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1805-1905

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(*Died 6th Nov.*, 1893, *and Sir. W. S. Church, Senior* [*Medical*] *Vice-President, acted as President until 1st March*, 1894.)
1894 JONATHAN HUTCHINSON, F.R.S.
1896 WILLIAM HOWSHIP DICKINSON, M.D.
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1900 FREDERICK WILLIAM PAVY, M.D., LL.D., F.R.S.
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1905 H.R.H. THE PRINCE OF WALES, K.G.

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.

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

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.

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

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.

1905 BARNES, ROBERT, M.D., F.R.C.P., Bernersmede, Eastbourne.

1905 GAIRDNER, SIR WILLIAM, K.C.B., M.D., F.R.S., LL.D., 32, George Square, Edinburgh.

1905 GASKELL, WALTER HOLBROOK, M.D., F.R.S., The Uplands, Great Shelford, Cambridge.

1905 LISTER, The Right Hon. BARON, O.M., F.R.S., D.C.L., LL.D., D.Sc., F.R.C.S., 12, Park Crescent, Portland Place.

1905 RAMSAY, SIR WILLIAM, K.C.B., F.R.S., LL.D., D.Sc., F.C.S., 19, Chester Terrace, Regent's Park.

1905 RAYLEIGH, The Right Hon. BARON, O.M., F.R.S., D.C.L., LL.D., Sc.D., Terling Place, Witham, Essex.

1905 WILKS, SIR SAMUEL, Bart., M.D., F.R.S., LL.D., F.R.C.P., 8, Prince Arthur Road, Hampstead.

FOREIGN HONORARY FELLOWS

Elected

1868 KÖLLIKER, ALBERT, Würzburg.

1878 BACCELLI, GUIDO, M.D., Rome.

1887 BILLINGS, JOHN S., M.D., D.C.L.Oxon., New York.

1887 VON ESMARCH, His Excellency FRIEDRICH, M.D., Kiel.

1896 VON BERGMANN, ERNST, Berlin.

1896 ERB, WILHELM, M.D., Professor of Clinical Medicine, Heidelberg.

1896 FOURNIER, ALFRED, M.D., Paris.

1896 KOCH, ROBERT, M.D., Berlin.

1896 KOCHER, THEODORE, M.D., Berne.

1896 LAVERAN, A., M.D., Paris.

1896 MARIE, PIERRE, M.D., Paris.

1896 MITCHELL, SILAS WEIR, M.D., Philadelphia.

1905 BOHR, CHRISTIAN, Copenhagen.

1905 KITASATO, Tokio.

1905 MARCHIAFAVA, ETTORE, Rome.

1905 NOTHNAGEL, H., Vienna [deceased, 1905].

1905 PAVLOFF, I. P., St. Petersburg.

1905 RAMON Y CAJAL, S., Madrid.

1905 WELCH, WILLIAM HENRY, Baltimore.

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OF THE

ROYAL MEDICAL AND CHIRURGICAL SOCIETY

OF LONDON

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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.

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 and underlined 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*.

[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 Secretary before the 1st of July in each year.]

RESIDENT FELLOWS

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.

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.

1865 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. *Referee*, 1905—.

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.

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.

1904 ARKWRIGHT, JOSEPH ARTHUR, M.D., The Garth, Kenley; and 13, Welbeck street, Cavendish square.

1902 ARMOUR, DONALD JOHN, M.B., Assistant Surgeon to the West London Hospital; Senior Assistant Surgeon to the Belgrave Hospital for Children; 89, Harley street.

1903 ASCHERSON, WILLIAM LAWRENCE, M.B., B.C., 37, Brunswick gardens, Campden hill.

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

Elected

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.

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. *Referee*, 1904—. *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.

1902 BARNARD, HAROLD L., M.S., 21, Wimpole street.

1893 BARRETT, HOWARD, 49, Gordon square.

1880 BARROW, A. BOYCE, Surgeon to King's College Hospital; 8, Upper Wimpole street, Cavendish square. C. 1903—4.

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

1904 BARWELL, HAROLD SHUTTLEWORTH, M.B., 55, Wimpole street.

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.

Elected

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. V.P. 1904-5. *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.

1905 BATTLE, WILLIAM HENRY, 49, Harley street, Cavendish square.

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., late Professor of the Principles and Practice of Medicine in King's College, London, and Consulting Physician to King's College Hospital; 6, Bentinck street, Manchester square. C. 1876-7. *Referee*, 1873-5. *Trans.* 1.

1897 BEDDARD, A. P., M.D., 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.

Elected

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.

1897 BLACKER, G. F., M.D., Obstetric Physician to University College Hospital and to the Great Northern Central Hospital; 45, Wimpole street, Cavendish square.

1901 BLAIKIE, J. BRUNTON, M.D., C.M., 80, Brook 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.

1902 BLUMFELD, JOSEPH, M.D., B.C., 7, Cavendish place, Cavendish square.

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

1903 BOLTON, CHARLES, M.D., 16, Devonshire street, W.

1905 BOSANQUET, WILLIAM CECIL, M.A., M.D., 117A, Harley street.

1882 **Bowlby,** ANTHONY ALFRED, C.M.G., Surgeon to St. Bartholomew's Hospital; 24, Manchester square. C. 1903—4. *Trans.* 8.

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

Elected

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. C. 1904—. *Referee*, 1895—1904. *Trans.* 1.

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

1904 BRANSON, WILLIAM PHILIP SUTCLIFFE, M.D., 59, Gordon square. *Trans.* 1.

1901 BREWERTON, ELMORE WRIGHT, 84, Wimpole street.

1904 BRIMACOMBE, RICHARD WILLIAM, M.D., Colebrook Lodge, Putney Heath.

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. V.P. 1905—. *Referee*, 1881-4, 1891-7. *Trans.* 5.

1905 BROWN, WILLIAM CARNEGIE, M.D., 32, Harley street, and 13, Hampstead hill gardens.

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.

Elected

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

1871 BRUNTON, SIR THOMAS LAUDER, M.D., D.Sc. LL.D., F.R.S., Consulting Physician to, and late 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.

1898 BRYANT, J. H., M.D., Assistant Physician to Guy's Hospital; 8, Mansfield street, Portland place.

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; 42, Norfolk 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.; 16, Devonshire street, Portland place. *Trans.* 1.

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.

1903 BURROWS, HAROLD, M.B., B.S., Caragh, Lyford road, Wandsworth common.

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. V.P. 1904—. *Referee*, 1893—1904. *Trans.* 4. *Pro.* 1.

Elected

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.D., 33, Harley street, Cavendish square.

1868 Buzzard, THOMAS, M.D., Physician to the National Hospital for the Paralysed and Epileptic; 74, Grosvenor street, Grosvenor square. C. 1885-6. V.P. 1905. *Referee*, 1887-1905.

1885 CAHILL, JOHN, M.D., 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.

1905 CAMMIDGE, PERCY JOHN, M.B., D.P.H., 2, Beaumont street, Portland place. *Trans.* 1.

1897 CANTLIE, JAMES, M.B., 140, Harley street.

1901 CARGILL, LIONEL VERNON, Assistant Ophthalmic Surgeon to King's College Hospital; Ophthalmic Surgeon to the Seamen's Hospital Society; Surgeon to the Royal Eye Hospital, Southwark; 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.

Elected

1903 CARRUTHERS, SAMUEL WILLIAM, M.D., C.M., 44, Central hill, Norwood.

1888 CAUTLEY, EDMUND, M.D., B.C., 15, Upper Brook street. *Trans.* 2.

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; 120, Queen's road, Richmond. C. 1888. *Referee,* 1886-7, 1899-1903. *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.

1868 **Cheadle,** WALTER BUTLER, M.D., 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. *Trustee* 1888-1903. *Marshall Hall Trustee* 1873-1903. S. 1886-8. C. 1890-91. V.P. 1904-5. *Sci. Com.* 1889-95. *Bldg. Com.* 1889-92. *Referee,* 1885. *Trans.* 1.

1879 CHEYNE, WILLIAM WATSON, C.B., 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, 1904—. *Lib. Com.* 1886-8, 1891-6. *Trans.* 1.

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.

Elected

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., Surgeon to the North-West London and City of London Orthopædic Hospitals; 18, Portland place.

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

1888 CLARKE, ROBERT HENRY, M.B., 9, St. James's road, Surbiton.

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. *Lib. Com.* 1905—. *Trans.* 1.

1905 CLOGG, HERBERT SHERWELL, M.B., M.S., 117A, Harley street, Cavendish square.

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.

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

Elected

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.

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. C. 1903-4. *Trans.* 3.

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

1905 CROSS, WILLIAM FOSTER, 80, New Cavendish street.

1888 CULLINGWORTH, CHARLES JAMES, M.D., D.C.L., LL.D., Consulting Obstetric Physician 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; 11, Park crescent, Portland place. *Trans.* 1.

Elected

1898 CURRIE, A. STARK, M.D., 20, Oxford terrace, Hyde 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. C. 1904-5. *Lib. Com.* 1902-4.

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.

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.A., M.B., B.Ch., 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, Surgeon to, and Lecturer on Surgery at, St. George's Hospital; 61, Brook street. C. 1890, 1905—. S. 1901-4. *Bldg. Com.* 1890-2. *Cent. Com.* 1904-5. *Referee*, 1892—1901. *Trans.* 7.

1891 DE SANTI, PHILIP ROBERT WILLIAM, Assistant Surgeon and Aural Surgeon to the Westminster Hospital; 15, Stratford place. *Trans.* 1.

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; 10, Stanhope place, Marble arch. P. 1896-8. C. 1874-5. V. P. 1887. *Referee*, 1869-73, 1882-6. *Sci. Com.* 1867, 1879, 1889-96. *Trans.* 16.

Elected

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.

1888 DONELAN, JAMES, M.B., M.C., Chevalier, Crown of Italy, 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.* 4.

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.D., 11, Devonshire place. *Trans.* 1.

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. 1902-4. *Referee*, 1885-97. *Trans.* 2.

1903 DUDGEON, LEONARD, 6, Powis gardens, Bayswater.

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., Consulting Surgeon to the North-West London Hospital; 52, Brook street, Grosvenor square.

1905 EASON, HERBERT LIGHTFOOT, M.D., M.S., The College, Guy's Hospital.

Elected

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

1893 ECCLES, WILLIAM MCADAM, M.S., Assistant Surgeon, St. Bartholomew's Hospital, to the West London Hospital, and to the City of London Truss Society; 124, Harley street.

1891 EDDOWES, ALFRED, M.D., 28, Wimpole 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 ENGLISH, THOMAS CRISP, B.S., Assistant Surgeon to St. George's Hospital and to the Grosvenor Hospital for Women and Children; 31, Grosvenor street, Grosvenor square. *Trans.* 1.

1902 EVANS, ARTHUR, M.S., Assistant Surgeon to, and Lecturer on Surgical Anatomy at, Westminster Hospital, Surgeon to the Seamen's Hospital, Royal Albert Dock; 84, Harley street.

1904 EVANS, JOHN HOWELL, 63, Grosvenor street.

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; 61, 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., Assistant Obstetric Physician to St. Thomas's Hospital; 60, Wimpole street.

Elected

1905 FAWCETT, JOHN, M.D., 66, Wimpole street.

1905 FAULDER, THOMAS JEFFERSON, 50, Welbeck street, Cavendish square.

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.

1900 FLEMMING, PERCY, M.D., B.S., Professor of Ophthalmic Medicine and Surgery in University College, London; 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 to St. Bartholomew's Hospital, Physician to the 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 and Rhynie, Haywards Heath, Sussex. *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. 1902-3. *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. C. 1904-5. *Trans.* 1.

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

Elected

1902 FRENCH, HERBERT, M.D., 26, St. Thomas's street. *Trans.* 1.

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

1898 FRIPP, SIR ALFRED DOWNING, C.B., M.V.O., M.S., Honorary Surgeon-in-Ordinary to H.M. the King; Kt. of Grace of St. John; 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.

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., Physician to the German Hospital, 39, 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.

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.

Elected

1886 GARROD, ARCHIBALD EDWARD, M.D., Assistant Physician to St. Bartholomew's Hospital; Physician to the Hospital for Sick Children, Great Ormond street; 9, Chandos street, Cavendish square. C. 1902-4. *Sci. Com.* 1889-1902. *Lib. Com.* 1896-1902, 1904—. *Trans.* 8.

1905 GASK, GEORGE ERNEST, 84, Wimpole street.

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—. *Cent. Com.* 1905. *Trans.* 11.

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

Elected

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. *Trans.* 1.

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. C. 1903–4. *Referee*, 1900-3. *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. V.P. 1904—. *Referee*, 1895-8. *Ho. Com.* 1892-8, 1904—. *Lib. Com.* 1891. *Cent. Com.* 1904-5. *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—.

1903 GRÜNBAUM, OTTO F. F., M.B., B.C., 30A, Wimpole street, Cavendish square.

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. C. 1903-4.

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 the Westminster Hospital; 47, Wimpole street, Cavendish square. C. 1901-3. *Referee*, 1893-7.

Elected

1891 HAMER, WILLIAM HEATON, M.D., 55, 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.

1905 HANDLEY, WILLIAM SAMPSON, 51, Devonshire street.

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

1901 HARMER, WILLIAM DOUGLAS, M.B., The Warden's House, St. Bartholomew's Hospital.

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

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

1904 HARRISON, CHARLES JOSEPH, M.D., B.S., 6, Swan Walk, Chelsea.

1901 HARTIGAN, T. J. P., 94, Harley street.

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.

Elected

1905 HEAD, HENRY, M.D., 143, Harley street, Cavendish square.

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.

1877 HERMAN, GEORGE ERNEST, M.B., Consulting Obstetric Physician to 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. C. 1904—. *Ho. Com.* 1904—.

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. C. 1903—4. *Lib. Com.* 1902-3 1904—. *Ho. Com.* 1903—4. *Trans.* 3.

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.

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

1904 HOBSON, H. OVERTON, M.D., 32, Upper Berkeley street; and Helouan, Egypt.

Elected

1856 **Holmes,** TIMOTHY, M.C., 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. C. 1904—.

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-HARTLEY, 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.

1902 HULBERT, ERNEST BEDDOE, M.D., 77, Welbeck street, Cavendish square.

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. 1894-6. *Referee*, 1876-81, 1883-94. *Lib. Com.* 1864-5. *Trans.* 15. *Pro.* 2.

Elected

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 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 55, Queen Anne street.

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.

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; 143, Harley 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. 1901-3. *Ho. Com.* 1902-3.

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

Elected

1882 KEETLEY, CHARLES R. B., Senior Surgeon to the West London Hospital; 56, Grosvenor street, Grosvenor square. C. 1901-3. *Lib. Com.* 1903—. *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., 120, Harley street.

1902 KERR, JAMES, M.D., D.P.H., 3, Hanger lane, Ealing.

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. *Referee*, 1905—. *Trans.* 4.

1902 KING, DAVID BARTY, M.A., M.D., Ch.B., 13, Queen street, Mayfair.

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, Surgeon Laryngologist, Mount Vernon Hospital for Consumption; Surgeon, Royal Ear Hospital; 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. C. 1904–5. *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. C. 1904–5.

Elected

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.

1893 LAWSON, ARNOLD, Ophthalmic Surgeon to the Children's Hospital, Paddington Green; 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., 18, Cheyne row, Chelsea.

Elected

1895 LESLIE, ROBERT MURRAY, M.D., 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. *Trans.* 1.

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

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

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. 1901-3. *Trans.* 5.

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. *Referee*, 1903—. *Trans.* 1.

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

1898 LYSTER, C. R. C., 70, Wimpole street, Cavendish square.

Elected

1905 McCann, Frederick John, M.D., 5, Curzon street, Mayfair.

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*, 1890–1905.

1899 Macdonald, Greville, M.D., 85, Harley street.

1898 McFadyean, Sir John, The Royal Veterinary College, Camden Town.

1894 Macfadyen, Allan, M.D., C.M., Lister Institute of Preventive Medicine, Chelsea bridge. *Referee*, 1905—.

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.

1902 Mackenzie, Hector William Gavin, M.A., M.D., 34, Upper Brook street.

1881 Mackenzie, Sir 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.

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.

Elected

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.

1890 MANSON, SIR PATRICK, K.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. *Referee*, 1904—.

1867 MARSH, F. HOWARD, Professor of Surgery, University of Cambridge, late Surgeon to, and Lecturer on Surgery at, St. Bartholomew's Hospital; 14, Hertford street, Mayfair. 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. *Referee*, 1904—.

1892 MASTERS, JOHN ALFRED, M.D., 94, Knightsbridge.

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.* 3.

Elected

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

1873 **Moore**, NORMAN, M.D., *Hon. Librarian*, 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. *Cent. Com.* 1904–5.

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, Consulting Surgeon to the Skin Department of, and late 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.

1905 MUECKE, FRANCIS FREDERICK, M.B., B.S., 63, Wellington road, St. John's Wood.

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

Elected

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

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, Lecturer on Clinical Medicine, and Joint Lecturer on Medicine 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.

1904 NESS, THOMAS MATHESON, M.B., 80, Brook street.

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

1880 OGILVIE, GEORGE, M.B., B.Sc., Senior Physician to the French Hospital, and to the Hospital for Epilepsy and Paralysis, Maida Vale; 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.

1860 OGLE, WILLIAM, M.D., late Superintendent of Statistics in the Registrar-General's Department, Somerset House; 10, Gordon street, Gordon square. *Marshall Hall Trustee*, 1873-1903. S. 1868-70. C. 1876-7. V.P. 1887. *Lib. Com.* 1871-5. *Trans.* 5.

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

Elected

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.

1905 ORMOND, A. W., 37, Queen Anne street, Cavendish square.

1879 OWEN, EDMUND, M.B., Consulting Surgeon to St. Mary's Hospital; Consulting Surgeon to the Hospital for Sick Children, Great Ormond street; Surgeon-in-chief to the French Hospital; 64, Great Cumberland place, Hyde park. C. 1896-7. *Trans.* 4.

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, *Hon. Secretary*, Surgeon to the West London Hospital; Surgeon to the Throat and Ear Department of the Middlesex Hospital; 70, Harley street. S. 1904—. *Lib. Com.* 1902—4.

1905 PARDOE, JOHN GEORGE, M.B., 77, Wimpole street.

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

1904 PARKINSON, THOMAS WRIGHT, M.D., 77, Sloane street.

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.

Elected

1901 PATERSON, HERBERT JOHN, M.A., M.B., B.C., Assistant Surgeon to the London Temperance Hospital, 9, Upper Wimpole street.

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

1891 PATON, EDWARD PERCY, M.D., M.S., 53, Queen Anne street, Cavendish 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., 58, Harley street.

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, SIR EDWIN COOPER, M.D., Physician to 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.* 2.

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

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; 3, Upper Brook street, Grosvenor square. *Trans.* 1.

Elected

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.* 2.

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—.

1901 PLIMMER, HENRY GEORGE, 3, Hall road, St. John's Wood.

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.

1905 PORTER, HARRY EDWIN BRUCE, M.D., 6, Grosvenor street, Grosvenor square.

1867 POWELL, SIR RICHARD DOUGLAS, Bart., K.C.V.O., M.D. *President*, Physician Extraordinary to H.M. the King; Consulting Physician to, and late Lecturer on Medicine at, the Middlesex Hospital; Consulting Physician to the Hospital for Consumption, Brompton; 62, Wimpole street, Cavendish square. P. 1904—. S. (Oct.) 1883-5. C. 1887-8. V.P. 1902-4. *Referee*, 1879-83, 1886. *Trans.* 3.

Elected

1887 POWER, D'ARCY, M.A., M.B., Surgeon at St. Bartholomew's Hospital; Surgeon to the Victoria Hospital for Children, Chelsea; 10A, Chandos street, Cavendish Square. *Lib. Com.* 1896—. *Trans.* 3.

1905 PRICE, FREDERICK WILLIAM, M.B., 77, Wimpole street.

1900 PRICE-JONES, CECIL, M.B., Assistant to the Lecturer in Bacteriology, Guy's Hospital, Pathologist to the East London Hospital for Children, Shadwell; Guy's Hospital.

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, M.D., 4, Chesham street, Belgrave square. *Trans.* 1.

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

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. 1901-3. *Ho. Com.* 1902-3.

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. C. 1904—. *Cent. Com.* 1904–5.

Elected

1903 RICH, EVELYN ARTHUR, 12, Carlisle mansions, Carlisle place.

1887 RICHARDSON, GILBERT, M.A., M.D., 19, 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, Portland place. *Trans.* 1.

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.

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. *Trans.* 1.

Elected

1889 ROBSON, ARTHUR WILLIAM MAYO, Hunterian Professor of Surgery, Royal College of Surgeons; Consulting Surgeon, Leeds General Infirmary; Emeritus Professor of Surgery in the Yorkshire College; 8, Park crescent, London. *Trans.* 10. *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; Consulting Physician to the Samaritan Free Hospital for Women and Children; 14A, Manchester square. C. 1902-4. *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.

1900 RYALL, CHARLES, 51, Queen Anne street.

1903 RYAN, JOHN RUSSELL, B.A., M.D., B.Ch., B.A.O., 5, Bennett street, St. James's.

Elected

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.

1902 SAMBON, LOUIS W., M.D., London School of Tropical Medicine, Greenwich.

1899 SANDILANDS, JOHN EDWARD, M.B., 9, Launceston place, Kensington.

1903 SANDWITH, FLEMING MANT, M.D., Knight of Grace of the Order of St. John; Lecturer at the London School of Tropical Medicine; 31, Cavendish square. (April—Dec.) Cairo (Jan.—March).

1869 Sansom, ARTHUR ERNEST, M.D., Physician to the London Hospital; Consulting Physician, North-Eastern Hospital for Children; 83, Harley street, Cavendish square. C. 1887-8. *Referee*, 1889–1905. *Trans.* 3.

1902 SAUNDERS, EDWARD ARTHUR, M.A., M.B., B.Ch., D.Ph., Assistant Physician and Physician to the Department for the Medical Diseases of Children, West London Hospital; 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.

1905 SCHARLIEB, HERBERT J., C.M.G., M.D., B.S., 149, Harley street.

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.

1905 SCOTT, SYDNEY RICHARD, 44, Welbeck street, Cavendish square.

Elected

1863 **Sedgwick**, WILLIAM, 72, King Edward's gardens, Acton hill, Acton. 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, K.C.V.O., 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.

