated with Rheumatic Fever.*

While investigating the pathogenesis of rheumatic fever, our attention has been directed to certain cases of progressive heart disease which run a more or less prolonged course, and terminate almost invariably in death. After death it is found that the valves of the heart are very extensively diseased, and that the morbid process is often extremely active. Among such cases there is one group in which we were particularly interested, for previous to the fatal illness there had been a history of rheumatic fever; sometimes there had been repeated attacks, and

during the last illness symptoms had arisen which suggested that rheumatism of some unusual type was in reality the true excitant. The symptoms in these cases arose insidiously, and there was no local focus of suppuration, no wound or other demonstrable cause which may be considered to have been the starting-point of this progressive form of heart disease. This class of case is well recognised, for it is a comparatively common one in the large hospitals. It is possible in some instances to detect the nature of the disease even early in the illness, because of the persistently excited action of the heart and loudness of a systolic murmur; but on the other hand, even when death has occurred, several observers of equal acumen, and with the same advantages in the study of the case, may differ in their opinion as to whether the condition is one of rheumatic morbus cordis or so-called "infective endocarditis." No doubt the great majority of these cases as they progress diverge more and more from the appearance of rheumatic fever, and the force of the disease falls so exclusively upon the cardiac valves that it may be difficult in the end to detect any clinical resemblance; but it is equally certain that the more these cases are carefully studied, the more difficult it is to say where a distinctive line can be drawn between them and acute rheumatism. Anæmia, prostration, wasting, pyrexia, and infarction are very frequent and important symptoms in this disease, but there is not one of these which may not occur, to a lesser degree, in severe rheumatic fever. In these cases, again, suppuration does not occur even in the blood-clot of the aneurysms that may result, but numerous white infarcts are often found in the kidneys, lungs, or spleen after death.

(B) *"Malignant" Preferable to "Infective" as a Title for this Form of Endocarditis.*

The usual procedure in this country is to describe such cases as examples of "infective endocarditis," and if by

this term no suggestion were implied that rheumatic valvulitis was non-infective, the description would be an excellent one. It is unfortunate that such is not the case, but that through no fault in the term itself the name in question has been widely used in contradistinction to rheumatic valvulitis; and this is the more strange because for several years rheumatic fever has, in spite of the absence of actual proof, been looked upon as due to an infection.

For this condition the name malignant endocarditis seems to us preferable, for whether it proves fatal or not, the type is malignant.

(C) *Researches of other Investigators upon Malignant Endocarditis.*

An immense amount of research has been devoted to the study of malignant endocarditis, and it would be impossible in such a paper as this to mention the names of the many investigators. Their results have been of far-reaching importance. They have definitely settled the microbic origin of the condition. They have also shown that various micro-organisms may give rise to malignant endocarditis, but that the most usual cause is a streptococcus. Experiments upon animals have resulted in the reproduction of the disease, though not with constancy, and in some cases the cardiac valves have been damaged mechanically before valvulitis has resulted. It may be justly asserted that these investigators have elucidated the broad outlines of the pathology of malignant endocarditis, though there are several difficult problems yet to be solved, among which is the relation of such cases to rheumatic fever.

(D) *Renewed Investigation of Malignant Endocarditis Desirable.*

Heretofore it has not been possible to solve this problem, for there has been no agreement upon the cause

of rheumatic fever. The outcome of this limitation of knowledge has been the wide-spread belief that malignant endocarditis in a rheumatic patient is invariably a result of some secondary infection of the tissues injured by previous rheumatism. Nevertheless there have been some clinicians and pathologists who have felt this attitude to be too rigid, and have, without the means of bringing forward complete proof, believed that some cases are truly rheumatic in origin. At the present time so much evidence has been obtained in favour of rheumatic fever being the result of a diplococcus infection, that it seems a proper occasion to once more investigate this question of the relation of the two diseases.

(E) *The Result of the Authors' Investigations.*

It is this investigation with which our paper is concerned, and our conclusion can be shortly stated thus:—*That there is a group of cases of malignant endocarditis which is rheumatic in nature.* How comprehensive this group will prove to be further investigations alone can decide.

Before we summarise the reasons for this conclusion we are anxious to make clear the scope of our paper. We do not claim that the view that rheumatic fever is a cause of malignant endocarditis is an original one; we are well aware that others—as, for example, Ogle, Osler, Peter, Burkart, and Fernet—have entertained this opinion; that others before us have demonstrated that organisms similar in their morphology may occur in the two diseases, and have felt that in some instances no clinical distinctions can be drawn between simple and malignant valvulitis. Our paper, as its title claims, is but a contribution to the study of malignant endocarditis, and affords, we believe, a strong support of the view that malignant endocarditis may be of rheumatic origin.

(F) *Reasons for the Assertion that there is a Malignant Rheumatic Endocarditis.*

The chief reasons upon which we rely for support of our assertion can be summarised thus :

Firstly. The probability that some of these cases are rheumatic is in accord with clinical experience.

The clinical cases we record will bear out this statement.

Secondly. The probability that some of these are rheumatic is in accord with pathological experience.

The minute investigation of the morbid anatomy of the clinical cases we record supports this conclusion.

Thirdly. The probability that some of these cases are rheumatic is in our opinion in accord with bacteriological experience, for—

1. A diplococcus is a cause of rheumatic fever. The evidence in favour of this we have already dealt with in a series of papers published during the last two years.

2. A diplococcus can be isolated in pure culture from these cases of malignant endocarditis, which will reproduce the disease in rabbits.

3. The cultural and morphological characteristics of these two diplococci resemble one another so closely as to lead to the conclusion they are identical organisms.

4. The *Diplococcus rheumaticus* will produce malignant endocarditis, indistinguishable from that produced by the diplococcus isolated from certain cases of malignant endocarditis in man.

5. The *Diplococcus rheumaticus* may produce in a rabbit first a recoverable illness with the manifestations of rheumatic fever, and then on a second inoculation malignant endocarditis.

6. A diplococcus isolated from certain cases of malignant endocarditis in man will produce not only malignant endocarditis in rabbits, but a condition indistinguishable from the disease we believe to be rheumatic fever.

7. By these diplococci, every grade of valvulitis from simple to malignant, and from malignant to simple, can be produced, as our macroscopic specimens bear witness.

II. The Investigation.

(A) *Clinical, Experimental, and Pathological Observations.*

The first case will make clear the type we are engaged in studying.

Case 1.—A child, aged 11, was admitted to St. Mary's Hospital, under the care of Dr. W. B. Cheadle, upon October 22nd, 1897, and died November 12th. When three and a half years of age he had suffered from rheumatic fever, and when five and a half from scarlet fever. His mother had suffered from rheumatic fever. Five weeks before admission there had been swelling of the knees and ankles, and for five months there had been complaints of obscure pains in the chest and abdomen. There was no history of an injury, no suppurating focus, and no obvious cause which could be looked upon as an explanation of some secondary infection. Upon admission the boy was very anæmic, the temperature was 100·8°, pulse 100, respirations 28. The heart was much enlarged, there was a loud systolic mitral murmur, and also an aortic systolic murmur. The liver and spleen were enlarged, the urine was natural. Soon after admission crepitations were heard at the base of the left lung posteriorly, and there was pain in the left side.

Upon October 28th blood and albumen were found in the urine, and until death, upon the 12th of November, there was irregular pyrexia. Hæmaturia became persistent, and casts were found in the urine. There was pain over the spleen, and progressive enlargement of that organ. Purpura, vomiting, progressive anæmia, emacia-

tion, and sweating were prominent symptoms, and finally the pulse became irregular and intermittent, and death resulted from cardiac failure.

The necropsy showed recent pericarditis, with two ounces of fluid in the pericardium, which contained a few flakes. There was extensive ulceration of both flaps of the mitral valve, and exuberant granulations spread over the surface of the auricle. The valves upon the right side of the heart were not affected; the heart itself was hypertrophied and dilated. There were numerous white infarcts in the spleen, with perisplenitis; it was soft, and weighed five ounces. There were numerous white infarcts in the kidneys, but none found in the lungs. Numerous subserous hæmorrhages were visible along the intestines. There were no abscesses, but many white infarcts, as already stated. Numerous micrococci were found in the granulations.

We admit that secondary infections can occur during life without any demonstrable cause, but it seems to us legitimate to argue upon such a case as this in the following way:—Rheumatic fever is a bacterial disease, and one which apparently does not confer immunity. Evidence at present points to it as the result of a diplococcus infection, and it would appear that the diplococcus may exist for long periods in the body.

In such a case as the above there was a family and personal history of rheumatism, and such a child, as all clinical experience has shown, may be justly termed rheumatic. If, then, from such a case a diplococcus be isolated, it is as legitimate to assume that it may be the *Diplococcus rheumaticus*, under some unusual conditions, as to assume a secondary infection. The proof must rest upon an accurate study of the micro-organism which is isolated, by various methods, including among these the method of experiment.

Case 2.—The next case was that of a woman aged 50, who was admitted to St. Mary's Hospital, under Dr.

W. B. Cheadle, in June, 1898, for dyspnœa of some months' duration. The only cause that was given for this dyspnœa was an attack of rheumatic fever eight years previously. Upon admission she was cyanosed and short of breath, and complained of pain in the left side. There was orthopnœa. The temperature was 102·8°, pulse 103, respiration 40. The heart was much enlarged, and there was a mitral systolic murmur. The hands were deformed by previous attacks of rheumatism. The nature of the case remained quite in doubt, though towards the end irregular pyrexia, infarctions in the lungs and spleen, and purpura suggested the diagnosis of malignant endocarditis.

The necropsy showed recent pericarditis, adhesive in type, and also old adhesions, the result of a previous attack.

The mitral, tricuspid, and aortic valves showed extensive vegetative endocarditis, and there were vegetations over the surface of the left auricle. There were white infarcts in the lungs and spleen, but none in the kidneys. There was no suppuration. Numerous micrococci were visible in the granulations.

The necropsy disclosed malignant endocarditis of the characteristic type, yet clinically this case was most obscure, and resembled at first a severe rheumatic morbus cordis. It was not until the end of the illness that the malignant character of the disease became apparent.

Case 3.—A patient aged 16 was admitted into St. Mary's Hospital in January, 1900, under Dr. Lees, suffering from morbus cordis. When six years of age he had an attack of rheumatic fever, and since that time had suffered from three more definite attacks. His mother had suffered from rheumatic fever. The final illness had commenced insidiously, with pain round the heart, and three weeks before admission there had been pains in the ankles and knees. No cause was assigned for this illness,

and on special inquiry of the mother she volunteered that she had thought this was another attack of rheumatism, because it commenced just as the previous attacks had done.

On admission the patient was very anæmic and wasted, and there was irregular pyrexia, with well-marked mitral and probably aortic disease.

The course of the illness was progressive and malignant in type. Irregular fever, enlargement of the spleen, and hæmaturia, with progressive anæmia and emaciation, were the prominent symptoms, and throughout the time that the patient was in the hospital no doubt was entertained as to the nature of the illness.

The necropsy showed a few ounces of clear fluid in the pericardium; the mitral valve was fringed with numerous minute vegetations, varying in size from a pin's head to a pea. There were recent vegetations upon the aortic valve, but the right side of the heart was unaffected. In the spleen there were three white infarcts, and in the left kidney one.

There was no suppuration.

This case was, in one respect, the converse of the preceding. The clinical diagnosis was quite definite, but the post-mortem showed a condition which, without the clinical history for a guide, could have been explained as active rheumatic morbus cordis, and not as malignant endocarditis.

We investigated the bacteriology of this case, and at first included it (the resemblance was so striking) among our first series of cases of rheumatic fever published in the 'Lancet' in September, 1900; but we finally concluded, before publication, that it was better to rigidly exclude a border-line case of this kind, and have not made allusion to it until the present paper.

The bacteriological investigations resulted as follows:

Numerous diplococci growing in chains were demonstrated in films made from the granulations of the mitral

valve, and cultures were made with the following media: —agar, ascitic fluid, acid and alkaline bouillon, an alkaline pork medium, and in milk and bouillon slightly acidified with lactic acid. The liquid media were incubated both aërobically and anaërobically.

Twenty-four hours afterwards the results were as follows:

Upon agar, a poor growth of minute discrete colonies consisting of extremely minute diplococci. The pork medium and ascitic fluid were sterile. The alkaline bouillon showed a very poor growth of minute diplococci.

The acid milk, both aërobically and anaërobically, showed a vigorous growth of diplococci in chains. This diplococcus was grown in the milk medium by means of subcultures for two months. From the original tubes a subculture was made upon blood-agar, and upon two occasions Mr. Plimmer injected into the auricular vein of a rabbit the contents of one blood-agar tube. The result in each case was negative.

The isolation of a minute diplococcus from a case such as this is in accord with the experience of Professor Litten,[1] who also isolated a minute diplococcus from a condition which he termed the malignant non-septic form of rheumatic endocarditis. Such cases as these he considered to be characterised by pyrexia, infarction, pallor, and sweating, with hæmaturia and enlargement of the spleen, but no suppuration.

Professor Litten was inclined to the view that this diplococcus was probably not identical with the diplococcus described by Professor Wassermann[2] as the cause of rheumatic fever. We believe that the diplococcus we isolated in this case is identical with the diplococcus

[1] "Ueber die maligne (nichtseptische) Form der Endocarditis rheumatica," 'Berliner klinische Wochenschrift,' 1899, No. 29, p. 644.

[2] "Ueber den Infectiosen-Charakter und den Zusammenhang von acuten Gelenkrheumatismus und Chorea," 'Berliner klinische Wochenschrift,' 1899, No. 29, p. 638.

which we have now isolated from twenty cases of rheumatic fever.

Case 4.—This fourth case, although a case of rheumatic fever, we mention here because it resembled malignant endocarditis in this respect, that during life upon two occasions diplococci growing in chains were isolated from the blood. The patient was under the care of Dr. D. B. Lees, and the case was published in full in the 'Mirror of the Lancet,' October 28th, 1899, and was the first from which we isolated the diplococcus of rheumatic fever from the blood. We naturally thought at that time that the case was one of malignant septic endocarditis, because we isolated streptococci from the blood during life, though previous investigations had made us somewhat doubtful of the validity of this conclusion. The clinical history, the course and character of the disease, and the result of the necropsy proved conclusively that it was a case of severe rheumatic fever. Though a most severe case of rheumatic fever with numerous and severe *local* lesions there was no suppuration, and yet during life there was a streptococcus, or, to be more exact, a diplococcus which grew in chains, circulating in the blood-stream.

Case 5.—A boy aged 10 was admitted to St. Mary's Hospital in April, 1900, for heart disease, under the care of Dr. W. B. Cheadle. Six weeks before admission he had suffered from pain over the heart, sweating, and attacks of diarrhœa. A year previous to this he had been in the hospital for an attack of rheumatic fever, and one brother had also suffered from rheumatic fever. On admission aortic and mitral valvulitis were discovered, and a very noticeable feature was extensive muscular wasting.

Upon April 30th he developed pericarditis.

In May there was arthritis, the ankles and knees being affected. There were also diarrhœa and vomiting. In June crepitations were detected in the lungs. In July

infarction, sweating, and wasting were prominent, and death occurred in July, after eighty-eight days of irregular pyrexia.

This appeared to us during life to be a classical case of rheumatic malignant endocarditis.

There was unfortunately no opportunity of obtaining a complete necropsy, but the heart was removed, and the pericardium was found generally adherent. The heart itself was very little enlarged, but upon the mitral and aortic valves and on the wall of the left auricle there were extensive and exuberant granulations. The right side was not affected. Films showed minute diplococci in chains. Aërobic cultures in the milk medium were obtained and transferred to blood-agar. A series of important experimental results followed.

The growth from six tubes was intra-venously injected into a rabbit on July 28th, and upon the 31st and 1st of August the left knee-joint and left shoulder-joint were swollen. The animal died suddenly upon the fifth day. The necropsy showed exuberant granulations upon identical valves, namely, the aortic and mitral. The micro-organisms were demonstrated in great numbers in the damaged valves.

In thus reproducing malignant endocarditis without any previous injury to the cardiac valves, we confirmed the classical investigations of Dreschfeld, Ribbert, Bonomé, Roux, Mannaberg, and others. It will also be apparent that in the course of this investigation we have confirmed the results of other observers by the experimental production of infarction and hæmorrhages.

Upon August 8th a second inoculation, from a culture obtained from this rabbit, was made into a smaller animal. Death occurred upon the fifth day from vegetative aortic valvulitis. No other valve was affected.

The cultures from this rabbit were contaminated with the *Bacillus coli*, so recourse was had to the original culture, and a third inoculation made with a smaller quantity of the organism.

Death occurred on the nineteenth day. There was arthritis of the right knee and diarrhœa, but no clinical evidence of valvulitis or pericarditis. Death occurred from gradual cardiac failure due to dilatation and fatty degeneration of the heart muscle with ante-mortem thrombosis. In this case it will be observed there was no manifestation of malignant endocarditis, but the necropsy showed a simple endocarditis.

A larger quantity of the original culture was used for a fourth injection.

Death occurred on the tenth day. During life there were noted diarrhœa, heart disease, and arthritis of the right shoulder-joint. The necropsy showed well-marked malignant mitral valvulitis, white infarcts in both kidneys and in the spleen, but no pericarditis (Plate 6, figs. 1 and 2).

A smaller quantity of the original culture was injected into a fifth rabbit, which was killed—for it was moribund —upon the tenth day. During life diarrhœa, pericarditis, and arthritis were noted.

The post-mortem confirmed that this condition was one of rheumatic fever.

The culture from this case was injected into a sixth rabbit, and death occurred upon the tenth day. There was arthritis, but no valvulitis. The heart's action was, however, extremely rapid, and for some days there was a mitral systolic murmur.

It is evident from this series of inoculations that in three nstances definite malignant endocarditis resulted, in two death occurred from cardiac failure—without malignant endocarditis,—and in one case death occurred from pericarditis.

Arthritis was frequent. One symptom occurred which we have not noticed in rabbits inoculated with the *Diplococcus rheumaticus* from rheumatic fever, namely, diarrhœa; and this we know occurs not infrequently in man during the course of malignant endocarditis, and was a prominent symptom in the case from which this organism was isolated.

Case 6.—A boy aged 13 was admitted into St. Mary's Hospital, November, 1900, for morbus cordis, under the care of Dr. Lees. Six years before he had suffered from enteric fever, and three years before from pneumonia and rheumatic fever. He had been ailing for two months previous to admission, and had suffered from pains in the chest and abdomen. The boy was pale and sallow, but well nourished; there were mitral and aortic disease, and an enlarged spleen. He remained in the hospital until his death in January, and during that time there was usually irregular pyrexia, though sometimes for days the temperature remained normal. Death was sudden.

The necropsy showed general pericardial adhesion, and fungating masses of vegetation upon the mitral and aortic valves. There were petechiæ under the capsule of the liver. The spleen weighed fifteen ounces, was tough in consistence, and contained one recent infarct. There were numerous small hæmorrhages in the cortices of both kidneys. There was no suppuration. Two hours after death the mitral valve was exposed, and four tubes of the acid milk medium inoculated with fragments of the granulations. In two out of four there was a pure growth of very small diplococci growing in chains. Two were sterile.

Upon January 24th the growth from six small tubes was injected into a strong rabbit at 1 noon.

At 3 o'clock the temperature had risen to 105·2°, and a blowing systolic murmur was audible.

During the rest of January the temperature was raised, there was some diarrhœa, and the heart was rapid.

During February there was improvement, but occasional fever.

During March improvement continued.

Upon April 8th the hind limbs were found completely paralysed, and there were complete incontinence and loss of tone of the anal sphincter. The diplococci were isolated from the urine and the animal was killed. There was no definite valvulitis or pericarditis, and nothing to be

found in the other viscera of importance except a hæmorrhage into the pia mater some quarter of an inch in vertical extent immediately above the lumbar enlargement.

It will be noticed that whether because the resistance of the animal was unusually great, or the initial inoculation not sufficient, the disease was not reproduced; but the length of time (ten weeks) that the diplococci remained active in the body is a point of much interest.

Another inoculation from the original culture was made upon January 25th, a day after the former inoculation, into a rabbit of smaller size. The animal was killed upon the tenth day; during life there were pyrexia and morbus cordis.

The post-mortem showed well-marked vegetative mitral valvulitis (Plate I, fig. 1), petechiæ in the heart wall, a white infarct which was softening in the left kidney, also white infarcts in the right kidney and spleen.

A pure growth of the diplococcus was obtained from the blood in the heart.

The third rabbit was inoculated from a culture from the preceding, and died in the night of the third day. The heart throughout the illness was extremely excited.

The necropsy showed the nearest approach to septicæmia we have seen with this diplococcus. Except for a minute granulation upon the aortic flap of the mitral and early peritonitis, there was no local lesion to be seen. Numerous diplococci were present in the granulation. There was excess of fluid in the pericardium, and numerous diplococci were present in the pericardial tissues.

The liver was pale; the kidneys pale; the spleen large, soft, and dark. The lungs showed no definite changes.

A fourth rabbit was inoculated from a culture from the preceding, and death ensued upon the sixth day. The necropsy showed pericarditis, with a fibrino-cellular exudation; slight mitral valvulitis, a small white infarct in the left kidney, and some perisplenitis—a condition of rheumatic fever.

A fifth rabbit was inoculated with a culture from the fourth, and died on the fourteenth day of severe pericarditis. The necropsy showed general recent pericardial adhesion, and a condition which resembled the severe general plastic pericarditis in the rheumatic fever of childhood.

There was no valvulitis.

Thus again it will be seen that both malignant endocarditis and a condition we believe indistinguishable from rheumatic fever had been produced by inoculations of this diplococcus.

This concludes our clinical investigations, though we would emphasise the fact that in some of these cases of malignant endocarditis in rheumatic subjects rigors may occur for many weeks, yet after death not a trace of suppuration be found, and infarcts be discovered to be cicatrising. We have also obtained from post-mortem records thirty cases of malignant endocarditis without the mention of an abscess in any one, and all of them giving a previous history of rheumatic fever.

The next experiment illustrates that a culture originally obtained from a case of rheumatic fever may produce the malignant type of endocarditis after it has been passed through several rabbits (13).

In June, 1901, an intra-venous injection was made into a rabbit from a culture which was the direct descendant of the original one obtained from the pericardial fluid of a fatal case of rheumatic fever in March, 1900.

A very loud mitral murmur developed upon the seventh day, and the animal died upon the eighth. The necropsy showed that there was a large fungating vegetation upon the mitral valve, with white infarcts in the kidneys and spleen and one small one in liver. The condition was one of characteristic malignant endocarditis (Plate I, fig. 2).

The next experiment proves that a rabbit may survive a first inoculation with the diplococcus of rheumatic fever, recover completely, except for a slight thickening of the

mitral valve, and then may die from malignant endocarditis, the result of a second inoculation.

The first injection was made from a culture of the diplococcus obtained from a boy suffering from acute rheumatic pericarditis. Treatment had necessitated a venesection, and the organism was isolated from the blood of the living patient.

The injection was made upon March 27th, 1900, and four days afterwards there was arthritis of the right knee-joint. Later the animal became thinner and irritable, both knee-joints were affected, and the heart sounds were very rapid and weak. In May recovery commenced, and eventually the animal regained health.

Six months after recovery from the previous illness the second inoculation was made, upon January 30th, 1901. The culture used was from the pericardial exudation of a fatal case of rheumatic pericarditis. The original growth had been obtained in the pericardial fluid itself in March, 1900. This organism had repeatedly caused rheumatic fever in rabbits, and two specimens of simple valvulitis caused by it are shown among the macroscopic specimens.

For some months the organism had been kept growing in the acid milk medium, but it had not of late been passed through an animal, and we were doubtful whether it had not completely lost all virulence.

The organism was transferred to blood-agar tubes in the usual manner, and an exceptionally large amount used for inoculation.

The temperature upon the next day was 103°, but until the fourteenth day we noticed no change at all, and then we found the heart very excited. This in a rabbit is not a reliable sign of cardiac disease, and as there was no murmur we somewhat hastily concluded that there was no result to be expected. The animal was found dead on the nineteenth day.

The necropsy made the cause of death quite clear. The heart was large and the cavity of the left ventricle dilated. Vegetative endocarditis of the aortic valve had

practically closed the lumen of the aorta, and the aortic ring was thickened. Minute beads were found fringing the mitral valve, and its aortic cusp was thickened by previous endocarditis.

From the aortic vegetations the diplococcus was isolated, and was demonstrated in the sections of the valve. There were no infarcts. It must, we think, be allowed that this was a very remarkable and suggestive result.

Two macroscopic specimens of rabbits' hearts are also shown, one resulting from an injection with the *Diplococcus rheumaticus*, and one from the diplococcus obtained from a case of malignant endocarditis, which illustrate the transitional phases of the valvulitis (Plate I, fig. 3), and also a third specimen showing primary malignant tricuspid endocarditis produced by the diplococcus of rheumatic fever.

The remainder of the series of experimental investigations we must record very briefly. These investigations were made with the *Streptococcus pyogenes*, and emphasise, we believe, the salient points of our previous results.

Upon two occasions virulent cultures of the *Streptococcus pyogenes* obtained from a case of puerperal fever were supplied to us from the Jenner Institute. The virulence had been increased by passing the organism through a series of rabbits, and the cultures that we received may be looked upon as characteristic of the virulent *Streptococcus pyogenes*.

We treated this micro-organism in the same way that we did the *Diplococcus rheumaticus*, that is, transferred it first to the acid medium, and thence to blood-agar. The only difference in detail was the use for inoculation of a small part of the growth from one tube instead of the growth from some four or six tubes. With such a small quantity as this, in our experience, no result is obtained with the diplococcus of rheumatic fever.

The rabbits died in every instance within twenty-four hours of inoculation. The post-mortem appearances

differed widely from those which we have previously described. There were hæmorrhages from the mucous surfaces. The blood was fluid, the spleen large, dark and soft, the kidneys pale and extremely friable. There were no local lesions, such as arthritis or valvulitis. Microscopic examination of the organs showed great numbers of streptococci in the blood capillaries and tissues.

On each occasion this condition of septicæmia resulted, and although we cultivated the streptococcus for a week in the acid medium (a medium which is not considered to be a favourable one), the result on inoculation was the same.

It may be objected to these results that the virulence of the streptococcus had been artificially raised, and that they are not therefore comparable to our previous investigations, but this objection cannot be raised against the next case. A woman was admitted to St. Mary's Hospital, suffering from septic absorption from a suppurative phlebitis. An operation cured her, and from the pus the *Streptococcus pyogenes* was isolated and cultivated in the acid medium, and then transferred to blood agar. Intravenous inoculation of a rabbit resulted in death within twenty-four hours from a condition of septicæmia of the same nature as that which resulted from the streptococcus sent to us from the Jenner Institute.

The last experiment was made with a streptococcus isolated from the pus of a suppurative pericarditis. The patient, a boy, had died from a streptococcus pyæmia, the result of a punctured wound of the right knee-joint.

The same procedure was adopted as before, and on this occasion the rabbit lived for five days, during which time arthritis of the right carpal joint developed.

The post-mortem showed purulent arthritis, small abscesses in the liver and both lungs, a clear exudation in the pericardium, and a fibrino-cellular exudation in the pleuræ. There was no valvulitis.

These investigations with the *Streptococcus pyogenes* serve to show more distinctly the definite character of

the results we have obtained with the diplococcus of rheumatic fever and the diplococcus isolated from certain cases of malignant endocarditis. We do not pretend for a moment that they settle the question of the relation of these various processes to one another, but they demonstrate that, as in man, characteristic rheumatic fever and this type of malignant endocarditis, and pyæmia and septicæmia from the *Streptococcus pyogenes*, are different conditions, and suggest that there must be some very definite reason for such differences.

These clinical cases, the experimental investigations, and our specimens show, we believe—

Firstly, that the probability that some of these cases of malignant endocarditis are rheumatic is not contrary to clinical experience.

Secondly, that a diplococcus is the cause of some of these cases of malignant endocarditis.

Thirdly, that this diplococcus will reproduce in rabbits malignant endocarditis, and also a condition we consider to be rheumatic fever.

Fourthly, that the *Diplococcus rheumaticus* will produce malignant endocarditis.

(B) *Histology.*

The minute anatomy of the two conditions is the next consideration.

If a necropsy is made upon a characteristic case of rheumatic fever and upon a case of malignant endocarditis of the type under consideration, the most striking feature in which they differ is found to be the condition of the damaged cardiac valves.

In acute rheumatism there are minute vegetations, in malignant endocarditis there are as a rule large exuberant masses, with possibly also ulceration of the valve substance and rupture of chordæ tendineæ. Yet these large vegetations, save in one respect, do not differ in their microscopic structure from the minute ones. There is the same

necrosis, the same cell infiltration, the same swelling of the connective tissue.

If a careful search is made in the damaged valve of rheumatic fever, the diplococci may be found in regions where the process has not reached the limit of necrosis (Plate III, fig. 2), though the search is not easy because the fibrous framework of the valve is not an easy structure to examine minutely. If search is made in the necrotic part of the vegetation, all attempt to demonstrate the micro-organisms may and probably will be met with failure; they have been for the most part destroyed. But in malignant endocarditis they are found in masses, sometimes fringing the free edge of the vegetation, sometimes buried in the necrotic tissue (Plate IV, figs. 1 and 2).

This then, we believe, is the essential difference in the morbid anatomy of the two conditions. Hence it is that in rheumatic fever, death does not occur from acute valvulitis but from peri- and myocarditis, whilst in malignant endocarditis death occurs almost invariably from valvulitis and its secondary results; though occasionally during the illness, sometimes within the last few days of life, pericarditis may develop. Hence it is that numerous white infarcts occur in the malignant form, and are exceptional in the simple. The white infarcts need no detached clot or fragment of vegetation for their formation, it is sufficient that a considerable mass of the micro-organisms be carried to the spot, and there set up by their poisonous action the phenomenon of coagulation necrosis and those other changes which make these lesions, as it were, visceral nodules. Upon innumerable occasions these organisms, which grow so vigorously in the vegetation, are scattered in every direction by the blood-stream, and give rise to the irregular fever, the sweating, the prostration and wasting. In the heart the process steadily advances, but it by no means follows, and indeed does not follow, that the secondary foci in the various viscera will also of necessity steadily progress. The place of election in this disease is the heart, and no one can seriously doubt that

the chemistry of each particular organ of the body must be in some measure peculiar, and it is not strange that while the process is spreading in the heart an infarct in the kidney, for example, may be healing.

The clinical distinction between a characteristic rheumatic fever and malignant endocarditis is wide, and the difference in the vegetations in the two conditions is equally wide, but just as the two clinical conditions merge the one into the other, so too do these vegetations. In some cases of rheumatic fever there may be many diplococci in the valves (Plate III, fig. 2). In some cases of malignant endocarditis the vast majority of the micro-organisms are destroyed. The first represent cases of rheumatic fever, which toward the end approach the type of malignant endocarditis; the second represent those cases of subacute malignant endocarditis of long duration in which the virulence appears to be low in intensity, but persistent in character.

There does not appear to us to be any essential difference in the morbid anatomy of the two conditions other than this, that for some occult reason the micro-organism in the malignant type, instead of being destroyed in the vegetation, survives and multiplies. It also seems unlikely to us that the organisms *select* a previously damaged valve,—the results of experiment, indeed, decided against this; it is more probable that there is in this type, as in rheumatic fever, that same tendency for the diplococci to attack the cardiac valves, and that damaged valves from lack of a full power of resistance permit the rapid and continual growth of the micro-organisms, and in this way predispose to the malignant type of the disease.

(c) *Bacteriological Details.*

To turn now to some of the bacteriological details. We have no knowledge of the occurrence of the diplococcus we have isolated from these cases of malignant endocarditis outside the body, except in so far as we

have studied it in culture. In the body it is present in the local lesions which characterise the disease, and in these situations it may be discovered by staining sections of those morbid structures with appropriate dyes, though more readily still by treating films made from scrapings of these tissues.

The organism is stained best by the aniline dyes, but in our experience, though it stains by Gram's method, it does not retain the stain with tenacity. It may be present in the vegetations upon the cardiac valves in enormous numbers where it can be seen in the substance of the vegetation, and also in large masses in direct contact with the blood-stream.

In this situation it is present as a minute diplococcus, measuring 0·5 or even less in diameter. We have isolated it in pure culture by the incubation of scrapings of the vegetation in a mixture of bouillon and milk slightly acidified with lactic acid, a medium such as we used for isolating the diplococcus from cases of rheumatic fever. When cultivated in this manner it resembled very closely the latter organism, though it is slightly smaller, and may grow in longer chains in fluid media, and form more definite masses upon the solid ones. Such differences as these, we believe, can be explained by its more rapid growth.

In sub-cultures made upon blood agar, which is very suitable for maintaining its virulence, the difference between these two organisms is hardly discernible. Both form upon this medium discrete colonies of minute size, the smaller and younger of which are translucent, the older and larger opaque, and of a yellowish colour. On ordinary media the growths of the two organisms are strikingly similar. Thus in bouillon they form a slightly granular deposit on the sides and bottom of the tube, while the supernatant fluid remains clear. On gelatine both form discrete non-liquefying colonies, but these media are not suitable for its growth.

Both these organisms coagulate the milk medium, forming a firm coagulum, but the diplococcus from the

malignant type the more rapidly. By both, alkaline media are rendered very distinctly acid.

This acid-producing property is a well-known feature in the growth of many bacteria. Dr. Sidney Martin, in his important researches upon the poisons of infective endocarditis, attributed this to a non-proteid body. When we recall how much attention has been directed to the possibility of some acid-producing process in the metabolism of rheumatic affections, and when we bear in mind the wide-spread belief in the value of treatment by alkalies, we are led to wonder whether sufficient attention has been given by clinicians to this result of bacterial growth in rheumatic and gouty affections. Is it not possible that in a gouty subject an attack of gout may result from an infection with these acid-producing bacteria ?

The pathological action of the organism was studied by isolating it in the milk and bouillon medium and then transferring it to blood agar. Injections were made directly into the circulation of rabbits through the auricular vein.

III. Concluding Observations.

(a) *Upon the Specific Nature of Rheumatic Fever.*

Finally, it remains for us to touch very briefly upon some of the considerations that arise if it be true that some cases of malignant endocarditis are rheumatic in origin. In these considerations, much must turn upon the question whether rheumatic fever is a specific disease. If it be a specific disease the processes involved must be specific, and the problem arises to what extent this specific character is due to the poisons which are formed by the micro-organism, and to what extent to the peculiar tissue reactions. As knowledge upon this problem is gained, the mode of origin of the malignant type may become apparent. Again, whatever the nature of the poisons that are formed in rheumatic fever, those of this type of

malignant endocarditis would be allied to them, a conclusion which, in the future, may have a close bearing upon the treatment of the disease.

(B) *Upon the Relation of Septic to Rheumatic Processes.*

An interesting point which arises from this question of the specific nature of rheumatic fever is the relation of rheumatic to septic processes. Are these distinct in their essence, or is rheumatic fever an infection with attenuated pyogenic cocci, as Singer maintains?[1]

We have been compelled to differ from Singer because we have isolated only one organism from rheumatic fever; nevertheless, this question must still arise in a slightly modified form thus:—Is this diplococcus we have isolated the attenuated *Streptococcus pyogenes*, and rheumatism a result of this attenuation? In reply to this we would ask, can the term "attenuation" be applied in this sense?[2]

Chemical pathology will, we suppose, solve this question, and meantime we are driven back to clinical experience, and ask of it once more, is rheumatic fever a specific disease? If it is, the diplococcus, call it what you will, must be to this extent specific, that it has produced a constant disease. It is to be hoped that this problem of the relation of rheumatic to septic processes will be solved before very long, and if the diplococcus described by ourselves and others prove to be the only cause of rheumatic fever, this solution will mark another step in the progress of our knowledge.

(C) *The Position that this Type of Malignant Endocarditis occupies.*

Again, if the two processes, rheumatic and septic, are essentially different, then, we suppose, mild acute rheuma-

[1] "Weitere Erfahrungen über die Aetiologie des acuten Gelenkrheumatismus," 'Wiener klinischen Wochenschrift,' Jahrgang 1901, No. 20.

[2] Rheumatic fever may prove fatal with the evidences of great virulence.

tism corresponds to the milder forms of pyæmia, virulent rheumatism to severe pyæmia; this form of endocarditis to malignant endocarditis with suppuration, while septicæmia perhaps finds a parallel in some cases of rheumatism with profound toxæmia.

We have undertaken some investigations into this subject—starting from the assumption that the micro-organisms isolated from *distinct types* of rheumatic and septic diseases should, if placed under the same conditions out of the body, produce also distinct types of disease in susceptible animals.

Thus from rheumatic fever, puerperal fever, suppurative phlebitis, pyæmia, and cellulitis, we have isolated the organisms, and have endeavoured, as far as possible, to maintain their virulence by transferring them at once to blood agar. Rabbits have been injected with these cultures, but as yet pyæmia has not resulted from the *Diplococcus rheumaticus*, or rheumatic fever from the pyogenic organisms. Triboulet records the same experience.[1] We do not pretend these investigations are by any means sufficient to settle this question; but we make allusion to them because it does seem an important point in the study of the large group of pathogenic cocci to choose typical examples of the disease of which they are thought to be the cause, and then to put these organisms to the test of experiment under similar conditions, rather than to deal with cultures, the virulence of which has been artificially raised, or with organisms that have been placed upon various media far removed from their natural soil.

(D) *Local Malignancy in other Rheumatic Manifestations.*

To the assertion that these cases of malignant endocarditis are rheumatic the fair criticism may be raised that such persistent local processes should be met with also in other of the rheumatic manifestations. It cannot be supposed

[1] 'Le Rhumatisme Articulaire Aigu,' 1901.

that any lesion, except of the heart or great blood-vessels, would produce the same condition of blood infection as does the malignant endocarditis, for there will not be that same relation of the local lesions to the general blood-stream.

1. *Malignant pericarditis.*—Yet it is well recognised that there is in childhood a persistent intractable malignant form of pericarditis. This may smoulder on for months, the process throughout being a repeated local pericarditis, never an acute general inflammation. In such a condition as this, if the organisms in the pericardium had the same easy access to the general circulation that they have in the vegetation of a valve, we should suspect the similar character of the two conditions would be at once apparent.

2. *Malignant arthritis and rheumatoid arthritis.*—It is probable, too, that the same process occurs sometimes in the joints, and gives rise to chronic destructive lesions of one or more, a condition included in the disease of joints known as rheumatoid arthritis. Such a condition of the joints need not react to treatment by salicylate of soda any more than do the conditions of endocarditis or pericarditis.

We have isolated, cultivated, and demonstrated in the synovial membrane from a knee-joint which contained two drachms of clear fluid, in which the cartilage had been eroded and the synovial membrane had been much thickened by prolonged and chronic disease, a diplococcus which reproduced severe arthritis in a rabbit, and in one instance a monarticular osteo-arthritis. The patient, an old man, had died from an acute poison (carbolic acid), and no history had been obtained of the origin of this condition of rheumatoid arthritis. Nevertheless this demonstrated the fact that destructive non-suppurative lesions of the joints may exist in the human being as a result of the presence of a diplococcus indistinguishable in morphology from the *Diplococcus rheumaticus*, and this has a very important bearing upon the pathology of rheumatoid affections of the joints.

(E) *The Insidious Onset of Rheumatic Fever.*

Another criticism of the investigation that may be raised from the clinical side is, that though malignant endocarditis and rheumatic fever may in their course sometimes resemble one another, their modes of onset are widely different. The onset of rheumatic fever, it may be said, is comparatively acute; of this type of malignant endocarditis almost invariably very gradual.

Rheumatic fever, no doubt, does very often commence somewhat acutely, but in childhood we are repeatedly met with the history that before the definite rheumatic symptoms were noticed the child had been out of health, was becoming anæmic and irritable, and was losing flesh. If the temperature is taken it may be found to be raised during this period. The onset of rheumatic fever is then often insidious, as Sir William Church emphasised in his article upon acute rheumatism in Professor Clifford Allbutt's 'System of Medicine.' It appears to us that observations in every direction tend to strengthen the view that this diplococcus may live for long periods in the body, as it certainly does in culture outside of the body. Possibly it may remain latent; often it produces indeterminate symptoms, and finally it may produce characteristic symptoms. The repeated relapses of the chronic types of rheumatic fever are most probably to be explained in this resistance of the micro-organism to complete destruction.

We must once more thank Dr. W. B. Cheadle and Dr. D. B. Lees for leave to make use of their clinical cases. It would be impossible, if it were not for this assistance, to collect sufficient data for the generalisations which are essential in investigations of this nature. To Mr. H. G. Plimmer, Pathologist to St. Mary's Hospital, we must also again express our indebtedness.

FIG. 1.

THE HEART OF A RABBIT. Exp. No. (2). Case No. VI.

The mitral valve is exposed, and upon it is seen a large vegetation.

FIG. 2.

THE HEART OF A RABBIT.

The mitral valve is exposed and shows a large vegetation. The animal was inoculated intravenously with a culture of the diplococcus which had been obtained from a case of rheumatic pericarditis and passed through thirteen rabbits.

FIG. 3.

THE HEART OF A RABBIT.

The tricuspid valve is exposed, and shows several small vegetations. The inoculation was made with a diplococcus isolated from malignant rheumatic endocarditis. (Case No. V.)

Whittingham and Sons Limited, London.

FIG. 2. FIG. 1.

FIG. 3. FIG. 4.

Photograph of agar plates prepared with vaccine material *four weeks* after GLYCERINATION; showing inhibitory effect of different percentages of glycerine.

FIG. 1.—20 per cent. glycerine. FIG. 3.—40 per cent. glycerine.
FIG. 2.—30 per cent. glycerine. FIG. 4.—50 per cent. glycerine.

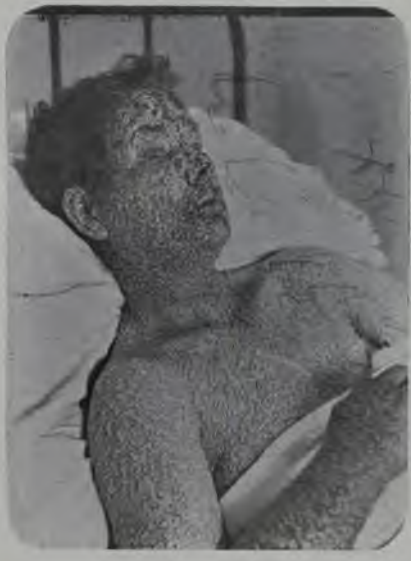


FIG. 5.

Photograph, by Dr. GORNALL, of a case of confluent Small-pox, in the vesicular stage. (Small-pox material for each series of experiments, illustrated in the following plates, was obtained from a similar case to that shown in this photograph.)

Bale and Danielsson, Ltd.

FIG. 1.

Photograph of monkey, showing result, on the eighth day, of inoculation with human small-pox lymph. Series I.

FIG. 2.

Photograph of calf, showing result, on the sixth day, of inoculation with variolo-vaccine lymph. Series I.
(Third remove from the monkey shown on Fig. 1.)

Bale and Danielsson, Ltd.

Copeman: Vaccination. Plate IV.

Photograph of child, taken on the eighth day of vaccination with lymph of variolous origin obtained from Calf No. 3, Series I. (Plate III.)

Bale and Danielsson, Ltd.

FIG. 1.
Photograph of Monkey, showing result, on the eighth day, of inoculation with glycerinated human small-pox lymph. Series III.

FIG. 2.
Photograph of calf, showing result, on the sixth day, of inoculation with variolo-vaccine lymph. Series III.
(Fourth remove from monkey shown above.)

Bale and Danielsson, Ltd.

Photograph of child, taken on the eighth day of vaccination with lymph of variolous origin, obtained from calf No. 4. Series III. (Plate V.)

Bale and Danielsson, Ltd.

DISCUSSION.

Dr. Leonard Dobson referred to the difficulties met with in the production of calf lymph. In his experience the best lymph was obtained from calves with light hair. The results, however, as tested by vaccination, varied greatly with the condition of the animal. In some instances, for example, good vesicles that had developed on the fourth day, on the fifth day dried up, leaving scabs alone. Lymph would become inert from many causes; if the glycerine were acid, sooner or later it would become inert; similarly it would do so if it were exposed to light or heat. It was unfortunate that there was no test for the activity of lymph except the result on the calf and on the child; and, moreover, lymph which produced a good result in the calf might be ineffective for the child. There was no test known at present by which the activity of lymph could be standardised. Recent complaints as to the efficiency of lymph were mostly in regard to the secondary vaccination of adults; the present epidemic gave the first general experience of this. In some cases it was possible to produce good vesiculation in the same person again and again within a short time from the use of different lymphs. He knew of the case of a child who in the space of four months had been vaccinated successfully no less than three times. In his own case, after having been successfully vaccinated with lymph from the Government laboratories, he shortly afterwards accidentally inoculated his finger with lymph from another source, with the result that a typical vesicle developed. What efficient re-vaccination really was was not yet established.

Professor Sims Woodhead, in resuming the discussion introduced by Dr. Copeman, on December 10th, 1901,[1] said that he had done many experiments with the object of observing the effect of adding glycerine to calf lymph, and that he could corroborate Dr. Copeman's observations on the action of glycerine on vaccine lymph, and on micro-organisms. The non-sporing organisms, with a few exceptions, were almost entirely eliminated by a 50 per cent. solution of glycerine; even in such a resistent form as the *Streptococcus pyogenes aureus* a large number were eliminated, sometimes only about 5 per 1000 being left. The spore-bearing organisms were almost unaffected. Thus some indication as to the nature of the vaccine organisms, which might be so small as to be invisible with the highest powers of the microscope now available, was given. The organisms

[1] 'British Medical Journal,' December 14th, 1901, p. 1736.

present in lymph were chiefly skin organisms and non-sporing, and hence were amenable to glycerinisation. It had been pointed out by the Special Commission on Glycerinated Calf Vaccine Lymphs that when non-spore bearing organisms were present in large numbers this was due to imperfect glycerination, but that when spore-bearing organisms were in excess in any lymph it was an index that that lymph had not been sufficiently carefully collected. He alluded to the effect of glycerinisation on the activity of the lymph, and expressed the opinion that little or no weakening occurred within three or four weeks. After a longer time weakening probably did, to a slight extent, occur, but the deterioration was more likely to be due to under-glycerinisation than to over-glycerinisation. When organisms that grow best at the body temperature were in excess glycerinisation had a more active influence. The local inflammatory effect of vaccine had been considered of late by many to be greater than formerly, but this was probably due to imperfect glycerinisation, the greater part of the local effect being always due to extraneous organisms. He suggested that the Local Government Board might be able to prepare statistics from their returns to decide that point.

Dr. T. D. ACLAND said that whilst acting as medical officer to the Royal Commission on Vaccination he had had an unusual opportunity of seeing the methods of vaccination practised throughout the country, and of forming an opinion as to the manner in which the operation was carried out, and the cause and extent of vaccinal injuries. In the course of the same inquiry he had been brought intimately into relation with the Medical Department of the Local Government Board, and was able to appreciate the manner in which its officers carried out the difficult and often thankless task of maintaining efficient vaccination. There could be no question that the country owed much to Dr. Copeman for his researches into the origin and purity of vaccine lymph, and for his share in the establishment of an institute able to supply calf lymph on a scale quite impossible a few years ago. Dr. Acland hoped that the Government would take steps to establish a laboratory on a far larger scale than at present, so that all practitioners in the kingdom might be able to obtain lymph from a laboratory fitted with every requisite for perfect work, unfettered by economies necessary in an establishment run solely for trade purposes. He thought that it would be well also if all supplies of vaccine lymph were placed under Government control, and expressed regret that there should be two "Jenner Institutes" in this country—the one formerly known as the "British Institute of Preventive Medicine" in Chelsea, where the National Vaccine Establishment was temporarily situated, the other the Jenner Institute for calf lymph in Battersea, to which the name by priority

belonged. It was inevitable that confusion should arise between the two establishments, and it had arisen.

The Nature and Origin of Vaccine Lymph.

Vaccination, although it was the first protective inoculation practised on a large scale throughout the whole civilised world, had, in all things except the extent of its use, been outstripped in the scientific details of its technique by many of its successors in the department of protective inoculations as now carried out. The practical question now was whether a protective virus could be obtained from bovine animals by the inoculation of existing lymph, or of the virus of *inoculated* smallpox (which differs in many important particulars from the natural or exanthematous form). It would seem that Dr. Copeman, by transmitting smallpox through the monkey, and by inoculating bovines with the virus so obtained, had produced a lymph which was of a similar, and possibly of the same nature as that which would be obtained from cows who had accidentally contracted local pocks from a milker suffering from inoculated smallpox. Lymph derived from such a source, and definitely derived from smallpox, seemed to be capable of producing "vaccinia" in man, whilst it had lost its power of transmitting smallpox. Whether this lymph was identical with what was called vaccine lymph could only be decided by further experiment. The lymph now in use in this country, whatever its origin, was not merely an attenuated smallpox virus, since, if this were the case, some amongst the many millions of vaccinated children would be likely to develop smallpox instead of vaccinia. Dr. Acland was not aware that any such case had been recorded. The generalised eruption, which in rare instances followed vaccination, was generalised vaccinia, not variola.

The Preparation of Lymph.

Glycerine had long been used to dilute the lymph, and to serve as a medium for triturating the lymph products obtained from the calf, but no experiments were made previous to Dr. Copeman's to show that the treatment by glycerine was capable of producing a sterile lymph, that was a lymph freed from all extraneous organisms. Dr. Copeman's researches had at any rate shown that lymph freed from all organisms capable of cultivation on ordinary media did not lose its specific property. This answered the question which had been raised whether erysipelas was a stage in the evolution of inoculated cow-pox.

Standardising Lymph.

The standardisation of vaccine lymph had hitherto presented practically insuperable difficulties. It was possible that the

facts demonstrated by Drs. Calmette and Guérin [1] might enable this difficulty to be overcome. They had shown that the rabbit was susceptible to the vaccine virus, and consequently the potency of any particular batch of lymph might readily be tried before it was sent out. The method had been tested successfully for two years at Lille, with the great advantage that it was found possible to eliminate inert batches of lymph prepared for vaccination.

The Actual Methods of Vaccination.

Vaccination as practised was open to two main objections: (1) that the necessity for causing a local sore created a definite point of vulnerability in the individual vaccinated, and formed a possible starting-point for various inflammatory complications; (2) that it created a certain amount of opposition, since in the homes of the poor a vaccinated child imposed a burden on the often overworked mother which was hardly realised by those who had no practical acquaintance with the facts; the gain to the community was impersonal and remote, while the sick child was a present and very real source of anxiety and difficulty.

The necessity for producing a local pock seemed to depend upon the fact that so far all efforts to isolate and cultivate a "vaccinia" organism *in vitro* had failed, so that the only practicable method of administering vaccine lymph medicinally was to create a local pock of a certain area, arrived at by experience, and to leave the production of the immunising bodies to take place in the person of the individual vaccinated.

The Production of an Antitoxin in the Body by Local Inoculation (Vaccination).

Dr. Acland recognised two views as to the means whereby protection was attained by vaccination. (*a*) "That by the local inoculation a body, presumably an organism, is introduced into the tissues, which by its multiplication, after absorption, produces the desired antitoxins." According to this hypothesis, the number or extent of superficial vesiculations is immaterial, if only sufficient of the original virus was introduced at the point of inoculation to overcome the resistance of the individual. Dr. Acland knew of no evidence corroborative of this hypothesis; on the contrary, such evidence as there was went to show that (*b*) under ordinary circumstances a given area of vesiculation was necessary, and that the immunising effect of vaccination bore a more or less definite relation to the area of the vesicles. This would follow if it were a fact, as now generally supposed.

[1] 'Annales de l'Institut Pasteur,' 1901, vol. xv, p. 161.

that the area of vesiculation bore a direct relation to the amount of antitoxin produced. From these considerations several important questions—practical, therapeutical, and political—arose. 1. Why was it necessary to produce on the arm of a newly born infant the same number of vesicles as on a fully grown man? It might be urged as a matter of clinical experience that infants required a larger dose of vaccine lymph as of other remedial substances, such as arsenic and belladonna, because their tissues were more active, they were more susceptible to the infection of smallpox, and the changes (increase) in their body-weights had no parallel in the adult, all of which circumstances necessitated a proportionately larger reserve of the immunising material if the dose was to be effective. 2. How was it that different standards of efficient vaccination were permitted? Ought there not to be an irreducible minimum below which no certificate of successful vaccination should be given? If satisfactory immunity—a variable quantity for every individual—could only be procured by a certain standard of vaccination, it was surely desirable that there should be a uniform certificate of successful or efficient vaccination. At present it was mainly those vaccinated by the public vaccinator who were efficiently protected according to the official standard. Those vaccinated in private might have one, two, three, or four vesicles, according to the conscience of the operator or the insistence of the mother. The medical profession ought to endeavour to create a public opinion in favour of a really efficient standard of vaccination, and not give its tacit approval to its performance as a piece of therapeutic ritual which had to be got over as cheaply as possible. 3. Did the number or area of scars really give any indication as to the efficiency of protection? This was a most difficult question, but fortunately one to which a great deal of attention has been directed, notably by Dr. Coupland, whose work was a model of thoroughness, and by Dr. Barry, whose report on the smallpox epidemic at Sheffield was well known. It would appear from the figures given in the report of the Royal Commission on Vaccination, 1896, that in nearly 7000 cases the mortality was 6·2 amongst those with one mark, and 3·2 amongst those with four. These figures emphasised the fact that a minimal standard of efficient vaccination was much required if the community was to take full advantage of the protection of vaccination against smallpox. Vaccinia or immunity to subsequent vaccination had been produced by many experimenters without the formation of superficial vesicles both in man and animals. Burckhart vaccinated six children whose mothers had been successfully vaccinated whilst pregnant. The operation was unsuccessful in all of them. Kellock vaccinated 38 women in various stages of pregnancy, and found that the infants resisted vaccination directly as the stage of preg-

nancy at which the mother was vaccinated. In 21 cases the children of women who were vaccinated during the latter half of pregnancy proved insusceptible of vaccination; whilst in 14, whose mothers were vaccinated before the seventh month, vaccination was successfully performed. Smallpox contracted by a mother before her confinement might be transmitted to the fœtus, and a child whose mother had contracted smallpox during the third week before delivery might prove insusceptible to vaccination. Straus and Beclard, Chauveau and Ménard, had also made experiments which confirmed these clinical observations, since they had found that the serum of calves taken during the height of vaccinia produced a measure of immunity in other animals of the same species; when injected intra-venously the serum injections acted at once, whilst immunity was not secured in the calf by vaccination before the eleventh or twelfth day, or in the pig two days earlier. This date, according to Cory, was approximately the period after vaccination at which immunity began to be secured in man. The time varied very much in different individuals of the same species; in man probably the receptivity to successful vaccination diminished gradually during the second week, and became extinct before the fourth. Dr. Acland had recorded a case of vaccinia generalised by auto-inoculation, in which the pocks continued to develop for four weeks certainly. Such evidence tended to show (1) that the serum of a vaccinated animal was capable of modifying and possibly destroying the susceptibility of another animal of the same species to vaccination; (2) that it was not essential for the immune animal to have been actually vaccinated in order that it might be rendered refractive against further inoculations of the same virus; and (3) that whereas serum injections had a rapidly immunising power, vaccination was a comparatively slow process, a fact which had an important bearing on the question as to whether it served any useful purpose to vaccinate an individual who had already been exposed to the infection of smallpox. Dr. Acland then referred to the attempts which had been made to treat variola by the antitoxin of vaccinia. Kinyoun[1] had recorded two cases of variola treated with serum taken from a calf vaccinated four weeks previously. The observations were inconclusive. Each case received 15 c.c. of serum followed later by 20 c.c. more. One case died in seventy-two hours. In 1896 MM. Beclard, Chauveau, and Ménard had recorded[2] observations made on seventeen cases of variola of all ages treated by subcutaneous injections of serum taken from a vaccinated calf. In one case no less than a litre and a half was injected without causing any local or general disturbance, and

[1] 'Philadelphia Med. News,' February 2nd, 1895.

[2] 'Ann. de l'Institut Pasteur,' vol. x, 1896, p. 1.

the patient recovered. These cases had been alluded to by Dr. Copeman in his Milroy Lectures, but in a later communication the same observers had given an account of further experiments on the immunising power of the blood of man and animals, after vaccination, against the vaccinal or variolous infections, and they came to the definite conclusion that not only did the serum of a vaccinated calf taken on the fourteenth day after inoculation possess certain powers both in the direction of conferring immunity and assisting the cure in the body, but that it also acted on vaccine lymph in the test-tube, rendering it inert. They also concluded that the serum of convalescents from variola had the same destructive power over the vaccinal virus. All these experiments seemed to indicate the general truth of Jenner's hypothesis, and to supply the scientific basis which was lacking from his empirical practice—a practice which, although empirical, had stood the test of time and of rigid experimental test. They also served to indicate the direction in which the advance might be made. Dr. Copeman had made it possible to obtain from the calf a supply of antitoxin directly derived from variola; might it not be that this would afford a means of combating variola itself? It might well be asked that Dr. Copeman, should he be willing, should be granted the opportunity of testing the accuracy of the scientific facts by utilising them for the treatment of variola.

Dr. Acland then gave notice of the following resolution:

> "That, in the opinion of this meeting of the Fellows of the Royal Medical and Chirurgical Society, it is desirable that the Government should without delay make such additions to the National Vaccine Establishment as shall enable it to supply glycerinated calf lymph to all medical practitioners who may desire to use it; and that, in view of the national importance of the subject, steps should be taken to test and regulate the sale of all imported lymph, and to inspect the manufacture of all that is made in this country."

Professor Haccius (Geneva) acknowledged the debt conferred on universal medical science by Jenner, and also later by Dr. Copeman. In 1892, when in Switzerland there was difficulty in procuring good calf lymph, he transmitted variola to a calf, from this to six other calves, and eventually with success to children, typical vaccine vesicles being formed. Professor Chauveau attacked these experiments on the ground of accidental inoculation with vaccinia in the laboratory, saying that the disease was vaccinia and not modified variola; but this source of fallacy was eliminated by transmitting the inoculated smallpox from calf to calf before inoculating it on children. Professor Chauveau had inoculated a cow with variola, and from the vesicle produced had inoculated a child who developed typical variola. This, however,

was to be explained by the direct transference of variola virus to the child, the cow being merely a stage in its transference. In Munich and Stettin smallpox had been transmitted to calves, the inoculations being performed apart from vaccine establishments. It was recognised that in many instances variola could not be thus transmitted to the calf, but he believed that the identity of variola and vaccinia would be established. His results on children were quite good, there being no generalised eruption. Allusion was made to glycerinated lymph and to powdered dry lymph—the one being successful, the other not. The collection of calf lymph was now conducted under better conditions than formerly, thus diminishing from the first the number of extraneous organisms. It was hoped that in a short time vaccination would become compulsory in Switzerland.

The discussion was adjourned.

ADJOURNED DISCUSSION, January 28th, 1902.

Dr. SIDNEY COUPLAND, in resuming this discussion, referred to the great improvement that had supervened in modern vaccination methods, mainly as a result of the introduction of glycerinated lymph, the use of which had been made obligatory by the Act of 1898. The objects of recent improvements had been to obviate the risks of the process, and there had been the even greater advance in having vaccination performed at home, and in raising the age of the children required to be vaccinated. The vaccinal injuries had been in the past, perhaps, too lightly considered by the profession. In the majority of cases with ill-effects it had not been the lymph which had been at fault, but some accidental complication. He quite agreed with Dr. Acland that full control of all the vaccine lymph used in this country should be in the hands of the Government. Large unsightly scars were unnecessary, and were often associated with inefficient vaccination. The scars from purified lymph were often small, and did not even present the size which was formerly supposed to be an essential index of good vaccination. Indeed, probably now the number of the scars was the best evidence of efficient protection. The question was probably one of dose, as in the use of antitoxin in cases of an infectious disease. The scars were numerically a permanent record of the dose, whilst the statistics of Marson, Gayton, Barry, and others prove that the efficiency of vaccination in mitigating an attack of smallpox is in direct relation to the number of insertions. Thus Barry found, at Sheffield, the proportion of mild attacks to be 20 per cent. in persons who had been vaccinated in *one* place only, and 52 per cent. in those who had *four* marks. It was admitted that vaccination was a good prophylactic against smallpox, but it had its limitations. When smallpox invaded

a house or other small community where all were equally exposed to infection, some escaped and others were attacked. Among those who had had smallpox previously, personally collected statistics showed that 1 in 20 were attacked a second time, and in the epidemic at Warrington, investigated by Dr. J. D. Savill, an even larger proportion, namely, 9 out of 41, had second attacks. But a single vaccination did not afford this degree of protection. Of those who had been only vaccinated once in childhood, about 25 per cent. were liable to contract the disease when living in a smallpox atmosphere. Recently vaccinated persons were much more powerfully protected than the more remote; about 1 in 7 of those who were vaccinated for the first time on their being exposed to smallpox infection contracted the disease. Revaccination properly performed conferred practically absolute immunity; even in a person who had had smallpox the immunity could be prolonged by revaccination. In children under ten vaccinated in infancy, liability to attack was about 1 in 11 or 12; but 2 out of 3 of unvaccinated children were likely to take the disease on being exposed to infection for the first time. These results, it was pointed out, were based on a relatively few cases—some three epidemics. It was probable that even recently vaccinated persons were not all absolutely immune, owing to their vaccination with various strains of lymph, some of which had become inert.

Dr. ALBERT E. COPE stated that he represented the body of public vaccinators, and that he wished to describe his own practice as an illustration of the way in which the actual work of vaccination was carried out. The arm in the case of infants, which had been washed by the mother, was first rubbed with a pledget of boric wool saturated in rectified spirit, and then dried with a pledget of dry wool. For revaccination a 20 per cent. carbolic soap was used with which to wash the arm. The water was boiled, a pledget of wood-wool was used to apply the soap, the excess was rapidly washed off with water or rectified spirit, and the arm was dried with a similar pledget. The instrument he had found most useful was a lancet made of platinum, hardened with iridium, so that it might be flame-sterilised. The best ejector of the lymph from the tube was a solid rubber bulb with a funnel-shaped perforation through which the tube was passed, and the outer aperture closed by the thumb before compression. Lymph was placed on the arm and the skin was lightly scratched through it, the faintest trace of blood being drawn. An interval was allowed until an erythematous reaction was visible, when a boric pad was applied and strapped on. This was allowed to remain for a week, then a powder of boric acid, zinc, and starch was applied under another boric pad. The most troublesome complication he had had was an eczema occurring under the strapping, which

subsided readily under the application of calamine lotion. Complications of any kind were very rare. Revaccination was obtainable at about fourteen after good vaccination in infancy; it would be well to arrange for it to be done on children before leaving school, so that later it should not interfere with the daily work of adult life. This might well be made a Government regulation.

Mr. WILLIAM F. BLAKE asked what constituted a successful revaccination. If two persons were vaccinated from the same tube, and one took but the other did not, could it be asserted that the person who did not take was protected against smallpox? He also asked what was meant by the statement that had been made earlier in the discussion that successful vaccination might occur several times within a few months in the same person.

Dr. F. CHURCHILL asked if there was any age beyond which vaccination might safely be discarded. It was probable that persons of all ages were liable to contract the disease. It was a question whether the present epidemics of smallpox were really so effectively controlled by improved methods of vaccination and isolation as in the earlier epidemics by rougher measures. It was possible that the more elaborate procedures acted as a deterrent against vaccination being universally had recourse to, because the poor have their infected children so amply provided for at the expense of the State.

Dr. BERNARD O'CONNOR asked whether a second or third revaccination was necessary; theoretically he had thought revaccination should be continued until it failed to produce any result.