1900 SEQUEIRA, JAMES HARRY, M.D., Physician to the Skin Department and Lecturer on Dermatology at the London Hospital, 63, Harley 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. *Referee*, 1903—.

1884 SHEILD, ARTHUR MARMADUKE, M.B., B.C., Assistant Surgeon to St. George's Hospital; 4, Cavendish place. C. 1905—. *Referee*, 1897-1905. *Trans.* 6.

1903 SHERREN, JAMES, 40, Devonshire street.

1896 SHORE, THOMAS WILLIAM, M.D., Woodlawn, 6, Kingswood road, Upper Norwood.

1899 SHUTTLEWORTH, GEORGE EDWARD, M.D., Ancaster House, Richmond Hill, and 27, New Cavendish street.

Elected

1899 SIMPSON, WILLIAM JOHN RITCHIE, M.D., 13, Queen Anne street, Cavendish square.

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., 25, Welbeck 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., K.C.V.O., Honorary Sergeant-Surgeon to H.M. the King; Consulting Surgeon to St. Bartholomew's Hospital; 5, Stratford place, Oxford street. *Marshall Hall Trustee*, 1877—1903. S. 1870-2. C. 1875-6. V.P. 1887-8. *Referee*, 1873-4, 1880-6. *Sci. Com.* 1867. *Trans.* 4.

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.

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—.

Elected

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. *Referee*, 1905—. *Lib. Com.* 1904—.

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., Ophthalmic Surgeon to St. Bartholomew's Hospital; Surgeon to the Royal London Ophthalmic Hospital (City road, late Moorfields); Consulting Ophthalmic Surgeon to the Metropolitan Hospital; 5, Wimpole street, Cavendish square.

1903 SPRIGGS, EDMUND IVENS, M.D., 48 Bryanston street, Portman square.

1885 SQUIRE, JOHN EDWARD, C.B., M.D., Physician to the Mount Vernon Hospital for Consumption; 5, 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, C.M.G., 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. C. 1904—.

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., 3, Chester place, Hyde Park square.

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. *Referee*, 1904—.

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., Physician to, and Lecturer on Medicine at, Guy's Hospital; Consulting Physician to the Evelina Hospital for Sick Children; 20, Wimpole street, Cavendish square. *Trustee*, 1893-1903. 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.

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

1904 THOMPSON, HENRY EDMUND SYMES, M.D., Assistant Physician Royal Hospital for Diseases of the Chest; 33, Cavendish square. *Trans.* 1.

1905 THOMPSON, RALPH, Superintendent's Office, Guy's Hospital.

1862 Thompson, REGINALD EDWARD, M.D., 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., Assistant Physician to the Middlesex Hospital and Physician to the Hospital for Epilepsy and Paralysis, Maida Vale; 34, Queen Anne street. *Trans.* 3.

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.

1892 THORNE, WILLIAM BEZLY, M.D., 2, Harley street.

1899 THURSFIELD, JAMES HUGH, M.D., 84, Wimpole street. *Trans.* 2.

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. C. 1905—. *Referee*, 1902-5. *Sci. Com.* 1896-1902.

Elected

1879 **Treves**, SIR FREDERICK, Bart., G.C.V.O., C.B., LL.D., Sergeant-Surgeon in Ordinary to H.M. the King; Surgeon in Ordinary to H.R.H. the Prince of Wales; Kt. of Grace of St. John; Consulting Surgeon to the London Hospital; 6, Wimpole street, Cavendish square. C. 1895-6. *Referee*, 1890-95. *Sci. Com.* 1889-95. *Trans.* 7.

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

1897 TUNNICLIFFE, FRANCIS WHITTAKER, M.D., 129, Harley street.

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; 41, Half Moon street, Piccadilly. C. 1903—4. *Trans.* 1.

1898 TURNER, WILLIAM, M.B., M.S., Assistant Surgeon, Westminster Hospital; 17, Harley 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; 18, Harley street. *Trans.* 3.

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.

1876 VENN, ALBERT JOHN, M.D., 3, Hanover court, Hanover square.

Elected

1870 VENNING, SIR EDGCOMBE, 30, Cadogan place. C. 1898-1900. V.P. 1902—4. *Ho. Com.* 1903—4.

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 Patients, Throat and Ear Department, Great Northern Central Hospital; 45, Upper Brook street.

1884 WAKLEY, THOMAS, jun., 16, Hyde Park Gate. C. 1905—.

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

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

1900 WALKER, JOHN WILLIAM THOMSON, M.B., Assistant Surgeon to the North-West London Hospital, and to St. Peter's Hospital; 8, Cavendish place. *Trans.* 1.

1883 WALLER, AUGUSTUS, M.D., F.R.S., Lecturer on Physiology, St. Mary's Hospital; Weston Lodge, 32, Grove End road, St. John's Wood. *Referee* 1895—. *Trans.* 1.

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.

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

Elected

1894 WARD-HUMPHREYS, GEORGE HERBERT, 26, Charles street, St. James's.

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. *Referee*, 1905. 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. *Referee*, 1905—.

1903 WATSON, CHARLES GORDON, 44, Welbeck street, Cavendish square.

1861 **Watson**, WILLIAM SPENCER, M.B., 44, Chepstow place, Kensington. C. 1883-4. *Trans.* 1.

1891 **Weber**, FREDERIC PARKES, M.D., Physician to the German Hospital, Dalston; 19, Harley street. *Trans.* 4. *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.

1903 WEST, CHARLES ERNEST, 132, Harley street.

Elected

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.

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.

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

1897 WHITE, CHARLES POWELL, Pathological Department, St. Thomas's Hospital.

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., 21, Bentinck street, Manchester square. *Trans.* 1.

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

1902 WIGHTWICK, FALLON PERCY, M.D., 9A, Upper Brook street.

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.

Elected

1865 **Willett**, ALFRED, Surgeon to St. Bartholomew's Hospital; Surgeon to St. Luke's Hospital; 36, Wimpole street, Cavendish square. *Trustee*, 1892—1903. C. 1880-1. V.P. 1890-1. P. 1902—4. *Referee*, 1882-9, 1892-1902. *Bldg. Com.* 1889-92. *Ho. Com.* 1892-8. *Cent. Com.* 1904-5. *Trans.* 2.

1887 WILLETT, EDGAR, M.D., 22, Queen Anne street, Cavendish square. *Trans.* 1.

1902 **Willett**, JOHN ABERNETHY, M.B., 36, Wimpole 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, Agar street, Strand. C. 1905—. *Trans.* 1.

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

1905 WILLIAMSON, HERBERT, M.B., B.C., 45, Weymouth street, Portland place.

1903 WILLIAMSON, OLIVER K., M.A., M.D., 50, Upper Berkeley street.

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. *Trans.* 1.

Elected

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.

1892 WRIGHT, ALMROTH EDWARD, M.D., Ch.B., 7, Lower Seymour street. *Trans.* 1.

1890 WYNTER, WALTER ESSEX, M.D., Physician to the Middlesex Hospital; 27, Wimpole street.

1904 YOUNG, ROBERT ARTHUR, M.D., B.Sc., 41, Wimpole street.

LIST OF RESIDENT FELLOWS

ARRANGED ACCORDING TO

DATE OF ELECTION

1848 John Clarke, M.D.
1849 C. H. F. Routh, M.D.
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.
1859 Wm. Howship Dickinson, M.D.
Richard Barwell.
1860 William Ogle, M.D.
Thomas Bryant, M.Ch.
John Couper.
1861 William Spencer Watson, M.B.
1862 Lionel Smith Beale, M.B., F.R.S.
Edmund Symes Thompson, M.D.
Reginald Edward Thompson, M.D.
George Cowell.
1863 Sydney Ringer, M.D., F.R.S.
Sir Thomas Smith, Bart., K.C.V.O.
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., LL.D.
Frederick W. Pavy, M.D., F.R.S.
1865 John Langton.
Alfred Willett.
Sir Alfred Cooper.
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., K.C.V.O., M.D.
F. Howard Marsh.
1868 H. Charlton Bastian, M.D., F.R.S.
Sir W. H. Broadbent, Bart., K.C.V.O., M.D., F.R.S.
Thomas Buzzard, M.D.
Walter Butler Cheadle, M.D.
T. Henry Green, M.D.
George Eastes, M.B.
1869 Joseph Frank Payne, M.D.
Arthur E. Sansom, M.D.
Thomas Laurence Read.
1870 J. Warrington Haward.
Sir Edgcombe Venning.
Clement Godson, M.D.
Reginald Harrison.
1871 William Cayley, M.D.
Sir T. Lauder Brunton, M.D., F.R.S.

1871 J. Hughlings Jackson, M.D., F.R.S.
Philip Frank, M.D.
1872 Charles S. Tomes, M.A., F.R.S.
Sir William Bartlett Dalby, M.B.
1873 Frederick Taylor, M.D.
Norman Moore, M.D.
Sir William R. Gowers, M.D., F.R.S.
Jeremiah MacCarthy.
Henry T. Butlin.
1874 Alfred Lewis Galabin, M.D.
John Mitchell Bruce, M.D.
Henry Morris, M.A.
William Laidlaw Purves.
William Harrison Cripps.
Herbert William Page, M.C.
Frederic Durham, M.B.
William Robert Smith, M.D.
1875 Thomas Crawford Hayes, M.D.
Waren Tay.
Fletcher Beach, M.B.
Henry Cooper Rose, M.D.
1876 Sir Thomas Barlow, Bart., K.C.V.O., M.D.
Albert J. Venn, M.D.
1877 Sir Felix Semon, K.C.V.O., M.D.
Sidney Coupland, M.D.
Francis Warner, M.D.
William Ewart, M.D.
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, K.C.V.O.
1878 Sir Jas. Crichton-Browne, M.D., F.R.S.
Fred. T. Roberts, M.D.
Clinton T. Dent.
John H. Morgan, C.V.O.
Donald W. Charles Hood, C.V.O., M.D.
1879 Edward Woakes, M.D.
Malcolm A. Morris.
A. E. Cumberbatch, M.B.
Edmund Owen.
Arthur E. J. Barker.
Sir Fredk. Treves, Bart., G.C.V.O., C.B.
Andrew Clark.
1879 Francis Henry Champneys, M.D.
William Watson Cheyne, C.B., F.R.S.
George Henry Savage, M.D.
Henry Hugh Clutton, M.B., M.C.
Frederic S. Eve.
E. Noble Smith.
William Henry Allchin, M.D., F.R.S.
1880 Robert Alex. Gibbons, M.D.
David Ferrier, M.D., F.R.S.
Edmund Distin Maddick.
Jas. John Macwhirter Dunbar, M.D.
James William Browne, M.B.
William Appleton Meredith, M.B., C.M.
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.
Sir Stephen Mackenzie, M.D.
William Hale White, M.D.
Eustace Smith, M.D.
Percy Kidd, M.D.
Oswald A. Browne, M.D.
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 Ernest Clarke, M.D., B.S.
George Robertson Turner.
Howard Henry Tooth, C.M.G., 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., K.C.B., M.D.
1883 Edwin Clifford Beale, M.A., M.B.
James Kingston Fowler, M.D.
James Frederic Goodhart, M.D.
Walter H. H. Jessop, M.B.

1883 Walter Edmunds, M.C.
Sir Victor A. Horsley, F.R.S.
Dudley Wilmot Buxton, M.D.
John James Pringle, M.B.
Henry Roxburgh Fuller, M.D.
Wilmot Parker Herringham, M.D.
Augustus Waller, M.D., F.R.S.
William Pasteur, M.D.
John Bland-Sutton.
Robert Marcus Gunn, M.B.
1884 George Newton Pitt, M.D.
Charles Stonham, C.M.G.
Stanley Boyd, B.S.
William Arbuthnot Lane, M.S.
Arthur Marmaduke Sheild, M.B.
Sidney Harris Cox Martin, M.D., F.R.S.
Thomas Wakley, jun.
F. Swinford Edwards.
James Johnston, M.D.
William Duncan, M.D.
George Richard Turner Phillips.
Bilton Pollard.
1885 Alexander Haig, M.D.
Theodore Dyke Acland, M.D.
Frederick Walker Mott, M.D., F.R.S.
James Berry, B.S.
John Cahill, M.D.
John Poland.
A. C. Butler-Smythe.
Charles Alfred Ballance, M.S.
Walter S. A. Griffith, M.D.
John Edward Squire, C.B., 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 H. N. Lewers, M.D.
1887 Walter George Spencer.
Thomas Outterson Wood, M.D.
Edgar William Willett, M.D.
Henry Lewis Jones, M.D.
Francis George Penrose, M.D.
1887 Hugh Percy Dunn.
Frederic William Hewitt, M.V.O., 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.
1888 Robert Henry Scanes Spicer, M.D.
Jonathan Hutchinson, jun.
Campbell Williams.
James Donelan, M.B., M.Ch.
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.
David Henry Goodsall.
Raymond Johnson, M.B.
John Fletcher Little, M.B.
Henry Work Dodd.
George Lindsay Turnbull, M.D.
Sidney Phillips, M.D.
Henry Percy Dean, M.S.
William Hunter, M.D.
J. Inglis Parsons, M.D.
Bernard Pitts, 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.
Charles D. B. Hale, M.D.
Sir Edwin Cooper Perry, M.D.
Morton Smale.
Frederick Willcocks, M.D.
William T. Holmes Spicer, M.B.
Henry Walter Syers, M.D.
Seymour Taylor, M.D.
William Alfred Wills, M.D.
W. G. O. White-Cooper, M.B.
William A. F. Bateman.
James Jackson Clarke, M.B.
Leonard G. Guthrie, M.D., B.Ch.

1890 G. William Hill, M.D., B.Sc.
Edward Law, M.D., C.M.
Sir Patrick Manson, K.C.M.G., M.D., C.M., F.R.S.
Humphry D. Rolleston, M.D.
Walter Essex Wynter, M.D.
Edward Lawrie, M.B.
1891 Herbert P. Hawkins, M.D., B.Ch.
Cyril Ogle, M.A., M.B.
Arthur F. Voelcker, M.D., B.S.
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.
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.
Philip R. W. De Santi.
Percy W. Dove, M.B.
William J. Gow, M.D.
Paul Frank Moline, M.B.
Edward Percy Paton, M.S.
James Samuel Risien Russell, M.D.
Charles Percival White, M.B., B.C.
W. Page May, M.D.
Charles Ernest Baker, M.B.
1892 J. Dundas Grant, M.D.
R. J. Bliss Howard, M.D.
Thomas Horrocks Openshaw, C.M.G., M.S.
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.
St Clair Thomson, M.D.
Henry Rayner, M.D.
H. Marmaduke Page.
Almroth Edward Wright, M.D.
1893 James Taylor, M.D.
Howard Barrett.
Robert Cozens Bailey, M.S.
Henry Albert Caley, M.D.
Arthur Edward Giles, M.D., B.Sc.
1893 D. Watkin Roberts, M.D.
Leonard A. Bidwell.
Frédéric F. Burghard, M.D., M.S.
John H. Drysdale, M.D.
William McAdam Eccles, M.S.
Vaughan Harley, M.D.
George Herschell, M.D.
Arnold Lawson.
Guthrie Rankin, M.D.
1894 Richard Gill.
Joseph Sefton Sewill.
Thomas Vincent Dickinson, M.D.
Alexander Morison, M.D.
L. Hemington Pegler, M.D.
Herbt. Furnivall Waterhouse, C.M.
Percy Furnivall.
R. L. Langdon-Down, M.B., B.C.
Allan Macfadyen, M.D.
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.D.
James Galloway, M.D.
David Bridge Lees, M.D.
Arthur G. Phear, M.D.
Edward Erskine Henderson, M.B.
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., M.Ch.
Percival Horton-Smith Hartley, 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.
William Edward Lee, M.D.
1897 Comyns Berkeley, M.B., B.C.

1897 William Arthur Brailey, M.D.
James Cantlie, M.B.
Raymond H. Payne Crawfurd, M.D.
Francis Whittaker Tunnicliffe, M.D.
Arthur Whitfield, M.D.
Edward Stainer, M.B.
Alfred G. Levy, M.D.
A. P. Beddard, M.D.
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.
Charles Powell White.
1898 J. H. Bryant, M.D.
L. A. Dunn, M.S.
E. Hurry Fenwick.
Sir A. Downing Fripp, C.B., M.V.O., M.S.
A. Corrie Keep, M.D.
A. C. Latham, M.D.
J. B. Lawford.
Sir John McFadyean.
H. Murray Ramsay.
J. F. H. Broadbent, M.D.
A. Stark Currie, M.D.
James Morrison, M.D.
Thomas J. Horder, M.D.
F. W. Robertson, M.D.
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, C.B., M.D.
Thomas Herbert Kellock, M.D.
1899 James Hugh Thursfield, M.D.
Lindley Marcroft Scott, M.D.
Louis Bathe Rawling, M.B.
John Edward Sandilands, M.B.
Arthur J. Whiting, M.D.
Edward Farquhar Buzzard, M.D.
Greville Macdonald, M.D.
George Jones, M.B.
Herbert Campbell Thomson, M.D.
Thomas Morison Legge, M.D.
William John Ritchie Simpson, M.D.
1899 Karl Fürth, M.D.
Purves Stewart, M.D.
George Edward Shuttleworth, M.D.
1900 Clive Riviere, M.D.
H. Roe Walker.
Richard Lake.
Percy Flemming, M.D., B.S.
John Shields Fairbairn, M.B.
Aslett Baldwin.
Charles Ryall.
William Hern.
Cecil Huntington Leaf, M.B.
James Harry Sequeira, M.D.
Harold Batty Shaw, M.D.
Charles Herbert Thompson, M.D.
John William Thomson Walker, M.B.
Cecil Price-Jones, M.B.
1901 Sir Hugh Reeve Beevor, Bart., M.D.
J. Brunton Blaikie, M.D.
John Patrick Henry, M.D.
Herbert John Paterson, B.C.
George Henkell Drummond Robinson, M.D.
Elmore Wright Brewerton.
Thomas Rupert Hampden Bucknall, M.S., M.D.
William Douglas Harmer, M.B.
Henry George Plimmer.
Lionel Vernon Cargill.
T. N. Kelynack, M.D.
Leonard Williams, M.D.
T. J. P. Hartigan.
1902 J. P. Lockhart 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.
Herbert French, M.D.
Holland John Cotton, M.D., C.M.
Arthur Evans, M.S.
James Kerr, M.D., D.P.H.
Donald John Armour, M.B.
David Barty King, M.D., Ch.B.
Hector William Gavin Mackenzie, M.D.
John Abernethy Willett, M.B.
Thomas Crisp English, B.S.
Louis W. Sambon, M.D.
Joseph Blumfeld, M.D., B.C.
Ernest Beddoe Hulbert, M.D.

1902 Harold L. Barnard, M.S.
Fallon Percy Wightwick, M.D.
1903 Edmund Ivens Spriggs, M.D.
Otto F. F. Grünbaum, M.B., B.C.
Samuel William Carruthers, M.D.
John Russell Ryan, M.D., B.Ch.
Oliver K. Williamson, M.D.
William Lawrence Ascherson, M.B., B.C.
Harold Burrows, M.B., B.S.
Leonard S. Dudgeon.
Evelyn Arthur Rich.
Fleming Mant Sandwith, M.D.
Charles Gordon Watson.
Charles Ernest West.
Charles Bolton, M.D.
James Sherren.
1904 Harold Shuttleworth Barwell, M.B.
William Philip Sutcliffe Branson, M.D.
John Howell Evans.
Thomas Matheson Ness, M.B.
Henry Edmund Symes Thompson, M.D.
Joseph Arthur Arkwright, M.D.
Richard William Brimacombe, M.D.
H. Overton Hobson, M.D.
Thomas Wright Parkinson, M.D.
1904 Robert Arthur Young, M.D., B.Sc.
Charles Joseph Harrison, M.D., B.S.
1905 William Carnegie Brown, M.D.
Percy John Cammidge, M.B.
Herbert Lightfoot Eason, M.D., M.S.
John Fawcett, M.D.
Herbert J. Scharlieb, C.M.G., M.D., B.S.
William Henry Battle.
Herbert Sherwell Clogg, M.B., M.S.
William Foster Cross.
Thomas Jefferson Faulder.
George Ernest Gask.
William Sampson Handley.
Henry Head, M.D., F.R.S.
Frederick John McCann, M.D.
Harry Edwin Bruce Porter, M.D.
Sydney Richard Scott.
Ralph Thompson.
Herbert Williamson, M.B., B.C.
William Cecil Bosanquet, M.D.
Francis Frederick Muecke, M.B., B.S.
A. W. Ormond.
John George Pardoe, M.B., C.M.
Frederick William Price, M.B.

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.* 4.

1903 *Amsden, Walter*, Lexden House, Seaford, Sussex.

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

1905 *Anderson-Berry, David*, 23, Grosvenor crescent, St. Leonard's-on-Sea.

1880 *Appleton, Henry*, M.D., Charlbury, Oxford.

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.

1905 *Bain, William*, M.D., Straythorpe, York place, Harrogate.

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

1896 *Ball, Sir 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.

Elected

1900 *Bardswell, Noel Dean*, M.D., King Edward the Seventh's Sanatorium, Midhurst, Sussex. *Trans.* 2.

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

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

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

1896 *Belben, Frank*, M.A., M.B., Redlands, Knyveton 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: 10, Rodney street, Liverpool.

1901 *Bisshopp, Francis R. B.*, M.D., Parham House, Tunbridge Wells.

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

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

1904 *Bossan, Emile*, M.D., 19, Boulevard Dubouchage, Nice, France.

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

Elected

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

1870 *Bowles, Robert Leamon*, M.D., Consulting Physician to the Folkestone Hospital and Physician to St. Andrew's Home, Folkestone; Prior's Mesne, Lydney, Gloucestershire. C. 1897-9. *Sci. Com.* 1896-1902. *Trans.* 3.

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*, Oriel Lodge, Cheltenham.

1899 *Bremridge, Richard Harding*, Bangkok, Siam (c/o R. Bremridge, 17, Bloomsbury square).

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

1867 *Bridgwater, Thomas*, M.B., LL.D., Hooke Hall, Uckfield, Sussex.

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.

1904 *Butcher, William Deane*, Holywood, Cleveland road, Ealing.