Dr. COPEMAN, in reply, stated that dilute glycerine had a gradual inhibitory effect on micro-organisms that were non-spore-bearing; most of the pathogenic organisms were non-spore-bearing, and the spore-bearing organisms at all likely to be present were of no pathogenic importance. Even if such organisms as those of erysipelas and tubercle were intentionally added to the lymph in considerable quantity, the subsequent addition of glycerine gradually eliminated them, so that after an interval of about a month the lymph became entirely freed from infective qualities. The same dose or amount of vaccination was probably desirable in the child as in the adult. Probably a greater effect, however, relatively to the size of the individual was obtained in the adult. There was at present no efficient method of standardisation of lymph; the only useful test was the clinical result. It was not possible as yet to afford the same degree of protection by injecting an antitoxic serum into the system. With the use of glycerinated lymph there was not the same destruction of skin tissue as even in what was considered perfect vaccination of years

ago. It was essential that all persons should be revaccinated at least once in later life. There was no doubt that a person might contract smallpox twice or even three times. In referring to the high degree of immunity that vaccination gave, he said that he had on two occasions accidentally inoculated himself with smallpox, with the result that no infection took place. If no effect was observed to follow the use of lymph which subsequently was suspected of being inert, an interval of at least a month should be allowed to elapse before revaccination, because a slight local immunity might be produced even by inadequate vaccination. In recent methods there was probably no weakening of the lymph by mixing with glycerine as compared with the old arm-to-arm method, for it was believed that the microbes of vaccinia were contained in the epithelial cells which were removed in the pulp from the calf, while in vaccination from the human arm only the fluid from the loculi was taken. Dr. Copeman, in conclusion, referred to the possibility of protection against smallpox by internal administration.

CLINICAL AND EXPERIMENTAL OBSERVATIONS INTRODUCING A DISCUSSION

ON THE

REGENERATION OF PERIPHERAL NERVES

AN ADDRESS

BY

CHARLES BALLANCE AND PURVES STEWART

With Lantern and Microscopical Demonstration

Read February 25th, 1902

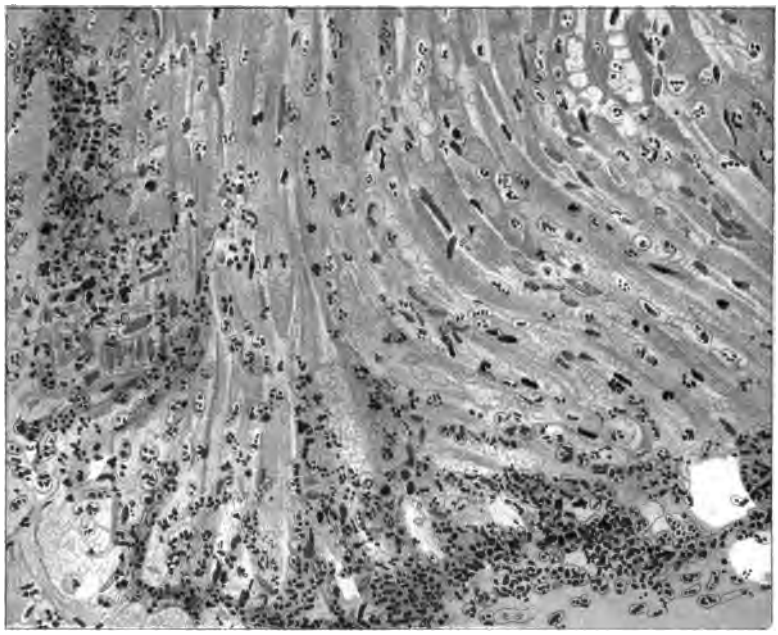
SYLLABUS.

1. Preliminary Remarks—The "central" school—The "peripheral" doctrine.
2. Changes following the division of a peripheral nerve.
    - A. Degeneration.
        - (1) Changes in the axis-cylinders and medullary sheaths.
        - (2) Cellular proliferation.
            - (*a*) Of leucocytes.
            - (*b*) Of connective-tissue cells.
            - (*c*) Of neurilemma cells.
    - B. Regeneration.
        - (1) In the proximal segment of a divided nerve which has not been reunited.
        - (2) In the distal segment of a divided nerve reunited to the proximal segment by sutures.
        - (3) In the distal segment of a divided nerve whose cut ends have been left widely separated.
3. Nerve grafts.

demanded up to some 8000 tubes *per diem* can be at once despatched to the National Vaccine Establishment at Whitehall, from whence the lymph is distributed to public vaccinators.

7. *Recording the results of vaccinations by public vaccinators.*—Each public vaccinator receives, in response to application made to the National Vaccine Establishment, a consignment of lymph, together with a schedule in which to record the results of its use, and these schedules, after having been examined at the National Vaccine Establishment, are sent to the laboratories. The schedules indicate the series number of the lymph, the date of its despatch from the National Vaccine Establishment, the name of the public vaccinator to whom it was supplied, the number of tubes sent, the dates when the several tubes were used, the number of persons vaccinated, the number of scarifications made, and the number of vesicles obtained. All these details are recorded at the laboratories, and from the last two items information as to the success which has resulted, both as regards individuals vaccinated and insertions of lymph made, is obtained and set forth, both in full and in the form of a percentage. In addition to these records a register is kept stating the particulars of the calves employed, the details of the lymph obtained from each calf, including the results of the bacteriological examinations, the results of the use of the lymph at the Animal Vaccine Establishment, and also the number of tubes of each series despatched to the National Vaccine Establishment.

During the first year of operations nearly 500,000 tubes of glycerinated lymph were sent out from the Government Laboratories. Notwithstanding the difficulties that had naturally to be overcome in the inauguration of work of a character entirely new to practically all those engaged upon it, the success attending the use of the lymph at the hands of public vaccinators throughout the country was distinctly gratifying, the returns made by them to the National Vaccine Establishment showing that a case



Sciatic nerve of cat. Two days after division. Longitudinal section of lower end of proximal segment, showing infiltration of the nerve-fibres by proliferated leucocytes. The lower part of the figure represents the site of operation.

M. H. Lapidus, del. *Bale and Danielsson, Ltd., Chromo-Lith.*

A. Degeneration.

B. Regeneration.

The two processes overlap in point of time, regenerative changes commencing before degeneration is complete.

Degeneration.

Degenerative changes affect the lower end of the proximal segment and the whole extent of the distal segment. They occur simultaneously in these parts, and do not spread from the centre to the periphery, nor *vice versâ*.[1]

1. *Changes in the axis-cylinders and medullary sheaths.*—Fragmentation commences on the fourth day after the traumatism. The smallest axis-cylinders and medullary sheaths are more resistent than the larger ones, and remain unbroken until the fifth day (see 'Healing of Nerves,' plate 1, fig. 2). After that date, however, all the axis-cylinders and medullary sheaths, small as well as large, become completely fragmented.

The broken-down axis-cylinders and medullary sheaths form globular or ovoid masses, which gradually become absorbed. The process of absorption is practically complete by the end of five weeks, though a few scanty remains of fatty *débris* can often be detected for a long period afterwards.

2. *Cellular changes:*—(A) *Leucocytes.*—Diapedesis begins immediately after the injury, and is well marked for the first three days (see plate). It remains evident for two weeks, after which it gradually subsides.

(B) *Connective-tissue cells.*—These commence to proliferate on the second day. They have an absorbent action on the fragmented myelin and axis-cylinders (see 'Healing of Nerves,' plate 15, fig. 3). This process of absorption having been completed, they then proceed to the formation of fibrous tissue. Thus the degenerated

[1] The illustrative plates to which reference is made in the subsequent part of this paper are to be found in 'The Healing of Nerves,' by Charles Ballance and Purves Stewart (Macmillan, 1901).

nerve-trunk becomes denser in consistence than in the normal condition.

(c) *Neurilemma cells.*—These cells proliferate at a somewhat earlier time than the connective-tissue cells, and in a patchy fashion (see 'Healing of Nerves,' plate 15, fig. 2). A possible explanation of the earlier proliferation of the neurilemma cells as compared with that of the connective-tissue cells may be found in the fact that the neurilemma cells are nearer to the degenerating elements, and therefore receive the chemical stimulus first. After a short period, during which they have an absorbent action on the medullary sheaths, they relinquish this function to the connective-tissue cells, and commence the regenerative process by arranging themselves in closely packed longitudinal columns.

Regeneration.

(1) *In the proximal segment of a divided nerve which has not been reunited.*—Changes occur which result ultimately in the formation of the well-known so-called "end-bulb"—a dense, club-shaped swelling in which are numerous young nerve-fibres, coiled and intertwisted in all directions, embedded in fibrous tissue. According to the "central" theory of nerve regeneration, these new fibres were regarded as outgrowths from the central end, which, unable to find their way into the distal segment, had turned back on themselves in a futile manner like the apex of a fountain. This, however, is not so. Every new fibre of the permanent end-bulb is laid down in separate short links, each in apposition to a neurilemma cell, thickest near the nucleus of the cell and tailing off at each extremity. These short lengths of young nerve-fibres are, at first, separated by some distance from the ends of the central axis-cylinders. The end-bulb is not formed by a downgrowth and recurving of axis-cylinders, but by the development of new fibres in a structure which we have named the "*primitive end-*

bulb"—a mop-like protuberance which is formed immediately the nerve-trunk is divided, the result of the curling back upon themselves by the divided nerve-fibres (see 'Healing of Nerves,' plate 1, fig. 1). In this "primitive end-bulb" degeneration occurs as above described, and is followed by regeneration, a process carried out by the proliferated neurilemma cells, which secrete small islands of axis-cylinders and medullary sheaths. These islands later overlap in an imbricating fashion and ultimately fuse together to form a long nerve-fibre continuous with one of the nerve-fibres of the proximal segment above.

(2) *In the distal segment of a divided nerve, reunited to the proximal segment by sutures.*—The proliferated neurilemma cells arrange themselves in longitudinal columns, separated by strata of proliferated connective-tissue cells. The neurilemma cells have a neuroblastic function and proceed to form new axis-cylinders and medullary sheaths.

At the end of three weeks (with the Golgi method) neuroblastic action is first detected (see 'Healing of Nerves,' plates 8, 9, and 10). All through the distal segment scattered neuroblasts are seen, from whose opposite poles young axis-cylinders grow out longitudinally, stretching out towards similar processes of adjacent neuroblasts in the same longitudinal column, but not yet reaching them. By the end of four weeks these young processes have grown in length so as to overlap and fuse into long axis-cylinders (see 'Healing of Nerves,' plates 10, 11, and 12). In sections stained by the Weigert and Stroebe methods respectively the same process can also be studied, and the new axis-cylinders and medullary sheaths are seen to be secreted by the cells of the neurilemma. In the earliest stage of this process the young fibre is deposited along one side of the body of the cell in the vicinity of the nucleus. It grows in length and assumes a spindle form, thickest in the neighbourhood of the nucleus and tapering off at each pole (see 'Healing of Nerves,' plate 2, fig. 5, and plate 14, fig. 6). These

all vaccinators, public and private alike, shall conform to a definite standard. The further suggestion has been made that every medical man should become a public vaccinator, to the extent that he should have the right of claiming a fee from public funds for every vaccination performed by him, provided that he was willing that his work should be subject to inspection on behalf of the Government. But the originators of this idea can hardly have realised the magnitude of the inspectorial staff that would be required if such an arrangement were to be put in force.

The treatment of the arm, at the time of vaccination and subsequently during the progress of the case, is another subject which has aroused considerable controversy, and concerning which much divergence of opinion would appear to exist. Thus, in some quarters, the initial cleansing of the arm is said to be objected to by the parents as a reflection on the care, or want of care, on their part, as regards the condition of their children; but in general it is found that a little tactfulness in explaining the difference between ordinary and surgical cleanliness has sufficed to overcome the difficulty. In addition to this aspect of the case the friction employed in the process is of value in causing a slight capillary dilatation which undoubtedly contributes to the success of the operation. Water, soap and water, spirits of wine, or antiseptic solutions, of greater or less potency, containing boric or carbolic acid, lysol or perchloride of mercury, for instance, are employed by different operators for the purpose, of which, in all probability, a warm solution of boric acid is the most generally useful,—a stronger antiseptic, such as corrosive sublimate, unless removed by the subsequent use of sterilised water or alcohol, being liable to exert a somewhat deleterious effect upon the lymph.

The method to be employed at the operation and during the maturation of the vesicles for the protection of the vaccinated area from extraneous infection has not been defined by the regulations, for the reason that it

appeared probable that each man would best attain the desired end by the same methods that he would ordinarily employ in the treatment of any other case of minor surgical injury. As was to be expected, therefore, the means adopted for the protection of the vaccination wounds have been very various, and different trade firms have undoubtedly reaped an extensive harvest by the introduction and energetic advertisement of special dressings of one and another kind. In Paris, at the time of my official visit, a semi-transparent material, known as "taffetas Marinier," not unlike thin isinglass plaster, and which adheres to the skin when moistened with water, was, I found, invariably employed to protect the vaccinated area during the first few days following the operation; and a somewhat similar substance, advertised by an English firm, is, I believe, at present utilised to a considerable extent in this country. But during the second week of the process it is essential that some dressing of an absorbent nature should be employed, as it is during this period that oozing from the vesicles occasionally supervenes.

The means employed for retaining the dressings in position are almost as numerous as the latter themselves. At the Government Station in Lamb's Conduit Street a dressing composed of a couple of layers of boric lint, kept in place by means of pieces of rubber strapping which do not entirely encircle the arm, is applied at the time of vaccination, and this is replaced by another exactly similar dressing when, a week later, the case returns for inspection of the result. But, whatever be the nature of the dressing, the free use beneath it of a dusting powder of boric acid has a most beneficial effect in preventing any undue amount of inflammatory reaction.

Concerning the nature of the instrument best adapted for the purpose of vaccination I desire to offer a few remarks. Here, again, each operator will probably attain the greatest measure of success with that instrument to

CLINICAL AND EXPERIMENTAL OBSERVATIONS INTRODUCING A DISCUSSION

ON THE

REGENERATION OF PERIPHERAL NERVES

AN ADDRESS

BY

CHARLES BALLANCE AND PURVES STEWART

With Lantern and Microscopical Demonstration

Read February 25th, 1902

SYLLABUS.

1. Preliminary Remarks—The "central" school—The "peripheral" doctrine.
2. Changes following the division of a peripheral nerve.
   - A. Degeneration.
     - (1) Changes in the axis-cylinders and medullary sheaths.
     - (2) Cellular proliferation.
       - (*a*) Of leucocytes.
       - (*b*) Of connective-tissue cells.
       - (*c*) Of neurilemma cells.
   - B. Regeneration.
     - (1) In the proximal segment of a divided nerve which has not been reunited.
     - (2) In the distal segment of a divided nerve reunited to the proximal segment by sutures.
     - (3) In the distal segment of a divided nerve whose cut ends have been left widely separated.
3. Nerve grafts.

4. Clinical observations: (1) Primary suture; (2) Secondary suture; (3) Nerve grafts.

5. Topics suggested for discussion: (1) The mode of regeneration of peripheral nerve tissue; (2) The absence of regeneration in the spinal cord and brain; (3) The bearing of this on the neuron theory; (4) Indications and contra-indications for operative interference; (5) Comparative advantages of various operations.

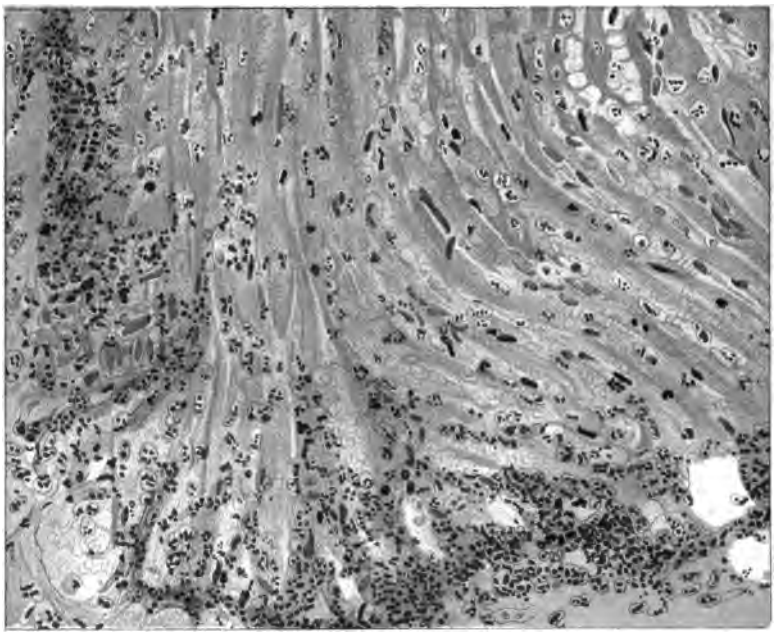
THE fact has long been admitted that regeneration can occur in peripheral nerves if their opposing ends, previously divided, are brought into apposition.

As to the process whereby this regeneration is accomplished, however, there have been two schools of opinion, which may be termed the "central" and "peripheral" respectively.

According to the "central" school (supported by Ranvier, Waller, His, Vanlair, Stroebe, Howell and Huber, and others), the new nerve-fibres which occur in the distal segment of a reunited nerve-trunk are formed by a process of downgrowth from the proximal segment, and thread their way gradually along the neurilemma sheaths of the distal segment, already rendered empty by the degeneration of the old axis-cylinders and medullary sheaths. The new fibres are thereby guided ultimately to the periphery.

The "peripheral" doctrine, hitherto a less popular one (amongst whose chief supporters may be mentioned Tizzoni, Cattani, Bethe, Kennedy, and Galeotti and Levi), teaches that the new nerve-fibres are formed locally in the distal segment from pre-existing elements there. This, in our opinion, is the correct view, and its accuracy is demonstrable by the lantern slides and microscopic sections illustrating this paper, the results of our experimental observations in the lower animals and clinical observations in man.

If a peripheral nerve be cut across, certain changes occur in the lower part of the proximal segment and throughout the entire extent of the distal segment. These changes consist of—



Sciatic nerve of cat. Two days after division. Longitudinal section of lower end of proximal segment, showing infiltration of the nerve-fibres by proliferated leucocytes. The lower part of the figure represents the site of operation.

M. H. Lapidge, del. *Bale and Danielsson, Ltd., Chromo-Lith.*

A. Degeneration.

B. Regeneration.

The two processes overlap in point of time, regenerative changes commencing before degeneration is complete.

Degeneration.

Degenerative changes affect the lower end of the proximal segment and the whole extent of the distal segment. They occur simultaneously in these parts, and do not spread from the centre to the periphery, nor *vice versâ*.[1]

1. *Changes in the axis-cylinders and medullary sheaths.*—Fragmentation commences on the fourth day after the traumatism. The smallest axis-cylinders and medullary sheaths are more resistent than the larger ones, and remain unbroken until the fifth day (see 'Healing of Nerves,' plate 1, fig. 2). After that date, however, all the axis-cylinders and medullary sheaths, small as well as large, become completely fragmented.

The broken-down axis-cylinders and medullary sheaths form globular or ovoid masses, which gradually become absorbed. The process of absorption is practically complete by the end of five weeks, though a few scanty remains of fatty *débris* can often be detected for a long period afterwards.

2. *Cellular changes*:—(A) *Leucocytes.*—Diapedesis begins immediately after the injury, and is well marked for the first three days (see plate). It remains evident for two weeks, after which it gradually subsides.

(B) *Connective-tissue cells.*—These commence to proliferate on the second day. They have an absorbent action on the fragmented myelin and axis-cylinders (see 'Healing of Nerves,' plate 15, fig. 3). This process of absorption having been completed, they then proceed to the formation of fibrous tissue. Thus the degenerated

[1] The illustrative plates to which reference is made in the subsequent part of this paper are to be found in 'The Healing of Nerves,' by Charles Ballance and Purves Stewart (Macmillan, 1901).

nerve-trunk becomes denser in consistence than in the normal condition.

(c) *Neurilemma cells.*—These cells proliferate at a somewhat earlier time than the connective-tissue cells, and in a patchy fashion (see 'Healing of Nerves,' plate 15, fig. 2). A possible explanation of the earlier proliferation of the neurilemma cells as compared with that of the connective-tissue cells may be found in the fact that the neurilemma cells are nearer to the degenerating elements, and therefore receive the chemical stimulus first. After a short period, during which they have an absorbent action on the medullary sheaths, they relinquish this function to the connective-tissue cells, and commence the regenerative process by arranging themselves in closely packed longitudinal columns.

Regeneration.

(1) *In the proximal segment of a divided nerve which has not been reunited.*—Changes occur which result ultimately in the formation of the well-known so-called "end-bulb"—a dense, club-shaped swelling in which are numerous young nerve-fibres, coiled and intertwisted in all directions, embedded in fibrous tissue. According to the "central" theory of nerve regeneration, these new fibres were regarded as outgrowths from the central end, which, unable to find their way into the distal segment, had turned back on themselves in a futile manner like the apex of a fountain. This, however, is not so. Every new fibre of the permanent end-bulb is laid down in separate short links, each in apposition to a neurilemma cell, thickest near the nucleus of the cell and tailing off at each extremity. These short lengths of young nerve-fibres are, at first, separated by some distance from the ends of the central axis-cylinders. The end-bulb is not formed by a downgrowth and recurving of axis-cylinders, but by the development of new fibres in a structure which we have named the "*primitive end-*

bulb"—a mop-like protuberance which is formed immediately the nerve-trunk is divided, the result of the curling back upon themselves by the divided nerve-fibres (see 'Healing of Nerves,' plate 1, fig. 1). In this "primitive end-bulb" degeneration occurs as above described, and is followed by regeneration, a process carried out by the proliferated neurilemma cells, which secrete small islands of axis-cylinders and medullary sheaths. These islands later overlap in an imbricating fashion and ultimately fuse together to form a long nerve-fibre continuous with one of the nerve-fibres of the proximal segment above.

(2) *In the distal segment of a divided nerve, reunited to the proximal segment by sutures.*—The proliferated neurilemma cells arrange themselves in longitudinal columns, separated by strata of proliferated connective-tissue cells. The neurilemma cells have a neuroblastic function and proceed to form new axis-cylinders and medullary sheaths.

At the end of three weeks (with the Golgi method) neuroblastic action is first detected (see 'Healing of Nerves,' plates 8, 9, and 10). All through the distal segment scattered neuroblasts are seen, from whose opposite poles young axis-cylinders grow out longitudinally, stretching out towards similar processes of adjacent neuroblasts in the same longitudinal column, but not yet reaching them. By the end of four weeks these young processes have grown in length so as to overlap and fuse into long axis-cylinders (see 'Healing of Nerves,' plates 10, 11, and 12). In sections stained by the Weigert and Stroebe methods respectively the same process can also be studied, and the new axis-cylinders and medullary sheaths are seen to be secreted by the cells of the neurilemma. In the earliest stage of this process the young fibre is deposited along one side of the body of the cell in the vicinity of the nucleus. It grows in length and assumes a spindle form, thickest in the neighbourhood of the nucleus and tapering off at each pole (see 'Healing of Nerves,' plate 2, fig. 5, and plate 14, fig. 6). These

short lengths of new nerve-fibre grow in length until the processes of adjacent cells overlap in an imbricating fashion. They then fuse together to form a continuous undulating fibre in whose course are numerous bead-like swellings, corresponding to the neurilemma cells from which it has been secreted (see 'Healing of Nerves,' plate 3, fig. 12). As time goes on, the new axis-cylinders and medullary sheaths (both secreted by the neurilemma cells) gradually increase in diameter, the bead-like swellings disappear, and ultimately the adult form of fibre is attained.

The new fibres in the distal segment have a longitudinal direction from the outset, whilst in the intermediate scar-tissue between the proximal and distal segments their direction is wildly irregular (see 'Healing of Nerves,' plates 7 and 8). But the mode of formation is the same in every case.

(3) *In the distal segment of a divided nerve whose cut ends have been left widely separated.*—According to the "central" theory, it being impossible for new fibres to reach the distal segment from the proximal, regeneration cannot occur. But such is not the case. Regeneration can occur in the distal segment of a nerve even although widely separated from the proximal segment, and the process is exactly the same as that which occurs in a reunited distal segment. It commences, however, at a later date (four or five weeks after division, instead of three weeks, as in a reunited nerve), and progresses more slowly. Moreover the new fibres do not attain beyond the sinuous, beaded stage characteristic of incomplete maturity (see 'Healing of Nerves,' plate 4, fig. 13 and plate 14, fig. 9). If, however, such a distal segment, already partially regenerated, be sutured to the proximal segment, the new fibres quickly attain to adult proportions, thus illustrating the common truth that physiological activity is necessary for anatomical perfection.

Cases where the distal segment is in a condition of inflammatory sclerosis—such neuritis being usually of

microbic origin—may exhibit great delay or even total inhibition of the regenerative process.

Nerve Grafts.—In a number of cases, both in animals and in man, we have inserted a graft of fresh nerve tissue to join the proximal and distal segments of a divided nerve when separated too widely for suture. The longest graft that we have successfully employed in man has been two inches in length.

In such cases the transplanted portion of nerve serves to restore conductivity in the divided nerve-trunk. But it does so by acting simply as a scaffold into which there migrate successively leucocytes, connective-tissue cells, and neurilemma cells. Alongside the ingrowing blood-vessels of the new living tissue replacing the graft, the neurilemma cells (derived both from the proximal and from the distal segments) advance into the substance of the graft. There they proceed to arrange themselves into columns and secrete new axis-cylinders and medullary sheaths in the usual manner, whereby the proximal and distal segments become functionally reunited. In a microscopic section of a graft in the sixth week after operation, the neurilemma cells are found in greatest abundance by the side of newly formed blood-vessels of the young connective tissue replacing the graft. They appear around the vessels, under a low power, like a dense shoal of minnows (see 'Healing of Nerves,' plate 3, fig. 11). None of the original cells of the graft take part in this process of regeneration.

Clinical Remarks.—From what has been already stated it follows that in every case of accidental division of a nerve-trunk its reunion ought to be attempted either by—

(1) Primary suture at the time of injury;

(2) Secondary suture at a later date; or

(3) Transplantation of a portion of nerve from another animal.

In such operations the most satisfactory suture is one of the finest silk. It should be inserted in the fibrous sheath of the nerve, three or four sutures at least being used.

(1) *Primary suture.*—In this the surfaces, being already newly rawed, do not require to be "refreshed" unless they are ragged. Thus little or no shortening of the nerve-trunk results.

It should be remembered, however, that immediate return of function is not to be expected after primary suture, since before regeneration can occur degeneration must first take place. Return of function therefore occurs only after some weeks, the earliest date (in the lower animals) being the end of the fourth week.

(2) *Secondary suture.*—The immediate results of this procedure depend largely upon the length of time which has elapsed since the primary injury. If the period has been long enough (four weeks at least) to permit of regeneration in the distal segment to be fairly advanced, an immediate return of sensation in the previously anæsthetic area is often observed. There is no reason to suppose that any interval is too long to attempt secondary suture. In cases, however, where suppuration or microbic infection has occurred in the distal segment as a result of the injury, an interstitial neuritis may be set up sufficient to entirely prevent regeneration. This consideration would explain the striking success of certain cases of secondary suture and the equally conspicuous failure of others.

The inspection of the portion of the distal segment exposed at the operation gives no clue to the surgeon as to whether or not regeneration has occurred, but a microscopical examination of the small portion of tissue removed from the distal segment (in order to raw it previous to suture) may demonstrate that regeneration has taken place. The surgeon then may confidently predict the success of his operation. Thus, though operation may be advised in all cases, a successful result

cannot be predetermined, and is not assured (if the primary wound healed after suppuration) until sensation returns after the operation, or until a microscopical examination proves that regeneration has taken place in the distal segment.

In successful cases sensation always returns before motor power. Thus, for example, in one of our cases of complete division of the external popliteal nerve four months before by a bullet (of which a portion was found at the time of operation, between the proximal and distal segments), sensation had returned by the time the patient recovered consciousness after the anæsthetic, whereas motor recovery had not commenced five weeks later, though the muscular atrophy was distinctly less.

(3) *Nerve grafts.*—These are to be recommended only in cases where apposition of the proximal and distal segments is surgically unattainable. In our opinion nerve grafting is a preferable operation to that of turning a flap from one segment into the other, inasmuch as the making of such a flap diminishes the size of the nerve-trunk from which it is derived, whereas a graft unites undiminished ends.

The following subjects are suggested as a possible basis for discussion:

(1) The mode of regeneration of peripheral nerve tissue.

(2) The absence of regeneration in the spinal cord and brain after injuries, and its association with the absence of neurilemma cells in the central nervous system.

(3) The bearing of this on the neuron theory.

(4) Indications and contra-indications for operative interference.

(5) Comparative advantages of various operations.

[Dr. PURVES STEWART, after demonstrating by means of the epidiascope a number of drawings and photographs illustrating the processes of degeneration and of regeneration, added:]

If the views which we have maintained are correct, they entail a reconstruction of our conceptions as to the architecture of the nervous system. According to Waldeyer's neuron theory, which has for the last eleven years practically held the field, every nerve-fibre is a mere outgrowth from a nerve-cell, which outgrowth degenerates if cut off from its parent cell, and can only regenerate again by a process of downgrowth from that cell. According to the neuron theory, regeneration ought to be impossible in the distal segment of a divided nerve whose ends have remained widely separated. But our observations show that such is not the case. The neuron theory therefore, so far, at any rate, as the peripheral nervous system is concerned, must be discarded.

With regard to the clinical side of the question, if our views are correct, operative reunion of peripheral nerves ought to be attempted in almost every case. But are there any contra-indications for such operation? Firstly, there may be anatomical difficulties. Thus, for example, if the whole of the roots of the brachial plexus have been ruptured close to their exit from the intervertebral foramina, the surgeon might perhaps hesitate before diving down into a deep and dangerous dissection at the root of the neck. Another contra-indication might be the total loss of galvanic excitability in the atrophied muscles. Obviously it is not worth while reuniting a motor nerve if there are no muscle-fibres left for it to innervate. But such total disappearance of muscle-fibres is certainly less frequent than is commonly supposed. Thus I have examined the electrical reactions of muscles in a case sixteen years after division of their motor nerve, and still obtained some reaction to galvanism.

DISCUSSION

Professor C. S. SHERRINGTON thought the paper proposed a revolution in the teaching as to structure of nerves which was opposed to the neuron theory. It had been suggested that a nerve-fibre was not a single nerve-cell process, but a series of nerve-cells. The paper reminded him of experience as to the absence of regeneration after experimental lesions of the central nervous system, even after section of the posterior columns of the cord, which were hardly spinal fibres, but rather the direct continuation of peripheral nerves. After removal of the posterior ganglia he had, however, on one occasion found apparently new-formed nerve-fibres in the spinal part of the tract fifty-five days after the operation. Physiological difficulties in regard to the nature of nerve action—on an electrical type—were perhaps more easily explained by the new linked chain theory giving retardation of the impulses at the intervals between the *quasi*-electrical conductors. Similarly, the difference in resistance between the longitudinal and transverse axes of the nerve was accounted for to some extent, as were also the facts of polarisation, by the idea that the axis-cylinders were not continuous fibres, but apposed short lengths. However, this theory was perhaps somewhat difficult to accept in view of the degeneration in the peripheral nerves after removal of the trophic centres in the spinal cord. The regeneration of centripetally conducting fibres was even more difficult to get over.

Dr. R. KENNEDY (Glasgow) considered that these researches fully corroborated the results of his own investigations, published by the Royal Society some five years ago, by which the theory of the regeneration of nerves by a downgrowth from the peripheral end was combated. He referred to the view that the higher up a nerve was divided the longer was the time taken for the restoration of sensation. In his experience this had not been the case, and restoration of sensation had occurred as rapidly when the nerve was divided at one point as at another. This theory could not explain the rapid return of sensation after apposition of the divided nerves, occurring as it might even on the day following operation. The demonstration of newly formed fibres in the peripheral segment which had been for some time totally separated from the central segment was first made by Philipeaux and Vulpian in 1859, and had been confirmed by Bowlby and himself, and by other observers. These young fibres, however, never attained maturity while unconnected with the central end, for the reason that they had remained without an opportunity of performing their function of the normal trans-

mission of impulses from the nerve-cell. Dr. Kennedy then referred to the healing of divided nerves, and the restoration of the normal paths for the transmission of impulses. If the non-corresponding ends of nerve-fibres were brought into apposition and united, the consequence was that the peripheral terminations became in connection with centres in the central nervous system, to whose innervation they did not by nature belong. To determine this question he divided the sciatic nerve in dogs, and reunited it so that the fibres on the external aspect of the central end were in contact with those on the internal aspect of the peripheral end, and *vice versâ*. The result was that function was completely restored, and that as rapidly as in control animals in which the nerve had been divided and united as accurately as possible. A second series of experiments were then undertaken. The median, ulnar, and musculo-cutaneous nerves were divided above the elbow in dogs, and the musculo-spiral was divided at the same point; the central end of the musculo-spiral was then attached by suture to the peripheral ends of the three nerves which supplied the flexor muscles, and *vice versâ*. The result was that the animals in which these cross-sutures were made regained co-ordination of movement perfectly. In these animals, after co-ordinated function had been restored, the condition of the cerebral cortical areas associated respectively with flexion and extension of the paw were examined by cortical stimulation, and it was found that the relative positions of the two areas had become reversed. He also referred to a case in which he had for facial spasm divided the facial nerve and united the peripheral end to the spinal accessory, with the result that the face recovered its power of movement to a great extent, but that whenever the patient suddenly lifted the right arm a spasm of the face was produced.

Dr. R. A. Fleming (Edinburgh) said that he was still, to some extent, an upholder of the "central" view of regeneration, and he did not see why it should not be compatible with the finding of new-formed fibres in the distal segment of a divided nerve. He referred to some experiments which he had performed on rabbits in which he had ligatured the sciatic nerve in two places, and he had been able to demonstrate young axis-cylinders both in the part between the two ligatures and also in the peripheral part eighteen weeks after the operation. He asked Mr. Ballance and Dr. Stewart what method of fixing they had adopted, and in what medium the sections had been cut. Stroebe's method was particularly apt to give fallacious results when applied to celloidin specimens. He did not agree with their observations that the fine nerve-fibres degenerated more slowly than the larger fibres, but he thought, on the contrary, that they degenerated more rapidly. He had for long held that the neurilemma nuclei acted as trophic agents to the

axis-cylinder which they protected. It was therefore, from his standpoint, not improbable that regeneration should take place from these cells. When old neurilemma sheaths were found to contain small new fibres, it was always in the central end of a divided nerve; this he held to be in favour of the central theory, and he considered that the sketches in Mr. Ballance's and Dr. Purves Stewart's book bore out this contention. To hold the peripheral theory it was not necessary to absolutely reject the central. It was a matter for future experiment whether union of nerve by first intention without previous degeneration of the peripheral segment was possible, but present researches seemed almost uniformly to point to such primary union being very problematical.

Dr. Fleming stated that his specimens above referred to were cut in paraffin and stained by a modification of Stroebe's method.

Mr. W. Thorburn (Manchester) referred to the extraordinary way in which the central nervous system adapted itself to the new conditions after reunion of divided nerves. In a case in which a portion of practically the whole of the brachial plexus was excised, so that it was impossible to unite each nerve to its corresponding trunk, a more or less indiscriminate union was performed, and there was as a result but little confusion in the weak movements that returned.

Adjourned Discussion, March 11th, 1902.

Prof. J. N. Langley (Cambridge), in resuming the adjourned discussion on this subject, remarked that the primary question was whether a nerve separated from its central connections could regenerate of itself; from his own experiments and observations he had come to the conclusion that a peripheral nerve might regenerate of itself. His own observations had had to do with the sympathetic system; after a sympathetic ganglion had been removed in the cat, two years later there was apparently regeneration of medullated and non-medullated fibres. It was very difficult to distinguish with certainty between non-medullated nerve-fibres and strands of connective tissue, and therefore non-medullated fibres might possibly exist between the peripheral ends and their centres, which were undiscoverable by the microscope; and thus it was possible that the severed nerves might receive a stimulus from the central ganglia by fibres that could not be traced. After extirpation of the superior cervical ganglion at the end of a year the peripheral end was stimulated without effect, then another portion of the central end was excised, but after an interval of days there was no degeneration in the peripheral end, thus apparently excluding the possibility of nerve stimuli reaching

the peripheral end from its own central end. It was possible that the sympathetic system recovered more easily than the ordinary peripheral nerves; but even in them, although there was return of histological structure, there was no return of function. This was possibly due to the greater vulnerability of the terminations of the sympathetic nerve as compared with the fibres themselves. The variation in recovery would probably differ in different nerves and in different animals. As to the manner in which the actual regeneration occurred, the neuroblast view of the authors of the paper was not convincing; the observations made were on sections, and it was not possible to follow a nerve-fibre by this method; the method of teasing was necessary. The Golgi stain was erratic, and osmic acid stains were certainly preferable both to it and the Weigert stain. His observations had rather revealed the idea of a long multinucleated cell than a series of short cells as the authors had maintained. The difficulty of admitting the phagocytic action of the connective-tissue cells for the medulla was obvious in view of the fact that they were separated by a membrane—the neurilemma. The earlier disappearance of the medullary substance of the large fibres was probably due to the staining agent. In his experience this change was earlier in the small than in the larger fibres. The rapid return of sensation (from an hour to a day) in cases of secondary suture was not cleared up by the paper. If recovery of function did not correspond with regeneration of structure how could it be explained? The nerve could not be cut without the stump of the central end degenerating, and this in itself negatived the rapid recovery on the basis suggested in the paper, as seven to ten days at least were required for their recovery. He would suggest as a theory that the division of one or more nerve-fibrils might happen just between two nerve segments, and thus the two in contact end to end might escape injury, and being in anatomical continuity might transmit sensory impulses. In the surgical operations for the repair of a divided facial nerve it was usual partially to divide the spinal accessory nerve and to graft the facial on the central end of the spinal accessory. He would suggest that it might be better to cut the spinal accessory nerve right across, to split it, and to unite one portion thus divided to the facial and the other to the peripheral trunk of the spinal accessory.

Dr. F. W. Mott congratulated the authors of the paper on affording still further proof that regeneration of a divided nerve took place from the periphery. For some time past he had been engaged with Professor Halliburton in making a series of observations upon the chemical changes occurring in nerves undergoing degeneration and regeneration after their division. The inquiry was not conducted for the purpose of ascertaining

how regeneration took place; therefore a positive opinion in the paper which was published in the 'Philosophical Transactions' was not expressed, but Dr. Mott, from the histological examination of the nerves, came to the conclusion that new axis-cylinders were formed by the proliferation of the cells of the neurilemmal sheath. Subsequent observations which he had made confirmed this opinion. He stated that he relied upon teased preparations rather than sections, as a most valuable method of studying both degeneration and regeneration, because individual fibres could thus be seen in their entirety. He used the direct Marchi method for fixing and hardening the tissues, which stained the myelin a greenish grey and the degenerated myelin black. He considered that this was a more valuable method than the Weigert, which stained both myelin and degenerated myelin blue. The protoplasmic substance of the new axis-cylinders and the nuclei of the neurilemmal cells were subsequently stained by the Ströbe method and logwood. In this way he was enabled to see the proliferation of the neurilemmal cells, their phagocytic action upon the degenerated products, and formation of the axis-cylinder process and new sheaths by a process of differentiation of their protoplasm. He purposely did not use the term "secretion of axis-cylinders" which the authors of the paper had employed. He was of the opinion that the axis-cylinders were formed more especially by the nuclear protoplasm of the cells. Frequently in the neighbourhood of the nuclei of these teased preparations he had seen the products of degeneration of the myelin stained black, mingled with highly refractive, much lighter stained globules which appeared like the new myelin. He stated that he was at present engaged with Professor Halliburton in making further inquiries regarding the process of degeneration and regeneration under the following conditions:—(1) the process of regeneration of the divided ulnar nerve after section of a sufficient number of posterior roots on one side to produce paralysis; (2) the conditions of the nerve terminations in the skin and muscles. So far the observations were not sufficiently numerous to make any definite statement, but they tended to show that stimulus played an important part in regeneration. He asked the authors of the paper whether they had in their numerous experiments tested the conductivity of the nerve above and below the seat of division and union with the faradic current. Dr. Mott considered the Golgi method, which the authors of the paper had used, unreliable for pathological purposes. The experiments of Dr. Kennedy, of Glasgow, had practically established the fact that regeneration of nerves took place from the periphery, and this had now been confirmed by the researches of Mr. Ballance and Dr. Purves Stewart.

Mr. Mayo Robson was sorry that he had been unable to be

present to hear the opening paper, but he had had the opportunity of becoming acquainted with what had passed at the last meeting of the Society. The experiments proved conclusively all that has been advanced by the authors, but he thought that the clinical observations he had reported some years ago, and which bore very pertinently on some of the questions, had not quite received the notice they merited. The questions he had advanced could only be partly settled experimentally, and he hoped, now that he had a "pied à terre" in London, to find time to work at the subject from this point of view. Some of the points could, however, be better settled by clinical research. He believed that he had been the first to perform nerve grafting, and, so far as he knew, the idea had not been previously conceived. His first example had been fully reported in the 'Transactions of the Clinical Society of London' for January, 1889.

The case was that of a young girl from whom he removed a tumour of the forearm the size of a small orange, which involved the median nerve, after which sensation and movement in the parts supplied by the nerve were found to be absent. It had occurred to him that if he could substitute a fresh portion of nerve for the part removed he might be able to restore the lost function; and the day after the first operation he reopened the wound and transferred a portion of posterior tibial nerve directly from a young man's leg amputated in an adjoining theatre by a colleague, the interval between the amputation and the grafting being merely momentary, the graft being transferred in normal saline solution. The interesting point now came in, for, much to his astonishment, sensation in all the parts supplied by the median nerve had been good when tested the following day, and remained so throughout the convalescence, though the motor functions were only restored later.

Now, according to the authors of the paper, degeneration in the distal segment of a divided nerve did not begin until four days subsequent to the accident, and what he wanted to know was—in case of an aseptic division of nerve and an immediate restoration of continuity either by immediate union or by union of an aseptic graft—whether or not degeneration of the divided segment was a necessity,—whether, in fact, the restoration of physiological activity by the re-establishment of nervous impulses along it might not prevent degeneration and so lead to anatomical perfection; or, to put it in another way, whether the immediate establishment of anatomical continuity might not keep up physiological activity, and so prevent degeneration of the distal segment.

Facts were stubborn things, and those that he had given required an explanation. Some physiologists had chosen to ignore his observations, doubtless because they could not

explain them, and possibly thinking them due to inaccurate observation; but he was positive of his facts, and if his explanations were not correct they would have to be proved inaccurate by further experiments and clinical observations before he could be satisfied.

Now, as to the time when regeneration of the distal segment was possible, he could give an example, also published ('British Medical Journal,' October 31st, 1896). In this case, seen in January, 1890, seven months after a scythe accident, a man aged 29 had been sent to him with a useless arm. There was a large scar over the inner and lower end of the arm just above the elbow, and the parts supplied by the median and ulnar nerves were absolutely paralysed, so that the forearm was a useless flail. He determined to try to save the limb, and to this end he exposed the nerves and excised the bulbous ends; with slight tension he secured end-to-end union of the ulnar nerve, but the extremities of the median were separated by a space of fully three inches, into which he inserted the spinal cord of a rabbit, thus securing continuity. In ten days sensation began to return in the median which had been united by a graft, but in the ulnar which had been directly united, a return of sensation was much longer delayed, as was return of muscular power. Ultimately complete recovery ensued. So complete was the recovery that the man returned to his work as a platelayer on the Midland Railway.

This case was interesting as showing that so long after division as seven months was not too late to hope for restoration of function in the distal segment of a divided nerve. As to the material for suture, to his mind fine xylolised catgut was better than silk or any non-absorbent material. The authors of the paper put the limit of a successful graft as two inches; in the cases he had related the graft was fully three inches long. In venturing on these criticisms he would at the same time congratulate the authors on the excellent work they had done.

Dr. W. Aldren Turner discussed the attack made in the paper on the neuron theory in relation to the peripheral nerves. He did not think it would lead to the neuron theory being discarded, as there were too many facts in its favour. There were not only the facts of Wallerian degeneration, but there was an important change affecting the central end of the divided nerve extending up to the nerve-cell which had not been dealt with. It was, indeed, a defect of the paper that the condition of the whole of the peripheral nerve (including its cell) after section was not described. The fact that new fibres did not reach maturity until the divided ends were sutured rather supported than negatived the neuron theory. He asked what occurred in the peripheral nerves in acute anterior poliomyelitis; if regeneration did not occur the neuron theory was

present in lymph were chiefly skin organisms and non-sporing, and hence were amenable to glycerinisation. It had been pointed out by the Special Commission on Glycerinated Calf Vaccine Lymphs that when non-spore bearing organisms were present in large numbers this was due to imperfect glycerination, but that when spore-bearing organisms were in excess in any lymph it was an index that that lymph had not been sufficiently carefully collected. He alluded to the effect of glycerinisation on the activity of the lymph, and expressed the opinion that little or no weakening occurred within three or four weeks. After a longer time weakening probably did, to a slight extent, occur, but the deterioration was more likely to be due to under-glycerinisation than to over-glycerinisation. When organisms that grow best at the body temperature were in excess glycerinisation had a more active influence. The local inflammatory effect of vaccine had been considered of late by many to be greater than formerly, but this was probably due to imperfect glycerinisation, the greater part of the local effect being always due to extraneous organisms. He suggested that the Local Government Board might be able to prepare statistics from their returns to decide that point.

Dr. T. D. ACLAND said that whilst acting as medical officer to the Royal Commission on Vaccination he had had an unusual opportunity of seeing the methods of vaccination practised throughout the country, and of forming an opinion as to the manner in which the operation was carried out, and the cause and extent of vaccinal injuries. In the course of the same inquiry he had been brought intimately into relation with the Medical Department of the Local Government Board, and was able to appreciate the manner in which its officers carried out the difficult and often thankless task of maintaining efficient vaccination. There could be no question that the country owed much to Dr. Copeman for his researches into the origin and purity of vaccine lymph, and for his share in the establishment of an institute able to supply calf lymph on a scale quite impossible a few years ago. Dr. Acland hoped that the Government would take steps to establish a laboratory on a far larger scale than at present, so that all practitioners in the kingdom might be able to obtain lymph from a laboratory fitted with every requisite for perfect work, unfettered by economies necessary in an establishment run solely for trade purposes. He thought that it would be well also if all supplies of vaccine lymph were placed under Government control, and expressed regret that there should be two "Jenner Institutes" in this country—the one formerly known as the "British Institute of Preventive Medicine" in Chelsea, where the National Vaccine Establishment was temporarily situated, the other the Jenner Institute for calf lymph in Battersea, to which the name by priority

ration of their results in his description of the nerve-fibre as a series of linked cells, each of them containing in solution electrolytes, thereby explaining the retardation of electrical stimuli in their passage along a peripheral nerve. In the course of his remarks he had also referred to the occurrence of regeneration in the posterior roots five weeks after the excision of the root-ganglion, and he now showed them, for him, a photograph of such regeneration taken by him in 1894. They had examined nerves from several cases of anterior poliomyelitis in which Dr. Batten had cut sections, but had failed in these particular instances to find evidences either of degeneration or of regeneration; but in this connection there was an interesting point figured by Ziegler in his 'Pathology' (Bd. ii, fig. 194, 6th German edition, 1890). This picture was described as showing advanced degeneration in a peripheral nerve after atrophy of the anterior horns, but in their opinion it might equally well be described as showing the beaded stage of regeneration so frequently illustrated in their series of drawings. With regard to Dr. Fleming's question as to whether primary reunion ever occurred after division and immediate suture, they would unhesitatingly answer in the negative. Professor Langley's experiments on regeneration in the cervical sympathetic were of great interest, and afforded another corroboration of the results obtained by themselves. He seemed inclined to doubt the phagocytic action of the proliferated connective-tissue cells on the theoretic ground of the supposed continued integrity of the neurilemma sheath. They had, however, satisfied themselves that the proliferated connective-tissue cells did contain myelin, and that process of phagocytosis was identical with that observed in all other injured tissues. As to the immediate return of sensation in certain cases of secondary suture, the clinical fact was beyond question, not only from their own observations but from those of many observers. The most ingenious explanation of those cases in which sensation was alleged to return immediately after primary suture appeared to be that offered by Professor Langley himself, namely, that there were probably a number of fibres in which the severance happened to occur exactly at a node of Ranvier, and in which no degeneration needed necessarily to occur proximal to the site of injury. He was not inclined to accept Professor Langley's suggestion that in facio-accessory anastomosis for the treatment of facial palsy the accessorius should be partly divided (rather than merely incising its sheath) in order to obtain end-to-end anastomosis with the stump of the facial. Such a procedure would be based upon the old theory of the outgrowth of new fibres from the proximal end, which was no longer tenable. The chemistry of nerve regeneration, to which Dr. Mott, in conjunction with Professor

Halliburton, had made such valuable contributions, was a point which they did not venture to discuss. The conclusions, however, at which those authors had arrived were in many respects strikingly similar to their own histological work. The discussion of the neuron theory he would leave to Dr. Stewart.

Dr. PURVES STEWART, in reply, said that Mr. Ballance had already dealt with a number of points raised by the various speakers, and that he would only allude to those not already referred to by him. A very interesting question was raised by Professor Sherrington when he asked why, if the nerve-fibre was a linked chain of cells, did the whole chain degenerate and not merely the injured links? To this he would venture to suggest that the explanation was to be found in the loss of impulses from the rest of the nervous system which necessarily occurred when a nerve-fibre was divided. Dr. Fleming had inquired as to the fixing methods employed by them in the course of their research. In most cases the nerves had been fixed in Müller's fluid; they had found that formalin-hardened specimens did not take the aniline blue stain properly. Stroebe's stain was uncertain in its results, but when it was successful the pictures yielded by it were of great value. But the bulk of their results did not depend upon the observations made with the Stroebe stain; they were based chiefly on the Weigert-stained series. Dr. Fleming still held that in the process of degeneration the finest fibres broke down earlier than the larger ones. They could not agree with that statement, and in some sections of degenerating nerve stained by Dr. Batten, to which he had recently had access, a similar survival of the finest fibres was also quite clear, as described by them in their research. The Golgi method, as Professor Langley pointed out, was rather an uncertain one, but in their cases it had been most successfully carried out for them by Dr. David Orr, of Prestwich. He did not think that the cells described by them as neuroblasts could be connective-tissue or glia cells, for they had been totally absent in the distal segment at the end of one and two weeks respectively, and abundantly present at the third and fourth weeks, and their axis-cylinder processes had been many times longer at the fourth week than at the third. Both Dr. Batten and Dr. Turner had properly pointed out that to gain a proper conception of the processes of degeneration and of regeneration, it was not sufficient to confine one's examination to the immediate neighbourhood of the injury, but that one should examine the whole extent of the nerve. In many cases this had been done by them, and in every instance the changes had been identical throughout the entire peripheral extent of the nerve. The well-known delay in the return of motor functions as compared with the early recovery of sensation was probably referable to the muscular atrophy which had

to be recovered from, in addition to the recovery of the nerve-trunk.

But the various views which had been expressed in the course of this discussion had not shaken the essential fact from which they had started, viz. that regeneration occurred in a nerve-fibre even when it was permanently cut off from its corresponding nerve-cell. The question then arose, "On what theory could these facts be explained?" The neuron theory did not explain them. According to the neuron theory the nervous system was made up of innumerable units called "neurons." Each neuron is supposed to consist of a nerve-cell, with its various processes, the nerve-fibres, all of them being mere outgrowths from the cell. But this theory did not fit all the facts. Thus, for example, it did not explain the absence of regeneration in the central nervous system after injury, nor did it account for the occurrence of regeneration in the peripheral nerves when cut off from the central nervous system. The logical procedure was not to shut their eyes to the facts and cling to the moribund neuron theory, but to stick to the facts and throw the inadequate theory overboard.

And if so, what hypotheses were they to substitute which would harmonise with the facts? It was perfectly obvious that the central nervous system exerts a profound influence upon all nerve-fibres, including those of the peripheral nerves. Degeneration undoubtedly did occur in a nerve-fibre if separated from its corresponding nerve-cell; but the probable reason for this degeneration was that the function of the nerve-cell was to divert impulses from other parts of the nervous system into the nerve-fibre; and the nerve-fibre degenerated, not because it was cut off from the nerve-cell as a cell, but because it was cut off from the impulses reaching it from the rest of the nervous system.

The most reasonable explanation appeared to be that offered by Apàthy, and supported by Bethe, Nissl, and others, according to which the essential elements of the nervous system were the nerve-fibrils. Each nerve-fibre was made up of a bundle of these nerve-fibrils. The fibrils of different parts of the body were connected one with another through the central nervous system, which was a huge "exchange" in which the nerve-cells acted as convenient shunts or depôts, transmitting impulses from one set of fibrils to another. When an outlying nerve-fibre was cut off from the central exchange it was no longer functionally active, and therefore degeneration set in. But the central segment, which remained connected with its corresponding nerve-cell or nerve-shunt, still received impulses from other fibrils (through the cell), and did not degenerate. Later on, the detached distal segment was regenerated by the activity of

the neurilemma cells, and was ready to resume its function if linked on to the central nervous system. The essential elements of the nervous system, therefore, were the nerve-fibrils; the nerve-cells were accessories. Their conception of the nervous system should be that of a vast and delicate interlacement of nerve-fibrils, with nerve-cells interspersed at convenient intervals to act as depôts or shunts for nerve impulses.

ATELEIOSIS

A DISEASE CHARACTERISED BY

CONSPICUOUS DELAY OF GROWTH AND DEVELOPMENT

BY

HASTINGS GILFORD, F.R.C.S.Eng.

Received April 5th, 1902—Read June 11th, 1902

Introduction.

In 1868 Professor Schaaffhausen, of Bonn, wrote an account of a dwarf who died at the age of 61, and whose appearance and proportions were those of a child. This dwarf showed no signs of cretinism, rickets, or any other of the known causes of stunted growth. Fourteen years later Schaaffhausen was able to obtain the skeleton, when he found that the development of the bones corresponded with the development of the soft parts, for most of the epiphyses were still ununited. No other contribution of any importance to this particular type of dwarfism appeared until 1891, when Dr. Arnold Paltauf described another case in his monograph on "Zwergwuchs." A third instance was recorded in the same year by Dr. Schmidt, of Munich. Dr. Manouvrier, of Paris (1896), and Dr. Joachimsthal, of Berlin (1899), then followed with other cases. It is also probable that the dwarf briefly described by Professors

Ranke and von Voit (1885), and another by Dr. A. A. Bouchard (1884), were of the same type. Further examples of the disease have been incidentally referred to by Professor Quetelet, Dr. N. W. Kingsley, Drs. Gould and Pyle, Geoffrey St. Hilaire, and others. We also find instances in the writings of certain semi-scientific and popular authors, such as E. J. Wood, E. Garnier, P. O. Barnum, and " Count " Boruwlaski.

By far the most important of these contributions is that by Dr. Paltauf, who gives the results of a *post-mortem* examination of his case, and compares it with other forms of dwarfism. I myself have been able to examine two skeletons, and four living cases, and to make one *post-mortem* examination.

Though the details given of many of the cases which have been recorded are somewhat meagre, yet there appears to be sufficient material to enable us to form a fairly complete picture of the disease. This disease cannot be accounted for by any of the known causes of dwarfism. Cretinism, syphilis, microcephaly, achondroplasia, rickets, and congenital heart disease, can be excluded. In short, the morbid condition has a definite individuality. Its most striking feature appears to be the delay in growth and development. Though other diseases may have a retarding effect upon these processes, yet in none of them does this one feature stand out so prominently. In cretinism, for example, though the delay of development may be of equal degree, yet it differs in that there are certain other morbid conditions of mind and body which are quite as conspicuous. This delay of growth and development is so evidently the main feature, that I have suggested that the disease should receive a name which emphasises this fact. I have proposed that it should receive the name of Ateleiosis (ἀτελείωσις, not arriving at perfection). It should be noticed that usually neither growth nor development is arrested, though both are indefinitely retarded. Those who are affected may, in fact, grow slowly up to the age of 30 years, or even later.

The examples recorded seem to be capable of division into groups or classes, according to the age at which the disease first declares itself. Like cretinism, it may appear either before birth (Group I), during infancy (Group II), or not until the later stages of development (Group III). The subjects of the disease are, therefore, not necessarily dwarfs. Most of the cases began during infancy or early childhood, and consequently belong to the second class, when the disorder apparently exhibits its most characteristic features. We may find eventually that there is a fourth class, for it is not improbable that it may also begin after puberty.[1] In all likelihood the disease, as a rule, becomes so ill-defined when it appears in these later years that it can only be recognised as a mere eccentricity of normal development, or is lost altogether in those variations to which all life is subject.

I now propose to divide my subject into two parts, first giving a short account of the cases which have been reported up to this time, with fuller details of those which have come under my own observation. There will then be a description (in Part II) of the chief features of the malady, followed by remarks on its diagnosis and pathology. The description of a disease which has no name is so inconvenient that I have ventured to use the name I have suggested throughout the rest of this article.

PART I.

GROUP I.—*Ateleiosis beginning during Fetal Life.*

CASE 1.—In the Museum of the Royal College of Surgeons of England is the "skeleton of a female child of unusually stunted growth and arrested osseous development." It is the skeleton of Caroline Crachami, who was exhibited in London in 1824, under the name of "the Sicilian dwarf," and at the time of her death was said to be of the age of 9 years. A portrait of her in oils is also

[1] A possible example may be found in König's case on p. 343.

the neurilemma cells, and was ready to resume its function if linked on to the central nervous system. The essential elements of the nervous system, therefore, were the nerve-fibrils; the nerve-cells were accessories. Their conception of the nervous system should be that of a vast and delicate interlacement of nerve-fibrils, with nerve-cells interspersed at convenient intervals to act as depôts or shunts for nerve impulses.

ATELEIOSIS

A DISEASE CHARACTERISED BY

CONSPICUOUS DELAY OF GROWTH AND DEVELOPMENT

BY

HASTINGS GILFORD, F.R.C.S.Eng.

Received April 5th, 1902—Read June 11th, 1902

Introduction.

In 1868 Professor Schaaffhausen, of Bonn, wrote an account of a dwarf who died at the age of 61, and whose appearance and proportions were those of a child. This dwarf showed no signs of cretinism, rickets, or any other of the known causes of stunted growth. Fourteen years later Schaaffhausen was able to obtain the skeleton, when he found that the development of the bones corresponded with the development of the soft parts, for most of the epiphyses were still ununited. No other contribution of any importance to this particular type of dwarfism appeared until 1891, when Dr. Arnold Paltauf described another case in his monograph on "Zwergwuchs." A third instance was recorded in the same year by Dr. Schmidt, of Munich. Dr. Manouvrier, of Paris (1896), and Dr. Joachimsthal, of Berlin (1899), then followed with other cases. It is also probable that the dwarf briefly described by Professors

Ranke and von Voit (1885), and another by Dr. A. A. Bouchard (1884), were of the same type. Further examples of the disease have been incidentally referred to by Professor Quetelet, Dr. N. W. Kingsley, Drs. Gould and Pyle, Geoffrey St. Hilaire, and others. We also find instances in the writings of certain semi-scientific and popular authors, such as E. J. Wood, E. Garnier, P. O. Barnum, and " Count " Boruwlaski.

By far the most important of these contributions is that by Dr. Paltauf, who gives the results of a *post-mortem* examination of his case, and compares it with other forms of dwarfism. I myself have been able to examine two skeletons, and four living cases, and to make one *post-mortem* examination.

Though the details given of many of the cases which have been recorded are somewhat meagre, yet there appears to be sufficient material to enable us to form a fairly complete picture of the disease. This disease cannot be accounted for by any of the known causes of dwarfism. Cretinism, syphilis, microcephaly, achondroplasia, rickets, and congenital heart disease, can be excluded. In short, the morbid condition has a definite individuality. Its most striking feature appears to be the delay in growth and development. Though other diseases may have a retarding effect upon these processes, yet in none of them does this one feature stand out so prominently. In cretinism, for example, though the delay of development may be of equal degree, yet it differs in that there are certain other morbid conditions of mind and body which are quite as conspicuous. This delay of growth and development is so evidently the main feature, that I have suggested that the disease should receive a name which emphasises this fact. I have proposed that it should receive the name of Ateleiosis (ἀτελείωσις, not arriving at perfection). It should be noticed that usually neither growth nor development is arrested, though both are indefinitely retarded. Those who are affected may, in fact, grow slowly up to the age of 30 years, or even later.

The examples recorded seem to be capable of division into groups or classes, according to the age at which the disease first declares itself. Like cretinism, it may appear either before birth (Group I), during infancy (Group II), or not until the later stages of development (Group III). The subjects of the disease are, therefore, not necessarily dwarfs. Most of the cases began during infancy or early childhood, and consequently belong to the second class, when the disorder apparently exhibits its most characteristic features. We may find eventually that there is a fourth class, for it is not improbable that it may also begin after puberty.[1] In all likelihood the disease, as a rule, becomes so ill-defined when it appears in these later years that it can only be recognised as a mere eccentricity of normal development, or is lost altogether in those variations to which all life is subject.

I now propose to divide my subject into two parts, first giving a short account of the cases which have been reported up to this time, with fuller details of those which have come under my own observation. There will then be a description (in Part II) of the chief features of the malady, followed by remarks on its diagnosis and pathology. The description of a disease which has no name is so inconvenient that I have ventured to use the name I have suggested throughout the rest of this article.

Part I.

Group I.—*Ateleiosis beginning during Fetal Life.*

Case 1.—In the Museum of the Royal College of Surgeons of England is the "skeleton of a female child of unusually stunted growth and arrested osseous development." It is the skeleton of Caroline Crachami, who was exhibited in London in 1824, under the name of "the Sicilian dwarf," and at the time of her death was said to be of the age of 9 years. A portrait of her in oils is also

[1] A possible example may be found in König's case on p. 343.

preserved on the walls of one of the staircases in the Museum. Through the kindness of the Council of the Royal College of Surgeons I have been able to obtain a photograph of both the skeleton and the picture. Her portrait shows that she was of a thin and delicate figure, and that her hands and feet were in proportion to the rest of her body. A cast of her face, left ankle and foot, and a portion of one upper extremity, which are included in the case with the skeleton, exhibit the same features. Her attitude is indicative of weakness or fatigue, and the expression on her face is dull and heavy. She looks much older than her years, but this is partly due to the prominence of her nose, which would have been remarkable even in an adult. Her hair is plentiful, and the nails are well formed.

After her death an examination of her body was made by Sir Everard Home,[1] who has written the following account of her:

"An Italian woman, aged 20 years, when, by her reckoning, three months gone with her child, was travelling in a caravan with the baggage of the Duke of Wellington's army. In the middle of the night, in a violent storm, when she was fast asleep, a monkey that had been chained on the top of the caravan, in its fright found its way into it, and, as the warmest berth it could find, got under her clothes. Half asleep, she put her hand down to scratch herself, but on scratching the monkey it bit her fingers and threw her into fits. She did not miscarry, but went her full time. The child, when born, weighed one pound, and measured seven inches in length. It was reared with difficulty, and was carried by its parents to Ireland, where it became consumptive.

"It was brought to London and shown as a curiosity. It died just after it completed its ninth year. I saw it several times while alive, and it came into my possession after death.

"On examination of the body after death the fontanelle

[1] 'Lectures on Comparative Anatomy,' 1828, vol. v, p. 191.

Gilford: Ateleiosis. Plate I.