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

Elected

1900 CARLYON, T. B., Brinklow, near Coventry.

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; 18, Palmeira square, Hove, Sussex.

1885 CHAPMAN, PAUL MORGAN, M.D., Physician to the Hereford General Infirmary, 1, St. John street, Hereford. *Trans.* 1.

1881 *Chavasse*, SIR 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.

1890 CHILDS, CHRISTOPHER, M.D., D.P.H., Knight of Grace of St. John, Boscarn, Looe, Cornwall.

1896 CHRISTOPHERSON, JOHN BRIAN, M.D., B.C., late Assistant Demonstrator of Anatomy at St. Bartholomew's Hospital; late Surgeon to Seamen's Hospital, Albert Dock; c/o P.M.O., Egyptian Army, Cairo.

1892 CLARK, JAMES CHARLES, 10, Kimbolton road, Bedford.

1897 CLARK, W. GLADSTONE, Buluwayo.

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.

1905 COLLINSON, HAROLD, 24, Park Square, Leeds.

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.

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.

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., 500, Lordship Lane, Dulwich.

1902 *Douty, E. H.*, M.D., La Madeleine, Cannes.

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

1898 *Dreschfeld, Julius*, Stanley House, Wilmslow road, Withington, 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.

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

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

Elected

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.

1906 *Edgecombe, Wilfrid,* M.D., Rodney house, Victoria avenue, Harrogate.

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

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.

1903 *Etlinger, Frederick Kincaid,* Cotswold Sanatorium, nr. Stroud, Gloucestershire.

1875 *Fagan, John,* Consulting Surgeon to the Belfast Royal Hospital; Graigavenue, Monasterevan, Queen's Co.

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

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

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; Kt. of Grace of St. John; Lamorna, Falmouth. C. 1888. *Referee,* 1881-7.

1902 *Fennell, Charles Henry,* M.A., M.D., County Asylum, Hellingly, Sussex.

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

1903 *Ferguson, George Bagot,* M.D., M.Ch., Cheltenham.

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

1903 *Forbes, Norman Hay,* Drumminnor, 7, Boyne Park, Tunbridge Wells.

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

Elected

1892 *Foster, Michael George*, M.A., M.D., Villa San Giovanni, San Remo.

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

1903 *Freeborn, John C. R.*, 38, Broad street, Oxford.

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

1885 *Gamgee, Arthur*, M.D., LL.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*, Edge Mount, Altrincham.

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. *Trans.* 1.

1898 *Granville, Alexander*, The Sanitary Department, Ministry of the Interior, Cairo, Egypt.

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*, 19, Gold Tops, Newport, Mon.

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.

Elected

1905 *Grünbaum, Albert Sidney Frankau*, M.D., The Drive, Roundhay, Leeds.

1889 *Gubb, Alfred S.*, M.D., Mustapha Supérieur, Algiers.

1904 *Hall, Arthur John*, M.A., M.B., B.C., 342, Glossop road, Sheffield.

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.

1880 *Harris, Vincent Dormer*, M.D., Consulting Physician to the City of London Hospital for Diseases of the Chest, Victoria Park; the Royal National Hospital for Consumption and Diseases of the Chest, Ventnor; the Metropolitan Dispensary and the Morley House Convalescent Institutions, St. Margaret's Bay and Sandgate, etc.; Woodrouffe House, Milford-on-Sea, near Lymington, Hants. *Referee*, 1899—.

1905 *Hathaway, Frank John*, M.D., Langholm, Osborne road, Windsor.

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; 16, Blakesley avenue, Ealing. C. 1878-9.

1891 *Hayward, John Arthur*, M.D., 23, The Grange, Wimbledon Common. *Pro.* 1.

Elected

1899 *Hillier, A. P.*, M.D., Markgate Cell, near Dunstable, Beds.

1899 *Hind, Henry*, Blytheholm, Harrogate.

1900 *Hobhouse, Edmund*, M.D., 12, Second avenue, Brighton.

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.

1905 *Hort, E. C.*, San Remo, Italy.

1905 *Howard, Alan Campbell Palmer*, M.D., C.M., Montreal, Canada.

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

1903 *Hulbert, Henry Louis Powell*, M.A., M.B., B.C., The Vicarage, Towcester, Northants.

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

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

1883 *Jacobson, Walter Hamilton Acland*, M.Ch.Oxon., Surgeon to Guy's Hospital; Lordine Court, Ewhurst, Hawkhurst. C. 1902-3. *Referee*, 1895-1902. *Lib. Com.* 1896-1902. *Trans.* 2.

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.

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; 10, Ferndale, Teignmouth.

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

Elected

1884 KESER, JEAN SAMUEL, M.D., Villa Colatel, Chemin Vinet, Lausanne, Switzerland.

1898 KLEFSTAD-SILLONVILLE, O., M.D., Aix-les-Bains, Savoie.

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.

1905 *Lawson*, DAVID, M.D., Nordrach-on-Dee, Banchory, Scotland. Trans. 1.

1880 LAYCOCK, GEORGE LOCKWOOD, M.B., C.M., Melbourne, Victoria, Australia.

1892 LAZARUS-BARLOW, WALTER SYDNEY, M.D., Fernholme, Woodside Park, Finchley. *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.

1905 LEEDHAM-GREEN, CHARLES, M.D., 31, Frederick road, Edgbaston, Birmingham.

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.

1905 LEWIS, FRANK BENJAMIN, Springfield House, 144, London road, St. Leonard's-on-Sea.

1898 LINDSAY, JAMES, M.A., M.D., 15, College square East, Belfast.

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

Elected

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.)

1876 *Mackey, Edward*, M.D., Physician to the Sussex County Hospital; Senior Physician to the Royal Alexandra Hospital for Sick Children; 1, Ventnor villas, Hove.

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

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. 1902-4. *Referee*, 1884-90, 1895-7. *Lib. Com.* 1886-90.

1891 *Manby, Sir 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.

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

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

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

1898 *Millard, William Joseph Kelson*, M.D., Trediden, Hewlett road, Cheltenham.

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

Elected

1904 *Mitchell, Thomas Walker*, M.B., C.M., Hadlow park, Tonbridge, Kent.

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

1904 *Morgan, William Pringle*, M.B., B.Ch., Rostrevor, Seaford, Sussex.

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. *Trans.* 2.

1899 *Mundy, Herbert*, Florida road, Durban.

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.

1905 *Nettleship, Edward*, Nutcombe Hill, Hindhead, Haslemere.

1902 *Newland, Henry Simpson*, M.B., Ch.B., Adelaide, South Australia.

1895 *Newsholme, Arthur*, M.D., 11, Gloucester place, Brighton. *Trans.* 1.

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

1905 *Norman, George*, M.B., B.S., "Brendon," Palmerston road, Buckhurst hill, Essex.

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

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

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

Elected

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.* 2.

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, Sir Lambert 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.

1894 *Osborn, Samuel*, Knight of Grace of St. John; Maisonnette, Datchet, Bucks.

1904 *Osler, William*, M.D., Regius Professor of Medicine in the University of Oxford; 7, Norham gardens, Oxford.

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

1887 *Pardington, George Lucas*, M.D., Glynlin, Tunbridge Wells.

1873 *Parker, Robert William*, Consulting, late Senior Surgeon to the East London Hospital for Children and 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.

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

Elected

1903 *Pearson, S. Vere*, M.B., The Sanatorium, Mundesley, Norfolk.

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.

1867 **Pick**, *Thomas Pickering*, Consulting Surgeon to St. George's Hospital; The Nook, Great Bookham, Surrey. C. 1884-5. V.-P. 1893-4. *Referee*, 1882-3, 1891-93. *Sci. Com.* 1870, 1889—. *Lib. Com.* 1879-81.

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

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.

1905 *Plummer, Harry Beddoes Wetherell*, 54, Birmingham road, West Bromwich.

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

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

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

Elected

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.

1905 *RASHLEIGH, JOHN COSMO STUART*, M.D., Throwleigh, Okehampton, Devon.

1902 *RAW, NATHAN*, M.D., B.S., 66, Rodney street, Liverpool.

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.

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. Thomas Cook and Son, 9, Old Court House street, Calcutta.]. *Trans.* 3.

1863 *ROWE, THOMAS SMITH*, M.D., Consulting Surgeon to the Royal Sea-Bathing Infirmary; Union crescent, Margate, Kent.

Elected

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

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.

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—. *Trans.* 1.

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

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

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

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

1891 *Smith, G. Cockburn*, M.D., 14, South road, Newton Abbot.

1902 *Smith, Harry Lyon*, M.D., Woodfield House, Uppingham, Rutland.

1886 *Smith, Howard Lyon*, Buckland House, Buckland Newton, Dorchester.

1894 *Smith, Robert Shingleton*, M.D., B.Sc., Senior Physician to the Bristol Royal Infirmary, and Emeritus Professor of Medicine, University College, Bristol, also Consulting Physician to the Bristol Dispensary ; Deepholm, Clifton Park, Clifton, Bristol.

Elected

1894 *Smith, Thomas Rudolph*, M.B., B.C., Blytheholm, Stockton-on-Tees.

1873 Smith, *W. Johnson*, Consulting Surgeon to the Seamen's Hospital Society, Woodlands, Willingdon, Sussex.

1868 *Solly, Samuel Edwin*, Colorado Springs, Colorado, U.S.A.

1875 *Spitta, Edmund Johnson*, 41, Ventnor Villas, Hove. C. 1903-5.

1891 *Stevens, Surg.-Capt. Cecil Robert*, M.B., B.S., I.M.S., Eden Hospital, Calcutta.

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].

1897 *Strangeways, T. S. P.*, Department of Medicine, The University, Cambridge.

1871 *Strong, Henry John*, M.D., J.P., Consulting Surgeon to the Croydon General Hospital; Colonnade House, The Steyne, Worthing.

1905 *Sutcliffe, William Greenwood*, 7, Dalby square, Margate.

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.

1891 *Thomson, John Roberts*, M.D., Monkchester, Bournemouth.

1904 *Thorburn, William*, M.B., B.S., 2, St. Peter's square, Manchester. *Trans.* 1.

Elected

1883 *Thursfield, Thomas William*, M.D., Physician to the Warneford and South Warwickshire General Hospital; Selwood, Beauchamp square, Leamington.

1880 *Tivy, William James*, 5, Victoria square, Clifton, Bristol.

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, Ryde, 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.

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.

1861 *Waters, A. T. Houghton*, M.D., Consulting Physician to the Royal Infirmary; 69, Bedford street, Liverpool. *Trans.* 3.

Elected

1874 *Wells, Harry*, M.D., San Ysidro, Buenos Ayres, S. America.

1882 *Wharry, Charles John*, M.D., 14, Ewell road, Surbiton, Surrey.

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.

1859 *Williams, Charles*, Senior Surgeon to the Norfolk and Norwich Hospital; 48, Prince of Wales road, Norwich.

1872 Williams, *Sir John*, Bart., K.C.V.O., 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; Plâs Llanstephan, Carmarthenshire. C. 1891. *Referee*, 1878-90. *Lib. Com.* 1876-82.

1903. *Williamson, Richard Ernest*, M.B., C.M., Romagna House, Otley, Yorkshire.

1887 *Wilson, Arthur Hervey*, M.D., 504, Broadway, Boston, U.S.A.

Elected

1889 *Wise, A. Tucker*, M.D., Montreux, Switzerland.

1885 *Wolfenden, Richard Norris*, M.D., Rougemont, Seaford, Sussex.

1905 *Wollaston, Alexander Frederick Richmond*, M.B., B.C., c/o Messrs. Smith, Mackenzie & Co., Mombasa, British East Africa.

1892 *Woodhead, German Sims*, M.D., Professor of Pathology in the University of Cambridge; 6, Scrope terrace, Cambridge.

1899 *Wynter, Andrew Ellis*, M.D., Oakfield House, Clifton, Bristol.

1905 *Young, Eric Ernest*, M.B., M.S., The Infirmary, Stoke-on-Trent.

SERVICE FELLOWS.

1903 BENTHAM, ROBERT, Royal Naval Hospital, Malta.

1905 HOLT, MAURICE PERCY, 67B, Shooter's Hill, Blackheath, and c/o Messrs. Holt, 3, Whitehall place.

1904 KELLIE, GEORGE JEROME, Sirur, Poonah District, c/o Messrs. H. S. King & Co., 9, Pall Mall.

1905 KILKELLY, CHARLES RANDOLPH, Osborne, Isle of Wight.

1903 LEICESTER, JOHN HOLDICH, M.D., B.S., c/o Messrs. Grindlay & Co., 11, Hastings street, Calcutta.

1904 *Pollock*, CHARLES EDWARD, Royal Naval Hospital, Malta [c/o Messrs. Holt & Co., 3, Whitehall Place].

1903 RANKING, ROBERT MAURICE, M.B., B.C., Hanover House, Tunbridge Wells.

1903 SMITHSON, ARTHUR ERNEST, M.B., B.C., Barberton, Transvaal, c/o Sir C. R. McGrigor, Bart., & Co., 25, Charles Street, St. James's Square.

1905 SPENCER, CHARLES GEORGE, M.B., Kent Lodge, 52, The Avenue, Kew Gardens.

1903 TAYLOR, SIR WILLIAM, K.C.B., M.D., C.M., 29, Duke street, St. James's.

Corrected to 30th December, 1905.

ANNUAL GENERAL MEETING.

Wednesday, March 1st, 1905, at 5 p.m.

Present—37 Fellows.

SIR RICHARD DOUGLAS POWELL, Bart., K.C.V.O., M.D., President.

G. NEWTON PITT, M.D., STEPHEN PAGET, } Hon. Secs.

The PRESIDENT nominated Dr. Bowles and Dr. Wightwick to act as scrutineers of the ballot, and declared the ballot open until 6 o'clock.

The SENIOR HON. SECRETARY (Dr. Newton Pitt) read the report of the Council.

REPORT OF THE COUNCIL.

THE Council has every reason to congratulate the Fellows that the last financial year of the Society's Centenary finds its affairs in a more flourishing condition than at any previous period of its history. This is the more noteworthy, as it coincides with the falling in of the Berners Street lease, from which the Society enjoyed a net annual income of £436.

During the past year 14 new Honorary Fellows (7 Foreign and 7 British) and 26 new Ordinary Fellows

have been elected, including 15 Resident, 9 Non-resident, and 2 Service Fellows. On the other hand, the Society has to record the loss, by death or resignation, of 1 Honorary Fellow and 35 Ordinary Fellows. This number includes several Non-resident Fellows, who have passed away during the last few years, whose representatives did not announce their decease to the Society. At the present time the roll stands as follows:

Honorary Fellows—British	. . .	12	
	Foreign . . .	20	
			— 32
Ordinary Fellows—Resident	. .	538	
	Non-resident. . .	279	
	Service . . .	11	
			——828
			860

It was hoped that a larger number of candidates would have applied for the Service Fellowship, but it takes some time for a new privilege of this kind to become generally known, and it may be considered desirable to take some special steps for announcing to the Members of the Services that the Society welcomes them on terms of special privilege. The interest in the meetings during the past session has been well maintained, and, although there is still room for improvement in the matter of actual attendance, the value of the discussions is evidenced by the reports, which are now printed in the 'Transactions.' The Council appointed a Special Committee to consider what steps might be taken to improve the attendance at meetings, and as a result of this Committee's report, the Council has adopted the following measures, which it trusts will be appreciated and used to their fullest extent by the Fellows:

(1) Abstracts of the papers to be read will be issued in advance to any Fellows who apply for them, and Fellows who desire it can register their names to receive them regularly by post.

(2) Authors are invited to supply lists of all persons they think likely to be interested in their papers, including members of the profession who are not Fellows of the Society, and abstracts are also sent to these, with invitations to be present at the meeting.

(3) The Council has also decided to hold at least one special discussion in each session. The first of these was held yesterday on "The subsequent course and later history of cases of appendicitis after operation," and its undoubted success, both as regards the large attendance and the high quality of the discussion, fully justifies the action of the Council.

The Congress on School Hygiene, held in Nuremberg last year, was attended on behalf of the Society by Sir Lauder Brunton and Dr. Clement Dukes, who were delegated for this purpose, and reported on the work of the Congress to the Council. At the Sanitary Institute Congress, the President of the Society, Sir Richard Douglas Powell, kindly undertook to represent the Society. At the Congress of School Hygiene for this year, held last month, Dr. Clement Dukes was again appointed delegate of the Society, and will report thereon in due course. The War Office, in connection with its special Committee of Inquiry on the subject of Venereal Diseases, invited the Society to assist it by expert evidence. The Council appointed Mr. Edgcombe Venning to represent them for this purpose, and he appeared before the Committee and gave evidence.

It is, perhaps, worthy of mention that our Secretary was specially invited as one of a select few, representing European librarianship, to attend the International Library Conference at St. Louis, but, unfortunately, the condition of Mr. MacAlister's health made it impossible for him to accept the invitation.

The never ending problem of book storage has been solved, for a few years at least, by the construction of a large book store behind the Meeting Room, which will hold at least 10,000 volumes, and the addition of two large bookcases in the corridor. Within the last few weeks, a contract has been entered into for the ventilation

of the North Room and the corridor on the Glover Lyon system.

The Special Report upon Suspended Animation in the Apparently Drowned, drawn up by Professor Schäfer for the Committee appointed to investigate this subject, has been published, and forms a most valuable addition to our knowledge on this difficult subject. It is to be sold to the public at 5*s.*, but can be purchased by Fellows at cost price, viz. 3*s.* 6*d.*

Centenary.—Immediately after the last Annual Meeting the Council appointed a special Committee to draw up a programme to arrange for the celebration, in a fitting manner, of the Society's first Centenary. The Committee consists of—

The President.—Sir Richard Douglas Powell.
Dr. Newton Pitt, } *Hon. Secretaries.*
Mr. Stephen Paget, }
Sir William S. Church, } *Hon. Treasurers.*
Mr. Warrington Haward, }
Mr. Alfred Willett, *Ex-President.*
Mr. Clinton T. Dent.
Mr. Rickman J. Godlee.
Mr. A. Pearce Gould.
Dr. Norman Moore.
Sir James Reid.

The Fellows have already been briefly informed by circular of some of the proposals of the Committee, but they may be repeated here :

The Issue of a Centenary Volume.—This volume, which is being edited by Dr. Norman Moore and Mr. Stephen Paget, will contain an account of the chief incidents in the history and development of the Society. It will form a substantial volume of about the same size as the 'Transactions,' and will be presented to each Fellow.

A Special Meeting will be held on the Centenary day,

May 22nd, when a special address will be delivered by the President, and he will welcome in the name of the Society the new Honorary Fellows who were elected at the last Ballot, viz.:

Foreign

Professor H. NOTHNAGEL (Vienna).
„ W. H. WELCH (Baltimore).
„ E. MARCHIAFAVA (Rome).
„ I. P. PAVLOFF (St. Petersburg).
„ KITASATO (Tokio).
„ CHR. BOHR (Copenhagen).
„ S. RAMON Y CAJAL (Madrid).

British

The Right Hon. Baron LISTER, O.M., F.R.S., D.C.L., LL.D., D.Sc., F.R.C.S.

The Right Hon. Baron RAYLEIGH, O.M., F.R.S., D.C.L., LL.D., Sc.D.

Sir WILLIAM RAMSAY, K.C.B., F.R.S., LL.D., D.Sc., F.C.S.

Sir SAMUEL WILKS, Bart., M.D., F.R.S., LL.D., F.R.C.P.

Sir WILLIAM GAIRDNER, K.C.B., M.D., F.R.S., LL.D.

W. H. GASKELL, M.D., F.R.S.

ROBERT BARNES, M.D., F.R.C.P.

The Marshall Hall Prizeman, Dr. Henry Head, will give an address on his recent work, under the title "The Afferent Nerves under a New Aspect," on Tuesday, May 23rd, at 5 p.m., in the Society's Hall.

A Banquet will be held on Monday, May 22nd, at the Hotel Cecil, when the Society will entertain H.R.H. The Prince of Wales and a number of distinguished guests.

A Conversazione will be held on the evening of Wednesday, May 24th.

During the week there will be an Exhibition in the Society's rooms illustrating the progress of medicine in the year 1805.

Report of the Honorary Treasurers :

"The Hon. Treasurers can congratulate the Society on its financial position.

"The past year was the last in which the Society will receive the rent of its former quarters in Berners Street; the net loss owing to the termination of the lease amounting to £436 10*s.* 5*d.* It has for some years been a source of anxiety to the Treasurers as to how the Society would bear this diminution of income. Owing to the changes which have been made in our premises, enabling us to let more of our rooms, and other arrangements also proposed and in great measure carried out by our Secretary, the loss of the Berners Street rent has been made good, and a net increase of £320 per annum has during the last two years been secured.

"The receipts during the past year, including a legacy from the late Dr. George Thin of £100, have exceeded the expenditure by £604 15*s.* 5*d.*

"W. S. CHURCH.
"WARRINGTON HAWARD."

Report of the Honorary Librarians :

"The changes affecting the Library Catalogue and the shelf arrangement of the books are nearing completion. The card catalogue is ready and the cards are arranged in the drawers of a cabinet which will shortly be fitted up in the Library to the left of the catalogue desk. The new catalogue will be in working order by the beginning of May, three weeks before the Centenary Celebration.

"The re-numbering of the books is now practically completed, and the transference of the acccesion number to the new cards is now commencing.

"The room acquired at the back of the Meeting

Room alluded to in the last report has been fitted up with shelves and is now in use.

The number of books and pamphlets added to the Library during 1904 was 615, of these 287 were presented by Fellows and others.

"The number of issues of books, apart from those used for reference purposes in the Library, was 3927, an increase upon the figures of the previous year. The actual number of Fellows and others visiting the Library was 4701, giving an average of 427 per month, in the course of the year of eleven months.

"Three hundred and seventy-six volumes were borrowed from Lewis's Library, as against 473 in 1903, and 331 in 1902. This is the highest figure that can be reached under the existing subscription for sixty volumes.

"It has been decided to incorporate in the proposed Centenary exhibition a selection from the Library of all books, published in 1805, which it possesses."

Mr. WARRINGTON HAWARD (Hon. Treasurer) read and explained the Accounts and Balance-sheet.

The PRESIDENT moved that the report of the Council, together with the accounts as audited, be adopted. (Carried unanimously.)

The President then delivered the Annual Address (see p. cvii).