A. B.

Ateleiosis Group i. Case 1. Skeleton of Caroline Crachami (A) standing by the side of normal skeleton of a child of 15 months (B). The ossification of the former is less advanced than it is in the latter. The skeleton, which is in the Royal College of Surgeons' Museum in London, is distorted by the passage of an iron rod through its cerebro-spinal axis. [*For portrait see end of paper.*

Bale & Danielsson, Ltd., London.

was closed.[1] There was no fat in any part but in the sockets of the eyes, behind the balls. The uterus had not been developed beyond that contained in a fœtus of four months. The bladder was distended with urine to the size of a hen's egg. As the child had never made water freely from its birth, the bladder probably had been injured at the time the monkey alarmed the mother. On comparing the ovaria with those of an abortion of three months, they were nearly of the same size.

"The child, when I saw it, could walk alone, but with no confidence. Its sight was very quick, much attracted by bright objects, delighted with everything that glitters, mightily pleased with fine clothes; had a shrill voice and spoke in a low tone; had some taste for music. Was very sensible of kindness, and quickly recognised any person who had treated it kindly. The mother has had a fifth child in Ireland, which, like her first three children, is naturally formed."

In the accounts of her death in the newspapers[2] of the day, it is stated that she had been "for some time afflicted with a cough," and that "on Thursday last she was exhibited as usual, and received upwards of 200 visitors; towards the evening a languor appeared to come over her, and on her way from the exhibition room she expired."

The skeleton has been but indifferently mounted, for a rod has been so run through the cerebro-spinal cavity as to obliterate all the natural curves, dislocate the atlas and skull off the axis, and project through the anterior fontanelle.

From this cause it is impossible to obtain accurate measurements of some parts, and the whole aspect of the skeleton is distorted. The clavicles are horizontal, the scapulæ are dragged away from the thorax, and the shoulders are raised so as to appear stiff and square, and very different from their rounded appearance in the paint-

[1] This is incorrect.

[2] 'The Newspaper,' June, 1824.

ing. Owing to this elevation of the shoulders one does not notice at first the disproportionate length of the upper extremities, inasmuch as they do not reach down too low on the thigh. The pelvis, also, is made to project forward so that its brim is almost horizontal, while the lower limbs are dragged up to such an extent as to diminish the height appreciably.

Another source of inaccuracy of measurement is due to shrivelling of the epiphysial cartilages.

Height, 49 cm. = 19⅓ inches.

Middle point of total height, 1·3 cm. = ½ inch, above the symphysis pubis.

Length of spine, 5·8 cm. = 2¼ inches.

Clavicle, 4·8 cm. = 1⅞ inches.

Acromion to elbow, 8·9 cm. = 3½ inches.

External condyle to lower end of radius, 7 cm. = 2¾ inches.

Femur, 12 cm. = 4¾ inches.

Tibia, 9 cm. = 3½ inches.

Total length of lower extremity, 23 cm. = 9 1/16 inches.

Foot, 6·8 cm. = 2⅝ inches.

Hand, 6 cm. = 2⅜ inches.

Skull: greatest length, 12·6 cm. = 5 inches; greatest breadth, 9·4 cm. = 3⅝ inches; vertex to base, 8·55 cm. = 3⅜ inches; circumference, 35 cm. = 13¾ inches.

The skull is very thin and delicately formed. The anterior fontanelle is open for a length of 1·6 cm. (½ inch) and a width of 1·2 cm. (5/12 inch), but the posterior fontanelle is closed. The lower half of the frontal suture is closed, but there is a small unossified oval area 6 mm. (¼ inch) long, near the superior angle of the occipital bone close to the left lambdoid suture. Another is present at the bottom of each occipital groove. The basi-sphenoid and basi-occipital bones are not united. There is no thickening of the sutures. The skull is unopened, but on placing a lighted match in the occipital foramen the bones of the calvarium are seen to be of fairly even thickness, and on looking through the open fontanelle the sella

turcica and the other parts of the base are also found to be normal. The ear bones are of adult size, but the auditory process is a mere ring, like that which is met with in infants. The palate is of normal shape, but the alveolar border of the upper jaw is very irregular, and, owing to absorption of the outer part of the alveolus from the canine teeth backwards, descends lower in front than behind. The mandible is ill-formed. Its angle and ramus are like those of an infant, and while the alveolar portion is unduly thick, the chin is ill-developed, and therefore recedent. The dental formula is as follows, viz. :

Upper jaw, 2 m. × 1 c., 1—2 in., 1 c. × 2 m.

Lower jaw, 2 m. × 1 c., 1—2 in., 1 c. × 2 m.

The teeth are all of the temporary set, and are of natural size as a rule, but very irregularly placed. All four of the second molars can be seen, though they do not project to the level of the gums. The two upper first molars, as well as the right upper second molar, are carious, but the others are sound.

Ossification throughout is greatly delayed. This is shown both in the backward condition of the epiphyses, and in the smallness and delicacy of the shafts. There is but a trace of roughness to show where muscles were attached, and the position of several of the muscular prominences is not visible. The long bones possess hardly any curve, and are narrowest at the middle of their shafts, and from thence increase in width towards their ends. The ribs have very abrupt curves at their angles, but are almost straight from thence forwards, and those on the right project more and are more horizontal than those on the left. This last peculiarity of position is possibly due to the way in which the specimen is mounted. There are no centres visible for the spinous and transverse processes of the vertebræ, and the atlas is not united behind. The sacral vertebræ are not joined together. There are no laminæ for the lateral surfaces, and the anterior parts of the uppermost sacral vertebræ are not united to the posterior. There are no coccygeal centres. There are no centres for the crests of

the ilia, and the three parts of the ossa innominata are widely separated by cartilage, while there is a very distinct interval between the ramus of each pubis and ischium. There are no centres for the tuberosities of the ischia, for the symphysis pubis, or for the anterior inferior spines. The centres for the heads of the femora are small, and there is none for the trochanters. There is also none for the patellæ. There is a small area of bone in the cartilage cap over the lower end of each femur, but it seems to be present in the outer condyle only. Both the upper and lower ends of the tibiæ also contain centres, which have apparently grown to about a third of their proper size. The ends of the fibulæ have no ossification centres. These bones are bent in towards the tibiæ so as almost to touch them at their lower ends. Each os calcis and astragalus is of fair size, but of the four other internal bones the external cuneiform alone contains a centre. Traces of centres are present in the heads of the metatarsals.

The sternum has four distinct centres, though the lower of these seems to be partially joined. The uppermost is much the largest. There is no trace of a fifth centre for the gladiolus. The scapulæ have no centres for the acromion processes; that for each coracoid is present, but not united. Small centres are present in the heads and tuberosities of the humeri, and there is a small one also for each radial head. There is no epiphysis for the radius or ulna of either side. Of the carpal bones, that for the os magnum and cuneiform are alone present. There are no centres for the heads of the metacarpals. Ossification is almost equal on the two sides.

It will be seen that, judging from these centres which are present, the ossification is about equal to that of a child during its second year. The centres for the lower ends of the radii, of the fibulæ and of the gladiolus, which ordinarily appear in the second year, are not present, whereas those of the lower end of tibiæ which appear in the second year are well represented. There are also traces of the heads of metatarsal bones which do not

usually appear until the third year. Ossification is therefore somewhat irregular, but, on the whole, is not more advanced than that of most children between one and two years of age. The dentition is that of a child who has attained the age of two years. But on comparing the proportions of Caroline Crachami with those of a normal child, we find that the height of her skeleton (49 cm. = 19⅓ inches) is a little less than the measurement which Spiegelberg[1] gives of the height (51 cm. = 20$\frac{1}{16}$ inches) of an average mature fœtus; while the length of the head of Caroline Crachami is 12·6 cm. (5·4 inches), as compared with 11·75 cm. (4½ inches) of the normal newborn infant. The breadth of the skull in Caroline Crachami is 9·4 cm. (3¾ inches), as compared with 9·25 cm. (3$\frac{5}{8}$ inches) of the newborn child. In other words, if we add on a little to the measurements of Caroline Crachami in consideration for the loss in height which is produced by the method of mounting, and for the measurements having been taken from a skeleton, we may say that while Caroline Crachami was of the height of a newborn baby of average size, her skull was a little larger, especially in the longitudinal diameter.

Remarks.—Caroline Crachami is an example of one of two possible diseases. We must either regard her as what the French term a sub- or demi-microcephalic imbecile, or as an instance of ateleiosis occurring in fœtal life. There seems to be no question that she was an imbecile. Her inability to walk with confidence, her quickness of sight, attraction to bright objects, and pleasure in music and fine clothes are all points which would claim attention in an imbecile. The fact that they are recorded implies that they were the most conspicuous features of her mental condition, and that indications of a higher intelligence were absent. This view is corroborated by the facial appearance of the child, which is eminently characteristic of microcephalism. The nose is much too large in proportion to the face, and the mandible is too small, giving a ferretty or rat-like

[1] 'Text-book of Midwifery,' (N.S.S. translation), vol. ii, p. 121.

appearance to the countenance, such as is commonly seen among small-headed idiots.

On the other hand, the head is distinctly not microcephalic. Its size is, on the contrary, rather over than under that which is natural to infants whose development corresponds with that of Caroline Crachami. Moreover, observations which have been made on the bones of idiots show that there is no delay of development in their case.[1]

There is more to be said in favour of the case being one of ateleiosis. Not only is there marked delay of development of the skeleton and a comparatively large skull, but we have the very important fact that the internal sexual organs were still more backward in their development than were the bones. This, we shall presently see, is a feature of most cases of ateleiosis. There is, therefore, good reason to believe that Caroline Crachami's condition was not due to primary hypoplasia of the brain, but was the result of a more wide-spread developmental error. In other words, the hypoplasia of the brain was only part of a general hypoplasia. At the same time we must recognise that it was probably this defective development of the brain which gave the case its peculiar features. Though the virtual arrest of development of the brain of an infant may not interfere with the intellectual faculties beyond keeping them more or less childish; yet, if the arrest takes place at an earlier age, there must be a period at which development cannot be virtually stopped without imbecility resulting. In that case, what is more likely than that the condition will present the facial as well as the mental characters of primary microcephaly? It should be noted that according to Boyd's tables[2] the weight of the brain at the age of nine years (1154 grammes = 40½ oz.) is not far from its highest (1244 grammes = 43 oz.), whereas in the case of Caroline Crachami, we must regard the brain as but slightly heavier than that of a newborn child (283 grammes = 10 oz.).

[1] Dr. Placzch, 'Zeitschrift f. Ethnol.,' 1901, p. 335.

[2] 'Quain's Anatomy,' vol. iii, part 1, p. 178.

Doubtful Cases.

There is a very pronounced form of infantilism which cannot be accounted for by any of the recognised causes of delayed development. The stature is so diminutive, and the proportions so good, that these dwarfs are in great demand for the purposes of exhibition in shows. It is probable that they are examples of ateleiosis beginning in fœtal life.

Among the best known of these was Frank Flynn, or "General Mite," who was examined by Virchow,[1] who found that he was 80·7 cm. (31⅜ inches) in height, while the head was 41·5 cm. (16⅓ inches) in circumference. His head, therefore, as Virchow pointed out, was a little too large for his body, though he was in other respects of excellent proportions. Professors Ranke and von Voit,[2] who examined him one year later, found that he then weighed 6570 grammes (14 lbs. 7½ oz.), and was 824 mm. (32½ in.) in height. He was of quick apprehension and good memory, while his intelligence, in spite of the extreme smallness of his head, was excellent. Ranke considered him to have the proportions of an adult, though the head and feet were a little too large, and his arms a little too short.

Nothing is said of the sexual organs, except that they were undeveloped.

Another example of the same condition was also examined by Ranke and von Voit.[3] This was that of Millie Edwards, who was of the age of 12 years. She was described as being of a very quick and lively disposition, and of excellent intelligence and memory. She was 72 cm. (2¼ feet) high, and weighed 6601 grms (27 lbs.), but no other measurements were given, though it was stated that her proportions were correct.

A still more striking case was that of Pauline

[1] 'Zeitschrift f. Ethnologie,' 1883–4, Bd. xv.

[2] 'Arch. f. Anthrop. Braunschweig,' 1885–6, Bd. xvi, s. 228.

[3] Loc. cit.

Muster, a Belgian dwarf, who was no more than 30 cm. (11¾ inches) in length at birth. She was examined by Virchow[1] at the age of 3 years, when she weighed 3·63 kilos. (8 lbs.), was 538 mm. (21⅕ inches) high, and measured 363 mm. (14⅓ inches) round the head. At the age of five she was examined by Dr. A. A. Bouchard,[2] who found that she was then 550 mm. (21⅞ inches high, and that the circumference of her head had increased to 390 mm. (15⅜ inches). She had twenty-two teeth, but no particulars are given of the state of dentition. Virchow said of her, that she talked with quickness and volubility, and was exceedingly bright and lively. She was described by others as being in perpetual movement and of good intelligence.

No examination of the bones was made in any of these cases; we cannot therefore say definitely to what class of dwarfism they belong, though they are evidently examples of the same morbid condition, and that condition is one of infantilism. It will be noticed that the intelligence was good in all three, though the heads were very small. The size of the head and length of the body of Pauline Muster were very near to those of Caroline Crachami, but it should be observed that there was in reality a difference between them, for Caroline Crachami, at the reputed age of 9 years, was of about the same size as Pauline Muster at three. Their photographs show them to be of the same facial type, and in some respects they resembled microcephalic dwarfs, for different observers seem to have been much struck with their quick movements and extreme restlessness.

Group II.—*Ateleiosis beginning during Infancy or early Childhood.*

Case 2.—According to his birth certificate, T. L. Spooner was born in Layer Breton, Essex, on February 23rd, 1874, and is therefore now twenty-eight years of age. The

[1] 'Zeitschrift f. Ethnologie,' Bd. iv, s. 215.

[2] 'Journ. d. Méd. de Bordeaux,' 1884-5, vol. xiv, pp. 276—279.

Gilford: Ateleiosis. Plate II.

Ateleiosis. Group ii. Case 2. T. L. S., age 28 years, height 3ft. 7in. (1.096m.) standing between a normal adult man and a normal boy of 6 years. The physiognomy and proportions are childish and the sexual organs infantile, while the attitude, expression and the markings of the face are suggestive of age.

Bale & Danielsson, Ltd., London.

photographs of his father and mother and of some of his brothers and sisters show that they are all of ordinary size and development. Of five brothers and two sisters one only is dead (from pneumonia). He was of average size when born, and cut his teeth at the usual age. He had measles in childhood, and during the last three years has had influenza twice. Nothing is known of the closure of the fontanelles. It was first noticed that he was not growing when he was between one and two years old, though there was no illness nor any other circumstance to account for it. He went to school at ten, and left at sixteen, after passing the sixth standard. I first saw him when he was just over twenty-three years of age. He was then 1·078 m. (3 feet 6 inches) in height. One year afterwards, when I measured him again, he was 1·086 m. (3 feet 6¾ inches) high, and three years later he had still further increased to 1·096 m. (3 feet 7⅛ inches). On February 21st of this year, or one year after he was last measured, he was still of the same height. Other measurements on the last occasion were as follows, viz.:

Head, 49·5 cm. = 19½ inches in circumference; 17 cm. = 6⅔ inches in greatest length; 13·9 cm. = 5½ inches in greatest breadth.

Round chest in nipple line, 62·6 cm. = 24⅝ inches inspiration; 61 cm. = 24 inches expiration.

Round abdomen, 56 cm. = 22⅛ inches; middle point of total length, 1 cm. above the pubes.

Upper extremity.—Arm from acromion to elbow, 20·75 cm. = 8⅛ inches; forearm, external condyle to styloid process, 17·3 cm. = 6¾ inches; length of hand, 11·75 cm. = 4$\frac{7}{12}$ inches.

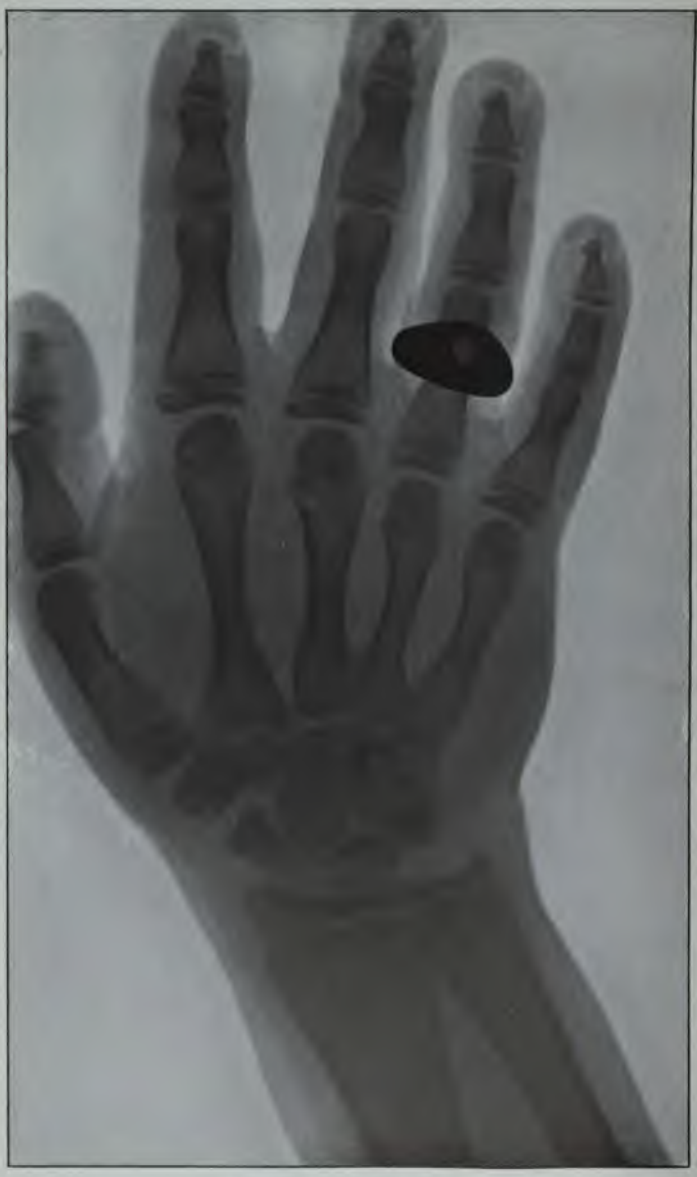
Lower extremity.—Great trochanter to external condyle, 28·5 cm. = 12 inches; lower end of femur to internal malleolus, 24 cm. = 9 $\frac{7}{10}$ inches; length of foot, 17 cm. = 6⅝ inches.

Weight, 25 kilos. = 4 st. 1 lb.

These measurements vary very little from those which I made four years before.

the peripheral end from its own central end. It was possible that the sympathetic system recovered more easily than the ordinary peripheral nerves; but even in them, although there was return of histological structure, there was no return of function. This was possibly due to the greater vulnerability of the terminations of the sympathetic nerve as compared with the fibres themselves. The variation in recovery would probably differ in different nerves and in different animals. As to the manner in which the actual regeneration occurred, the neuroblast view of the authors of the paper was not convincing; the observations made were on sections, and it was not possible to follow a nerve-fibre by this method; the method of teasing was necessary. The Golgi stain was erratic, and osmic acid stains were certainly preferable both to it and the Weigert stain. His observations had rather revealed the idea of a long multinucleated cell than a series of short cells as the authors had maintained. The difficulty of admitting the phagocytic action of the connective-tissue cells for the medulla was obvious in view of the fact that they were separated by a membrane—the neurilemma. The earlier disappearance of the medullary substance of the large fibres was probably due to the staining agent. In his experience this change was earlier in the small than in the larger fibres. The rapid return of sensation (from an hour to a day) in cases of secondary suture was not cleared up by the paper. If recovery of function did not correspond with regeneration of structure how could it be explained? The nerve could not be cut without the stump of the central end degenerating, and this in itself negatived the rapid recovery on the basis suggested in the paper, as seven to ten days at least were required for their recovery. He would suggest as a theory that the division of one or more nerve-fibrils might happen just between two nerve segments, and thus the two in contact end to end might escape injury, and being in anatomical continuity might transmit sensory impulses. In the surgical operations for the repair of a divided facial nerve it was usual partially to divide the spinal accessory nerve and to graft the facial on the central end of the spinal accessory. He would suggest that it might be better to cut the spinal accessory nerve right across, to split it, and to unite one portion thus divided to the facial and the other to the peripheral trunk of the spinal accessory.

Dr. F. W. Mott congratulated the authors of the paper on affording still further proof that regeneration of a divided nerve took place from the periphery. For some time past he had been engaged with Professor Halliburton in making a series of observations upon the chemical changes occurring in nerves undergoing degeneration and regeneration after their division. The inquiry was not conducted for the purpose of ascertaining



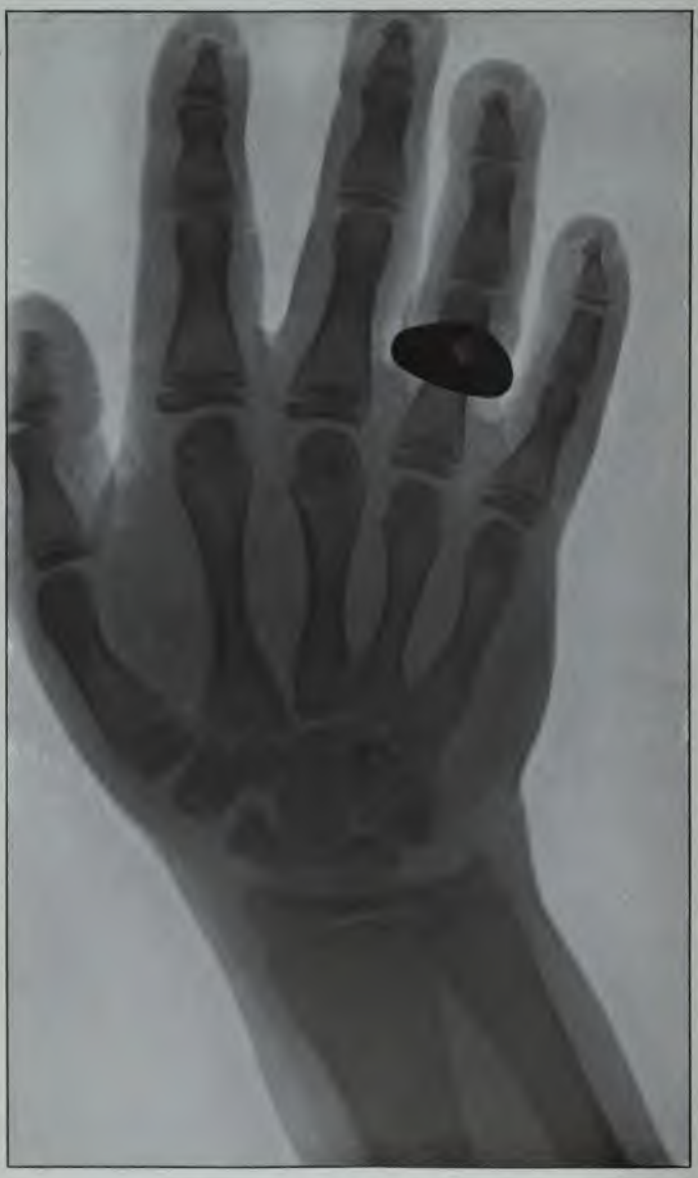
Ateleiosis. Group ii. Case 2. Radiogram of hand of Plate II., age 28. The ossification resembles that which is usual at from 10 to 12 years. Note the size of the hand.

Bale & Danielsson, Ltd., London.

immaturity, there are marks of age upon his face, and a certain air of assurance in his manner which prevent his being mistaken for a child. His mental development and tastes are somewhat childish, for though he does not care to associate with children, he is inclined to indulge in childish games. He is fond of reading, and is capable of steady application to his daily work. He earns 3*s*. a week as an under-gardener. His muscles seem to be of the size and strength of those of a child of his height. The pulse averages about one hundred beats to the minute. The heart is of normal size, but a faint soft murmur can be heard after the second aortic sound with an interval between them. The arteries are in good condition, and there is no arcus senilis. The organs of special sense appear to be normal. He sweats freely when he exerts himself to any unusual extent.

Genital and urinary organs.—The breasts look prominent, but no gland tissue can be felt. The external organs of generation are in size and appearance like those of a child of about three years of age. The testicles are undescended, though each forms a projection and can be felt in its canal. They are of infantile development, but testicular sensation is well marked. From what I can gather there has been none of the emotional evidences of puberty. The breasts are not developed. The urine is normal in quality, but I cannot speak as to its quantity.

Osseous system.—The fontanelles are closed. There appears to be no irregularity of ossification anywhere. All the bones are slightly formed. A radiogram of one hand taken four years ago showed that ossification corresponded with that of a boy of about the age of ten years, though the bones themselves are smaller than usual, and the muscular prominences are not easily detected. A radiogram which I had taken a year ago shows that ossification had made but little advance during the previous three years. The bones are, as a rule, perhaps a little thicker or a little longer, but there appears to be no more progress in the fusion of the epiphyses. The hand is apparently just



Ateleiosis. Group ii. Case 2. Radiogram of hand of Plate II., age 28. The ossification resembles that which is usual at from 10 to 12 years. Note the size of the hand.

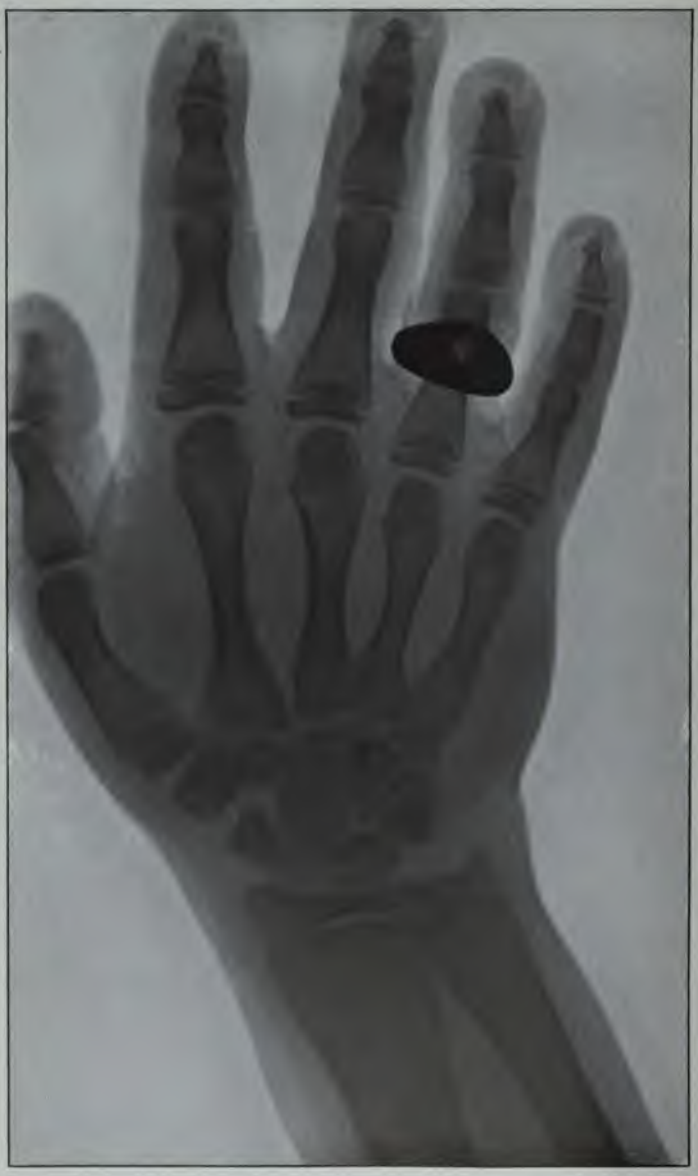
Bale & Danielsson, Ltd., London.

immaturity, there are marks of age upon his face, and a certain air of assurance in his manner which prevent his being mistaken for a child. His mental development and tastes are somewhat childish, for though he does not care to associate with children, he is inclined to indulge in childish games. He is fond of reading, and is capable of steady application to his daily work. He earns 3*s*. a week as an under-gardener. His muscles seem to be of the size and strength of those of a child of his height. The pulse averages about one hundred beats to the minute. The heart is of normal size, but a faint soft murmur can be heard after the second aortic sound with an interval between them. The arteries are in good condition, and there is no arcus senilis. The organs of special sense appear to be normal. He sweats freely when he exerts himself to any unusual extent.

Genital and urinary organs.—The breasts look prominent, but no gland tissue can be felt. The external organs of generation are in size and appearance like those of a child of about three years of age. The testicles are undescended, though each forms a projection and can be felt in its canal. They are of infantile development, but testicular sensation is well marked. From what I can gather there has been none of the emotional evidences of puberty. The breasts are not developed. The urine is normal in quality, but I cannot speak as to its quantity.

Osseous system.—The fontanelles are closed. There appears to be no irregularity of ossification anywhere. All the bones are slightly formed. A radiogram of one hand taken four years ago showed that ossification corresponded with that of a boy of about the age of ten years, though the bones themselves are smaller than usual, and the muscular prominences are not easily detected. A radiogram which I had taken a year ago shows that ossification had made but little advance during the previous three years. The bones are, as a rule, perhaps a little thicker or a little longer, but there appears to be no more progress in the fusion of the epiphyses. The hand is apparently just

Gilford: Ateleiosis. Plate III.



Ateleiosis. Group ii. Case 2. Radiogram of hand of Plate II., age 28. The ossification resembles that which is usual at from 10 to 12 years. Note the size of the hand.

Bale & Danielsson, Ltd., London.

immaturity, there are marks of age upon his face, and a certain air of assurance in his manner which prevent his being mistaken for a child. His mental development and tastes are somewhat childish, for though he does not care to associate with children, he is inclined to indulge in childish games. He is fond of reading, and is capable of steady application to his daily work. He earns 3*s*. a week as an under-gardener. His muscles seem to be of the size and strength of those of a child of his height. The pulse averages about one hundred beats to the minute. The heart is of normal size, but a faint soft murmur can be heard after the second aortic sound with an interval between them. The arteries are in good condition, and there is no arcus senilis. The organs of special sense appear to be normal. He sweats freely when he exerts himself to any unusual extent.

Genital and urinary organs.—The breasts look prominent, but no gland tissue can be felt. The external organs of generation are in size and appearance like those of a child of about three years of age. The testicles are undescended, though each forms a projection and can be felt in its canal. They are of infantile development, but testicular sensation is well marked. From what I can gather there has been none of the emotional evidences of puberty. The breasts are not developed. The urine is normal in quality, but I cannot speak as to its quantity.

Osseous system.—The fontanelles are closed. There appears to be no irregularity of ossification anywhere. All the bones are slightly formed. A radiogram of one hand taken four years ago showed that ossification corresponded with that of a boy of about the age of ten years, though the bones themselves are smaller than usual, and the muscular prominences are not easily detected. A radiogram which I had taken a year ago shows that ossification had made but little advance during the previous three years. The bones are, as a rule, perhaps a little thicker or a little longer, but there appears to be no more progress in the fusion of the epiphyses. The hand is apparently just

8 mm. ($\frac{1}{3}$ in.) longer. The long bones of the arm and hand are perhaps more lightly and delicately formed than are those of most children of ten years.

Case 3.—The following is a female case in which I was able to make a complete examination of the whole body, and to obtain a radiogram of the forearm and hand. This dwarf was born in Paris eighteen years ago. Her mother, who is of ordinary stature, sought my advice for obesity. She told me that there were two other children beside this dwarf, but that they and the father are of ordinary stature, and she does not know that any other member of the family has ever had any abnormality of growth. This dwarf child was small at first, but grew at an ordinary rate until she was a little over two years of age. After this period she still continued to grow at a fairly uniform, but much diminished, rate. She has had attacks of bronchitis in childhood, but with that exception has been healthy. She presents no sign of approaching puberty, but, on the contrary, in almost every respect resembles a child. She differs in that her intelligence is certainly more mature than is usual in a child of her height. She is of a lively disposition, but is not restless. She sweats on taking any unusual exertion. Her hair is fine, eyes full, nose depressed at the bridge, lips thin, and chin small. The appearance of her face is spoiled when she smiles by the projection of her teeth, which are also very irregular. Teething began, it is said, at the eighth month, but nothing is known of the time of onset of the second dentition. The teeth form two irregular rows and are much crowded and displaced. Some belong to the permanent, and some to the temporary set. In the upper jaw are four permanent incisors, of which the two lateral are only just through the gums, while two of the temporary set still remain. There are also two temporary canines, two premolars, and four permanent and decayed molars, while one decayed temporary molar has been crowded out. In the lower jaw are only

Ateleiosis. Group ii. Case 3. Age 18 years, height 2ft. 9½in. (·850m.). A normal adult hand is introduced for the sake of comparison. Note the characteristic physiognomy, and the crowded teeth. A radiogram showed that ossification was equal to that which is usual at six years.

Bale & Danielsson, Ltd., London.

two small and imperfect incisors, two temporary canines, four crowded premolars, and two molars, one of which is decayed to the roots. The appetite is said to be excellent, she has six meals a day, and altogether her food is thought to be equal in quantity to that taken by an average child of the age of from twelve to fourteen years. The mother states that the anterior fontanelle did not close until she was seven or eight years of age. Measurements of her head and body are as follows, viz. :

Total height, 850 mm. = 33½ inches.

Head circumference, 445 mm. = 17½ inches.

Round the chest in the nipple-line between inspiration and expiration, 475 mm. = 18¾ inches.

Round abdomen at umbilicus, 460 mm. = 18⅛ inches.

Arm, 153 mm. = 6 inches.

Forearm, 140 mm. = 5½ inches.

Hand to extremity of middle finger, 107·5 mm. = 44¼ inches.

Thigh, 230 mm. = 9 inches.

Leg, 250 mm. = 9⅞ inches.

Foot, 125 mm. = 5 inches.

A radiogram of the hands shows that the ossification is a little more advanced than that of a child of six years, as shown in Mr. Poland's atlas. Ossification is more forward in the right hand than in the left. Her manner is childish; she has not the slightest objection to being stripped of her clothes and examined. She possesses no hair on her body, and her breasts and sexual organs are undeveloped. The pelvis is that of a child, and there has never been any sign of the onset of menstruation. A faint systolic murmur can be heard over the upper part of the sternum. The heart appears to be normal in other respects.

She possesses little muscular strength, but is certainly able to endure far more fatigue than a child of her height. Her mother declares that she can walk 3 kilometres (2 miles), and has once walked 6 kilometres (3¾ miles) without undue fatigue. She gains her living on the stage and is an expert dancer.

two small and imperfect incisors, two temporary canines, four crowded premolars, and two molars, one of which is decayed to the roots. The appetite is said to be excellent, she has six meals a day, and altogether her food is thought to be equal in quantity to that taken by an average child of the age of from twelve to fourteen years. The mother states that the anterior fontanelle did not close until she was seven or eight years of age. Measurements of her head and body are as follows, viz. :

Total height, 850 mm. = 33½ inches.

Head circumference, 445 mm. = 17½ inches.

Round the chest in the nipple-line between inspiration and expiration, 475 mm. = 18¾ inches.

Round abdomen at umbilicus, 460 mm. = 18⅛ inches.

Arm, 153 mm. = 6 inches.

Forearm, 140 mm. = 5½ inches.

Hand to extremity of middle finger, 107·5 mm. = 44¼ inches.

Thigh, 230 mm. = 9 inches.

Leg, 250 mm. = 9⅞ inches.

Foot, 125 mm. = 5 inches.

A radiogram of the hands shows that the ossification is a little more advanced than that of a child of six years, as shown in Mr. Poland's atlas. Ossification is more forward in the right hand than in the left. Her manner is childish; she has not the slightest objection to being stripped of her clothes and examined. She possesses no hair on her body, and her breasts and sexual organs are undeveloped. The pelvis is that of a child, and there has never been any sign of the onset of menstruation. A faint systolic murmur can be heard over the upper part of the sternum. The heart appears to be normal in other respects.

She possesses little muscular strength, but is certainly able to endure far more fatigue than a child of her height. Her mother declares that she can walk 3 kilometres (2 miles), and has once walked 6 kilometres (3¾ miles) without undue fatigue. She gains her living on the stage and is an expert dancer.

Case 4.—There is a skeleton in the Barclay collection of the Royal College of Surgeons of Edinburgh which shows the changes which characterise the bones of these ateleiotic dwarfs, and I have to thank the Museum Committee for permission to describe and photograph this specimen. I could find no description beyond that which is given on the label. This sets forth that it is "the skeleton of the dwarf Bobbie Fenwick, who died in 1815, upwards of fifty. The bones of the ossa innominata remain disunited, as do all the epiphyses, except the right upper end of ulnar. Lines of junction can be seen in most vertebræ, ribs, sacrum, fingers, and toes. Several of the second set of teeth have never protruded."

The measurements of the skeleton are as follows, viz.:

Height, 3 feet 10 inches = 1188 mm.

Length of spine, 35 inches = 890 mm.

Skull: greatest length, $7\frac{1}{4}$ inches = 185 mm.; greatest width, $5\frac{3}{4}$ inches = 145 mm.; circumference, $19\frac{7}{8}$ inches = 505 mm.

Biacromial, $11\frac{1}{5}$ inches = 285 mm.

Humerus, $13\frac{3}{4}$ inches = 350 mm.

Radius, $9\frac{7}{8}$ inches = 250 mm.

Hand, $5\frac{1}{8}$ inches = 130 mm.

Femur, $18\frac{1}{2}$ inches = 470 mm.

Tibia, 13 inches = 330 mm.

Foot, 6 inches = 153 mm.

Pelvis, antero-posterior, $2\frac{1}{2}$ inches = 65 mm.; transverse, $3\frac{3}{8}$ inches = 85 mm.

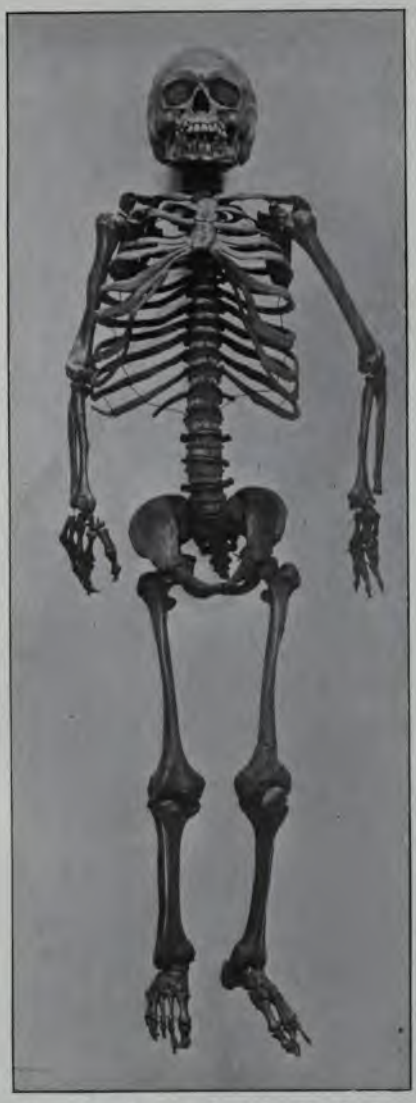
Between anterior superior spines, $6\frac{1}{3}$ inches = 160 mm.

Crest to crest, $6\frac{3}{4}$ inches = 173 mm.

The bones are small and slightly formed, and the muscular prominences are not well marked, though they can be readily recognised. The proportions are, apparently, faulty, but allowance must be made for the very liberal supply of inter-vertebral substance[1] which has been put in

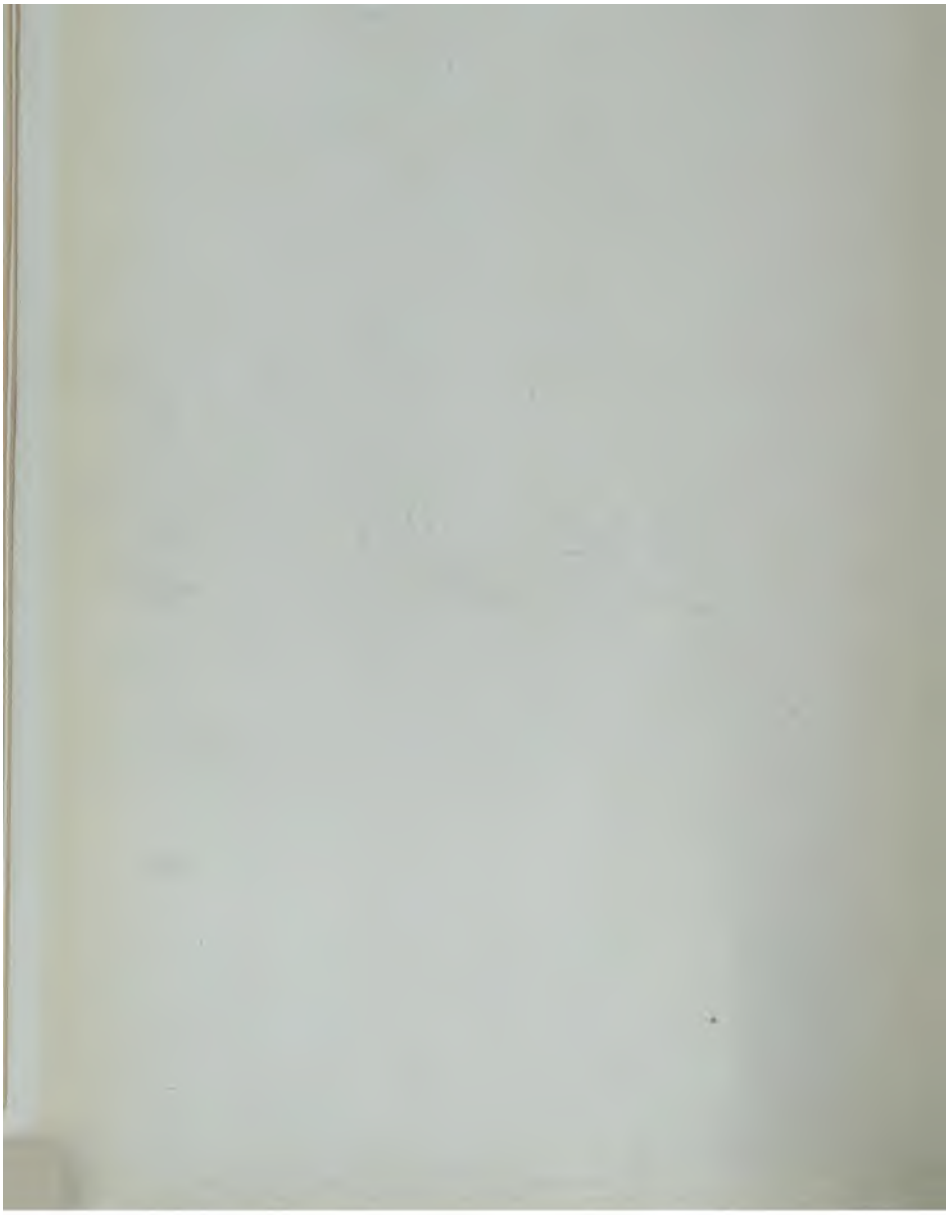
[1] According to Mr. Morris ('Anatomy of the Joints,' p. 69) the inter-vertebral substance does not normally exceed one quarter of the total length of the spine, but in this skeleton it must be nearly half that length.

Gilford: Ateleiosis. Plate V.



Ateleiosis. Group ii. Case 4. Age 50. Height 3ft. 10ins., 1.188m. Skeleton of Bobbie Fenwick, from the Royal College of Surgeons' Museum, of Edinburgh. The bones are delicately formed, the epiphyses about the knee joint are enlarged and many of the epiphyses are not united. The spine is too long owing to the insertion of excess of intervertebral cement.

Bale & Danielsson, Ltd., London.



by the articulator. In some parts, indeed, its depth equals or even exceeds that of the vertebræ between which it is placed. This accounts for much of the disproportionate shortness of the limbs, and also for the fact that the middle point of total length is at the sacral eminence, whereas it was probably during life a little above the pubes. Something should also, for the same reason, be discounted from the height, which probably did not amount to more than 1·100 mm. during life.

The skull, it will be noticed, is a little below the average size for an adult. It is, apparently, of normal thickness, and shows no irregularities of ossification. The sella turcica is neither too small nor too large. The basi-sphenoid and basi-occipital are not yet united. The angle of the lower jaw is somewhat open, and the body is shallow, behind the canine teeth. The palate is flat, and the dentition is very irregular.

Its formula is—

$$\begin{matrix}\text{Upper } \frac{4}{3}\,\text{i.}\ \frac{2}{2}\,\text{c.}\ \frac{4}{4}\,\text{pr.}\ \frac{6}{4}\,\text{m.}\\ \text{Lower}\end{matrix}$$

In the upper jaws the right canine has evidently erupted at a much later date than its neighbours, and has been crowded out by them. One of the premolars, though visible, has not descended. Of the three right molars, two have their crowns flush with the alveolus, as if they also had not erupted, and the same is the case with the last of the corresponding teeth on the left side. Another is decayed to the roots, and the third has been broken off. In the lower jaw the left lateral incisor has not yet been "cut," and the same is the case with the two canines. Two of the premolars are very little above the level of the alveolus. There is a cavity left by an absent first right molar, and the second has just erupted. All the teeth are of average size, but are very irregularly placed, some projecting unduly, while others are below their proper level. Another noticeable peculiarity in the lower jaw is the presence of two fissures, one running downwards just

outside the right central incisor, and the other from the root of the right canine, so as, apparently, to separate the bone between them from the rest of the jaw. The spinal and sacral epiphyses are all united. The sternum is short, and the upper pieces are still separate, while the third and fourth show an evident groove in their line of fusion. The coracoid and acromial processes are still separate. The rib epiphyses are all united. Ossification is, as a rule, more advanced on one side than on the other; thus the line of union of the right upper epiphysis of the humerus can be seen only on close examination, while, on the left side, it is evident at once. The stage of ossification is not symmetrical. This is especially true of the radii, for the head is still separate on the right side, while it has, apparently, been recently united on the left. The line of junction at the lower ends of each humerus can be seen only on near inspection. The lower ends of the radii are still separate, and so apparently are those of the ulnæ. The pelvis is flat and shallow, and the ossa innominata are still divided into their three constituent bones by well-marked cartilaginous divisions. The crest of the ilium is rough, and the edges are sharp, showing that its epiphysis had not united and has been detached. The cotyloid cavity is shallow and its lip irregular. The natural curve of the femur is not exaggerated. The head of the bone is flattened and roughened at its upper part, as if the cartilage there had been unusually thick. It is either ununited, or is quite recently united, to the neck on the right side, but it is difficult to see the line of junction on the left. There is no adventitious deposit about this or any other joint. The neck is unusually short and runs into the shaft at an angle of about 120 degrees. The lesser trochanter is very prominent. A noteworthy point about the femur is the relative enlargement of each lower epiphysis, which is rather conspicuous. The lower epiphysis on the left side is detached, but on the right it is joined, and the epiphysial line can be seen with difficulty. In no case does there appear to be any cartilage in the epiphysial

lines, and it is possible that the epiphyses are not always separate when they appear to be so.

Case 5.—Another example of the same disease has been described by Professor Schaaffhausen[1].

This was a male dwarf who died at the age of 61. His height was 94 cm. ($3\frac{1}{2}$ feet); weight 45 Pfund (2250 grms.). His parents and one sister were tall, and two brothers were about 5 feet (152·5 cm.) high. There were three other dwarfs in the family, two of whom were alive, one being of his own height, and the other 5 inches (12·7 cm.) taller. One brother, who was dead, had presented the same characters as himself in regard to voice and beard. "He had an old head on a childish body." The head was of circumference of 520 mm. ($20\frac{1}{2}$ inches), or about that of a boy of five years of age. "It seemed as if the growth of the head had remained childish, whilst at the same time most of the characters of the body were also childish." His intelligence was good, but not exceptional. The head was 170 mm. ($6\frac{3}{4}$ inches) long, as in the first year of life, whilst its greatest breadth was 150 mm. ($5\frac{7}{8}$ inches). The internal surface of the skull showed the impression of many deep convolutions. He had a squeaky voice, he was neither bald nor grey, though hair was absent from all parts except the head. Although he looked old, his face retained the characters of childhood, as was shown by the bulging forehead, the undeveloped nose, thick upper lip, and weak chin. The skull had a very childish form, which was especially shown in the lack of prominence of the parietal eminences, in the open condition of all the sutures, and in the serration of the bones, which resembled that of a child in the first year of life. The internal organs were not larger than those of a child of six years. He appeared not to be virile, and there was cryptorchism on both sides. In other respects he showed marks of age. He had lost most of his front teeth, and

[1] 'Verhand. d. Naturhist. Verein. d. preuss. Rhein. u. Westphal.,' 38 Jahr. Erstes Heft., Bonn, 1868, S. 26.

appearance to the countenance, such as is commonly seen among small-headed idiots.

On the other hand, the head is distinctly not microcephalic. Its size is, on the contrary, rather over than under that which is natural to infants whose development corresponds with that of Caroline Crachami. Moreover, observations which have been made on the bones of idiots show that there is no delay of development in their case.[1]

There is more to be said in favour of the case being one of ateleiosis. Not only is there marked delay of development of the skeleton and a comparatively large skull, but we have the very important fact that the internal sexual organs were still more backward in their development than were the bones. This, we shall presently see, is a feature of most cases of ateleiosis. There is, therefore, good reason to believe that Caroline Crachami's condition was not due to primary hypoplasia of the brain, but was the result of a more wide-spread developmental error. In other words, the hypoplasia of the brain was only part of a general hypoplasia. At the same time we must recognise that it was probably this defective development of the brain which gave the case its peculiar features. Though the virtual arrest of development of the brain of an infant may not interfere with the intellectual faculties beyond keeping them more or less childish; yet, if the arrest takes place at an earlier age, there must be a period at which development cannot be virtually stopped without imbecility resulting. In that case, what is more likely than that the condition will present the facial as well as the mental characters of primary microcephaly? It should be noted that according to Boyd's tables[2] the weight of the brain at the age of nine years (1154 grammes = 40½ oz.) is not far from its highest (1244 grammes = 43 oz.), whereas in the case of Caroline Crachami, we must regard the brain as but slightly heavier than that of a newborn child (283 grammes = 10 oz.).

[1] Dr. Placzch, 'Zeitschrift f. Ethnol.,' 1901, p. 335.

[2] 'Quain's Anatomy,' vol. iii, part 1, p. 178.

described by His and Schauta. The former of these two was a cretin. Schauta's case may also have been one of cretinism; it will be referred to among the doubtful cases. Another case of operation cretinism by Grundler is then quoted, and some cases are referred to which occur in Otto's 'Lehrbuch' and are described by Naegele. The latter alludes to the Edinburgh skeleton (Case 2). Paltauf believes that the infantile pelvis of obstetricians is not a local phenomenon only, but is part of a wide-spread condition of infantilism, such as is found in ateleiosis and cretinism. He quotes details of the descriptions of some of these pelves, in each of which there was some noticeable delay in the process of ossification. Three were apparently of cretinous or myxœdematous persons; in another the bones were thick, light, and spongy. One belonged to a dwarf woman of the age of 31 years, who was of childish intellect, and may possibly have been a cretin. She gave birth to a child of 5 lbs. 6 oz. (2·4 kilos.) in weight.

Paltauf then describes the microscopical appearances of the ossification zones, and compares the disease with natural dwarfism, rickets, so-called fœtal rickets, congenital osteoporosis and cretinism, and with normal childish growth. He then alludes to some well-known dwarfs, such as "General Mite," Jeffrey Hudson, Boruwlaski, and "Admiral Piccolomini," and finally goes into the question of the influence of the genital organs on growth. Paltauf, in his paper, does not clearly distinguish between ateleiosis and cretinism and other causes of defective development, though he fully recognises that his case (Mikolajek) stands apart from these secondary forms of dwarfism.

Case 6.—Paltauf's own case of ateleiosis, was a male named Mikolajek, of the age of 49, who died from acute disseminated tuberculosis in Professor Kahler's clinik in the Vienna hospital. He was born in Galicia. His parents and brothers and sisters were of medium size. At one time he suffered from a rheumatic affection of the right knee-joint, but subsequently recovered. Later,

while occupied as a gardener, he developed the same complaint in the left knee. At this time, and again four years before his death, he became affected with general œdema, but was at each time well in a few weeks. Three weeks before his entrance into the hospital he again became dropsical, and at the same time suffered from breathlessness and cough. He had had no other diseases. The bones were small, though he was of comparatively great muscular development. The genital organs resembled those of a child. The prepuce was phimotic, and though the left testicle was in the scrotum, the right was still in the inguinal canal. The chief measurements were as follows:

Height, 112·5 cm. = 44½ inches.

Head: circumference, 54 cm. = 21¼ inches; mento-occipital, 22·5 cm. = 8⅞ inches; bi-parietal, 15 cm. = 5⅞ inches.

Chest at nipple line, 67 cm. = 26⅜ inches.

Abdomen midway between xiphoid process and symphysis, 79 cm. = 31⅛ inches.

Extremities from acromion to end of middle finger, 52 cm. = 20½ inches.

Olecranon to styloid process of ulna, 18·5 cm. = 7⅓ inches.

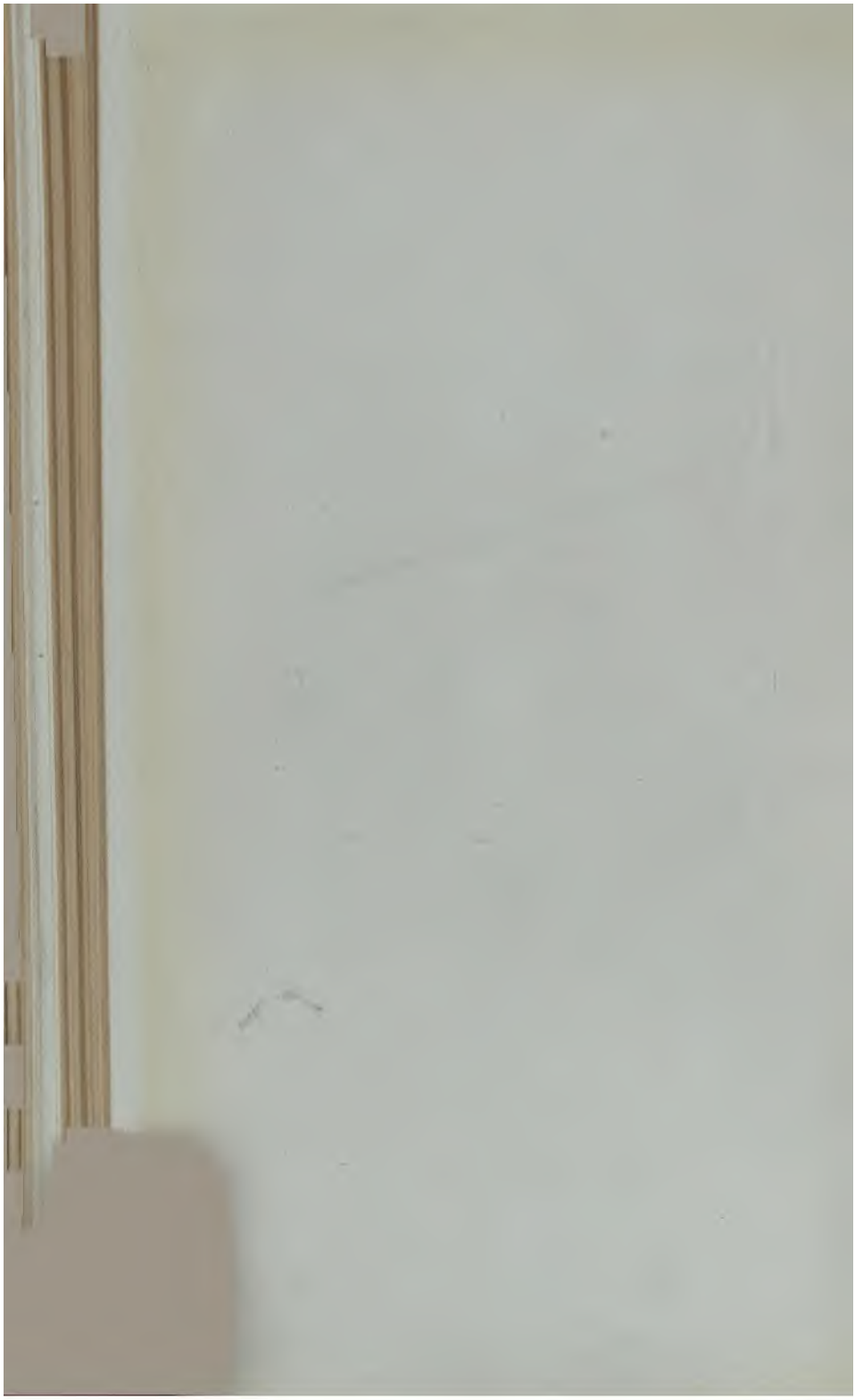
Great trochanter to external malleolus, 56 cm. = 22 inches.

Great trochanter to external condyle of femur, 26 cm. = 10¼ inches.

Internal condyle to internal malleolus, 28 cm. = 11 inches.

Nothing is said of his intelligence, but inasmuch as he was for twenty-one years valet to a colonel in the army, it is not likely that it was defective. The sella turcica was "peculiarly large and deep." It measured 17 mm. (⅔ inch) in length and 16 mm. (⅔ inch) in breadth, or 6 mm. and 3 mm. (⅙ and $\frac{1}{10}$ inch) respectively more than that which is usual in the adult. The thyroid gland was found at the *post-mortem* examination to be very small and pale

red in colour. Some of the glands of the left side of the neck were of the size of hazel-nuts, and were hard and yellowish and dry on section. The left lung was free and the right adherent. The left lung had three lobes. Both lungs contained disseminated tubercles. The heart was enlarged on the right side, but the valves were normal. The spleen was slightly enlarged, but the liver, kidneys, adrenal capsules, and stomach were normal. The teeth were well developed, not carious, and without sign of rickets or other disease. All were of the permanent set; and except that there were only eight fully cut molars, their number, size, and arrangement were as in the adult. The third upper molar on either side was only just appearing through the gums. The spinal column was 425 mm. ($16\frac{3}{4}$ inches) long, and showed slight scoliosis, with the upper convexity to the left. There was also lumbar lordosis. The ends of the spinous and transverse processes were still cartilaginous, and there were no signs of bone centres. The ribs showed no evidence of rickets. The sternum was in four pieces, and the ensiform process consisted of pure cartilage. There was slight bending of the clavicles, but it was not of the same character as that which is seen in rickets. The epiphyses of the sternal ends contained no traces of bone. They were 90 mm. long, that of a seven-year-old child being 94 mm. ($3\frac{3}{8}$ inches). The shoulder blades were like those of a child, and the epiphyses were cartilaginous and not joined. The condyles of the humerus were somewhat enlarged, especially the internal, though the whole of the lower end was bigger than usual. In the trochlea the beginning of bone formation could be seen. The top of the radius and the end of the olecranon were not united to their respective diaphyses. The carpal bones were of the usual number, but were smaller than in the normal adult, and their shape was sharply defined. The epiphyses of the long bones of the hands were either quite free, or were united by cartilage or by slight bony union only. The three bones of the pelvic girdle were separate; the sutures between the pubes and ischia were



shaped, or irregular, and hardly looking like cartilage-cells. Their groups lay free in the ground substance. The next stratum was that of the calcified cartilage of the diaphysis. This, too, was very abruptly defined, of smooth outline towards the cartilage, but very irregularly toothed towards the shaft of the bone. The cells were arranged in more or less regular vertical lines as in normal ossification. These lines were continued into the cartilage zone, where they soon spread out, and their cells became more scattered. Finally, in the cancellous bone of the shaft, thin, delicate septa divided off large spaces filled with secondary marrow. The cartilage was not only present on the ends of the bone, but followed the diaphysis for a short distance under the periosteum.

Paltauf insists that the calcification of cartilage in the two strata above mentioned must not be confounded with true ossification. It is rather to be compared with the deposit of mineral salts in the cartilage of the aged, such as occurs in the rib and laryngeal cartilages of old people. Next to these, in the child, is a layer of developing bone, but in the dwarf, though there is a formation of bone, it is thin, fibrous, and broken.

Case 7.—Dr. Manouvrier[1] has also given an account of a case of this disease. His patient, Auguste Tuaillon, was normal at birth, and walked at the age of 13 months. He fell downstairs at the age of 3 years, but impairment of growth was not noticed until he was 4½ years old. At this same age he had a very hard, prominent abdomen, and was always very constipated.

Dr. Manouvrier believes that the anomaly of growth really took place earlier, and was due to some injury of the brain produced by the fall. He points out that growth was delayed, and not brought to a sudden stop, for there was satisfactory evidence that growth to the extent of 4 cm. had taken place between the ages of 17 and 21. At the age of 20 he was ·95 m. (3⅛ feet)

[1] 'Bull. Soc. Anthrop.,' 4th series, tome vii, 1896, April 2nd, pp. 264—290.

high, and 17 kilos. (37½ lbs.) in weight. At the age of 23, when the account was written, he still weighed only 17 kilos. (37½ lbs.), but had increased in height to ·99 m. (3¼ feet). Nothing is said of the state of his ossification. The wisdom teeth had not been cut, and some of the milk teeth were still persisting. There was no appearance of rickets. His muscular strength was about equal to that of a child of six. His appearance, proportions, and gait were childish, and he was often mistaken for a child, but his manner and conversation were not childish. He appeared to be of average intelligence. The condition of the genital organs was infantile. At the age of 14 or 15, according to his own account, he showed some of the psychical phenomena of puberty, but there appears to have been no real evidence on this point. The skull measured :—Greatest length, 178 mm. = 6⅝ inches; transverse diameter, 148 mm. = 5⅓ inches; vertical, 127 mm. = 5 inches; horizontal circumference, 530 mm. = 20⅞ inches.

Cases 8 to 11.—A short account of some instances of dwarfism are given by Dr. Joachimsthal,[1] of Berlin, four of which appear to be ateleiotic. He examined a troupe of German dwarfs, and selected some of them for detailed description. He also refers to other cases, and compares the disease with achondroplasia and cretinism. Of his six cases of dwarfism one commenced at the third year, and will therefore come under my second group In his second case, the abnormality was first noticed in the seventh year, in the third at the tenth year, in the fourth at the eighth year, but he says nothing of the time of commencement in the last two. The ages varied from 30 to 36 years, and half of them were male and half female. Measurements of height appear to have been made by the director of the troupe as each dwarf came under his care. These are compared by Dr. Joachimsthal with those which he himself made when he first saw them. In one of these the

[1] 'Deutsch. med. Woch.,' No. 17, 1899, s. 269.

first measurement was made in the fifteenth and another in the seventeenth years. It is therefore possible that the respective increase of 10 cm. (3¾ inches) in fifteen years, and 29 cm. (11½ inches) in fourteen years, was in part due to natural growth at the time of puberty. But the same can hardly be said of two other cases in which the first measurement was taken in the twenty-first and twenty-second year, respectively. In the first of these two, the rate of growth was 25 cm. (9¾ in.) in thirteen years, and in the other 22 cm. (8¾ in.) in fourteen years. It cannot be said whether growth in these cases was continuous or regular, though Joachimsthal mentions that in his second case, where the age was 36, there had been a noticeable increase in height during the last three years. If we compare the heights of these dwarfs with the heights of normal children, we find that, according to Quetelet's tables, the first case grew from the height of a child of 3¾ years, until within sixteen years he had reached the height of a child of 6 years. His second case grew in fourteen years from the height of a child of 6 to that of one of 10 years. His third in thirteen years from that of a child 8½, to that of one of 11 years; and his fourth from that of a child of 6 to that of one of 11 years.