Dr. POLLOCK.—I am sure, gentlemen, I shall only express the feeling of this meeting when I ask you to return thanks to the President for his able and interesting address. He has touched on subjects which I shall not presume to enlarge upon, but a reference caught my ear to the special interest which the public is taking now

in the investigation of disease. I know the President's sentiments on this subject, and it struck me that there could not be a better time than this for the whole of this profession—medical, surgical, and all—to unite together and form one great central body. This is not the time or place to discuss it, but I believe we could not have a more opportune season than now. These investigations into the prevention of disease, such as tuberculosis, and the cause of disease such as cancer, have received notice from the highest authority in the kingdom, and we know well that His Majesty is deeply interested in everything which concerns the health of the community. Perhaps this Society and others will see that this is a good time for uniting all the societies of London, that is the leading societies of medicine, into one body, and so obtain strength for the profession. I have therefore to propose, with your goodwill, that the best thanks of the Society be offered to the President for his address and that he be requested to allow its publication in the 'Transactions.'

Sir WILLIAM H. BROADBENT, K.C.V.O., F.R.S.—I have great pleasure in seconding this motion, and I may add I feel very great pride in following so distinguished a member of our profession as Dr. Pollock. We have all listened to this admirable address with great interest. One element in that interest is the remarkable number of distinguished physicians and surgeons whose memory has been commemorated, and commemorated in such admirable terms that one might almost say that they were fortunate in the opportunity of their death. The President has, very properly I think, made this an occasion for a new attempt at the co-ordination and reorganisation of the medical societies. It has been a question I have long had in my mind, and is a most important matter; and I am quite certain of this, that the occasion is favourable, and that the address we have heard to-day renders it—I was going to say for the first time—possible. It has certainly marked a very distinct advance in what

will be, I believe, a very great addition to the efficiency of the medical societies. The sketch which he has given of the work of a Royal Society of Medicine, the way in which it may be so organised as to promote efficiency and economy and advance, I think, has rendered a very great service to this idea, and I hope it will be carried out during this Centenary period.

Dr. POLLOCK.—Gentlemen, I have to ask you to signify your assent in the usual way.

Carried by acclamation.

Sir THOMAS SMITH.—Might I ask your permission, Mr. President, as we have passed that vote of thanks to you, to say a word about what interests me very greatly, viz., what you drew attention to in the last part of your address to which Dr. Pollock and Sir William Broadbent have alluded? I think I was the victim of the last attempt to combine all these societies in the year 1870 or 1871. Sir George Burroughs was President, and I and my colleague, as Secretaries, worked hard enough, but we accomplished nothing. We found that the jealousies and feelings of the various societies were so strong that it was almost impossible to reconcile them, and the thing dropped, but I agree with what Dr. Pollock said, that things are very different now. The public generally are very interested in medicine, from the Sovereign downwards, and every class of society, and both sexes, share this interest, and I think now is the time when I should hope the plan which you have just sketched out might be undertaken with great prospects of success. I believe that at the present time there would not be the least difficulty in getting a Charter for what might be called either a Royal Society of Medicine or a Royal Academy of Medicine, and if it is the general opinion of this meeting that such a thing were feasible I should be very glad on the present occasion to move a resolution. But it remains to be seen what the feeling of others is.

Mr. Goodsall.—Mr. President,—Should such a resolution be proposed, I shall be very pleased to second it, if I may be allowed to do so. There are several important points which the profession should take up, and which can be taken up thoroughly only by a large and powerful body. If the suggestion is carried out, I think the interests of the public would be greatly benefited, and I should have much pleasure in seconding such a resolution. But, in so doing, I should state that I do it as an individual, and not as the Treasurer of the Medical Society of London.

Dr. Theodore Williams.—One of your suggestions was exceedingly good, sir, and that is, instead of waiting to the end of the year for our 'Transactions,' we should have them bit by bit when they are freshest in our minds, and that would be a great gain. If it is possible to reduce the subscription that would be a great thing too. The subscriptions tell on the junior members of the Society, and very likely prevent certain of the junior members joining this great Society, which would be most useful to them. These would be the grounds upon which I should support this idea. I was one of those who voted on the occasion that Sir Thomas Smith alluded to; I voted for the formation of a Royal Society of Medicine, as it was called, and it was almost an accident that it was not carried. It was only a small majority against. And if you, sir, can manage to bring that about, you will cover yourself with undying fame.

Mr. Timothy Holmes.—Perhaps you will allow me to say a word, as I am a very old member of this Society, and once occupied the chair which you so worthily fill now, to express the pleasure which it has given me to see that this idea has been brought up again, and has been brought up in such a business-like and promising way. This is an idea which has recommended itself to me most strongly, and I was exceedingly sorry on the

occasion to which Sir Thomas Smith has alluded that it was compelled to be dropped, and dropped then, I believe, more on account of the varied opposition which came from the minor societies than from any unwillingness on the part of the two chief societies in this town, that is to say, the Medical Society and our own Society. But, sir, I am not perfectly certain what the resolution before the meeting is.

The PRESIDENT.—Sir Thomas Smith has not given it to me yet.

Sir THOMAS SMITH.—I beg leave to move—

> "That the Council of the Society be requested, with as little delay as possible, to invite the leading medical societies of London to arrange for a joint meeting for the purpose of considering the advisability of amalgamating, and to take the necessary preliminary steps for that purpose."

Mr. GOODSALL.—I have great pleasure in seconding the resolution.

Mr. TIMOTHY HOLMES.—Exactly, sir. That is the very form in which it ought to have been moved, and I am delighted to support it in the very humble way that a man so old can do. I cannot promise to serve on any committee or anything of that kind, but anything I can do to promote the idea I shall do with the utmost possible pleasure, and I congratulate you, sir, on having revived an idea which will, I believe, redound greatly to the profession in London, and which, I believe, will also tend to throw fresh lustre upon the Centenary of this Society.

Dr. ALLCHIN.—I also should like, sir, to be allowed to congratulate you on the feasible and practical manner in which you brought forward this proposal, and also, at the same time, express the very sincere hope that you

will be more successful in carrying it through than has occurred with your predecessors who introduced it. I feel certain that you will be successful in this endeavour, and that your period in office will be more distinguished even than it has already been. It is probable, in regard to the many different societies that might be thought likely to enter into such a scheme as this, that, owing to their differences in origin and constitution and aims, very different arguments are likely to influence them in the course which they would take. You have mentioned two of the important advantages which are likely to accrue from such an arrangement as you foreshadow, economy in finance, and economy in labour and scientific work. Those material arguments would appeal to some societies, no doubt. Others with a long history, such, particularly, as that of the Medical Society, of which you, sir, as I myself, have had the honour to be President in the past—with a long history and prestige attached to it, and, at the same time, in possession of considerable material wealth, are likely to be influenced by somewhat different considerations than those societies of another kind. And I cannot but think that it is the putting forward of those advantages that should occupy the most prominent position in the work which will be started to carry this out, and I think that, under some such arrangement as is indicated, there will be a very considerably enhanced dignity to the whole of the objects of the societies which will enter into it—much more dignified and much more important, and that their work will have much greater power under the amalgamation of those different units than is likely to be the case if they pursue their individual course. Such is the kind of argument, sentimental though it be, which will be likely to appeal to some, and I strongly suggest such arguments as those should be used in seeking to obtain the amalgamation, and they should be put in the forefront of what should be done. I cordially support the proposition of Sir Thomas Smith.

The PRESIDENT.—I will now put the resolution—

"That the Council of the Society be requested, with as little delay as possible, to invite the leading medical societies of London to arrange for a joint meeting for the purpose of considering the advisability of amalgamating, and to take the necessary preliminary steps for that purpose."

Carried unanimously.

The PRESIDENT.—I should like to express my thanks to the Society for the vote of thanks that they have passed to me, and to say that I am very hopeful at all events that we may be able to make a good attempt to bring about this desired result. Of course, we cannot promise more than that.

The scrutineers announced the result of the ballot to be as follows :

President.—Sir Richard Douglas Powell, Bart., K.C.V.O., M.D.

Vice-Presidents.—Sir William Henry Broadbent, Bart., K.C.V.O., M.D., F.R.S.; Thomas Buzzard, M.D.; Henry Trentham Butlin; Alfred Pearce Gould, M.S.

Hon. Treasurers.—Sir William Selby Church, Bart., K.C.B., M.D.; J. Warrington Haward.

Hon. Secretaries.—George Newton Pitt, M.D.; Stephen Paget.

Hon. Librarians.—Norman Moore, M.D.; Rickman J. Godlee, M.S.

Members of Council.—Dawson Williams, M.D.; Howard Henry Tooth, C.M.G., M.D.; George Allan Heron, M.D.; Donald William Charles Hood, M.D., C.V.O.; Sir James Reid, Bart., G.C.V.O., K.C.B., M.D.; Stanley Boyd, B.S.; Clinton Thomas Dent; Arthur Marmaduke Sheild; Charles Stonham, C.M.G.; Thomas Wakley, Junior.

Mr. WILLETT.—Amongst the changes brought about by the result of the ballot, we lose two of the Vice-Presidents. I move, therefore, "That the best thanks of the Society be given to the retiring Vice-Presidents for their services to the Society during their respective terms of office, viz. Dr. H. Charlton Bastian and Dr. W. B. Cheadle."

Dr. ROBERT L. BOWLES.—I have much pleasure in seconding that resolution. The fact that it has been proposed by Mr. Willett, who was our distinguished President here in the past year, and the names of Dr. Charlton Bastian and Dr. Cheadle, two such distinguished men, is sufficient to ensure that they have done their duty extremely well. They have been valuable Vice-Presidents of our Society, and I have much pleasure in seconding this resolution.

The PRESIDENT.—I feel sure that this resolution will be unanimously carried. One knows how much we owe to the Vice-Presidents for their efforts on the Council.

Carried unanimously.

Dr. ALLCHIN.—I move "that the best thanks of the Society be given to the retiring members of Council for their services to the Society during their respective terms of office: Dr. W. R. Dakin, Dr. T. Colcott Fox, Mr. W. Arbuthnot Lane, Mr. William Lang, Mr. E. J. Spitta."

Dr. EASTES.—I have very great pleasure in seconding that. I am sure that the work of the Council, especially in connection with the arrangements for the celebration of the Centenary of the foundation of the Society, must have given the Council much more work than usual, and I think, therefore, that in a special manner our thanks are due to them for the work that they have done in the past year.

The PRESIDENT.—I am sure that this vote, too, will be accepted unanimously. I am glad we have not the opportunity of thanking the Honorary Secretaries also for their strenuous labours during the past year, because fortunately neither of them retires this year.

Carried unanimously.

The PRESIDENT.—That concludes the business, gentlemen.

(A) Income and Expenditure Account for the Year ending 31st December, 1904

Expenditure

	£	s.	d.	£	s.	d.
Rent, Rates, Taxes, and Insurance ...	198	0	2			
Salaries of Staff and Accountant ...	704	5	0			
House Servants, Porters, Cleaners, etc.	352	8	11			
Lighting, Warming, and Cleaning ...	192	2	11			
Printing, Stationery, Stamps and Telegrams	251	4	3			
Meeting Expenses	43	9	8			
Miscellaneous Disbursements ...	55	4	2			
				1796	15	1
Repairs, Alterations, etc. ...				141	4	2
Depreciation of Furniture, Fittings, etc.				62	9	0
Library Purchases and Expenses ...	490	14	7			
Work on New Catalogue ...	98	2	6			
				588	17	1
Report of Committee on Suspended Animation				135	16	11
Interest on Debentures				1010	8	3
Benefactors' Memorial Brass ...				12	10	0
'Transactions'				401	17	6
Audit Fee				10	10	0
Architect's Fees				16	16	0
Law Costs				26	1	1
Bonus to Secretary				52	10	0
Telephone				15	17	3
Lift Charges				34	17	0
Balance, being excess of Income over Expenditure				604	15	5
				£4911	4	9

Income

		£	s.	d.	£	s.	d.
418 *Annual Subscriptions at*	£3 3s.	1316	14	0			
136 *do.*	£1 1s.	142	16	0			
					1459	10	0
Composition Fees					23	2	0
Entrance Fees					129	3	0
Rents Receivable					3057	15	5
Sale of 'Transactions'					74	0	11
„ *'Climates and Baths'* ...					4	19	2
Interest on New South Wales Stock ...					12	11	11
Legacy from the late Dr. George Thin					100	0	0
Miscellaneous Receipts					7	6	4
Fees for use of Epidiascope ...		51	7	0			
Less Operator's Fees		8	11	0			
					42	16	0
					£4911	4	9

LIABILITIES.

	£	s.	d.
3 *per Cent. First Mortgage Debentures* . .	33,600	0	0
[The Debenture Debt has been reduced by £100 since the previous statement, by cash paid in respect to Debentures redeemed.]			
Sundry Creditors	833	10	9
Balance, being Surplus of Assets over Liabilities viz.—	29,868	3	8
Balance, 31st December, 1903 £28,968 8 3			
Valuation of Epidiascope . 160 0 0			
Valuation of Stock of Suspended Animation Committee's Report . . 135 0 0			
Excess of Income over Expenditure for the year 1904 . . . . . 604 15 5			
£29,868 3 8			
	£64,301	14	5

ASSETS.

				£	s.	d.
Freehold and Leasehold Property				51,566	11	6
Fixtures, Fittings, and Furniture	1,406	0	9			
Less 5 per cent. written off for depreciation . . .	62	9	0			
				1,343	11	9
Engravings . . . . . (as per Valuation of Mr. F. B. Daniell, Aug. 19, 1896).				555	0	0
Contents of Library (as per Balance-sheet, Dec. 31, 1901)				8,792	14	7
Stock of 'Climates and Baths' .				320	15	9
Stock of Suspended Animation Committees' Report . .				135	0	0
Investment—"Permanent Endowment Fund" . . . (New South Wales 4 per Cent. Inscribed Stock).				326	7	3
Sundry Debtors for Rents and Outstanding Subscriptions .				349	1	4
Cash at Bank and in hand .				912	12	3
				£64,301	14	5

Audited and approved,

NEWSON-SMITH, LORD & MUNDY.

18th January, 1905.

W. SELBY CHURCH,
J. WARRINGTON HAWARD, } *Hon. Treasurers.*

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

SIR RICHARD DOUGLAS POWELL, Bart.

K.C.V.O., M.D.

PRESIDENT

AT THE

ANNUAL MEETING, MARCH 1st, 1905

Gentlemen,—The Report that has just been read, including the Treasurers' Report, shows that the Society is flourishing in this its hundreth year of existence, and that notwithstanding the lapse of our lease of the Berners Street house, by which we lose a rental of £450 a year, we have a handsome balance in our favour.

You will note with great satisfaction that His Royal Highness the Prince of Wales has intimated his intention of honouring the Society by his presence at the Centenary Banquet in May.

The Honorary Librarians' Report is of continued progress and expansion.

Of the 39 Fellows who are reported to have died, only a portion, still too large, have actually died within the twelve months. To their memory I must, as is customary in the President's address, devote a few words.

Sir Edward Sieveking, M.D., LL.D., F.R.C.P., Physician Extraordinary to the King; Consulting Physician, St. Mary's Hospital. Edward Sieveking was born in 1816, at St. Helens Place, in the City of London. He came of a good old North German stock, and was educated partly in England and partly in Germany. He studied medicine at Berlin, Bonn, University College London, and at the University of Edinburgh, where he took his M.D. degree. For some years after graduating he practised at Hamburg, chiefly among the English colony. He returned to London in 1847, and passed the examination for membership of the Royal College of Physicians, of which college he was in 1852 admitted a Fellow. In the preceding year he was appointed Physician to the Out-patients at St. Mary's Hospital, with which institution he remained actively connected for forty years. On his retirement he was elected Consulting Physician. He was for some time Physician to the Lock Hospital and to the Hospital for the Paralysed and Epileptic. In 1849 he issued his first publication in England, a pamphlet on nursing, and this advocacy had much to do with the great development which has resulted in the trained nurse of to-day. Sir Edward was one of the translators of Rokitansky's great work on pathological anatomy for the Sydenham Society, and afterwards, for the same society, translated Romberg's work on nervous diseases. In 1854 he published a work on 'Pathological Anatomy,' written in conjunction with his colleague, Dr. Handfield Jones. He also wrote one of the earliest and best works on life assurance. He was a Croonian Lecturer of the Royal College of Physicians in 1866, and in 1877 he delivered the Harveian Oration before the same college. In addition he was for some years editor of the 'Medico-Chirurgical Review,' a periodical founded by one of his most intimate friends, Sir John Forbes.

He was appointed Physician in Ordinary to His Royal Highness the Prince of Wales in 1863, Physician Extraordinary to Queen Victoria in 1870, and Physician in

Ordinary in 1888. He received the honour of knighthood in 1886. When King Edward VII came to the Throne, Sir Edward Sieveking was appointed Physician Extraordinary to His Majesty. At the tercentenary of the Edinburgh University he received the honorary degree of LL.D.; he was also a Knight of the Grace of the Order of St. John of Jerusalem. Sir Edward was elected to our own Society in 1848, was Vice-President in 1873–4, and President in 1888–9. It was during his Presidency that the premises we now occupy were purchased, and on his retirement he presented the Society with the President's badge. He contributed two papers to our 'Transactions,' and was Referee for a number of years.

Sir Edward Sieveking was a learned physician and a dignified figure in medicine. He married in 1849 the daughter of Mr. J. Ray, of Finchley, and had a large family. One of his sons, Mr. Herbert E. Sieveking, was formerly Surgeon to the Victoria Hospital, Cairo.

Sir Henry Thompson, Bart., M.B., F.R.C.S., Surgeon Extraordinary, H.M. the King of the Belgians; Consulting Surgeon, University College Hospital; a Fellow of this Society since 1862, a Councillor in 1869, and Vice-President 1888, and contributor of eight papers to our 'Transactions.' Henry Thompson was born in August, 1820, at Framlingham, in Suffolk, an only son of Mr. Henry Thompson of that place, his mother being a daughter of Samuel Smedly, the artist. He was originally intended for mercantile pursuits, and only entered at University College Hospital as a medical student in 1848, when already twenty-eight years of age. He was a brilliant student, and graduated as Bachelor of Medicine at the University of London in 1851, taking the Gold Medal in Surgery. He had become a Member of the College of Surgeons in 1850, and he took the Jacksonian Prize for an essay on "The Pathology and Treatment of Stricture of the Urethra" in 1852, and the Fellowship of the College in 1853. He married in 1851 Miss Kate

Loder, a lady of great reputation as a pianist. He commenced practice in Wimpole Street, where he lived all his professional and later life, and became surgeon to the St. Marylebone Infirmary. He was appointed Assistant Surgeon in 1853 to University College Hospital. In 1860 he again took the Jacksonian Prize for his work on 'Diseases of the Prostate.' He studied in Paris under the great French surgeon Civiale, who first practised the removal of stone from the bladder by crushing. He was thus ready for the great occasion of his life when it arrived. In 1863 he was asked by Sir William, then Dr., Jenner, with the approval of Sir James Clark, to go to Brussels to the King of the Belgians, who had long suffered from symptoms of calculus, and from whom a small calculus had been already removed by Civiale eighteen months previously. By his firm insistence and clear exposition Thompson at last prevailed upon the King to undergo the necessary examination, found, and completely removed a large calculus, and cured his august patient. He was appointed Surgeon Extraordinary to the King, and an officer of the Order of Leopold. He wrote, on this occasion, a graceful letter acknowledging his indebtedness to Civiale's teaching.

Success in professional position and practice and honours now rapidly came to him. He was elected full surgeon to the Hospital in 1863, Professor of Clinical Surgery in 1866, Surgeon Extraordinary to the Prince of Wales and Commander of the Order of Leopold in 1876, and the following year he received a knighthood.

In 1873 Sir Henry was called to operate upon another monarch for stone, but, under all the adverse circumstances of long existing disease, a broken constitution, and a nervous system depressed by great calamities, the Emperor Napoleon III died from uræmia before the final operation for complete removal of the calculus could be performed. In 1899 Sir Henry was created a baronet.

Sir Henry Thompson was a brilliant pioneer in the surgery of the bladder, and especially with regard to diseases

of the prostate and the removal of stone by lithotrity. His treatise on "Diseases of the Prostate" is a classic in our literature, and his lectures on "Diseases of the Urinary Passages" are models of simple and lucid exposition.

Sir Henry early cultivated a talent for painting, no doubt inherited from the maternal side. He studied carefully under the best masters, and exhibited from time to time at the Royal Academy pictures of considerable merit. A charming host, his delight was in his so-called octave dinners, his guests for each dinner being chosen to bring together men representing art, science, religion, literature, politics. Simple in his personal dietary, he was profoundly acquainted with foods and their preparation, and wrote some valuable works on methods of cooking. I was myself present at the 299th of these octave dinners, and I believe he gave three more afterwards.

Sir Henry Thompson read much, travelled not a little, and kept an open mind for the reception of modern ideas. It is rather singular that, with his thoroughness in this respect, he never grasped fully the methods of antiseptic surgery, although he never expressed any adverse opinions upon them. In 1874 he introduced cremation into this country by writing an article to the 'Contemporary Review,' and by holding at his house a succession of influential meetings, which resulted in the formation of a society for promoting cremation, of which he was the first president.

Sir Henry soon afterwards drew attention to the fact that death certificates, being unrecognised by any fee attached to them, were frequently drawn up in a very inadequate and irresponsible manner, and, although he did not effect any reform in this important unpaid duty of our profession, his advocacy will sooner or later bear fruit, for it is absurd that this valuable work for the State should have no emolument attached to it. In his later life Sir Henry wrote several novels, and his last accomplishment was motor-car driving. It was at the age of

eighty-four, three days after his last motor drive, that he slept away, leaving without suffering the world in which he had spent so strenuous and varied a life.

A very distinguished and many-sided man, he yet could not be said to possess the mark of true greatness. Nevertheless, the world has in many ways gained by his having lived in it, and, through his teaching and practice, a most distressing group of complaints has been brought more and more within surgical control. Lady Thompson only survived him by a few months. He was succeeded in the title by his only son, the present Baronet, a member of the Bar, who has gained distinction as an Egyptologist. He left also two daughters, the elder married to Archdeacon Watkins, of Durham, and the younger to the Rev. Henry de Candole.

Louis Leopold Jenner, M.B.Oxon., the fourth son of the late Sir William Jenner, G.C.B., died Thursday, June 2nd, at St. Thomas's Hospital, aged 38, of enteric fever contracted by infection from the bacillus which he was cultivating. A young life sacrificed in the battle of science against disease and for the welfare of humanity. He was educated at St. Thomas's and took the M.B., B.Ch.Oxon. in 1894, and the M.R.C.P.Lond. 1899. He was elected a member of our Society in 1897.

He contributed various papers on pathological matters, and introduced a useful blood-staining reagent.

Alexander Oberlin MacKellar, F.R.C.S., died on June 15th at Bella Vista, Royston Park, Pinner, aged 58. He was Surgeon to St. Thomas's Hospital, and Chief Surgeon to the Metropolitan Police. He was educated at Silcott's Grammar School and Owens College, Manchester; he also studied at Queen's College, Belfast, University College, London; Paris and Vienna. He took the diploma of L.S.A. and M.R.C.S.Eng., 1867, and graduated as M.D. and M.Ch., Royal University of Ireland in 1869.

After some years of travel he served in the Franco-

Prussian War as Ambulance Surgeon, and was present at the battles of Metz and Sedan. At the close of the war he accompanied Surgeon-General Langenbeck on his official inspection of the military hospitals in Germany and France occupied by German troops. For the important services he rendered he was made a Knight of the Military Order of Merit of Bavaria. In 1873 was elected Resident Assistant Surgeon to St. Thomas's. At the age of forty-one he became full Surgeon, and when he was forty-eight Senior Surgeon on the Staff. He held the posts of Lecturer on Practical Surgery and Teacher of Operative Surgery for twenty-two years. He was an excellent teacher and a skilful operator. In 1885 he succeeded Mr. Timothy Holmes as Surgeon-in-Chief to the Metropolitan Police, which appointment he held at the time of his death. Mr. MacKellar married late in life and left no family.

John Birkett, F.R.C.S., F.L.S., Consulting Surgeon to Guy's Hospital, Corresponding Member of the Société de Chirurgie of Paris. Died July 6th, of apoplexy, aged 89, at his residence in Sussex Gardens. He was a greatly-respected Fellow of this Society, having held the offices of Hon. Librarian, Secretary, Councillor, Treasurer, and Vice-President, and contributed eight valuable papers to the 'Transactions.'

Educated privately until his entry at the Medical School, Guy's Hospital, in 1831. He was elected a Member of the Royal College of Surgeons in 1844, and was its President in 1877. As a lecturer and teacher Mr. Birkett was exact and methodical, and his note-taking in the wards was a model of what such should be. Precise and somewhat cold and formal in his demeanour, and lacking in the sense of humour, yet he was, to those who knew him well, a pleasant companion and a sincere friend. He was a fair botanist, and a man of wide general culture. His wife predeceased him by some years. He had ten children, of whom six survive.

Sir John Simon, K.C.B., F.R.S., who died July, 1904, aged 88, will ever be remembered as a powerful and strenuous worker in that most beneficial branch of our profession which seeks to prevent disease and to improve the physical and moral conditions of life. Mr. Simon may be said to have inherited the French tongue from his two grandfathers, who were both Frenchmen who married English wives, and he acquired a knowledge of German by living as a pupil in a German pastor's family for ten months. He commenced his professional life at the early age of seventeen, as a pupil of Joseph Henry Green, both at St. Thomas's and King's College Hospitals, and at the latter hospital he attained the position of Assistant Surgeon, and the colleagueship of Bowman, Ferguson, and Partridge. He seems to have been greatly influenced by Bowman, by whom he was induced to withdraw from more abstract studies of metaphysics and oriental languages in favour of more definite professional work. And in 1844 he gained the Astley Cooper Prize for an essay on the "Physiology of the Thymus Gland," and the following year a communication on the "Comparative Anatomy of the Thyroid Gland," gained for him the Fellowship of the Royal Society. He then became more definitely attached to St. Thomas's as Lecturer on Pathology, and as Surgeon to the Hospital, and remained associated with that hospital to the end of his professional life. He was President of the Pathological Society in 1867, and I remember the impressive occasion on which he announced and illustrated by the results of his own experiments on rabbits the demonstration by Villemin in 1865–6, of the inoculability of tubercle.

The great work of his life, however, was taken up in 1848, when he was appointed the first Medical Officer of Health to the City of London, and seven years later Medical Adviser to that Department of the Government which developed into the Local Government Board. The brief, incisive and picturesque introductory essays to the successive Blue Books on the Public Health, which ap-

peared during Simon's long tenure of office, were delightful and instructive reading to all sanitarians, and will ever remain classics in that section of medical literature. Simon's writings, which were very numerous, of which that on English Sanitary Institutions is the most important, are characterised by a profound pathological knowledge, a penetrating philosophy, and a singularly lucid and picturesque style very rarely associated with this class of literature.

He was President of the Royal College of Surgeons, Vice-President of the Royal Society, Vice-President of the Royal Medical and Chirurgical Society, and he received countless honours in his long working life, all appropriately and well deserved.

Sir John Simon was a man gifted with true genius and inspired with the love of his kind. He enjoyed a restful and a dignified old age, and will ever remain a noble figure in the medicine of the nineteenth century, and will live in history as the apostle of sanitation.

He married the daughter of Deputy Commissioner-General O'Mara, who predeceased him by three years. He left no family.

T. Gilbart Smith, M.D., F.R.C.P., Physician to the London Hospital; Physician to the Royal Hospital for Diseases of the Chest, died suddenly of heart failure whilst cycling with his son in Devonshire. He graduated in Arts and Medicine at Trinity College, Dublin; then studied at St. Bartholomew's and Paris.

Of handsome and engaging presence and genial manner, generous to a fault, and gifted with that touch of humour which adorns so many of his nationality, Dr. Gilbart Smith was a very popular man at the London Hospital. He was a good teacher, shrewd in diagnosis, a sound and good physician. He contributed but little to the literature of medicine. He read a paper on "Pharyngeal Stenosis," which is published in our 'Transactions.'

He married Miss Bewley, a lady of Irish descent, and

had three sons who survive him, one of whom is in the profession, and one daughter who died a few years ago.

He had had one or two anginal attacks, of which he and a few friends were aware, and it was with an attack of anginal heart failure that he suddenly died. His later life had been much clouded by financial anxieties, partially owing to calls upon shares in an unlimited company which had failed.

Sir William Mitchell Banks, M.D., LL.D., F.R.C.S.Eng., J.P., Liverpool, elected a non-resident Fellow of our Society in 1886. By the death of Sir William Banks the profession has lost, in the maturity of active usefulness, a man whose attractive personality, versatile abilities, many accomplishments, and influence over other men had long secured for him a leading position in Liverpool. He was born in Edinburgh, in 1842, where his father was a writer to the signet, was educated there, and took the M.D. degree with honours at the University in 1864. His thesis on the "Wolffian Bodies" brought him into notice, and gained the Gold Medal. He was Prosector of Anatomy to Professor Goodsir, and a pupil of Syme, Simpson, and Hughes Bennett. After a short sojourn abroad he returned and became Demonstrator of Anatomy in the University of Glasgow, but soon afterwards again moved, to finally settle in Liverpool, the attraction to him there being an appointment as private assistant to Mr. Bickersteth. Mr. Mitchell Banks became Lecturer on Anatomy at the Medical School and Surgeon to the Royal Infirmary.

As Dean of the School he worked hard, with other distinguished contemporaries, most of whom survive him, to develop the School, and later to found the University College, and finally, last year, the efforts of himself and his colleagues were crowned by the achievement of a Royal Charter founding the University of Liverpool.

Sir W. Mitchell Banks, as a member of the General Medical Council, as a member of the Council of the Royal

College of Surgeons, Lettsomian Lecturer, Orator of the year to the Medical Society of London, and in many other prominent positions exercised an influence over his professional brethren at large. He was an advocate rather than a writer, influencing men by his magnetic personality rather than through the cold logic of the written page.

He was a Justice of the Peace for the City of Liverpool, was created a knight in 1893, and LL.D. of Edinburgh in the same year.

William Lee Dickinson, M.D., F.R.C.P., Assistant Physician, St. George's Hospital, London. The Hospital and School of St. George's Hospital have sustained a severe loss, and a career of high promise has been prematurely brought to a close by the death of Dr. William Lee Dickinson, which took place at his father's house, Trebrea Lodge, Tintagel, Cornwall, on September 6th. He was the son of a distinguished father, Dr. Howship Dickinson, and bade fair to succeed to no small part of his reputation. He was a member of Caius College, Cambridge, from which he came to study at St. George's Hospital in the year 1882. He took his M.B. degree at Cambridge in 1890. In the same year he was elected Medical Registrar to St. George's Hospital, and filled this office till 1893, when he was appointed Curator of the Museum, and was elected upon the staff on the retirement of his father. He was made a Fellow of the Royal College of Physicians in 1894. In 1898 he succeeded Sir Isambard Owen as Lecturer in Toxicology and Forensic Medicine. In 1895 he was appointed Assistant Physician to the Hospital for Sick Children, Great Ormond Street, again following in the steps of his father. Dr. Lee Dickinson was an indefatigable worker, and during the early years of his connection with St. George's Hospital contributed to medical literature a number of valuable papers. In October, 1898, he developed symptoms of tuberculosis of the lung, but after spending

a winter in Africa returned apparently cured, in greatly improved health. Although urged at this time to pursue a less strenuous professional life in the country or in one of the colonies, he deliberately preferred the shorter, more interesting, and perhaps more useful career in London in pathological and clinical work. It was whilst spending, in the midst of his work, a short vacation at his father's residence, in Cornwall, that, without any previous warning, a sudden and severe attack of hæmoptysis ended his life. We can but respect his heroism and tender our respectful sympathies to his father, a former President of the Society, in the loss of a son so worthy of him.

Herbert William Allingham, F.R.C.S., Surgeon to His Majesty's Household; Surgeon in Ordinary to the Prince of Wales. Herbert Allingham died in the zenith of his fame and apparently in the full tide of usefulness, the friend of most of us. An able teacher, a successful surgeon, a winning friend, in all his outer life and projects things went well with him. But the strain of the day's hard work and urgent anxieties had been for many years attended with private grief and restless anxiety owing to the long illness of his wife, to whom he was devotedly attached. The anxiety had ceased with her death, but the grief remained, and free from all responsibilities to others, without children dependent upon him, without indebtedness to any, the rest prematurely sought by the tired man carries only the reproach of having been sought before the life's work was fairly done; perhaps not even this, for in consequence of an accidental blood-poisoning, contracted in the course of professional duty, his health had failed; he was haunted with the dread of failing power of work, and the end came to him in a period of great gloom and depression.

He was the son of the very distinguished surgeon still living, but now retired from practice. He was educated privately and at University College School, and at St.

George's Hospital. He took the Fellowship of the Royal College of Surgeons in 1887, was elected Surgeon to the Great Northern Central Hospital, Demonstrator of Anatomy to St. George's Hospital. He next became Surgeon to St. Mark's Hospital and to St. George's Hospital in 1894.

Following his father's practice, he utilised his large opportunities for becoming expert in diseases of the rectum and bladder, so that he soon acquired a first-rate reputation in the surgical treatment of these maladies. With his attachment to St. George's, however, and the wider field he enjoyed there in practice and teaching, his reputation extended as an all-round surgeon. He contributed no paper to this Society, but he wrote a book on operative surgery, and contributed a valuable article to Professor Allbutt's 'System of Medicine' on "Diseases of the Rectum." He was appointed Surgeon in Ordinary to the Prince of Wales in 1902, and Surgeon to His Majesty's Household in the same year.

Mr. Allingham was a man of handsome presence and refined demeanour, gentle and tactful in manner, with a quiet reserve of power that inspired ready confidence. His memory remains dear to many, especially to students of St. George's Hospital who appreciated his teaching.

George Vivian Poore, M.D., F.R.C.P., Emeritus Professor of Medicine, University College, London. George Vivian Poore, the youngest son of Commander John Poore, R.N., was born at Andover on September 23rd, 1843, and was educated at the Naval School, New Cross, and at University College, London. After qualifying at the College of Surgeons, he went out in 1866 as Medical Officer to the "Great Eastern" on the voyage to lay the first Atlantic cable. On his return he finished his student's career at University College, gaining the Atkinson-Morley Scholarship, and taking the B.S. and M.B. at the London University with the University Scholarship in Medicine.

He became M.D. in 1871, and a Fellow of the Royal College of Physicians in 1877.

He travelled abroad with the Prince of Wales (now King Edward VII) after his recovery from enteric fever in 1872, and whilst abroad he received the Order of the Dannebrog from the King of Denmark for his attendance upon the Princess Thyra. From 1870 to 1877 he attended the late Duke of Albany (Prince Leopold). He next became Assistant Physician, Professor of Medical Jurisprudence, and subsequently full Physician, and Professor of Clinical Medicine at University College, of which, on account of his distinguished university career, he had already been made a Fellow.

Dr. Poore was a bachelor, and lived for many years with that distinguished surgeon, Mr. Marcus Beck. With adequate means, and rich in the poverty of his wants, he enjoyed a fair practice, but he more enjoyed leisure to pursue the studies which interested him. He was particularly interested in the science of hygiene, was a great authority on the disposal of sewage, and very eloquent on the wasteful measures adopted for our large towns. He had a small property at Andover, and his garden there was a miniature sewage farm, conducted on the most productive and hygienic principles.

Dr. Poore was a man of great ability, great versatility, and keen dramatic instinct. His long and close association with his able and inspiring friend, Marcus Beck, perhaps led him to acquire that gentle cynicism which gave a piquancy to his conversation without clouding his sincerity. With all his knowledge, his was a whimsical mind, quaint and humorous in its conceptions, but keen as the razor's edge. He had the great gift of exposition derived from his dramatic method of detecting the salient points of any subject and portraying them, often with just that touch of exaggeration of the caricaturist that makes the sketch stand out salient from the shading. He was much too proud a man to be self-seeking, a very sincere friend, and a charming companion. Amongst his varied and

useful contributions to the professional literature of his time, Poore will be remembered for his ardent advocacy of the great truth that it is through the microbic agency of the living humus that vital force is resuscitated from putrescent organic matter.

> "To my bosom I fold
> All my sons when their knell is knolled,
> And so with living motion all are fed,
> And the quick spring like weeds out of the dead."

So spoke the great Shelley for mother earth a hundred years ago, so was Poore never tired of teaching and demonstrating to his life's end. He died in his little country home, which he had made luxuriant by his scientific methods of cultivation.

Dr. Poore was elected a Fellow of this Society in 1871, had been on the Council, and was a Referee and Member of the Library Committee shortly before his death. He contributed two papers to the 'Transactions.'

Dr. Achille Vintras died November 9th, aged 75. Of French extraction and education, he was Physician to the French Embassy, and Senior Physician to the French Hospital. He studied medicine at St. Mary's Hospital and in Paris, and took the degree of M.D. at the University of St. Andrew's, 1856. He was an Officer of the Legion of Honour and an Officier d'Académie. He was the author of a Medical Guide to the Mineral Waters of France and its Wintering Stations, besides contributing to medical periodicals.

Dr. Vintras was greatly esteemed at the French Embassy, and enjoyed a large practice amongst the French residents and visitors in London.

Charles Douglas Fergusson Phillips, M.D.Aber., F.R.C.S. Edin., F.R.S.Edin. Dr. Phillips died on November 13th, aged 74. He graduated as M.D. at Marischal College, Aberdeen; in 1859 started practising in Manchester, and in 1867 moved to London. In 1878, owing

to an injury received in a railway accident at Nine Elms Junction he became paralysed and had to retire, receiving substantial damages from the railway company. In 1883 he again commenced to practice, and continued to do so until 1903. In 1892 he published a book entitled 'Materia Medica, Pharmacology, and Therapeutics,' a third edition of which was issued in 1903. Only a few months before his death, he was invited to stand as Parliamentary candidate in the Conservative interest for the Universities of Glasgow and Aberdeen, an honour which, owing to his advanced age, he declined.

Arthur Quarry Silcock, M.D., B.S.Lond., F.R.C.S.Eng., Surgeon and Joint Lecturer on Surgery to St. Mary's Hospital, and Surgeon to the Royal London Ophthalmic Hospital. The untimely death of Mr. Silcock from appendicitis at the comparatively early age of 49 removed from amongst us an able surgeon whose work as a teacher and whose strenuous and loyal performance of all his official duties renders his loss a severe one to St. Mary's Hospital. He was educated at University College, London, and took the M.D. degree, as well as the Bachelor of Surgery, at the University. He preferred surgery in practice, however, and became a Fellow of the Royal College of Surgeons. He was in 1884 appointed Pathologist and Assistant Surgeon to St. Mary's, and in due time Surgeon and Lecturer on Surgery.

He was elected a Fellow of this Society in 1886. Mr. Silcock has left behind him the reputation of having been an upright and a downright man, a very able Surgeon, a sound Pathologist, and a clear and successful Teacher. He was one of those Surgeons of whom there are few left, who, like the late Mr. Hulke and Mr. George Lawson, combined a profound knowledge of general surgery with a special study of ophthalmic diseases. Perhaps in these days of increased labour from greater knowledge the combination is well nigh impossible for a man of average strength and energy, and the hurry and stress

of work had probably much to do in producing the appendicitis of which he died.

Mr. Silcock was married, and his wife and two children survive him.

Dr. Dan Astley Gresswell, M.A., M.D.Oxon. and Melbourne. Dr. Gresswell was a native of Lincolnshire, and was born in 1853, the second son of the late Alderman Gresswell, at one time Mayor of Louth. He died at Melbourne on December 10th. He went to Oxford as the winner of an open scholarship at Christ Church, and took a First Class in Physical Science in 1875. After working at physiology at Bonn and Würzburg he entered at St. Bartholomew's, and in 1881 became M.R.C.S.Eng., and M.B.Oxon., and four years later D.P.H.Camb. He was elected a Fellow of this Society in 1882. He was appointed Medical Adviser to the Board of Health of the Colony of Victoria in 1890, and took up his residence in Melbourne, where he took a leading part in all measures of sanitation, and was Lecturer and Examiner on Therapeutics, Dietetics, and Hygiene at the University.

Dr. Rustomjee Naserwanjee Khory, M.D.Brux., L.R.C.P. Lond. Dr. Khory was of Persian descent and religion. He obtained the diploma of L.R.C.P.Lond. in 1870 and graduated M.D.Brux. He completed his course in the Grant Medical College, and became a Licentiate in Medicine of the Bombay University in 1864. He was Dean of the Faculty of Medicine of the University of Bombay, and was the author of a unique work entitled "Bombay Materia Medica and Therapeutics," published in 1887. He died December 29th, 1904, at his residence in London, aged 65.

Luther Holden, F.R.C.S., Consulting Surgeon to St. Bartholomew's Hospital, died at Putney, in February last, in his 90th year. He was of good old family. His father, a clergyman, had eight children, of whom Luther

was the second son. Mr. Holden never went to a boarding school, but was educated at home, chiefly by his father who was a graduate of Oxford, and had previously served in the Army. At the age of seventeen he went to St. Bartholomew's Hospital as a pupil of Stanley, then teacher of anatomy, whose attention and friendship he attracted by his skill in dissections. He spent a year in Berlin, and another in Paris, where he made a special study of anatomy. He became Member of the Royal College of Surgeons in 1838, and became very successful in preparing private pupils for the College examinations. He was in the first group of men, and was the last survivor of the group, who took the Fellowship of the College by examination. Holden was elected Demonstrator of Anatomy at St. Bartholomew's in 1846, Assistant Surgeon in 1860, and full Surgeon in 1865. He never distinguished himself as a surgeon, but was unequalled in his powers as a teacher of anatomy, regional anatomy, and clinical surgery. His work in life was educational, and his publications, with the exception of an original paper on the "Mechanism of the Hip-joint," were on educational lines.

His 'Manual of Dissections' and his work on 'Human Osteology' were universally read by students for thirty or forty years. He adopted an original plan of marking the attachment of muscles on his drawings of the skeleton. His treatise on 'Medical and Surgical Landmarks' has been a much-read book and a handy guide, both to the student and to the practical surgeon for more than one generation. He lectured on anatomy for thirteen years, and remained Surgeon to the Hospital until 1881, when he retired as Consulting Surgeon. He subsequently travelled much, visiting Australia and the Cape. In 1898, in his eighty-third year, he visited Johannesburg, and was warmly received by his medical brethren there.

Mr. Holden was for many years an Examiner at the Royal College of Surgeons. He was President in 1879,

and to a very late period of his life he frequently visited the College on special occasions.

He was twice married, and his second wife survived him. He had no children. Mr. Holden was a particularly distinguished-looking man, tall, upright, extremely handsome, of courtly manners, and an accomplished linguist. He was fond of country pursuits, and a good horseman, frequently seen in the hunting field. There is a fine presentation portrait of him by Millais in St. Bartholomew's Hall.

Dr. Mirza Ali, M.D., of Teheran (Mr. Mehor Khan, of the Persian Legation, has been kind enough to send me the following account of Dr. Mirza Ali's life and works, which he obtained from a Persian gentleman). Dr. Mirza Ali, M.D., of Teheran, was born in 1255 (about 1837) at Hamadam, in the East of Persia. At an early age the medical profession seemed to have a great fascination for him; his father, seeing this, sent him to the most learned physician of that day, and also had him instructed in the French language.

At the age of twenty-four he joined the Military College of Teheran. After taking his degree he became, in due time, Professor at the Medical School, also writing and translating books on medical subjects. Besides all this, the surveillance of all the public hospitals came under his domain.

At the age of forty-nine he became personal Physician to the late Shah, and was at the head of the medical profession in Persia. He visited London in 1866, returning to Teheran with all the latest improvements in everything belonging to his profession. He died there at the age of sixty-eight, missed by many, and very deeply regretted.

The works translated by the late doctor were chiefly on pathology and surgery, copies of which (or some of them) he presented to the Royal Medical and Chirurgical Society when he was in London seven or eight years ago. The

late Mirza Ali did also a good work in building one or two hospitals in Teheran for the poorest classes of the population. He took a great interest in these hospitals, to which he devoted much of his time and energy.

I recognise that the year upon which we are now entering will be a somewhat anxious and difficult one, as it will also be a very auspicious one, the opening year of a new century of work. I feel that we may safely rely upon the strenuous and loyal endeavours of your Hon. Secretaries, and upon the watchfulness and thoughtful initiative of your Council.