Radiograms are given of the hands of four cases, and the first also includes the greater part of the upper extremity. It is noteworthy that the radiograms of the first case show that, while the height was equal to that of a child of 6 years, the ossification was equal to that of a child between the eleventh and twelfth years. Similar delay in ossification is shown in the next three cases. But in the fifth and sixth there was complete epiphysial growth. These were two females of the age of 26 years, who had not been measured during the last ten years. They differ from the other female, who was of the age of 30, not only in ossification, but also in regard to menstruation. In both of them menstruation had been regular since their twentieth year. It is unfortunate that no photographs are given, other than radiograms, and very

high, and 17 kilos. (37½ lbs.) in weight. At the age of 23, when the account was written, he still weighed only 17 kilos. (37½ lbs.), but had increased in height to ·99 m. (3½ feet). Nothing is said of the state of his ossification. The wisdom teeth had not been cut, and some of the milk teeth were still persisting. There was no appearance of rickets. His muscular strength was about equal to that of a child of six. His appearance, proportions, and gait were childish, and he was often mistaken for a child, but his manner and conversation were not childish. He appeared to be of average intelligence. The condition of the genital organs was infantile. At the age of 14 or 15, according to his own account, he showed some of the psychical phenomena of puberty, but there appears to have been no real evidence on this point. The skull measured :—Greatest length, 178 mm. = 6⅝ inches; transverse diameter, 148 mm. = 5⅓ inches; vertical, 127 mm. = 5 inches; horizontal circumference, 530 mm. = 20⅞ inches.

Cases 8 to 11.—A short account of some instances of dwarfism are given by Dr. Joachimsthal,[1] of Berlin, four of which appear to be ateleiotic. He examined a troupe of German dwarfs, and selected some of them for detailed description. He also refers to other cases, and compares the disease with achondroplasia and cretinism. Of his six cases of dwarfism one commenced at the third year, and will therefore come under my second group In his second case, the abnormality was first noticed in the seventh year, in the third at the tenth year, in the fourth at the eighth year, but he says nothing of the time of commencement in the last two. The ages varied from 30 to 36 years, and half of them were male and half female. Measurements of height appear to have been made by the director of the troupe as each dwarf came under his care. These are compared by Dr. Joachimsthal with those which he himself made when he first saw them. In one of these the

[1] 'Deutsch. med. Woch.,' No. 17, 1899, s. 269.

first measurement was made in the fifteenth and another in the seventeenth years. It is therefore possible that the respective increase of 10 cm. (3¾ inches) in fifteen years, and 29 cm. (11½ inches) in fourteen years, was in part due to natural growth at the time of puberty. But the same can hardly be said of two other cases in which the first measurement was taken in the twenty-first and twenty-second year, respectively. In the first of these two, the rate of growth was 25 cm. (9¾ in.) in thirteen years, and in the other 22 cm. (8¾ in.) in fourteen years. It cannot be said whether growth in these cases was continuous or regular, though Joachimsthal mentions that in his second case, where the age was 36, there had been a noticeable increase in height during the last three years. If we compare the heights of these dwarfs with the heights of normal children, we find that, according to Quetelet's tables, the first case grew from the height of a child of 3¾ years, until within sixteen years he had reached the height of a child of 6 years. His second case grew in fourteen years from the height of a child of 6 to that of one of 10 years. His third in thirteen years from that of a child 8½, to that of one of 11 years; and his fourth from that of a child of 6 to that of one of 11 years.

Radiograms are given of the hands of four cases, and the first also includes the greater part of the upper extremity. It is noteworthy that the radiograms of the first case show that, while the height was equal to that of a child of 6 years, the ossification was equal to that of a child between the eleventh and twelfth years. Similar delay in ossification is shown in the next three cases. But in the fifth and sixth there was complete epiphysial growth. These were two females of the age of 26 years, who had not been measured during the last ten years. They differ from the other female, who was of the age of 30, not only in ossification, but also in regard to menstruation. In both of them menstruation had been regular since their twentieth year. It is unfortunate that no photographs are given, other than radiograms, and very

little is said of the general appearance and of the condition of the sexual and other organs. In the first case, that of a male, the voice was high pitched, and there was no hair, except on the head. They are said to have been well proportioned, but there are no measurements other than those of height. The first case was said to have been of good mental development. It is almost certain that the first four were instances of ateleiosis, while the last two were probably of the same nature.

At the end of his paper Joachimsthal alludes to Schaaffhausen's case, and also mentions the cases of Schauta and Paltauf. He refers also to a skeleton shown him by Waldeyer, of a female dwarf of the age of 65 years, and 119 cm. high (3 feet 10⅞ inches), in whom the dwarfism was perhaps of the same nature.

Doubtful Case.

A doubtful case is described by Dr. Thomson.[1] The patient was a girl who was of the age of four years and eight months when Dr. Thomson first saw her, and she died nearly ten months afterwards. The parents were healthy, but of ten children one was said never to have grown properly, and to have been always dull, while another was a well-marked sporadic cretin. At three and a half years the patient was 27 inches (68·5 cm.) high; one year and two months later she was 28½ inches (72·5 cm.) high, weighed 20 lbs. 7 oz. (9·34 kilos.), and measured 18⅛ inches (47 cm.) round the head. Nearly eleven months afterwards she was 30⅛ inches (76·4·cm.) high and weighed 21 lbs. 4 oz. (9·5 kilos.) First dentition began at the age of four months. At the age of four years and eight months the ossification of the carpus and hand resembled that of a child of two or three years. The anterior fontanelle was still open, but ten months after-

[1] "A Case of a Peculiar Form of Dwarf Growth," by John Thomson, M.D., with notice of *post-mortem* examination by Jessie Macgregor, M.D., 'Scot. Med. and Surg. Journ.,' March, 1900.

Ateleiosis. Group iii. Case 12. Martin Lane, aged 28 years, height 4ft. 9½in. (1.46m.) is standing on the right, next to him is his brother of 13 and on the left is a normal adult. The ateleiosis began at the age of 14. Note the absence of sexual hair, the childish sexual organs, and the youthful aspect and proportions, combined with the weathering of age.

Bale & Danielsson, Ltd., London.

wards it was nearly closed. The mental condition was normal. Improvement occurred during the use of thyrocol. Death took place as the result of syncopal attacks, which resembled those which accompany lymphatic hypertrophy. At the *post-mortem* examination the thymus gland was found to be hypertrophied, though it did not flatten the trachea. The thyroid gland and heart were normal. The surface of the brain was much congested. This case was probably one of infantilism due to lymphatic hypertrophy.

GROUP III.—*Ateleiosis beginning between the Ages of Infancy or early Childhood and Puberty.*

Cases which belong to the previous class evidently belong definitely to one group, for they are all stamped with the facial and other characters which belong to infancy and early childhood. But after this age we can no longer say that we have to deal with one distinct facial type, for as infancy is left behind so the face changes, and the proportions approach to those of the adult. The subjects of ateleiosis commencing during these later years, will therefore exhibit different features from those which are shown during infancy. They will not be so dwarfed; the delay of osseous development will not be so conspicuous; the physiognomy will not be so infantile; the proportions of the body and limbs will approach nearer to those of the adult, and they will be more likely to attain puberty. The following cases are given in illustration of this third class.

CASE 12.—Martin Lane, aged 28 years, was of the following measurements:

Weight, 35·6 kilos. = 79 lbs.

Height, 1·46 m. (4 feet 9½ inches).

Head: circumference, 52 cm. (20½ inches); length, 17·4 cm. (6⅝ inches); breadth, 14·2 cm. (5 7/12 inches).

Chest round nipple-line, 73 cm. (28¾ inches) to 75 cm. (29½ inches).

Abdomen at umbilicus, 69 cm. (27⅛ inches).

Upper extremity: acromia to elbow, 24·5 cm. (9⅝ inches); external condyle to styloid process of radius, 22 cm. (8⅔ inches); hand, 17·5 cm. (6 11/12 inches).

Middle point of total height, 2 cm. above pubes.

Lower extremity: great trochanter to external condyle, 39 cm. (15⅜ inches); external condyle to external malleolus, 36·3 cm. (14⅜ inches); hand, 25·3 cm. (10 inches).

History.—The father is a "small-made man," and rather below the medium height. The mother and six brothers and sisters are of average growth. There is a brother of the age of thirteen who is 1·34 m. (4 feet 2¾ inches) high. The mother believes that Martin was of about the same height when he was of the same age. It was noticed that Martin seemed to have stopped growing at about that time. He was then just leaving school. His mother cannot account for the circumstance, for he was quite healthy at the time and has had no illness either before or since.

General appearance.—The proportions and appearance are those of a lad of fourteen years. He was at one time shown at a meeting of the Reading Pathological Society, when nearly all those who were present judged him to be of that age. His occupation was that of a farm boy. It was not possible for him to get work as a man, and his mother said that she always kept his certificate of birth in hand, because no one would believe that he was other than a boy until she had shown it. I myself first saw him among the crowd at a village jumble sale, and at once recognised that he was an instance of delayed development. The skin of his face was more rough and weather-worn than one ever sees in a youth, though his manner and voice were in keeping with his size.

His intelligence was not good, though he answered questions intelligently and seemed to have a fair memory. He had passed the fourth standard at school, and could read and write as well as most boys of his walk of life. His mother told me that he was too stupid to do better

work than minding sheep, and that he was not worth the 3*s*. 6*d*. a week which was paid him. He was not quite so strong as his brother of thirteen, and on having him stripped it was evident that the muscles were not of good size. He was, however, capable of working all day and then taking a walk of four miles without feeling tired. His hands and feet were rather large, and there was slight kyphosis and lordosis of the spine. He was also knock-kneed and flat-footed to the same degree, so that his gait was somewhat awkward and shambling.

Though the skin of the body felt harsh and dry he said that he sweated when he became over-heated. The hair of his head was fine and thin. There was plenty of lanugo over the body and limbs. His teeth were sound and the dentition was regular. There was no sign of syphilis either in the teeth or in any other part. His appetite and digestion were good. He ate about as much as is customary for a youth of his age. The bowels were regular, the urine was of sp. gr. 1018 and was otherwise normal. The special senses were of ordinary acuteness. A radiogram of the right hand and wrist showed that the ossification was equal to that of a youth of fourteen or fifteen. The distal epiphyses of the radius and ulna which usually unite at from eighteen to twenty years were still separated by a narrow line of cartilage.

Auscultation of the heart revealed a slight basal systolic murmur, but no other abnormality. The blood-corpuscles counted by means of a Thoma-Zeiss hæmocytometer showed 4,800,000 red discs to the c. mm., while the leucocytes were in the proportion of 1 to 450 red. These white cells appeared to be normal, but no stained specimen was examined.

The external genital organs were of the size and appearance of those of a child of eight or nine years. There was no pubic hair. The right testicle was descended, and the left could be felt about half-way down the inguinal canal. Testicular sensation was present, but there was no sign of virility. He was of an unusually timid disposition and would

never come to see me without his thirteen-year-old brother. He was also very modest and greatly objected to exposure of his body.

Less than three months after these observations were made, Martin Lane was affected with an illness which was probably influenza. Pneumonia then set in and he was seized with a series of eclampsic attacks, in one of which he died. He was attended by Dr. Robinson, of Sonning, who most kindly informed me of his death and assisted me to make a *post-mortem* examination.

Result of post-mortem examination.—Fat was present in ordinary proportions. The lymphatic glands of the anterior mediastinum were enlarged to the size of peas or small beans. In other parts they could be detected with difficulty. The lower lobes of both lungs and middle lobe of the right were of a dark homogeneous plum colour, and broke down easily on pressure with the thumb, exuding quantities of frothy serum. There were no signs of tubercles.

The heart weighed 8½ oz. = 241·5 grms.

The mitral valves were crumpled and thickened with atheroma, but seemed to be fairly efficient. An old organised clot was present in the right ventricle and extended for some distance up the pulmonary artery. The wall of the ventricle round the coronary artery of the aorta was slightly atheromatous. A ductus arteriosus was present but was not open. The thyroid gland weighed 179 grains = 11·5 grms. Its two lobes were separate, no isthmus being present. Its structure was homogeneous, and on microscopic examination nothing abnormal was detected save a slight excess of interacinal fibrous tissue.

The brain weighed 2 lbs. 13 oz. = 1275 grms. No abnormality could be detected.

The hypophysis cerebri was examined and appeared to be in every way normal, but unfortunately through an oversight it was not weighed or taken away for microscopical examination.

The stomach and intestines were normal.

never come to see me without his thirteen-year-old brother. He was also very modest and greatly objected to exposure of his body.

Less than three months after these observations were made, Martin Lane was affected with an illness which was probably influenza. Pneumonia then set in and he was seized with a series of eclampsic attacks, in one of which he died. He was attended by Dr. Robinson, of Sonning, who most kindly informed me of his death and assisted me to make a *post-mortem* examination.

Result of post-mortem examination.—Fat was present in ordinary proportions. The lymphatic glands of the anterior mediastinum were enlarged to the size of peas or small beans. In other parts they could be detected with difficulty. The lower lobes of both lungs and middle lobe of the right were of a dark homogeneous plum colour, and broke down easily on pressure with the thumb, exuding quantities of frothy serum. There were no signs of tubercles.

The heart weighed 8½ oz. = 241·5 grms.

The mitral valves were crumpled and thickened with atheroma, but seemed to be fairly efficient. An old organised clot was present in the right ventricle and extended for some distance up the pulmonary artery. The wall of the ventricle round the coronary artery of the aorta was slightly atheromatous. A ductus arteriosus was present but was not open. The thyroid gland weighed 179 grains = 11·5 grms. Its two lobes were separate, no isthmus being present. Its structure was homogeneous, and on microscopic examination nothing abnormal was detected save a slight excess of interacinal fibrous tissue.

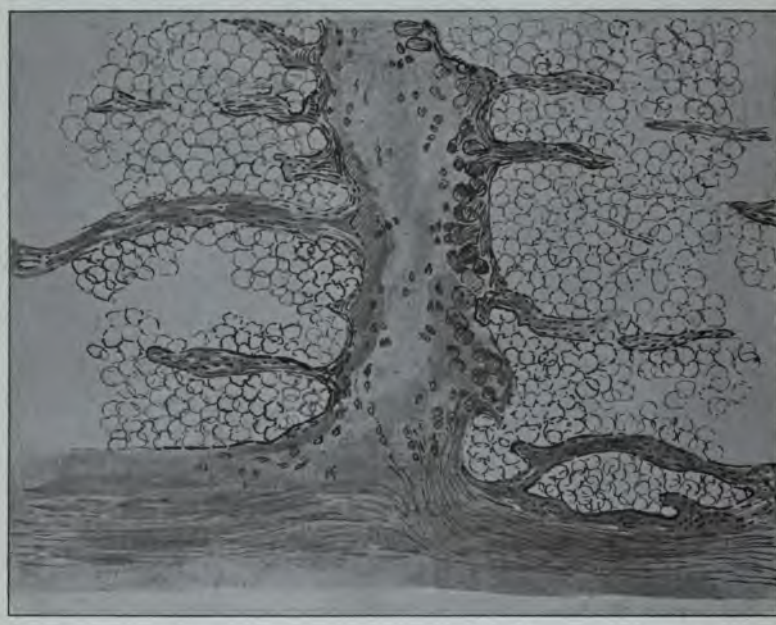
The brain weighed 2 lbs. 13 oz. = 1275 grms. No abnormality could be detected.

The hypophysis cerebri was examined and appeared to be in every way normal, but unfortunately through an oversight it was not weighed or taken away for microscopical examination.

The stomach and intestines were normal.

Gilford: Ateleiosis. Plate VII.

EPIPHYSIS. DIAPHYSIS.



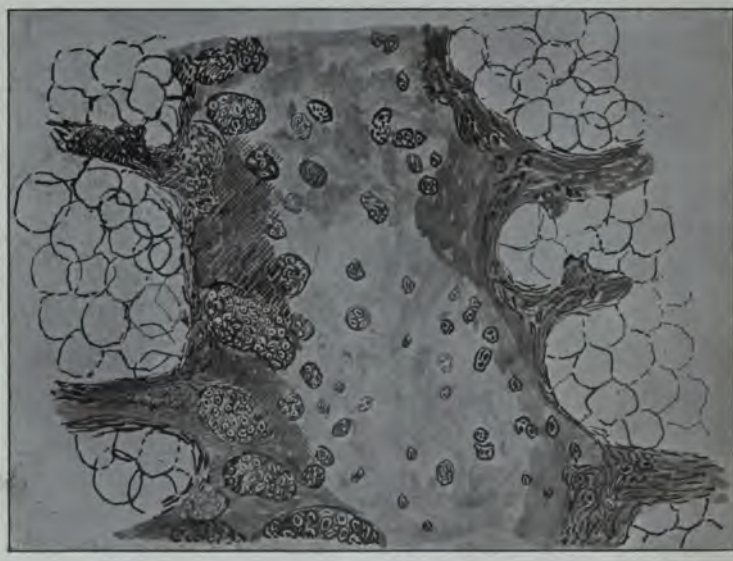
Case 12.—Section through ossifying zone of lower end of tibia as seen under $\frac{1}{3}$ objective.

At the lower part of the drawing is the periosteum, and running at right angles to it is the epiphysial cartilage dividing the epiphysis (on the left) from the diaphysis (on the right).

Bale & Danielsson Ltd., London.

Gilford: Ateleiosis. Plate VIII.

DIAPHYSIS. EPIPHYSIS.



Case 12. Part of section shown on previous plate under high power ($\frac{1}{2}$ objective). The positions of the diaphysis and epiphysis are reversed.

Bale & Danielsson, Ltd., London.

The liver was not weighed.

The kidneys weighed 7¾ oz. = 220·4 grms.

Supra-renal capsules weighed: spleen, 3¼ oz. = 92·25 grms.; pancreas, 2¼ oz. = 63 grms.

All the abdominal organs were examined microscopically and were found to be quite healthy, with the exception of some recent small cell infiltration in the kidneys and liver, which was evidently the result of the illness from which death resulted.

The testicles together weighed 15 grains (1 grm.); that on the right side was still in the inguinal canal, and was a little smaller than the left, which was descended.

A section of the left testicle was kindly examined for me by Mr. McAdam Eccles, who reported that there was no evidence of abnormality beyond the extreme delay of development; the organs resembled those met with in early infancy.

The several parts of the sternum were still separated by cartilage, and there was a small ossification centre in the ensiform cartilage; the ribs showed no beading.

The clavicles were fully ossified, and their muscular prominences were fairly well marked.

In the skull the frontal suture was obliterated, and the different pieces of the temporal and occipital bones were fused together, but the basi-sphenoid and basi-occipital were ununited. The os innominatum was still divided into its three constituent bones by thin lines of cartilage. The lower epiphysis of the tibia was separated from the diaphysis by a thin line of cartilage. A piece of this was afterwards examined under the microscope.

Microscopical characters of zone of ossification.—A section through the epiphysial cartilage of the lower end of the left tibia when compared with Paltauf's description of the epiphysial cartilage in his case shows the following characters:—The cartilage extends under the periosteum on the epiphysial side to such a distance that it probably embraces the whole of the ossified part of the epiphysis. The periosteum is, perhaps, a little thicker than normal.

The cancellous tissue of both the epiphysis and the diaphysis has a very open meshwork, causing the section to be extremely fragile and difficult to cut. The peripheral layer of calcified cartilage is of very unequal thickness; it consists of an irregular, shallow, abrupt, disconnected line, lying between a thin layer of imperfectly ossified bone on the one side, and the cartilage of the epiphysis on the other; its stain (logwood) is intermediate in tint between that of the bone and the cartilage; its margin is very uneven towards the bone and more even on the side of the cartilage. The cartilage cells are small and scattered, and of irregular shape, becoming first slowly, and then rapidly larger and rounder as they approach the diaphysis. Close to the diaphysial border they are collected together into large, round, or oval encapsuled masses, which take the logwood much better than any other part. A few of these masses are piled into somewhat oblique, irregular columns. Most of the columns are entirely surrounded by cartilage, but here and there are spaces containing marrow-cells, which are open towards the bone. Paltauf's stratum of calcified cartilage is so broken and indistinct that it is not easy to define.

Case 13.—Dr. Schmidt,[1] of Munich, describes an instance in Theresa Fend, a girl of the age of sixteen years, who was of normal size for the first half of her life, and in her ninth year was in bed for a fortnight with a severe illness, of which no details were obtainable.

From this time growth almost ceased, and at most did not amount to more than 1 or 2 inches (2·5 or 5 cm.). When Dr. Schmidt saw her at the age of sixteen years she had the appearance of a child of eight; this being the time at which she first gave evidence of delayed growth. The grandparents and two sisters were of normal size. She was not microcephalic, but, on the contrary, was of good intelligence, well-proportioned, and of good muscular and

[1] 'Zur Kenntniss des Zwergwuchses, Arch. f. Anthrop.,' Bd. xx, 1891, s. 59.

fatty development. She had broad shoulders, a well-formed thorax, and healthy lungs. There were no signs of the approach of puberty, and no hair on the pubes or armpits, though there was plenty on the head. The abdomen appeared to be inflated with gas, though there was no sign of abdominal tumour or of ascites. The teeth were of the permanent set, though the canines of the right side were only just appearing through the gum. They first appeared in the sixteenth year. The other teeth appeared to be normal. Five molars were erupted, and three others seemed to be about to break through. Her height was 1160 mm. (3 feet 9¾ inches), the height of an average girl of her age in Bavaria being 1520 mm. (5 feet). A photograph of the girl is given with the article.

Doubtful Cases.

Mr. Hutchinson[1] has described a case of apparent ateleiotic dwarfdom in a young man. The condition was associated with marked overgrowth of the gums. He was of the age of twenty-five, though he looked like a boy of twelve. The testes and penis were like those of a young child, while the voice was cracked and feeble, and there was no sexual hair. He was of good intelligence. A photograph which was taken of his face and head showed no peculiarity of physiognomy, except that he looked strongly prognathous, this appearance being due to the overgrowth of the gums. There was no indication of syphilis.

Dr. Kirk, of Glasgow,[2] has described a case of imperfect development in a male æt. 22 years. He was 4 feet ¼ inch (123 cm.) high, and a radiogram showed that the ossification was equal to that of a child of half his age. Nothing is said of the state of the sexual organs.

[1] "A Case of Hypertrophy of the Gums with General Dwarfdom." Jonathan Hutchinson, F.R.S., 'Edin. Med. Journ.,' n. s., vol. i, No. 2, p. 117.

[2] 'Lancet,' May 4th, 1901, vol. i, p. 1267.

also present, and the epiphyses of the crests of the ilia and of the other parts of the ossa innominata were not united. The cartilaginous lining of the acetabulum was thicker than usual. The femora were slightly formed; their lower epiphyses being of about the same thickness as those of a child seven years old. The head of each femur was small and shallow, and, with the neck, took a direction from the shaft which made a smaller angle than is usual with the child, but was more open than in the adult. The epiphyses of the tibiæ and fibulæ were not yet united. The patellæ were of ordinary form and size, and the bones of the feet, like those of the hand, corresponded in ossification with those of a seven-year-old child.

Dr. Paltauf made a microscopical examination of the epiphyses of several bones, and gives two illustrations of sections through the ossifying zone of the lower end of a femur. He found the periosteum thick and fibrillar. The most conspicuous features of these sections were two strata of calcified cartilage, one enveloping the diaphysial end, and another (the thinner) the epiphysial end of the bone. In cutting from below upwards through the epiphysial line, the following parts were cut through. First, the cancellous bone of the epiphysis with somewhat large marrow spaces and thin bony septa. Then a stratum of calcified cartilage, abrupt, of a bluish colour, shallow, and of irregular depth, so that in parts it was quite absent, the bone of the epiphysis coming in contact with the cartilage. It contained large, scattered, hyaline, and glistening cells, with oval or irregular nuclei. Next came the cartilage of the epiphysial line proper, which was hyaline, and, at this point, contained scattered cells in small irregular groups separated from each other by fine striæ of delicate fibrils. Some were long spindle-cells with nuclei and nucleoli; but the deeper cells showed much variation in size, form, and distribution. In the centre they resembled those which are found in the covering cartilage of joints. Those nearest the epiphysis were larger, plainer, more numerous, and in bigger groups. Some of them were long and spindle-

The transverse and other processes contained cartilaginous patches, and isolated bone centres occurred in the epiphyses of the vertebræ. The three bone centres of the great trochanter were surrounded by cartilage. This may have been an instance of ateleiosis combined with osteomalacia, but it seems more probable that the condition was a form of infantilism, the direct result of osteomalacia beginning in a young subject.

König's [1] case, which is quoted by Paltauf, was that of a girl who died at the age of 18 years, from "Cysticercus cerebri." She was above the average height of women and had a well-developed head. The bones of the pelvis were very thick and spongy, and light in weight. A Y-shaped cartilage separated each innominate bone into its three constituent bones. The iliac bones were greatly deformed. There was hypoplasia of the genital organs, the breasts being as small as those of a child, the mons veneris undeveloped and without hair, and the vagina narrow. The uterus resembled that of a new-born child, and the ovaries those of a girl of from 12 to 14 years. Some osteo-chondromata were found growing from the pelvis. The bones of the pelvis seem to have been the only bones examined.

This case may have been one of infantilism due to osteomalacia, as in the previous case, but the extreme hypoplasia of the sexual organs is suggestive of ateleiosis. It is interesting that osteomalacia should have occurred under such circumstances, seeing that removal of the ovaries is of so great effect in curing osteomalacia. It is also of interest in showing that it is possible for delay of bone development to be associated with normal stature.

Paltauf also gives a few details of another case of dwarfism, which seems to belong to this third class. On referring to the original account, it is evident that insufficient particulars were given to warrant its inclusion in my list. This case is one which is described by Dr. F.

[1] 'Beschreibung eines kindlichen Beckens und kindlicher Geschlechtstheile von einem 18 Jahre alten Mädchen,' Inaug-Diss., Marburg, 1855.

Rohrer,[1] of Zurich. The father was syphilitic, and the mother was a tall woman who had died of phthisis.

The patient was a man of the age of twenty, well proportioned, but thin, and looking old for his age. His facial appearance was not very intelligent. He was 120 cm. (3 feet 11¼ inches) high, and was normal at birth. In the second, and again in the eleventh year, he fell on his head, and in the twelfth year cut his head with a hatchet. Since this last accident there was incontinence of urine and "cessation" of growth and development. The voice remained a childish soprano. The sexual organs were undeveloped, like those of a child from five to seven years. There was no sign of sexual hair. The prepuce covered the penis, and the testicles were quite rudimentary on both sides.

In addition to these cases there are others which have been referred to by Professor Quetelet,[2] Sir G. M. Humphry,[3] Dr. N. W. Kingsley,[4] Messrs. Gould and Pyle,[5] and some information may also be gathered from the Natural History of Geoffrey St. Hilaire,[6] and from certain semi-scientific authors, such as E. Garnier,[7] Le Roux and J. Garnier,[8] and E. J. Wood.[9] We have also the autobiography of "Boruwlaski,"[10] who was himself one of these dwarfs, and a very candid account by Barnum.[11] Much that is "popular" in these writings must be regarded with suspicion, but in some respects they may certainly be relied upon. This is especially the case when no object is to be gained by deceit, and when the tale told in one case tallies with that told in another, or where it corresponds with facts which are already known. I have also myself seen many instances which have been exhibited at variety and

[1] 'Virch. Arch.' Bd. ci, s. 197.
[2] 'Anthropometrie.'
[3] 'On the Skeleton.'
[4] 'Oral Deformities.'
[5] 'Anomalies and Curiosities of Medicine.'
[6] 'Histoire Naturelle.'
[7] 'Les Nains et les Gèants.'
[8] 'Acrobats and Mountebanks.'
[9] 'Giants and Dwarfs.'
[10] 'Memoirs of Count Boruwlaski.'
[11] 'Life of P. O. Barnum.'

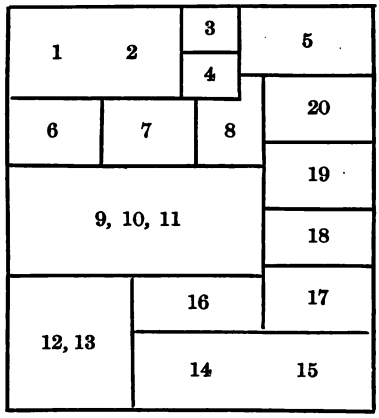
Heads of 20 dwarfs showing features of ateleiosis of the second group. Their proportions and facial characters are childish, though they show the superficial markings of age. The two heads at the right hand lower corner are those of "Tom Thumb" and his wife, and the bust in uniform below the figure at the opposite corner is that of Boruwlaski.

Bale & Danielsson, Ltd., London.

DESCRIPTION OF PLATE IX.

Ateleiosis: a Disease characterised by Conspicuous Delay of Growth and Development (Hastings Gilford, F.R.C.S.Eng.).

Group of dwarfs showing features of ateleiosis of the second group.



The features are those of stereotyped childhood. Hence the stature is small, the limbs short, the head large, and the face broad and flat; the bridge of the nose is undeveloped, and the distance from the ear to the vertex is unusually great. The facial type is so well defined in some cases (Nos. 5, 9, 10, 11, 14, 15, 16) as to obliterate the natural expression of character and produce a strong resemblance between dwarfs of different families But added to these childish features are the lines and superficial markings of age. In the case of No. 9 there is facial hair, and in at least four others (Nos. 6, 11, 14, 15) there was evidence of sexual maturity. All these dwarfs have been exhibited in variety shows, and are or were of good intelligence.

The most noteworthy of the group are Charles Stratton (No. 14), who was known as Tom Thumb, and his wife (No. 15) Lavinia Warren. They are said to have had one child. Charles Stratton died in 1883 at the age of 41. Minnie Warren (No. 11), sister of Lavinia, married George Washington Nutt (No. 10), who died in 1881 at the age of 33 years. No. 9 is known as Baron Magri. Boruwlaski (No. 6) was born in Poland in 1739, and died at Durham in 1837, aged 98. He married a lady of ordinary stature and had two children. He published a "memoir" of his life. The photograph is from a portrait in oils in the Hunterian Museum of London. No. 4 is the French dwarf described on p. 320; and No. 17 is described on p. 316. Nos. 12, 13, 19, and 20 are the German brothers Franz and Carl Rossow; No. 4 is Annie Nelson, and No. 16 the mulatto known as Chiquita.

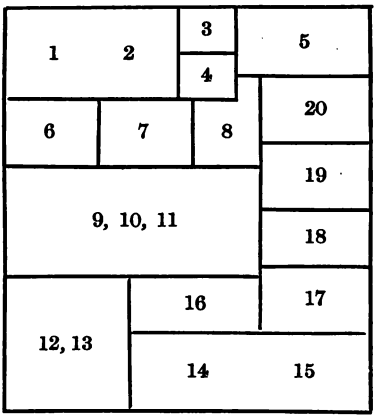
Heads of 20 dwarfs showing features of ateleiosis of the second group. Their proportions and facial characters are childish, though they show the superficial markings of age. The two heads at the right hand lower corner are those of "Tom Thumb" and his wife, and the bust in uniform below the figure at the opposite corner is that of Boruwlaski.

Bale & Danielsson, Ltd., London.

DESCRIPTION OF PLATE IX.

Ateleiosis: a Disease characterised by Conspicuous Delay of Growth and Development (Hastings Gilford, F.R.C.S.Eng.).

Group of dwarfs showing features of ateleiosis of the second group.



The features are those of stereotyped childhood. Hence the stature is small, the limbs short, the head large, and the face broad and flat; the bridge of the nose is undeveloped, and the distance from the ear to the vertex is unusually great. The facial type is so well defined in some cases (Nos. 5, 9, 10, 11, 14, 15, 16) as to obliterate the natural expression of character and produce a strong resemblance between dwarfs of different families But added to these childish features are the lines and superficial markings of age. In the case of No. 9 there is facial hair, and in at least four others (Nos. 6, 11, 14, 15) there was evidence of sexual maturity. All these dwarfs have been exhibited in variety shows, and are or were of good intelligence.