We have also in Mr. MacAlister, our Secretary, who so zealously manages our affairs and carries out the wishes of the Council, not only executive ability but a suggestiveness which in a permanent officer is most valuable. I anticipate, indeed I hope, in the coming year for developments in the constitution of our Society, which if wisely and sympathetically guided cannot fail to help on the great objects with which it was founded, viz. "for the cultivation and promotion of Physic and Surgery and of the branches of Science connected with them." With the strong Fellowship which the Society now presents, with its sound financial position, its able Council and energetic officers the Society should be in good keeping and guidance for any measures the Fellows may decide to take in hand.

In this, the last year of its Centenary, our Society has fairly maintained the high level of preceding years. To give a retrospect of the work of the Society in the past century would be out of place on this occasion, it would involve an account of the history of Medicine during that period. For there are few advances in Medicine or phases of medical thought which have not been foreshadowed or reflected in our transactions. How many great problems, most of them now solved, crowd themselves even within the recollection of my own professional lifetime, as having been initiated or developed at meetings

of this Society; ovariotomy, the antiseptic principle, the open air treatment of Phthisis, the treatment of the apparently drowned, the nature and significance of raised arterial pressures, the clinical use of the thermometer, the exact effects of the digitalis class of drugs upon the heart and arteries, the presence of leucocytosis in suppuration and its clinical value, the surgical treatment of appendicitis, are amongst them. But it is not now that I would deal in retrospect. We are not yet in our Centenary Festival, when a note of jubilation may fittingly be struck over the doings of the past. We have arrived at the eve of a new century of endeavour, and must perforce ask ourselves, Are we content with our present position and outlook? Shall we by continuing on the same lines be doing our best for the Medicine of the next century?

Our finances are flourishing, never more so, and we may justly congratulate ourselves upon our sound position in this respect. Were I the chairman of a financial or business concern, Medicine, Surgery, and Co., I should be content to make the usual flattering comments upon our sound prosperity. But whilst it is our duty to look to the upkeep and business interests of our property, we have behind us a great record to maintain in the promotion of every department of Medicine and Surgery and of the allied Sciences in relation to them. If, therefore, as may well happen, some modifications of our constitution or methods be required to meet the needs of the coming century we should give them our earnest thought. In the course of time the conditions of medical science and of professional life have changed. There has been great development is science, in practice, and in diffusion of knowledge.

Ours is the leading society of medicine enjoying Royal prestige. But many other societies have sprung up which are doing much the same work as we are doing, are catering for much the same papers as we are seeking. These societies—I myself am an original member of one

of the most vigorous of them—were called into existence to supply a want. Let us ask ourselves, did that want arise from any lack of watchfulness on our part or any defect in our adaptability to meet the needs of new developments and progress? We must not allow ourselves in our corporate being to grow old in a hundred years, however well qualified for euthanasia we may as individuals become in half that time. Well, we harbour several of these societies at a rental as tenants; I would ask ought they not rather to be our guests, or better still, ought we not to be of them and they of us? We are all working in harmony with the honest desire to advance our most interesting and beloved calling, but I confess I am inclined to think that the function of this Society, which is concerned with the mere reading of papers, and which it has in common with several other societies, might be usefully modified in some respects. The papers are of value as before, their merit is well sustained, but many to whom I have spoken admit, and it has, I believe, long been felt, that there is a great overlapping of work at many points, and a corresponding waste of force amongst the great central medical societies of London. Many important questions are dealt with in fragments, and there is a great want of co-ordination and economy.

Let me glance for a moment at the point of economy. A number of London men are paying annual subscriptions of from five to seven or nine guineas, besides initial entrance fees, in order that they may belong to societies representing several branches of work. Many others can only join one society when they would like to take a wider range, and we must remember it is the younger men, and often the best men, that are thus shut out; men full of enthusiasm and working power, and with time and opportunities for producing that output of original work that feeds our societies, and advances the front line of medical science. Our magnificent library and fine rooms are open to but a small proportion of these men, on account, mainly, of the large subscription. Yet by combination

the annual subscription admitting to all the central societies of London with library, reading and conversation rooms might perhaps easily come well within the subscription to this Society alone.

Then in point of economy of work; there must nowadays be more numerous divisions in medical and surgical work than 100 or even fifty years ago, but why should there be several departmental societies holding meetings to discuss the same, or much the same, questions and cases? Why should there not be a central Royal Society of Medicine open to all, with sections representing the great divisions of medicine, each section in the enjoyment of its own autonomy, yet co-ordinated with the central body? Would not cases and questions thus be discussed better and before larger and more representative audiences? And, instead of the central body also discussing papers in fortnightly meetings, might it not be more advantageous to have three or four general meetings each year for the full consideration of such larger questions as may from time to time ripen for such treatment in the course of the sectional work? We had a good example of the method I have in mind of dealing with such questions in the discussion on the 'After-consequences of Operations for Appendicitis,' which was introduced by Sir F. Treves last evening.

Again, as regards transactions, if, instead of the costly volumes now annually published by each society, which are placed on our bookshelves to remain there for the most part unopened, the transactions of the different sections were brought out in monthly fasciculi throughout the eight or nine months of the working year, a considerable economy would be effected, and a much more vital interest would attach to them. Perhaps, too, a kind of 'Medico-Chirurgical Review' might once or twice in each year be published, co-ordinating and collating the work of each section so as to review the position of science achieved.

Gentlemen, I have indicated, I trust not too lengthily,

at least one further step by which this Society, in conjunction with others might better carry on the objects of its foundation, viz. by bringing about a Federation of the central societies in London. I am aware that there is nothing new in the idea; it has been already attempted three times, two of the three occasions being within the recollection of many of us. Nevertheless, in my opinion, and in that of many Fellows of this and of other important societies, the present is a very favourable time for making another attempt. I do not think such an amalgamation has ever been thought out fairly on business lines, and I have reason to believe that it would be found to be entirely feasible on such lines. It can only be effected, however, with the full sympathy of all the societies concerned.

I would further say that I am only expressing my own views on this matter, with no mandate from the Council. It is, however, only after a careful consideration of the subject, and some careful inquiries that have been made into its merits, that I have ventured again to raise the question of a possible federation. I must leave to you the decision whether any movement of the kind should be started.

I very sincerely thank you for the kind attention with which you have favoured me.

THE SECRETARY'S OFFICIAL REPORT UPON THE SOCIETY'S CENTENARY, MAY 22ND, 1905.

20, HANOVER SQUARE, W.,
May 26th, 1905.

TO THE PRESIDENT AND COUNCIL OF THE ROYAL MEDICAL AND CHIRURGICAL SOCIETY.

GENTLEMEN,

I have great pleasure in reporting to you that the programme prepared by the Centenary Committee and approved by you has been carried out with entire success.

HISTORY OF THE SOCIETY

This volume of 337 pages, the work of Dr. Norman Moore and Mr. Stephen Paget, was delivered to Fellows on Saturday, May 20th, as a fitting introduction to the Centenary programme of the following week. It is perhaps worth mentioning that the entire volume was printed off and bound within seven days. The first plate is a reduced copy of the drawing[1] made for the Banquet Toast List by Mr. Bernard Partridge (son of a former President); the second is a reduction in photogravure of a mezzotint portrait (by L. Abbott) of its first President, from the Society's own collection. The remaining plates, illustrating the various homes of the Society, are from drawings specially made for the purpose.

On Centenary Day, Monday, May 22nd, at 11 a.m., the

EXHIBITION

(of which I submit a catalogue) was opened, and was inspected by a large number of Fellows and visitors.

The Catalogue was necessarily very hastily compiled, as some exhibits were received only two days before opening, and

[1] This original drawing has been framed and suspended in the Corridor.

it was entirely printed off and bound in one day, *viz.*, Saturday, May 20th. It was much appreciated, and I have received many requests for copies since the exhibition closed.

CENTENARY MEETING

At 4.45 p.m. the PRESIDENT received the Honorary Fellows and Fellows, and light refreshments were served.

At 5 p.m. the company entered the Meeting Room; the PRESIDENT took the chair, and invited the ex-Presidents who were present to take seats on the platform.

The PRESIDENT announced that—

HIS ROYAL HIGHNESS THE PRINCE OF WALES, K.G.,

had signified his willingness to accept the Honorary Fellowship, and he was forthwith unanimously elected an Honorary Fellow.

The PRESIDENT reported that as Lord Lister was not well enough to attend the Centenary Meeting, the officers of the Society had, on the previous Saturday, attended at Lord Lister's house, and in the name of the Society the President had admitted him to the Honorary Fellowship. Lord Lister had desired him to convey his affectionate greetings and congratulations to the Society, and his grateful thanks for the honour they had conferred upon him.

The HON. SECRETARIES introduced to the President the following newly-elected Honorary Fellows:

Professor CHRISTIAN BOHR (of Copenhagen).
Sir WILLIAM RAMSAY, F.R.S.
Dr. ROBERT BARNES.
Dr. W. H. GASKELL, F.R.S.

These signed the obligation and were formally admitted by the President, who presented each with his diploma.

The PRESIDENT delivered an address.

Dr. NEWTON PITT (Senior Hon. Secretary) read telegrams and letters of congratulation which had been received.

Professor CHIENE, President of the Medico-Chirurgical Society of Edinburgh, presented on behalf of that Society a beautifully illuminated address, and in a brief but eloquent speech offered his personal congratulations.

The congratulations of the following societies were also tendered:

The Medical Society of London, by its President, Mr. John Langton.

The Clinical Society of London, by its President, Dr. Frederick Taylor.

The Obstetrical Society of London, by Dr. Robert Boxall

(Hon. Secretary) in the unavoidable absence of the President.

The Laryngological Society of London, by its President, Mr. Charters Symonds.

Mr. JONATHAN HUTCHINSON, the senior ex-President present, moved a cordial vote of thanks to the President for his address, with a request that he would allow it to be printed.

This was seconded by Mr. THOMAS BRYANT (also an ex-President) and carried by acclamation.

The PRESIDENT briefly thanked the meeting, and proposed that a telegram of greeting be sent to the absent Honorary Fellows, and this proposal was unanimously accepted.

BANQUET

This was held in the Hotel Cecil in the evening of Centenary Day.

Before the banquet there was a special meeting of the Council in a room of the hotel, at which His Royal Highness the Prince of Wales was introduced by the President.

His Royal Highness signed the Obligation as an Honorary Fellow, and was formally admitted by the President, who then presented him with a specially-bound Diploma of Honorary Fellowship and also a very handsomely bound copy of the Centenary volume.

At 8 o'clock the Council, preceded by His Royal Highness and the President, adjourned to the Banquet Hall.

There were present 457 guests and Fellows. I submit plan of tables and list of those dining, together with copy of menu, for which Mr. Bernard Partridge designed the very beautiful symbolical frontispiece.

After dinner the Prince retired to another room with the Officers and Council, the new Honorary Fellows, and a few others whom he honoured with an invitation, and remained until nearly midnight.

TUESDAY, MAY 23RD.

MARSHALL HALL ADDRESS

The Ordinary Meeting of the Society, which fell on this day, was devoted to the delivery of an address by Dr. Henry Head, F.R.S., the Marshall Hall prizeman of 1903, on

THE AFFERENT NERVES UNDER A NEW ASPECT.

The address was illustrated by numerous diagrams projected by the epidiascope. There was a large attendance of Fellows, Hon. Fellows, and visitors.

After the meeting refreshments were served, and there was an informal reception by the President.

Wednesday, May 24th.

CONVERSAZIONE

This was held at the Natural History Museum, Cromwell Road.

The President and Lady Powell received the guests from 9.30 onwards, assisted by the Vice-Presidents and other officers and their wives. The Museum was especially illuminated for the occasion, and was beautifully decorated with flowers and palms.

In the Great Hall the Royal Artillery Band played, while in a side gallery the Meister Glee Singers sang through a charming programme. The refreshments were provided by Messrs. Lyons, who appear to have given entire satisfaction.

Upwards of 2,500 persons were present, and the last guests did not leave until after 12 o'clock.

This completed the Centenary Committee's programme, except that, for the sake of those who were too much engaged to visit it earlier in the week, the Exhibition was kept open throughout Thursday, and had numerous visitors.

The special thanks of the Council will doubtless be given to all those who lent objects of interest—some sent from long distances and of great value. All the exhibits were underwritten at Lloyds' and covered for full value (fixed by the exhibitors) during exhibition and transport till safe in the owners' hands.

I have prepared a detailed report of the Centenary Celebration for inclusion in the 'Transactions.'

The accounts are being prepared, and I hope to present a balance-sheet very shortly.

I have the honour to be, Gentlemen,

Yours very faithfully,

J. Y. W. MacAlister,

Secretary.

THE

ROYAL MEDICAL AND CHIRURGICAL SOCIETY.

PRESIDENT—SIR RICHARD DOUGLAS POWELL, BART., K.C.V.O., M.D.

CENTENARY MEETING

HELD AT THE SOCIETY'S HOUSE, No. 20, HANOVER SQUARE, LONDON, W., MONDAY AFTERNOON, MAY 22ND, 1905.

BEFORE the meeting there was an informal reception by the President and Officers to welcome the Honorary Fellows.

At 5 o'clock the President took the chair, supported on the platform by the ex-Presidents who were able to be present.

The PRESIDENT announced that

HIS ROYAL HIGHNESS THE PRINCE OF WALES, K.G., had signified his willingness to accept the Honorary Fellowship, and

His Royal Highness was unanimously elected.

The PRESIDENT reported that as Lord Lister was not well enough to attend the Centenary Meeting, the officers of the Society had, on the previous Saturday, attended at Lord Lister's house, and in the name of the Society the President had admitted him to the Honorary Fellowship. Lord Lister had desired him to convey his affectionate greetings and congratulations to the Society, and his grateful thanks for the honour they had conferred upon him.

The Hon. Secretaries introduced to the President the following newly-elected Honorary Fellows:

Professor Christian Bohr (of Copenhagen).
Sir William Ramsay, F.R.S.
Dr. Robert Barnes.
Dr. W. H. Gaskell, F.R.S.

These signed the Obligation and were formally admitted by the President, who presented each with his diploma.

The President then addressed the meeting.

Presidential Address.

Gentlemen,—It is naturally expected that the President should address a few congratulatory remarks on the attainment of the hundredth birthday of this great Society. Time only permits me to be very brief, for much has to be done to-day. I will therefore express, on the part of the past Presidents of this Society now living—Mr. T. Holmes, Mr. J. Hutchinson, Dr. W. H. Dickinson, Mr. Thomas Bryant, Dr. F. W. Pavy, and Mr. A. Willett—and on the part of the Council and myself, our most hearty congratulations to the Fellows of the Society on the completion of the hundredth year of its corporate life, and our most cordial welcome in the name of the Society to the distinguished Honorary Fellows and to the guests who honour us with their presence during this centenary celebration. The Royal Medical and Chirurgical Society was founded in 1805 for the purpose of conversation on professional subjects, for the reception of communications, and for the formation of a library. It was an offshoot from the Medical Society, the particular occasion for which could scarcely arise in modern times and from the effects of which that Society soon recovered. In 1808, indeed, showing that no ill-feeling was left behind by the severance, a proposal emanated from our sister, or rather our parent, Society for a reunion to form a new Society of Medicine. It is perhaps to be regretted that the union was not then effected, for a combination of the intellectual

and material forces of the two principal societies in London would doubtless have been for the greater service of Medicine. Might it not still be so? With the experience of the cause of this dissension, however, two very important resolutions were carried at the very first meeting of the Medical and Chirurgical Society under the presidency of Dr. Saunders, resolutions which have been in force ever since, were incorporated in the Royal Charter granted thirty years later, and have been adopted by most of the societies which have since arisen, viz.: (1) "That no gentleman be eligible to the office of president or vice-president for more than two years in succession," and (2) "That a certain number of the Council go out annually." The objects with which the Society was founded have been pursued with vicissitudes of fortune, but with a success which cannot but illumine our centenary retrospect with a glow of triumph and gratification, and which sheds a bright prospective hopefulness and encouragement upon the future of our work. I have here a volume, which has, I hope, reached every Fellow, in which the chronicles of the Society are most interestingly set forth by our honorary secretary, Mr. Stephen Paget, and which also contains a life sketch of each of the fifty Presidents by our honorary librarian, Dr. Norman Moore. It is most instructive reading, and I think the Council has done wisely to sanction the publication of the volume, and the Society is indeed gratefully indebted to the able authors for the labour and interest they have taken in its production. These chronicles and records are indeed an epitomised history of the Medicine and Surgery of the past century, a brilliant and an exhilarating history to read, a history of achievements in medical science with which the records of no previous century can compare. It would be tedious for me even to enumerate the subjects treated of; they are matters of medical history with which you are all very familiar. I would broadly characterise them as dealing in the first half of the century with many and great incidents of medicine and surgery, and

in the second with great developmental movements in pathology and the treatment of disease. Isolated cases and groups of cases of great intrinsic importance and, looking back upon them, of historical interest are brought forward in the earlier, and, indeed, in both, periods—many of them brilliantly suggestive, but not to find place and fulfilment until the larger knowledge and quickened inventions of later times permitted. Amongst such cases and papers I would mention Astley Cooper's ligature of the common carotid artery, Dundas's description of acute rheumatic peri- and endo-carditis, and Abernethy's account of mitral stenosis, all in the first year of the Society's formation. At the time when the Napoleonic wars were raging we were receiving papers on the health of the navy and army from our Fellow and later our President, Sir Gilbert Blane, whose introduction of lime-juice for the prevention of scurvy saved more lives and much more misery than were sacrificed or occasioned in all those cruel encounters. Sir B. Brodie in 1836, by his temperature records of a fatal case of luxation of the cervical vertebræ with destructive hæmorrhage into the upper cord, demonstrates what his earlier experiments of 1811 had led him to believe, and as had been guessed at by John Hunter, that fever was neither a mere matter of altered circulation, as the older physicians had thought, nor of oxidation, as the physiologists after Lavoisier were teaching, but that it was dependent upon some other controlling factor, probably of nervous origin. How otherwise could be explained a rise of temperature to 112° F. in a man dying with respirations reduced to less than six per minute? Here was the earliest use of clinical thermometry, fully to be established twenty-five years later by the careful labours of Wunderlich in Germany and Professor Ringer in this country. The clinical thermometer is now part of the pocket equipment of every nurse, and is to be found in every household, yet I remember when its value was roughly challenged by one of the greatest physicians of the time. It was during Bright's presidency,

in 1838, that Bostock read his paper on the clinical examination of the urine and so initiated an important development of clinical pathology. Dr. Webster's paper, in 1843, on the brutal treatment of the insane then prevailing was the forerunner of the gentler and more humane methods which Conolly so nobly advocated. The paper by Hamilton Roe and Thompson, in 1835, on "Paracentesis for Empyema" (preceded by 14 years by a single case recorded by Dobson), was the beginning of a great advance in thoracic therapeutics. Then we have John Hutchinson on the Spirometer, John Marshall on the Galvano-Cautery, Golding-Bird and Hilton on an Operation for Internal Strangulation in 1846-47, Kirkes on the Detachment of Fibrinous Clots from the Interior of the Heart and their Mixture with the Circulating Fluid, and Spencer Wells's first five cases of Ovariotomy a year or two later. These are amongst the more suggestive of the papers in the first half of the century, every one of which may be said to herald great future achievements in Medicine. The latter half of the century has also had its epoch-making papers. But in the fifty years between 1840 and 1890 there were four great events in medicine which would mark out for distinction any century—viz. Marshall Hall's exposition of the reflex function of the nervous system; the discovery of a safe anæsthesia; the discovery and acceptance of the antiseptic system in surgery; and the growing recognition of the action of bacteria in the etiology and pathology of the acute specific diseases. These four great advancements in medical science crowded into the short space of fifty years have an interdependence that might form the subject of an interesting discourse, upon which, however, I have no intention to enter. This Society has had something to do with them all, and they have permeated and dominated most of its later and most brilliant records. Without anæsthesia, in the absence of antiseptic methods or their more recent aseptic developments, and without a knowledge of bacteriology, no advance in visceral or joint surgery was possible, no further steps for the prevention

of epidemic diseases could be formulated, the explanation of many intricate disorders of digestive and excretory organs was lacking, and their satisfactory treatment was impossible. Most of the initiators of these advances in medical science which have led up to and accomplished such great results are, or have been, Honorary Fellows of the Society. The honoured names, Darwin, Pasteur, Marshall Hall, Villemin, Claude Bernard, Charcot, Lord Lister, Lord Kelvin, Koch, and others still living, are indelibly written upon the pages of science in its beneficent relations to humanity. Many others on our Honorary Fellows' list and amongst the Fellows of our Society are still heartily engaged in pressing on the work. It might not be in good taste to signalise them out, but they are well known to, and honoured by us all. There are other papers not directly connected with these four eventful discoveries which are of great practical and historical importance. Samuel Fenwick's paper on the "Microscopical Examination of the Sputum" and Marcet's paper on the "Inoculation of Sputum in the Diagnosis of Tuberculosis" followed close upon Villemin's discovery of the inoculability of tubercle in 1865, preparing us seventeen years beforehand for Koch's great discovery of the specific poison of that disease. Mr. Jonathan Hutchinson's paper on "Vaccino-Syphilis" in 1871 was of vast and far-reaching public importance. The report of a committee on the "Hypodermic Use of Medicines" in 1867 marked the time, which to some of us may seem almost prehistoric, when the hypodermic syringe was only coming into use. Dr. Ord's paper on "Myxœdema" in 1877 may also be especially mentioned. But the chief work of later years has undoubtedly been the development of visceral and especially of abdominal surgery, the necessary conditions for which did not previously exist. It was within the walls of this Society that Spencer Wells, in 1871, established the operation of ovariotomy, which led up to so many further developments of ovarian and uterine surgery; and that Sir Frederick Treves in 1888 read his first paper on "Relapsing Typhlitis

treated by Operation"; and it was here, too, that the operative treatment of appendicitis was brought up to date by a spirited discussion only a few weeks ago. I think that one of the attributes of our Society which may give us most unbounded satisfaction is the magnificent library we have acquired. Much of this has been due to the liberality of friends in sending us their works and in presenting to us rare books which have come their way. But to the enterprise of the Society and to the lavish expenditure of its scanty funds in former years it is chiefly due that we have perhaps the finest library of medical and scientific works in the world. I am not sure that the censure of our chronicler is all deserved for our illiberality with regard to publication of our proceedings in early years. For at that time the sale of our 'Transactions' was a large source of income provided by the work of our Fellows and wholly expended, not upon themselves, but upon new books for the library. The occasion and the practice have now long since passed. Perhaps the object which the Society had in view at its first foundation second to that of acquiring a library—viz. the conversational discussion of cases and subjects of medical and surgical interest—has been least cultivated, and perhaps amongst the amendments that must come in our proceedings in due time this point may be considered. Whatever future may be before us we can look back upon a vigorous past and a prosperous present. At no time has the position of the Society been better recognised than to-day; at no time has its material prosperity been greater. I would suggest that a grateful thought for the many distinguished men who formed the Councils of our Society in the past and have carried it on to its present prosperity may fitly occupy our minds during this centenary. Nor should we forget the permanent officers of the Society. We may some of us remember the genial courtesy of our late librarian, Mr. Wheatley, who spent the best years of his life at our former house in Berners Street; and to our friend Mr. J. Y. W. MacAlister, with his business talent,

wide knowledge, and zeal in the service of the Society, we owe much of our present prosperity, whilst to his great energy and that of his staff of assistants in carrying out the wishes of the Council and Special Committee is due the success which will, I hope, attend the celebrations which we commence to-day.

The PRESIDENT requested Dr. Newton Pitt, Senior Hon. Secretary, to read the numerous telegrams and letters of congratulation which had been received from various societies and institutions.

Professor CHIENE (President of the Medico-Chirurgical Society of Edinburgh) said:

Mr. President and Fellows,—By the unanimous desire of my colleagues in Edinburgh I, as President of the Medico-Chirurgical Society of that city, bring you a message of goodwill. We were founded in your early youth, and we have had one object in common during all these years, namely, the advance of medicine and surgery and the diminution of human suffering. It is therefore surely but right that we should rejoice with you to-day. You refer, sir, in your remarks, to the illustrious men who founded this Society. I, personally, like to think of them as present with us to-day. At the coming of age of anyone it is the grandparents who most rejoice, and I thoroughly believe that the founders and pioneers of this Society rejoice with us to-day. I am certain of this, that they take a lively satisfaction in the robust health of their child. One hundred years to them is but a moment, and they now know that the Temple of Knowledge was being built slowly, that the foundations have only yet been laid, and we have to build up the superstructure. They assure us that we can look forward with confidence to the future, because they tell us that there is no task ever given to men that is impossible of solution. The magnitude of the task is a tonic to us. I

think they will take special satisfaction in the library to which you allude. You spoke truly of that library, as I think, when you said it was the first library in medical science in this country. And if there are any of the Fellows who are younger than myself here I would only say to them, Dig, dig, dig in that library, because there is much there that requires to be rediscovered. These founders, then, being now in the light, send to us individually and collectively, as yet groping in the darkness, this message: "Be faithful to what is true and beautiful and good." Mr. President, long may your Society retain this high place as one of the great searchers out of truth. We are still asking Pilate's question. It is safe in your hands. And when you, sir, hand on the badge of office to your successor it will be said of you that you were guided by the best traditions of this noble Society. Personally, sir, I thank you for having given me this public opportunity of expressing to you the good wishes of your brothers in the North for continued usefulness and prosperity.

Professor Chiene then handed to the President an illuminated address of congratulation.

The PRESIDENT:

I am sure we are all deeply grateful to Professor Chiene for coming this long distance, and for presenting to us this very interesting document of congratulations, which we shall ever treasure amongst our most valued possessions. I beg to thank Professor Chiene for his most eloquent and sympathetic address.

Mr. JOHN LANGTON (President of the Medical Society):

Mr. President and Gentlemen, as the President of the senior Medical Society of London, I am deputed on behalf of myself and the Council to express to you our cordial congratulations on the Centenary of the Royal Medical and Chirurgical Society, and none the less to you also, who so worthily fulfil the office of President of the best traditions in medicine. Among other things to which I

think you incidentally alluded, Mr. President, was a sort of insinuation that there might be a combination or confederation of certain societies, if not of all London societies; and I can only hope, personally speaking, of course, that you may be still President when that wished-for amalgamation or confederation shall take place. That confederation will not be, I think, so much for the good of the older Members and Fellows of the different societies as it will be for the younger, and I hope therefore that in the interests of the younger generation of physicians and surgeons that confederation will soon be a matter of fact.

The PRESIDENT:

I can assure Mr. Langton, the President of the Medical Society, on the part of the Royal Medical and Chirurgical Society, that we greatly value the kind sentiments which he has expressed. Our respect for the Medical Society, as in a sense our founder, is unbounded, and I must also hope that at some time we may again unite our forces.

Dr. FREDERICK TAYLOR (President of the Clinical Society):

Sir, as President of the Clinical Society of London, I have the honour to offer the sincere congratulations of that Society to this that it has attained the hundredth anniversary of its foundation. In its useful and distinguished career during the hundred years of its existence the Royal Medical and Chirurgical Society has been the means of bringing before the Profession, and indirectly before the whole civilised world, the most beneficial and important additions to medical knowledge, and there are few of the leading members of the Profession who have not been included amongst its Fellows. No one who has read through the 'Transactions' of the Society, or looked at its numerous volumes or at the 'Proceedings' in which its discussions have been recorded, can fail to recognise the immense value of the contributions and records of work and research which have been made and carried out under

the Society's auspices, and published with their approval. And when one reflects on the fact that all these communications have been submitted to the criticism of your Committee of Reference and your Council before being published, and that their criticism has always been fearlessly exercised, I am sure he will feel and know that the merit of those communications is of the highest order. Of equal, if not more, importance are the advantages to which you, sir, have already referred of your magnificent library; and there are great numbers of writers in London and the British Isles who have availed themselves of the opportunity afforded by that library for the increase of knowledge, and they have enriched it by means of their labours. The Clinical Society cannot lay claim to the seniority which you are celebrating this day in your anniversary; indeed, the Clinical Society is one of the offspring of your Society—not the oldest nor the second in point of age, but still senior to many others. The Clinical Society required a separate existence for the consideration of communications which the venerable traditions of your Society did not make it expedient to include within the limits of their work. We look up to our parent Society, a Society in whose mansions we still have our home, with respect and with affection; and the Clinical Society, I may say, sir, will be second to none in the heartiness of its congratulations and in the sincere desire that it expresses for the success and prosperity of the Royal Medical and Chirurgical Society.

The PRESIDENT:

I am sure the Society is grateful to the President of our daughter Society—if I may venture to say so—the Clinical Society, of which I myself have the honour of being an original member, for the congratulatory and kind address which we have just heard. Our daughter Society has indeed been prolific in the good things she has done for medicine, and I am sure that we feel highly honoured to be congratulated by so illustrious and vigorous an offspring.

Dr. Boxall (Hon. Secretary of the Obstetrical Society):

On behalf of the Obstetrical Society of London, Mr. President, I have the pleasure to offer its most hearty congratulations to you and to this Society on attaining its centenary. Many of the works which have been included in your 'Transactions,' which are so widely read and generally known, have been from members who have been specially associated with obstetrics and gynæcology, and those who work in the special line dealt with by the Obstetrical Society have always found the greatest benefit from the library which has been collected together by such magnificent work under your auspices. Indeed, without that it would have been scarcely possible for many of these excellent works which have been brought, not only before your Society, but before the Obstetrical Society, to come into existence. On behalf of the Obstetrical Society, I beg to tender our most hearty congratulations on the Centenary.

The President:

I am sure Dr. Boxall will convey to the Obstetrical Society our warmest thanks for their very kind congratulations. I think the Obstetrical Society is one of the oldest societies which holds its meetings under our roof, and we hope they will long continue to do so.

Mr. Charters Symonds (President of the Laryngological Society):

Sir, the oldest societies having spoken, I have the honour at the moment to represent one of the newer societies which you shelter under this parental roof—namely, the Laryngological Society of London; and being a somewhat vigorous child, it was thought I might fitly offer to you its own congratulations on attaining the Centenary in addition to those other societies which owe you so deep a debt of gratitude. The subject of laryngology has been more than once instanced before your Society. So long ago as 1826 cases were published here. Czermak showed his laryngoscope in 1862; Durham, an old Guy's surgeon, first demonstrated the use of the laryngoscope

in operative surgery, and Morell Mackenzie showed Babington's laryngoscope. George Johnson also exhibited cases, and showed the value of the laryngoscope before this Society, as did Dr. Thomas Walker, still an active surgeon and laryngologist in Peterborough. You have seen, sir, many smaller societies start from this parent home, some of them showing a life perhaps more active, though not more useful, than the parent Society. And as we look at these various offshoots we must all acknowledge that your Society has lost nothing in dignity and nothing in usefulness. We smaller and more recent societies, I think, owe you a special debt of gratitude; for had it not been for the encouragement given to us by the privilege of meeting in your rooms we should never have been able to develop, and therefore it is that I specially bring to you the thanks and the congratulations of this Laryngological Society, which would never have been able to flourish as it has done if you had not allowed us to meet within your fine buildings. And now, as we enter upon this new period of the Society, we cannot but admire the action of the President, who is foremost in an attempt to unite these various offshoots and gather them into one whole, much as the Mother Country is attempting to unite her colonies. And I trust, as the President of the Medical Society has said, that it may be possible to complete this scheme while you, Sir, hold the chair which has been occupied by so many well-known and distinguished physicians and surgeons.

The PRESIDENT:

I am very much obliged to Mr. Symonds for conveying to us this very kind message from the Laryngological Society, and I can assure him we very greatly value the congratulations of that Society. Whatever future may be before us all with regard to amalgamation or otherwise, I am quite certain of this, that if we do not amalgamate we shall still go on working away as we do now, in the same amicable and friendly manner, for the good of medicine and of all society.

Mr. JONATHAN HUTCHINSON (former President of the Society) :

In virtue of my seniority, gentlemen, an exceedingly agreeable duty devolves upon me this afternoon, and one in which I feel certain I shall have the hearty concurrence of all present. It is that we return our very best thanks to our President for the address which he has been so kind as to give us. I am sure that we have all enjoyed the brief and very judicious summary of the history of the Society which he gave us, and that we have but one regret respecting it, and that is that it was so brief. I propose that we give the President our very best thanks for it, and that we join with it an expression of the wish that he will allow the address to be printed, for we all wish to read it.

Mr. THOMAS BRYANT (a former President of the Society) :

I have great pleasure in seconding the proposition of my friend Mr. Hutchinson.

The resolution was carried by acclamation.

The PRESIDENT :

I am extremely obliged to Mr. Jonathan Hutchinson and Mr. Bryant for the very kind expressions which they have given utterance to, and I regret that I could but give a very brief and sketchy address this afternoon. But I felt that the more important object before us, for which we specially met, was to congratulate our Fellows and also to confer honorary degrees, and I only intervened to say a very few words by way of epitomising some of the work which had been accomplished in the century. Of course I shall have pleasure in putting this very small production into print. I wish especially to draw attention to the very interesting exhibition which is now present in these rooms of rare and valuable mementos, in books, prescriptions, instruments, pictures, and other things, illustrative of medicine at the time of our foundation, which have been collected from many friends of the Society by the kind efforts of the Exhibition Committee of the Council. There is one other

matter. I think the Council feel that it might be a graceful thing if we were to send a telegram of greeting to our absent honorary Fellows, and if that is your pleasure it shall be done.

Agreed.

The PRESIDENT :

Gentlemen, that concludes the business.

CENTENARY BANQUET

Held at the Hotel Cecil, Monday, May 22nd, 1905

SIR RICHARD DOUGLAS POWELL, Bart., K.C.V.O., M.D., President, in the chair.

Principal Guest—H.R.H. the Prince of Wales, K.G.

The guests and Fellows present numbered 455.

Before dinner His Royal Highness was received by the President and Council in an ante-room, and after signing the Obligation as an Honorary Fellow, he was admitted in due form by the President.

TOASTS

"THE KING."

The PRESIDENT: Your Royal Highness, My Lords and Gentlemen, the first toast which is proposed in every assembly of Englishmen is that of His Majesty the King. The King is our Patron, and his honoured name is enrolled in the Fellowship of both the Royal Colleges of Physicians and Surgeons. His Majesty has ever shown the deepest solicitude in promoting the happiness of his people, in doing all that is possible for the present to promote the treatment and relief of suffering and disease amongst them. His Majesty, by his genial tactfulness, has lost no opportunity of promoting peace and goodwill amongst nations. Few, perhaps, in this generation know, or can know, all that His Majesty has done in this latter direction. My Lords and Gentlemen, I give you "His Majesty the King."

"Queen Alexandra, the Prince and Princess of Wales, and the other Members of the Royal Family."

The President:

I have now the honour to propose the toast of "The Queen, the Prince and Princess of Wales, and the other members of the Royal House." The Queen, since she first came amongst us, has won all hearts by the sympathy she has ever shown with all her subjects, with those who are rejoicing, and, still more, with those who are in sorrow or suffering. Her Majesty is not content, however, with an attempt to soothe the sorrow which Fate may bring: she has always exercised her vast influence in mitigating suffering. Almost immediately after the South African war the Queen interested herself in the registration of the Army Nursing Services of this country, and you are aware how eagerly Her Majesty has studied all the details connected with this service, of which Her Majesty is the head. Nor are there any other good works that Her Majesty has not in some way or another done her best to promote. The Prince of Wales, who has honoured us with his presence to-night, from the first took up the arduous duties of his position in a manner which has appealed to all Englishmen. Thoroughness has ever been the watchword of His Royal Highness—a watchword, I fancy, which he learnt on the quarter-deck. Thoroughness is his motto, whether it be in connection with sport or with philanthropy. As a sportsman we all know His Royal Highness to be a crack shot, both on the moors and at the coverts. His Royal Highness has equally the reputation for going straight to the heart of any public matter which he takes in hand. We who are present to-night perhaps best know the work of His Royal Highness as President of the King's Hospital Fund, which he inherited from his Father, who initiated it; we know the Prince of Wales as President of the Cancer Research Fund; and only those who are working on the committees of those two valuable institutions are aware how keenly His Royal Highness follows and studies everything connected with them, and how he brings his great influence to bear in promoting their work. It was indeed a gracious act on the part of His Royal Highness to accept the Honorary Fellowship of this Society. By doing so he has expressed his approval of the work which we have done in the past century and his sympathy with that work we may have to do hereafter. The Princess of Wales and other members of the Royal Family are ever thoughtful to "render the deeds of mercy," and to promote in all ways in their power that sympathy between the poor and the rich which they have so many oppor-

tunities of promoting from their high social position. I should like to express the earnest hope of this great assembly that the visit which is shortly to be paid by their Royal Highnesses the Prince and Princess of Wales to India may be most interesting to themselves, as it cannot fail to be of great advantage in bringing into closer touch the sympathies of the Eastern and Western peoples of this great Empire. We wish them a prosperous journey and a safe and happy return. I give you the toast of "Her Majesty the Queen and their Royal Highnesses the Prince and Princess of Wales and the other members of the Royal Family."

HIS ROYAL HIGHNESS THE PRINCE OF WALES:

My Lords and Gentlemen,—I am greatly touched by the words of Sir Richard Douglas Powell, and in the name of the Queen and the Princess of Wales and the other members of my family, and on my own behalf, I heartily thank him for the sentiments to which he has given expression, and this distinguished assembly for the kind manner in which they have received this toast. I am very happy to be present on this occasion, memorable in the history of the Royal Medical and Chirurgical Society, when we celebrate on the exact date, May 22nd, the anniversary of the day on which this Society was founded just one hundred years ago. I consider it a great honour to have been enrolled among its Honorary Fellows and to be associated with the names of men eminent both in our own nation and in almost every other nation of the world, and I am much pleased to meet some of them here to-night. It is a further satisfaction to me to be identified with a Society with which my family has been connected ever since its incorporation by Royal Charter in 1834. Sir Richard has kindly referred to my position as President of King Edward's Hospital Fund for London. I regard it as a precious trust transmitted to me by the King, its founder, and I shall always endeavour, to the best of my power, to further its work, the gradual but steady development of which I watch with keen interest and satisfaction. For it is my firm conviction that there is no better way in which we can help our poorer brethren than through the healing work and humanising influences of our great hospitals. With regard to the Imperial Cancer Research Fund, I am glad to say that we are working in close touch with similar undertakings in other countries. Considerable and valuable results have been attained during the past year, and I trust that we may look forward to the day when medical and surgical knowledge will have unravelled the mysterious cause of this terrible disease and have acquired the power of successfully dealing with it. I must con-

gratulate this famous Society on celebrating the 100th anniversary of its foundation. In spite of its years it is still full of the vigour, enterprise, and enthusiasm of youth. The advances which have been made in the science of medicine and surgery during the last hundred years are almost without a parallel in the history of human progress. During this period physiology has been established as a precise branch of learning. while the new science of bacteriology would seem to have laid bare the very foundations of disease. Antiseptics and the clinical thermometer have been invented, and methods of investigation introduced of which, I am sure, the medical profession of 1805 had never even dreamt. In the foundation of antiseptic surgery by Lord Lister this country can claim one of the greatest achievements in the cause of humanity. In public hygiene nothing short of a revolution has been effected. Our hospitals, which a hundred years ago were little more than mere refuges for the sick, have become institutions in which the most beneficent treatment is carried out with scientific thoroughness. With this great century of progress this Society has, indeed, been actively concerned, and it can look back upon its record of a hundred years with the assurance that it has taken no little share in a memorable scientific advance. Its deliberations can only result in a widening of the capabilities of medicine and surgery, and in a lessening of the mass of human suffering. With it also rests the grave responsibility of passing judgment upon new products and new theories, of directing investigation into right channels, and of keeping a jealous watch upon the scientific reputation of a great profession. I feel confident that the Royal Medical and Chirurgical Society will maintain this trust as faithfully in the future as it has done in the past. I heartily wish the Society continued prosperity. Gentlemen, I am most grateful for the cordial welcome you have given me, and I assure you it has given me the greatest possible satisfaction and pleasure to have been present on this memorable occasion.

"The Royal Medical and Chirurgical Society."

The President

then proposed the toast of "The Society," and said: I have already this afternoon touched upon the history of the great Society the centenary of whose existence we are now celebrating; in the chronicles of the Society and the personal records of the fifty Presidents who have presided over its proceedings that history has been most interestingly set forth by Dr. Norman Moore and by Mr. Stephen Paget in the volume which is now in the possession of every Fellow. These

chronicles not only give a faithful account of the difficulties the Society has passed through, and of its growth and mental and moral development through the century to its present ripe and distinguished and vigorous old age, but also very faithfully reflect the history of medicine in the last hundred years. The Society was founded at a meeting of physicians and surgeons one hundred years ago at the Freemasons' Tavern, with Dr. Saunders as its first President, for the purpose of conversation on professional subjects, for the reception of communications, and for the formation of a library. Abernethy, Babington, Blane, Astley Cooper, and others were present. Its field of work rapidly extended to the reading of papers, the receiving of communications for discussion from various parts of the world, and, above all, the accumulation of a library of English books and of foreign literature, in those early days difficult to obtain, bearing upon medicine, surgery, and the associated sciences. Premises were soon required for the meetings of the Fellows, and for the housing of the growing library; and in the year 1834 the Berners Street House was taken, and the Society attained its present position as the premier Society of Medicine, with a Royal Charter granted by His Majesty King William IV, who graciously consented to become its first patron. Since then it has never ceased to enjoy the patronage of the Sovereign, and to-day it has achieved the additional distinction of numbering your Royal Highness amongst its Honorary Fellows. The Society has always regarded the purchase of books as a primary object, and the library now contains more than 50,000 volumes, and its current literature includes the journals and proceedings of every country and learned medical society in the world. Its Fellowship includes the workers in the van of medical and surgical science in the metropolis and the provinces. There has been no advance in medicine that the Society has not been instrumental in promoting. Single cases of immense interest, and, looking back, of great historical importance, have been recorded in the first half of the century; they are as the streaks of light that usher in the dawn of great movements and discoveries in every department of science. To discuss the history of these and other landmarks of medical and surgical advance to which you, Sir, have alluded would be out of place here. The observations of the first half of the century may be said to culminate in three fundamental accessions to our knowledge, momentous and far-reaching in their consequences to the well-being of humanity: (1) The attainment of a safe anæsthesia in the later Forties; (2) the establishment of the antiseptic method in surgical practice; (3) the recognition of the microbic origin of specific diseases. It would be impossible even approximately to count the lives that have been saved, and to estimate the suffering that has been spared by the performance of operations now of

daily occurrence throughout the civilised world, not one of which could have been attempted thirty-five years ago—before our long-time Fellow, and now our Honorary Fellow, Lord Lister, interpreted, extended by elaborate researches, and reduced to terms of surgical procedure the great discovery by Pasteur of the essential *rôle* played by living organisms in fermentation and decomposition processes. In 1859 pyæmia accounted for 42 per cent. of fatal cases of amputation; its occurrence is now practically unknown in such cases, and death from amputation is almost unknown. Ovariotomy, which in the early days of Spencer Wells was protested against as unjustifiable from its enormous mortality, became within his time a fairly successful operation, owing to his cleanly methods, but still with a mortality in 1871 of 22 per cent. As the antiseptic method developed, however, the mortality fell in 1881 to 4 per cent., and it is now probably less. Numerous abdominal operations, including those for the radical cure of hernia and for appendicitis, have only become possible and safe within this period. I take abdominal surgery as but the type and illustration of the vast progress made in the relief of suffering throughout the world, brought about by the introduction of anæsthesia and antiseptic methods. On the same lines has medicine—and particularly preventive medicine—advanced. We were not acquainted in my student days with the exact nature of a single specific disease—small-pox, glanders, lockjaw, anthrax, diphtheria, cholera, typhoid, consumption, ague, plague, influenza, septicæmia, and the rest were equally obscure to us as regards the exact nature of the poison which gave rise to them. Now we can see projected on the screen the exact portraits of the particular organisms responsible for the majority of these diseases; the habits, food, appetite, degree of virulence, methods of multiplication, and mode of growth of each of these minute organisms are as well known to us as though they were beasts in a menagerie. We are learning to circumvent them as foes, and are beginning to police them as undesirable aliens; nay, we even recognise some of them as friends when restricted to their own proper sphere of activity. I have heard of grumbles at medical fees and at the increased costliness of hospital administration. But when you remember as I do the weeks of illness, with necessary doctor's visits, consequent upon a simple surgical operation thirty years ago, the bottles of medicine and the pints of wine consumed in the long and suffering days and nights of hectic fever spent in hospital or at home whilst the wounds were tardily healing; and when you contrast this suffering and loss with the smarter methods of to-day, dictated by more exact science, under which the most appalling wounds are healed and the patient up and about in a few days, it will be confessed at least that the labour and thought of a century have not been wasted. The average time of healing for a breast amputation

used in my early days to be from six to twelve weeks; it is now from one to three weeks, the bed accommodation in hospitals for such cases being thus increased fourfold. Similarly on the physicians' side whole regions, formerly the pleasure-grounds of the typhus, typhoid, cholera, diphtheria, and ague organisms, know them no more. Consumption is a diminishing, some even think a vanishing, disease; the rates of mortality and the prevalence of illness are steadily falling. And why? Because every doctor is a sanitarian and works loyally with those who organise State medicine for the prevention of disease. Methods of treatment are more precise, and the time seems rapidly approaching when the doctor's occupation will be gone, and he himself can neither live nor die. In a recent letter to the 'Times,' Dr. Ronald Ross, speaking of "another victory against malaria," achieved in one of our possessions in the Malay Straits, quotes the medical officer—Dr. Watson—in charge, who, as an illustration of the decline of fever in the locality, relates one fact which "he cannot help recording, although with less satisfaction, namely, that the fees resulting from his private practice in connection with malarial fever have fallen to zero!" Well, I may truly say that the Royal Medical and Chirurgical Society has taken a large share in bending to the service of medicine all the great discoveries in science of the century that have emanated from the fertile brains of Faraday, Darwin, Pasteur, Kelvin, Lister, Koch, and many more. Many of these great men are gone, others are developing and extending their work. I must here again allude to two of the greatest of them at the present time, our Honorary Fellows Lord Lister and Lord Kelvin, whose work has been more far-reaching in beneficence to humanity than any work done within their century, and who perhaps, alone of all men within the century, have lived to enjoy the sweets of immortality. We may be forgiven, then, on this unique occasion—not to be repeated within the lifetime of anyone here—a somewhat self-assertive and jubilant note; but in our hearts we are not unmindful of much undone that might have been achieved, of opportunities lost, of adventures unwisely checked; they are recorded in our chronicles. We fully acknowledge the strenuous labours in the same field of others—our sister Societies in the service of humanity. We are humbly mindful of the increasing responsibilities of the next century, and we are solicitous, if it may be, that by the union of forces that now overlap we may advance at a steeper gradient to higher realms of achievement.