The most noteworthy of the group are Charles Stratton (No. 14), who was known as Tom Thumb, and his wife (No. 15) Lavinia Warren. They are said to have had one child. Charles Stratton died in 1883 at the age of 41. Minnie Warren (No. 11), sister of Lavinia, married George Washington Nutt (No. 10), who died in 1881 at the age of 33 years. No. 9 is known as Baron Magri. Boruwlaski (No. 6) was born in Poland in 1739, and died at Durham in 1837, aged 98. He married a lady of ordinary stature and had two children. He published a "memoir" of his life. The photograph is from a portrait in oils in the Hunterian Museum of London. No. 4 is the French dwarf described on p. 320; and No. 17 is described on p. 316. Nos. 12, 13, 19, and 20 are the German brothers Franz and Carl Rossow; No. 4 is Annie Nelson, and No. 16 the mulatto known as Chiquita.

other shows, some of which I have been able to examine. From all these different sources it is possible to piece together a fairly connected account of the disease. Its main clinical and anatomical features may be summed up as follows:

PART II.

General Description of Ateleiosis.

Etiology.—In Dr. Schmidt's case (No. 13), in which dwarfism began at the age of eight years, there appears to have been some indication of a definite disturbance of health at the onset, but no details of the illness were obtainable. In one other instance arrest of growth seems to have dated from injury. This instance is reported by Rohrer: there had been three accidents to the head, but the case is not one of unquestionable Ateleiosis. There is no satisfactory evidence of the taint of syphilis in any of the cases. I can find no instance of its direct transmission from parent to offspring, though it undoubtedly occurs as a family disease among brothers and sisters. Boruwlaski was one of a family of dwarfs, and the wife of Charles Stratton, who was undoubtedly ateleiotic, had a sister who was a "midget." The conclusion that we arrive at is that the actual cause of the disease is unknown.

Facial and general appearance.—These vary with the age of onset of the disease. In its most characteristic form, *i. e.* when it begins during infancy or early childhood, ateleiosis may probably be invariably recognised by its remarkable perpetuation of childish characters. The head is large in comparison with the rest of the body, and is broad and high. The extremities are, as a rule, short, and the middle point of the body is therefore, as in childhood, above the pubic symphysis. The relative length of the segments of the limbs is also like those which are noticeable in childhood. The facial type is so distinctly childish that it is probable that ateleiotic dwarfs of the second class may be distinguished from all other dwarfs by their

physiognomy alone. The face is broad and flat. The nose is undeveloped, especially at the bridge, and is, as a rule, *retroussé*. The voice is usually thin or piping, and may be so high pitched as to be squeaky. It has appeared to me to be more treble among females than among males. In no case does there seem to have been any growth of hair on the face, and in none has there been any baldness.

From these general features it will be understood that these dwarfs are very like one another in appearance. They resemble each other in the same way that one baby resembles another baby. Their features lack that variety which is ordinarily produced by age, and which is, to a large extent, due to one part of the face growing more rapidly than another part. But, while the grosser features change very little, those finer changes, which mark the progress of age, appear to continue as in ordinary individuals. The face of a middle-aged ateleiotic dwarf of well-marked type is such as we may imagine would result if the features of an infant remained stereotyped through the succeeding periods of life. It retains its childish form while it undergoes the wrinkling and weathering of age. Indications of age are also present in the internal organs, as in Schaaffhausen's case (No. 5), in which death occurred at sixty-one.

Osseous and muscular systems.—The most conspicuous feature of the bones is the marked delay in the process of ossification. This is shown in three ways—by the late appearance of the centres of ossification, by their delayed fusion, and by lack of vigour of bone growth generally. The last is manifested in the smallness and delicacy of the bones, especially of the long bones, and in the ill-development of the muscular prominences.

On the other hand, Paltauf, in his account of his case, draws special attention to the prominence of the lines and ridges of bone produced by the attachment of muscles But these do not show very conspicuously in his portrait of the skeleton, and it is perhaps right to infer that he is comparing the skeleton, not with a man of the same age,

but with a child of the same size. These bony prominences probably vary greatly in accordance with the variation in the size of the muscles, but, as a rule, are more marked than is usual in children, and less marked than in adults. They are very different from the exaggerated lines and eminences which are seen in rickets. The different manifestations of osseous feebleness do not keep pace with one another, for it will invariably be found that the age, as determined by the ossification of the epiphyses, is in advance of that which is indicated by the height and proportions of the skeleton. The discrepancy is not, as a rule, great, but is noticeable in every case in which the state of epiphysial ossification is mentioned.

Muscular development is usually proportionate to the size. Martin Lane (Case 12) at twenty-eight was not so strong as his brother of fourteen. On the other hand, in one case that I have seen, the strength was undoubtedly excessive, but this was probably accounted for by the fact that the dwarf in question had been trained to perform as a "strong man." His shoulders and upper extremities were disproportionately big. It is also possible that the prominence of the bony eminences upon which Paltauf lays stress in his description of the skeleton of Mikolajek were to be explained by some exceptional growth of the muscles. But, as a rule, dwarfs of this type seem to be little, if at all, stronger than the children of their own size, though they are possessed of much greater powers of endurance. Many of them are skirt-dancers or actors, and all of those which I have seen have been able to carry on their work without undue effort and without detriment to health.

Dentition.—The teeth are, as a rule, decidedly backward in development, though they are of ordinary size. In some cases they are well preserved, and are regular in form and situation. In these it will, I think, be noticed that they have undergone but little wear, indicating that they have not long erupted, as was noticed in Paltauf's case. Room is then found for them in the diminutive jaw by the non-appearance of the back molars. In other instances they are,

other shows, some of which I have been able to examine. From all these different sources it is possible to piece together a fairly connected account of the disease. Its main clinical and anatomical features may be summed up as follows:

Part II.

General Description of Ateleiosis.

Etiology.—In Dr. Schmidt's case (No. 13), in which dwarfism began at the age of eight years, there appears to have been some indication of a definite disturbance of health at the onset, but no details of the illness were obtainable. In one other instance arrest of growth seems to have dated from injury. This instance is reported by Rohrer: there had been three accidents to the head, but the case is not one of unquestionable Ateleiosis. There is no satisfactory evidence of the taint of syphilis in any of the cases. I can find no instance of its direct transmission from parent to offspring, though it undoubtedly occurs as a family disease among brothers and sisters. Boruwlaski was one of a family of dwarfs, and the wife of Charles Stratton, who was undoubtedly ateleiotic, had a sister who was a "midget." The conclusion that we arrive at is that the actual cause of the disease is unknown.

Facial and general appearance.—These vary with the age of onset of the disease. In its most characteristic form, *i. e.* when it begins during infancy or early childhood, ateleiosis may probably be invariably recognised by its remarkable perpetuation of childish characters. The head is large in comparison with the rest of the body, and is broad and high. The extremities are, as a rule, short, and the middle point of the body is therefore, as in childhood, above the pubic symphysis. The relative length of the segments of the limbs is also like those which are noticeable in childhood. The facial type is so distinctly childish that it is probable that ateleiotic dwarfs of the second class may be distinguished from all other dwarfs by their

physiognomy alone. The face is broad and flat. The nose is undeveloped, especially at the bridge, and is, as a rule, *retroussé*. The voice is usually thin or piping, and may be so high pitched as to be squeaky. It has appeared to me to be more treble among females than among males. In no case does there seem to have been any growth of hair on the face, and in none has there been any baldness.

From these general features it will be understood that these dwarfs are very like one another in appearance. They resemble each other in the same way that one baby resembles another baby. Their features lack that variety which is ordinarily produced by age, and which is, to a large extent, due to one part of the face growing more rapidly than another part. But, while the grosser features change very little, those finer changes, which mark the progress of age, appear to continue as in ordinary individuals. The face of a middle-aged ateleiotic dwarf of well-marked type is such as we may imagine would result if the features of an infant remained stereotyped through the succeeding periods of life. It retains its childish form while it undergoes the wrinkling and weathering of age. Indications of age are also present in the internal organs, as in Schaaffhausen's case (No. 5), in which death occurred at sixty-one.

Osseous and muscular systems.—The most conspicuous feature of the bones is the marked delay in the process of ossification. This is shown in three ways—by the late appearance of the centres of ossification, by their delayed fusion, and by lack of vigour of bone growth generally. The last is manifested in the smallness and delicacy of the bones, especially of the long bones, and in the ill-development of the muscular prominences.

On the other hand, Paltauf, in his account of his case, draws special attention to the prominence of the lines and ridges of bone produced by the attachment of muscles But these do not show very conspicuously in his portrait of the skeleton, and it is perhaps right to infer that he is comparing the skeleton, not with a man of the same age,

but with a child of the same size. These bony prominences probably vary greatly in accordance with the variation in the size of the muscles, but, as a rule, are more marked than is usual in children, and less marked than in adults. They are very different from the exaggerated lines and eminences which are seen in rickets. The different manifestations of osseous feebleness do not keep pace with one another, for it will invariably be found that the age, as determined by the ossification of the epiphyses, is in advance of that which is indicated by the height and proportions of the skeleton. The discrepancy is not, as a rule, great, but is noticeable in every case in which the state of epiphysial ossification is mentioned.

Muscular development is usually proportionate to the size. Martin Lane (Case 12) at twenty-eight was not so strong as his brother of fourteen. On the other hand, in one case that I have seen, the strength was undoubtedly excessive, but this was probably accounted for by the fact that the dwarf in question had been trained to perform as a "strong man." His shoulders and upper extremities were disproportionately big. It is also possible that the prominence of the bony eminences upon which Paltauf lays stress in his description of the skeleton of Mikolajek were to be explained by some exceptional growth of the muscles. But, as a rule, dwarfs of this type seem to be little, if at all, stronger than the children of their own size, though they are possessed of much greater powers of endurance. Many of them are skirt-dancers or actors, and all of those which I have seen have been able to carry on their work without undue effort and without detriment to health.

Dentition.—The teeth are, as a rule, decidedly backward in development, though they are of ordinary size. In some cases they are well preserved, and are regular in form and situation. In these it will, I think, be noticed that they have undergone but little wear, indicating that they have not long erupted, as was noticed in Paltauf's case. Room is then found for them in the diminutive jaw by the non-appearance of the back molars. In other instances they are,

more or less, irregularly disposed, as was the case in "Tom Thumb," whose jaw was examined by Dr. N. W. Kingsley,[1] who found "a most marked mal-position of the teeth, so much so, that he had a double row of teeth all round." In the case of Bobbie Fenwick, who died after the age of fifty, dentition was very irregular, owing to the late eruption of several of the teeth. A third variety is found in some of the younger ateleiotic dwarfs, where the milk teeth persist side by side with the permanent ones, as was seen in my third case.

Nervous system.—The size of the head varies greatly, though, as a rule, it does not come far short of that of the average adult.

According to Quetelet, the circumference of the head of an adult male measures 564 mm. (22¼ inches). Martin Lane (No. 12) was 520 mm. (20½ inches), in Schaaffhausen's case (No. 5) the bare skull was of this same measurement, and in Manouvrier's (7) it reached to 530 mm. (20⅞ inches), but in my second case (female) it was so low as 445 mm. (17½ inches). Sir G. M. Humphry says that the circumference of the cranium of an adult male is 525 mm. (20¾ inches), and this is not much more than the 505 mm. (19⅞ inches) of the skull of Bobbie Fenwick. The variation in these measurements must largely depend upon the age at which the disease begins. Thus, in my third case, in which the disease seems to have commenced during the first year, the skull was smaller than it was in my second case, where it began in the second year. In the case of Caroline Crachami, in which the disease began during fœtal life, we have the smallest measurement of all (349 mm.). On the other hand, we notice that in Dr. Schmidt's case (No. 13), the skull of Theresa Fend had a circumference of only 505 mm. (19⅞ inches), which was the same as that of the bare skull of Bobbie Fenwick, though ateleiosis did not begin until the age of eight years. These variations are probably such as occur under normal circumstances in the sizes of different skulls.

[1] 'A Treatise on Oral Deformities,' p. 8.

It is not easy to say whether the intelligence of these dwarfs is greater than usual, but there can be no doubt whatever that it is, as a rule, in no way defective. They are very quick in comprehension, have good memories, and usually appear to have but little difficulty in learning foreign languages. Some have, undoubtedly, possessed mental abilities above the average.

Thus, Boruwlaski wrote an autobiography which was excellent, both in composition and style. Jacob Lehman was regarded in his time as a great artist, and Geoffrey Hudson was thought to be possessed of sufficient ability to be employed as a confidential messenger to the French Court by Charles I. The intelligence, however, is not always good, for Martin Lane (No. 12) was certainly somewhat deficient in this respect.

Sexual system.—The sexual organs are markedly undeveloped. This is true of every case in which these organs are mentioned. Indeed, the genitals are decidedly more backward than the rest of the body. This I have noted in my second case, and in Schaaffhausen's case (No. 4) there was cryptorchism of both sides. On the other hand, we have it on excellent authority that Boruwlaski married and became the father of three children. He writes with so much candour and simplicity that in reading his autobiography it is not easy to doubt his statements on this subject. In one of his portraits he is represented with his wife and one child. Yet this, and other portraits of him, one of which is in the Hunterian Museum, corroborate the impression which is conveyed by his memoir that he was undoubtedly an ateleiotic dwarf. "Tom Thumb," again, married a dwarf of the same kind as himself, who is said to have given birth to a child of average size who died in infancy. It is quite possible that there was some deception in this case. It may be observed, moreover, that the appearance and proportions of "Tom Thumb" and his wife, as shown in their photographs, are conspicuously infantile. We cannot, therefore, believe that it is possible for dwarfs of this

type to be virile until the evidence is stronger than it now is.

Condition of other organs.—There was some indication of disturbance of the *heart* in my third case. In the absence of other causes I attributed it to some congenital anomaly. Such an anomaly existed in Case No. 12, when a permanent but not patent ductus arteriosus was found after death. The mitral valves were crumpled and thickened.

The *thyroid* gland in Paltauf's case (No. 6) was small and pale red in colour. In my second case it seemed to be small, and at one time there appeared to be evidences of its defective action. In my third case, and again in Case No. 12, the gland was undoubtedly of good development.

The *pituitary* body was enlarged in Paltauf's case (No. 6), the sella turcica of Mikolajek being bigger even than that of the normal adult. Unfortunately Paltauf says nothing of the condition of the pituitary body itself. Still more unfortunately I myself failed to obtain this organ for microscopical examination at the necropsy (Case 12), though I noticed it appeared to be quite natural. In both of the skeletons I have examined (Cases No. 1 and 5) there was no disproportion in the size of the sella turcica. Schaaffhausen does not mention the subject. It is therefore probable that there was no conspicuous abnormality of the pituitary body in his case (No. 6). Dr. Byrom Bramwell found evidences of defective action of the pancreas in the case of infantilism which he examined. There is no evidence of disorder of the thymus, spleen, lymphatic glands, bone marrow, or of the suprarenal bodies.

Rate of growth.—Strictly speaking, it is not true that ateleiotic dwarfs are stereotyped children, even if we regard them from the physicial aspect alone, for growth changes take place which are of the same nature as those which occur in ordinary individuals, though they are much less in degree, and extend over a much longer period. Nevertheless, it seems certain that a time arrives after which

they grow no further, though this may vary in different cases. Thus it is explicitly stated of Boruwlaski that he ceased to grow at the age of thirty; while Geoffrey Hudson is said to have remained of the height of 18 inches (45·8 cm.) from the age of eight until the age of thirty, after which period he increased to 3 feet 9 inches (114·5 cm.), and then grew no further. No reliance can be placed upon the accuracy of these figures, but of the general circumstance of the cessation of growth before the attainment of average stature there seems to be no question. It is true that Joachimsthal shows that in one of his cases (No. 8), in which the age was thirty-six years, there had been noticeable increase in height since the age of thirty-three, yet no one has reported any case in which growth has continued after the age of forty; and the skeleton of Bobbie Fenwick shows that growth of the skeleton had practically ceased at about the age of fifty.

Duration of life.—"Tom Thumb" was said to be fifty-three when he died, and his companion, "Commodore" Nutt, forty-one; Paltauf's case lived to forty-nine years. There seems to be good evidence that ateleiotic dwarfs may live to a ripe old age. Thus Geoffrey Hudson is said to have lived to the age of sixty-two, while it is stated that Boruwlaski did not die until he had reached the age of ninety-eight. He was born in 1739, and was buried near to Stephen Kemble, in Durham Cathedral, in 1837.

Diagnosis.

The only diseases with which ateleiosis is likely to be confounded are those which retard growth and development. The chief of these are cretinism and myxœdema, syphilis, mongolianism, mitral disease, achondroplasia, rickets, microcephaly, and normal infantilism.

Cretinism, myxœdema, mongolianism, and *microcephaly* are distinguished by the fact that they affect the intelligence. It is, of course, quite possible for an ateleiotic dwarf to be an imbecile, but in that event the lack of in-

telligence is not part of the disease, but is one of those accidental accompaniments which may be met with in association with any disease. The only exception to this is to be found in those instances of ateleiosis which commence during intra-uterine life, and of which one example has been given (Case No. 1). But in that case the imbecility was the natural outcome of the very early period at which the disease began. The head was not disproportionately small, but was, on the contrary, a little too large for the body.

Cretinism and myxœdema, when well marked in the living subject, show characters which are quite unmistakable, but it is possible that the skeleton of a cretin may be very difficult to distinguish from the skeleton of an ateleiotic dwarf. There may be delay of development in both cases, and it seems that in cretinism this delay may be quite as conspicuous as it is in ateleiosis; but, as a rule, the skeleton of the cretin is not only immature, but is deformed, especially in the bones of the lower extremities. The long bones are thick, "the pelvis may be narrow as in rickets." Microscopically "Grawitz found in a typical case that all signs of columnar formation of the cartilage cells were absent."[1] Difficulties would arise of a still more puzzling nature should cretinism or myxœdema be associated with ateleiosis. This is by no means unlikely, for in one of my cases there were myxœdematous symptoms, and cases are sometimes reported as cretinism in which the intelligence is good. In these mixed cases it is possible that nothing but the history would be of any avail in coming to a decision as to the diagnosis, and it is also possible that the two diseases may be so intermingled that no one could say which has the priority.

Rickets and *achondroplasia* produce so much deformity that it is not likely that either of them can be mistaken for ateleiosis. The proportionate development of ateleiotic dwarfs, their well-formed figures, good intelligence, and

[1] 'Diseases of the Thyroid Gland,' Dr. Murray, p. 99.

conspicuous immaturity, cause them to stand apart from all diseases, which by affecting one part of the skeleton more than another part produce manifest disproportion in size.

Syphilis. — Mr. Hutchinson[1] says that "in certain cases arrest of growth occurs as a consequence of the inherited taint, and the patient remains a dwarf." "In most of these cases it would appear that there is arrest of sexual development also, but this is not invariable." Professor Fournier has paid a good deal of attention to this manifestation of congenital syphilis. On turning to the account of his observations[2] it is evident that the infantilism which is produced by syphilis is not so conspicuous as that which we meet with in ateleiosis. Professor Fournier gives measurements of six cases, and the shortest of them (1·33 m.) was, at the age of eighteen, much taller than the tallest of those who are in my second class. Moreover, development continues in these syphilitic cases until in course of time it is completed. They are not ateleiotic, because they do ultimately reach maturity. Menstruation or virility may be delayed "until the seventeenth, eighteenth, or nineteenth years, or even later." There appears to be some growth of sexual hair on the face or body. Lastly, the infantilism of syphilis seems to produce no special type of face, unless it be the type which is produced by the syphilis itself. Not one of the cases of ateleiosis I have seen has shown the usual evidences of the syphilitic taint.

Infantilism.—The word infantilism has been much used by French writers to signify arrested or retarded development. Though it is not a happy designation when applied to development which has become arrested during late childhood or youth, yet it is very convenient. Some such word is as much needed to express defective development, as the word dwarfism is needed to express defective growth. But it is unfortunate that some writers use the word as if it denoted a disease. Infantilism is not a disease, but a con-

[1] 'Twentieth Century System of Medicine,' vol. xviii, p. 390.

[2] 'Le Syph. Hered. Tardus,' 1886, p. 26.

dition or symptom. Thus Brissaud,[1] who says the word was introduced by Laségue and Brouardel, applies it to a condition which resulted from myxœdema. Lacomme[2] uses it for that form of immaturity which sometimes accompanies congenital heart disease, and is generally believed to be produced by defective nutrition. Lacomme is, however, of the opinion that the relation is not causal, and Giraudeau[3] and Ferrannini,[4] in describing other cases, express the same opinion. It has also been shown that infantilism may be an occasional feature of achondroplasia, and hydrocephalus. It may sometimes result from imperfect development of the sexual organs. It is possible that some of these cases are cases of ateleiosis combined with congenital heart disease, cretinism, or other disorder. But unless the facial, sexual, and osseous features of ateleiosis are well marked, such cases must at present be put on one side and not included among those of ateleiosis.

There is one other condition to which the word infantilism is appropriate, and that is a condition which may be termed normal infantilism. We recognise that growth varies greatly, and that it sometimes becomes excessive without being morbid. Just in the same way as there are normal giants, so there may be normal dwarfs, and in some of these dwarfs there is not only delay or arrest of growth but also of development. These constitute instances of normal infantilism. I am indebted to Mrs. Keith and Miss Keith for permission to examine one of the most striking cases of this condition which I have yet seen.

This case was that of a girl of the age of fifteen years and a half, whose stature was no greater than that of a girl of six, whose ossification and dentition corresponded to that of a girl of eleven, and whose sexual development was not more advanced than that which is usual at twelve. Though there appeared to be no cause for this condition it could be

[1] 'Leçons sur des Mal. Nerv.,' p. 606, Paris, 1895.
[2] 'Loire Méd.,' March 15th, 1899, p. 63.
[3] 'Arch. Général de Méd.,' tome viii, p. 547.
[4] 'Riforma Medica,' December 7th, 1800, pp. 162, 375, 687.

Normal Infantilism. Girl of 15½ (on the right) compared with normal girl of 5½. In ordinary Infantilism there is no peculiar type of face, while the delay of development is not so abrupt or conspicuous as it is in that special form of Infantilism which is termed Ateleiosis. Note that in this case though the stature is about ten years behindhand, the carriage, facial expression, proportions of the body, and development of the pelvis and sexual organs are not far short of those which are usual in girls of her age. See page

Bale & Danielsson, Ltd., London.

distinguished from the cases of ateleiosis which have been recorded in the following particulars:—There was no special type of face; the girl resembled her mother in appearance. The proportions of the body were adult rather than childish, the middle point being at the pubic symphysis, while the extremities were comparatively long. The intelligence was fully equal to that of most girls of her age. Lastly, though the condition had been first noticed when she was between two and three years old, the delay of development was not very conspicuous, and, above all, the pelvis and sexual organs were by no means infantile.

Pathology.

There can be no doubt that the most conspicuous feature of the condition, of which these cases are examples, is the delay of growth and development.

Now, the question we have to ask ourselves is, does this delay affect the whole body simultaneously, or does it, like cretinism, originate in one organ, and from thence produce a secondary effect upon the rest of the body?

At first it seems much more natural to look upon it as a primary affection of the body as a whole. In fact, this view seems to be taken by most of those who write upon the subject of these dwarfs, though it is true that Schaaffhausen is the only author who expressly states this opinion. But on examining the cases a little closer it soon becomes evident that we have to deal not with a physiological variation, but with a disorder. The process is not altogether uniform, for some parts are more affected than others; there is far greater variation than ever exists in health. Moreover, the hindrance to development is, as has already been shown, far too abrupt and pronounced to constitute a normal infantilism.

Having settled that ateleiosis is a disease, we have next to find out which organ or part is responsible for its appearance. The organs which we regard with most suspicion are the sexual organs, the thyroid gland, the

pituitary body, and the skeleton. It is also possible that the pancreas, the heart, or some other organ may play a part in the production of the malady, but inasmuch as their disorder is only occasional, and is certainly not a common feature, we can dismiss them. They may be causes of infantilism, but cannot be causes of ateleiosis.

Now, of all the organs which have been mentioned, the sexual organs seem to be most worthy of our attention. They were markedly backward in development in all the cases which have been reported. Moreover, we know that arrest of development of these organs does have an effect on growth. Those who are so circumstanced are sometimes of small stature, and of poor development generally. Indeed, some authorities [1] recognise an infantile type as the result of sexual ill-development. Further, those who are sexually precocious are nearly always also of premature development in other respects. Thus, I have now under observation a girl who began to menstruate when two and a half years old. At four her height and weight were equal to those of a girl of double her age, and on taking a radiogram of her hand it was found that ossification was also equal to that of a girl of eight. The sexual development was like that of a girl who was commencing puberty. If premature sexual development can give rise to so great acceleration of growth, it seems not improbable that the opposite condition of sexual immaturity may be capable of producing as striking an effect in hindering growth.

On the other hand, we know that the infantilism which sometimes results from sexual ill-development is but slight in degree, and that such a condition as ateleiosis has never been known to result from removal of the sexual organs of children. The fact that ateleiosis may occur in two or three members of the same family makes it exceedingly improbable that the disease may be a very rare and exceptional result of this malformation. Such a coinci-

[1] Dr. F. J. McCann, 'Amer. Journ. Med. Sci.,' vol. cxii, No. 4, October, 1896, p. 392.

dence would be almost too extraordinary to be possible. Moreover instances have been given of the procreation of children by these dwarfs. I myself have seen two of these sexually mature cases.

Mr. Hutchinson is of the opinion that the cause of this form of dwarfism will probably be found in some disorder of the pituitary body. We know that gigantism sometimes results from disease of this organ, and it is not improbable that some other affection of the same part, or a similar affection occurring in early life, might give rise to a disorder of an opposite nature. In support of this view is the fact that both acromegaly and ateleiosis are associated with imperfections of the sexual apparatus. Moreover, in Paltauf's case (No. 6), disease of the pituitary body was actually present, though we know nothing of its nature. The only indication of its presence was the very large size of the sella turcica. On the other hand, in the four other cases of ateleiosis, Nos. 1, 5, 6, and 12, which have been examined after death, no enlargement of the sella turcica or of the pituitary body was noticed. The sella turcica was certainly not abnormal in the three *post-mortem* cases which I have described.

Paltauf does not so much as mention the possibility of ateleiosis being due to some general anomaly of growth, but apparently takes it for granted that it must necessarily be, in the first place, a disease of the bones only. It is his opinion that the disease is confined to those bones which are of cartilaginous origin, and that it does not affect the membrane bones. In support of this view, he alludes to a case of Professor His', in which the bones of the skull continued to grow while the brain did not. This case, however, was one of cretinism, not of ateleiosis. But in infantile life ossification is naturally much more advanced in membrane than in cartilage bones, and the apparent difference to which Paltauf alludes must surely be due to this cause, and not to the exercise of any selection by the disease. In the same way we can explain the "cretinoid" type of face, which is also referred to by Paltauf.

The following considerations are in favour of Paltauf's view that the affection is primarily one of the skeleton. When the growth of the skeleton is prematurely stopped, as in rickets and achondroplasia, the muscles and other soft parts do not continue to grow as if the skeleton were normal, but, on the contrary, the growth of the soft tissues becomes adapted to that of the underlying bones. May not the disease with which we are now dealing be due to delay not of growth only, but of development as well, followed by a similar adaptation of the development of the soft parts? In other words, is ateleiosis a primary hypoplasia of the skeleton? And does the general immaturity merely indicate a physiological effort on the part of the soft structures to keep pace with this disorder of the skeleton? A great objection to this view is the fact that, if we may judge from the state of the epiphyses, the development of the bones is not less than, but is in advance of that of the soft parts; whereas were the disease primarily of the bones, to which the soft parts have accommodated themselves, we should expect the reverse. Moreover, there is evidence that the anomaly of the sexual organs may precede that of the skeleton by some years.

It is not yet possible to come to any conclusion as to the causation and nature of ateleiosis. The disease must, at present, be regarded as a form of infantilism. Yet it cannot be considered a sexual infantilism, that is, an infantilism produced by imperfect development of the sexual organs; for it seems clear that the sexual defect when it exists is not the cause of the general delay of development, but is merely its precursor. Moreover that form of infantilism which is known to be produced by maldevelopment of the sexual organs is quite distinct from ateleiosis.

Now infantilism is a condition which is associated with many abnormalities. Thus, in Mr. Hutchinson's doubtful case of ateleiosis, the infantilism was combined with hyperplasia of the gums. Similarly, in Dr. Thomson's case, thymic hypertrophy was present; while, in Paltauf's doubtful case,

there was osteomalacia. Dr. Byrom Bramwell has exhibited a patient in whom the infantilism was attributed to defective action of the pancreas, and Dr. Bryant has shown me the records of a case in which infantilism coexisted with pseudo-hypertrophic palsy. In Bobbie Fenwick's skeleton there was some curious anomaly of the mandible, and in Martin Lane the same tendency to anomalous manifestations of development showed itself in the presence of a persistent though not patent ductus arteriosus. Lastly, in the case of Mikalojek there was enlargement of the pituitary body. It seems not unlikely that in some instances in which infantilism is attributed to congenital heart disease or imperfect action of the thyroid gland, the relation between these conditions is not in reality one of cause and effect, but is due to some such similar association. There is, apparently, some common factor, with the nature of which we are as yet unacquainted. All that can be said is that in ateleiosis there is defective development of the body as a whole, that this is often preceded by hypoplasia of the sexual organs, and is prone to be associated with some growth anomaly of other parts.

Caroline Crachami. From a painting in the Museum of the Royal College of Surgeons of England. The ateleiosis began in fetal life. The portraits show the prominent nose and receding chin, which are characteristic of microcephalism.

INDEX

The Indices to the annual volumes are made on the same principle as, and are in continuation of, the General Index to the first fifty-three volumes of the 'Transactions.' They are inserted in the Library copy, where the entire Index to the current date may always be consulted.

dition or symptom. Thus Brissaud,[1] who says the word was introduced by Laségue and Brouardel, applies it to a condition which resulted from myxœdema. Lacomme[2] uses it for that form of immaturity which sometimes accompanies congenital heart disease, and is generally believed to be produced by defective nutrition. Lacomme is, however, of the opinion that the relation is not causal, and Giraudeau[3] and Ferrannini,[4] in describing other cases, express the same opinion. It has also been shown that infantilism may be an occasional feature of achondroplasia, and hydrocephalus. It may sometimes result from imperfect development of the sexual organs. It is possible that some of these cases are cases of ateleiosis combined with congenital heart disease, cretinism, or other disorder. But unless the facial, sexual, and osseous features of ateleiosis are well marked, such cases must at present be put on one side and not included among those of ateleiosis.

There is one other condition to which the word infantilism is appropriate, and that is a condition which may be termed normal infantilism. We recognise that growth varies greatly, and that it sometimes becomes excessive without being morbid. Just in the same way as there are normal giants, so there may be normal dwarfs, and in some of these dwarfs there is not only delay or arrest of growth but also of development. These constitute instances of normal infantilism. I am indebted to Mrs. Keith and Miss Keith for permission to examine one of the most striking cases of this condition which I have yet seen.

This case was that of a girl of the age of fifteen years and a half, whose stature was no greater than that of a girl of six, whose ossification and dentition corresponded to that of a girl of eleven, and whose sexual development was not more advanced than that which is usual at twelve. Though there appeared to be no cause for this condition it could be

[1] 'Leçons sur des Mal. Nerv.,' p. 606, Paris, 1895.
[2] 'Loire Méd.,' March 15th, 1899, p. 63.
[3] 'Arch. Général de Méd.,' tome viii, p. 547.
[4] 'Riforma Medica,' December 7th, 1800, pp. 162, 375, 687.

PRINTED BY ADLARD AND SON, BARTHOLOMEW CLOSE.

INDEX

The Indices to the annual volumes are made on the same principle as, and are in continuation of, the General Index to the first fifty-three volumes of the 'Transactions.' They are inserted in the Library copy, where the entire Index to the current date may always be consulted.