"Literature and Science."

Dr. Pye-Smith, F.R.S.: Mr. President, Your Royal Highness, My Lords and Gentlemen,—It is my privilege to propose for your

acceptance the toast of "Literature and Science." There has ever been a close connection between the medical profession and literature. In that golden period which intervened between the Wars of the Roses and the Reformation there were four men who directed the new learning and the national education. Their names were Erasmus, Sir Thomas More, Dean Colet, and Linacre. Linacre was the founder of the Royal College of Physicians, and was an eminent Greek scholar. He was followed by Dr. Caius, of Cambridge, and Datchet. In the seventeenth century the medical profession contributed to literature an author of the first rank—Sir Thomas Browne, who wrote 'Religio Medici,' 'Pseudo-doxia Epidemica,' 'The Garden of Cyrus,' and 'Urn Burial.' An eminent physician once said that Sir Thomas Browne was a more important man than his contemporary the great Harvey, because if Harvey had not discovered the circulation of the blood somebody else would certainly have done it, whereas if Browne had not written the 'Religio Medici,' no one else could. In the following century the great literary physician was Arbuthnot, who was the friend of Swift, of Pope, and of Gay. Arbuthnot contributed three of the greatest satires of our own language—'The History of John Bull,' 'The Conduct of the Allies,' and 'The Epitaph of Colonel Charters.' After the death of Arbuthnot, who it was feared died of cirrhosis of the liver, Pope confided the care of his health to the celebrated Dr. Mead, the collector of the finest library of the period, and to the great anatomist and surgeon Cheselden, both of St. Thomas's Hospital. And Pope did what we often wish our patients of the present day would do—he took what Mead and Cheselden advised. Johnson found a friend and physician in Heberden, whom he used to call "Ultimus Romanorum," the last of the learned physicians. Those who read Heberden's commentaries must still be struck with the conciseness of his style, the perfect clearness of his language and the abundance of his good sense. Johnson himself, in his "Lines on the Apothecary, Robert Levett," has portrayed the humble but useful life of the general practitioner, and thereby has proved his title to the name of poet. A medical writer who was also of high literary merit was Sir Thomas Watson. His art in telling cases was remarkable. We still have learned physicians amongst us. There is Dr. Joseph Payne, the Harveian Librarian, and Dr. Norman Moore, who tells sad stories of the deaths of kings. We also have learned surgeons—Mr. Tweedy, the President of the Royal College of Surgeons, Mr. Stephen Paget, and Mr. D'Arcy Power. But though literature is still a relaxation, a refreshment, and a delight to medical men, the art of medicine depends for its progress and its existence upon science. By science we have reached our present position of progress, and by science alone, persistently and ungrudgingly pursued, we shall reach new successes in the pre-

vention and cure of disease. How scientific the whole bent of medicine is we recognise, not only by the broad stream of authors like Harvey, Hunter, Jenner, Bichat, Laennec, Claude Bernard, Chauvaux, Johannes Müller, Ludwig, Pasteur, and Lister, but also by the work of many of our profession who have excelled in other branches of science—men like Gilbert, whose great work on the magnet has been lately published by Professor Sylvanus Thompson; men like Mayow, a young physician who in his short life established the theory of the mechanism of respiration and discovered the existence of oxygen; and also Thomas Young, physician to St. George's Hospital, who established the undulatory theory of light, deciphered the Rosetta stone, and earned the unstinted praise of Helmholtz—a praise, however, which has not been denied him by his own countrymen, as the great German supposed. Science is the very breath of life to medicine, and investigations must continually go on. Encouragement is not wanting, because much has been done towards the prevention of diphtheria, of malaria, and of many tropical diseases since a royal critic asked the question "Why are not preventable diseases prevented?" We all gratefully acknowledge the interpretation given by the eldest son of England to the motto of the Black Prince "Ich Dien," by serving on the Council of the Association which was formed for the purpose of studying the origin, and in time, it is to be trusted, the prevention, of that terrible disease which is rightly called "malignant."

Sir ARTHUR CONAN DOYLE: Mr. Chairman, Your Royal Highness, My Lords, Ladies, and Gentlemen,—The fact that I should be chosen to respond to the toast of "Literature" is an illustration of the very familiar rule observed in this country, that it is the understudy who has to do the hard work. I very much wish that Mr. John Morley had taken this task upon himself. I understand, however, that your kindness in coupling my name with the toast is due to the fact that I have the honour to be a medical man. It is true, as some unkind critic has remarked, that in spite of that fact no living patient of mine has ever yet been seen. Gentlemen, without desiring to expand upon the whole field of literature, there is perhaps one little corner upon which I might be permitted to say a word. Some authority has lately been treating the writers of fiction from a legal point of view and examining their law, and has come to the conclusion that the law of the novelist is even, if possible, more strange and more uncertain than that law with which we are familiar. I remember one aphorism, that if a man dies intestate his property goes to the nearest villain. But if the law of the novelist is strange, I think you will admit that his medicine is equally so. We only recognise in my calling, the writing of fiction, certain diseases; the

others are of no use to us. It is a remarkable fact that all these diseases are of the upper part of the body. The novelist never hits below the belt. We have not a lengthy list of ailments, and our treatment is strangely simple. There is, of course, phthisis. I do not know how we should get on with our heroines without it. We sometimes call it a decline, sometimes we call it a wilting away. This is most useful to us, and it ends usually in a complete cure in the second last chapter. The treatment, of course, consists in the bringing back of that great and good man who has been so cruelly misunderstood in Chapter IV. The symptoms of this disease are acute but variable. The most prominent one is extreme wasting, coupled with an almost ethereal beauty. Another disease which is most useful to us is fits. We do not descend to petty details; whether it is an apoplectic or whether it is an epileptic fit which eventually carries off the heavy and stertorous father is a matter which is between ourselves and our conscience. The symptoms as we describe them may belong to either, or to neither, or to both. But suffice it to say, that he falls down in a fit, usually with a pen in his hand, and in front of an unsigned will. The fit is opportune, and it is exceedingly effective. There is then that mysterious malady which is known as brain fever. What should we novelists do without that wonderful fever? What would she nurse him through during that anxious time, and how else could he, after many months of continual delirium, come to himself and proclaim himself a chastened and a better man? The novelist must upon these points deprecate your technical criticism. We have only a little, and we beg you to leave us that. We do not fly to extremes in our literary ailments. The only example which I know to the contrary is gout, which in all our pages only occurs in the ball of the big toe. For some reason it is usually treated as a semi-comic disease, which tends to prove that the novelist has not himself suffered from it. The gouty, irascible gourmand is one of our necessary puppets, and I am sure he has every reason to be irascible if contempt is invited for his very serious and painful malady. As to small ailments, gentlemen, we do not deal in them at all. No one in our pages is ever known to suffer from mumps, or from a sore throat, or from a nettle-rash. If we hit at all, we hit hard, but we are, on the whole, kindly disposed to the medical profession. Our types vary from the village practitioner, whose usual treatment seems to be a hearty slap on the shoulder and a kindly twinkle through his glasses, to that gloomy member of the upper hierarchy who comes down from town at the crisis of the novel, says nothing, shakes his head, and then, with a large fee in his pocket, returns again to the classical precincts of Cavendish Square. Mr. Chairman and gentlemen, you will excuse me if I have enlarged upon one little topic connected

with literature. I will not venture to treat the subject at large, but I will only say that if one is somewhat disheartened by seeing that the great trees of our profession have one by one during our days fallen, nevertheless, looking around, one sees that if we have no longer these great oaks, at least there is a considerable quantity of undergrowth and brushwood, and there may here and there be a young sapling pushing its way upwards, which in time may attain some dimensions. When I consider the general history of British literature, there have been many times when things have been quiet, when people have bemoaned the past, but it has invariably occurred that a fresh generation has arisen which has come up to the highest mark of its predecessors, and I cannot doubt that that will occur again.

Sir WILLIAM HUGGINS (President of the Royal Society): Your Royal Highness, My Lords, and Gentlemen,—The honour of responding to the toast of "Science," which you have so cordially honoured, falls to me as the representative of the Royal Society, which has now for two centuries and a half, with untiring energy and steadfast aim, strenuously pursued the great object for which it was founded—"the improving of natural knowledge." It is, indeed, not inappropriate that the Royal Society should be in evidence to night, for the connection between the Society and the Medical and Chirurgical professions has always been an intimate one. The College of Physicians smiled upon its birth, and of the original Fellows of the Royal Society, there were many physicians and surgeons. The roll of Fellows has always contained many names of men distinguished in your professions, of whom six have presided over the Society. I will mention only Sir Hans Sloane, Sir Benjamin Brodie, Professor Huxley, and last but not least, my immediate predecessor, that great man who, by the introduction of methods of science into the operating room, at once made comparatively safe operations which had hitherto been followed by very grave risks, and also, at the same time, made possible operations which it had not been thought prudent to attempt—a man whom, not only his own country, but mankind throughout the wide world honours, and to whom they are under lasting obligations—Lord Lister. Happily the time is long past when medicine and surgery were in the hands of illiterates and quacks. Nearly four hundred years ago the College of Physicians, and later the Medical Society, were founded to bring the science of the day to bear upon medical practice; how successfully this object has been carried out the valuable 'Transactions' of your Society abundantly testify. Still, even in comparatively modern days the paralysing idea prevailed that the living body was a sort of enchanted ground outside the sway of physical and chemical forces, where, in some mysterious way, vitality reigned supreme,

and was all in all. The wonderful progress, in recent times, of the art of healing followed closely upon the falling down of the walls of the fortress of Vitalism before the challenges of Chemistry, by the making of many substances which had been regarded as only possible by the transcendental processes of a living organism. During the last half-century many distinguished workers, notably on the Continent, have raised physiology and pathology to a place among the exact sciences by the introduction of the accurate methods of physics and chemistry into the clinical laboratories and the schools of medicine. Medicine came completely under the dominion of Science when the principle of the conservation of energy was found to hold in living beings and to determine the connection and sequence of their vital and physical forces and actions. In a word, the discoveries of Science, by bringing into practical medicine an increasing use of physical forces and methods, have largely contributed to the diminution of disease and to the prolongation of life. The great work of Darwin filled the intellectual world with a flood of light, and deposed man from his unique and central position in medicine, and so transformed the whole scheme of biological science. Again, the discovery of the great invisible host of the powers of the air, which make everything we touch and taste a possible Pandora's box full of insidious foes seeking to gain access to the citadel of life, has at the same time placed in our hands new powers of defence. The recent research so successfully carried out by the Royal Society on the mysterious and deadly diseases of tropical countries seems to show that it is in the power of man so to control and guard against the conditions of their propagation as to make it possible for Europeans to live and carry on their work in parts of the earth where hitherto the sacrifice of health, and even of life, has been fearfully great. In the past the power of modern medicine over disease has followed directly upon the improvement of Natural Knowledge; in the future it must be to a still more intimate acquaintance with Nature that we must look for further triumphs over disease and premature death, and so for the securing of a longer and fuller life to man.

"The Guests."

Mr. Butlin: Mr. President, Your Royal Highness, My Lords and Gentlemen,—When I was informed by our Secretary that I was to propose this toast, he was good enough to wish me well through it. He thought it would be a very difficult toast on account of the number and distinction of our guests. I confess that seemed to be an insufficient reason. I own that on one occasion I was seriously embarrassed by this toast, for there

were very few guests, and I could not find one good thing to say of any of them. But how different to-night! Was ever any medical society in this town or in this country so highly favoured as this Society now is? Has any society ever welcomed to its table so many and such distinguished guests? I would like to mention and thank them all by name, but that is impossible, and I must therefore speak in general rather than in personal terms. First, I must speak of our chief guest, His Royal Highness the Prince of Wales. We are twice indebted to him—for his presence here to-night, and also because he has become an Honorary Fellow of our Society. He is the first Royal Honorary Fellow of this Society, and it has taken us just one hundred years to achieve this honour; and I am sure the time has not been ill spent. His Royal Highness is to be envied in many things; but there is one thing of which we are all envious —that he is better heard in this room than any other speaker. Then there is his Grace the Duke of Northumberland. We are the more pleased to see him because we have no special claim upon his time. And there is my Lord Strathcona, on whom I think many persons would like to have a substantial claim, and Lord Cheylesmore, the Mayor of the Borough in which our house is placed. The House of Commons is so busy with its own affairs, that it has only been able to lend us two members, the Right Hon. John Morley and Sir Walter Foster, and on the latter we have a kind of prior claim. So, too, we have a claim on the representative of the Church, the Bishop of Oxford, for, some thirty years ago, his father was the President of our Society. The law has given us the Lord Chief Justice of England. We have distinguished representatives of science and literature; but where was art in the speech which was made just now? I cannot mention all the other distinguished persons; they are so many. But there is the Chairman of the London County Council; there are representatives of some of the greatest and most liberal of the City Companies; there are the Vice-Chancellor, the Principal and the Dean of the Faculty of Medicine of our own University, together with distinguished persons from the Universities of Cambridge and Oxford, Aberdeen, Glasgow, and Trinity College, Dublin. Of course we have the Presidents of our great Royal Colleges. Close by me sits the President of the British Medical Association, and in the distance I see the President of the General Medical Council. In presidents of societies we are wonderfully happy, for we have more than twenty of them, including the President of the Royal Society. Some come from London, some from the provinces, and some even from north of the Tweed—a great compliment. And that leads me to speak of two guests whom I would not on any account omit to mention here: one is from Denmark and the other is from France. Professor Pierre Marie, of Paris,

has been an Honorary Fellow of our Society for some years. He is very fond of England, for this is the second time he has visited us since October. Professor Bohr, of Copenhagen, has been made an Honorary Fellow this day. We accord a hearty welcome to both these gentlemen. I now ask the Fellows to drink the health of our guests; and with that toast I couple the name of His Grace the Duke of Northumberland, who naturally by his position should reply, but who is peculiarly fitted to do so because he is himself a man of science. He is not only a Fellow of the Royal Society, but he is also President of the Royal Institution and, what perhaps appeals still more to us, he is the very active President of the Institute of Sanitary Science. Gentlemen, I give you "Our Guests"; let us wish them long life and good health, and a very small acquaintance with our profession.

THE DUKE OF NORTHUMBERLAND: I thank the proposer of this toast very heartily in the name of the guests this evening for the kind words which he has used in presenting it to you, and you, gentlemen, for the extremely hearty way in which you have received it. There is one circumstance which strikes me as somewhat curious. There are, as we all know, in this great country four great professions standing at the head of all other professions—the navy, the army, the medical, and the legal professions. We honour them, we respect them, we value them; but we never wish, if we can possibly help it, to avail ourselves of their active services. There is a song which a good many of us used to sing some years ago about the army and the navy:

> "We don't want to fight, but, by Jingo, if we do,
> We've got the ships, we've got the men,
> We've got the money, too."

I confess I feel this evening as if I could sing the same song with somewhat different words:

> "We don't want the doctor, but, by Jingo, if we do——"

Well, gentlemen, I will not finish it, and so spare your blushes, but I venture to say we are very well furnished for the fight. But after all, I am not doing justice to these professions, because although in one sense we do not want to avail ourselves of their services, in another sense we do. There is a preventive side to all their energies; we feel safer for the care and the study which they give to the preservation of our interests, and if that side of their exertions is perhaps less known to the world than their showy and observable actions, we are very conscious of the debt we owe to them; and to none of the services do we owe more than to the science, the energy, the devotion, which is expended upon preventive medicine. I do not think,

gentlemen, you will expect me at this late hour to do full justice to the reply which I have to make. It would take far too long. I will only say one word about myself. I was quite overwhelmed when the proposer of this toast spoke of me as a man of science. I began to wonder whether the reflex action of the nervous system, of which your President spoke, meant the alarming sensations I experienced when I heard that. I will only say that, however undeserved such a compliment may be personally, there is not one of your guests who is not full of admiration of the value of those researches which, perhaps in consequence of his own ignorance, he is only able to follow at an immeasurable distance, and that all here present are anxious to aid in every way they can the great work which this Society and your profession have so magnificently discharged in the course of the past hundred years. I thank you once more, gentlemen, for the compliment you have paid me.

"THE PRESIDENT."

Lord ALVERSTONE (Lord Chief Justice of England): Your Royal Highness, my Lords, and Gentlemen,—I feel very much honoured that you have asked me to submit the last toast, which I shall propose in a few words, namely, that of your President this evening. I shall not at this hour deliver the oration which I had prepared to enable you to appreciate the merits of your President, because I have no doubt you appreciate him without any words from me. Were the hour not so late, inasmuch as this is the first time that a simple lawyer has been allowed to make any observations, I should have been tempted to reply to a few of the remarks of my friend, Sir Sherlock Holmes. He made some invidious comparison between the law of the novelist and the law of the nation. I must say, considering that his speech was delivered in the presence of His Royal Highness, who is very closely connected with the fountain of justice, I thought it rather ill-timed. But a lawyer is a long-suffering animal, and therefore I think it best to pass by any further reference. My Lords and Gentlemen, the occasion on which we meet Sir Richard Douglas Powell to-night is somewhat unique. Previous speakers have referred to the work of this great Society, and have mentioned the changes that have taken place in medical science during its life. It is remarkable that I believe this evening puts upon record also a new coincidence, namely, that the presidency of the Royal Medical and Chirurgical Society is held by a gentleman who is also the President of the Royal College of Physicians. I feel that the great position which Sir Douglas Powell holds in your profession will render, as I have already said, any words from me altogether

unnecessary. But when we remember the great predecessors in the position or office of President of the Royal College of Physicians, when for a moment one thinks of Sir Thomas Watson and Sir William Jenner, and when we remember that he is merely succeeding a very great man, present to-night, in the person of Sir William Church, I think you will say that the position which your President occupies is one which not only commands your respect, but it will induce you to give to the toast of his health a most hearty welcome. But, if I may be allowed to say so, as one who has crammed up sometimes a little medical knowledge and forgotten it as soon as the necessity for its use passed away, it was extremely interesting to hear that speech of the President to-night touching in such felicitous terms the landmarks of medical science and surgical science during these 100 years. And I cannot help feeling that in one who could expound in such a short time, in such terms, the advance of medical science, we have one fully worthy to represent the medical profession. My Lords and Gentlemen, I would make one personal appeal to the President, as the President of the Royal College of Physicians, and I make it with all the more sincerity because I have no longer a personal interest in the matter. I had the great privilege and honour of being selected, many years ago, as the Counsel of the Royal College of Physicians. It was in my early days at the Bar, and it was a great compliment paid by Sir William Jenner, and I thought my fortune was made and my success secured. I regret to say that the College contented themselves with regarding it as an honorary office, and I never had the honour of appearing for them in any litigation. I do not know what struggling counsel has received that honour at the present time, but I hope, as the legal profession has been spoken of in somewhat slighting terms this evening, Sir Richard Powell will think of it. I might say a great deal about Sir Richard Douglas Powell, of his position, of his great attainments, of his career at the university and in the hospitals, and all that which you in the medical profession know; I therefore content myself with expressing again the great gratification it gives me to be here and to listen to his eloquent speeches. We wish him long life and continued activity, and that he may long live to adorn that profession which he so well serves.

THE PRESIDENT:

Your Royal Highness, Lord Alverstone, my Lords and Gentlemen,—I will not detain you one moment further except to express my very hearty thanks for the very cordial manner in which my health has been proposed. I would only say that I trust this evening has been one of great success. I would express our thanks to the Dinner Committee and the Honorary Secretaries of this Society, and our thanks are greatly due to

the permanent Secretary of our Society, Mr. MacAlister, for his zeal in bringing about the success of this large gathering. I also think the thanks of the Society are due to Mr. Ashbee for the very fine and artistic design which he has made for the honorary diplomas. Our thanks are also very sincerely given to Mr. Bernard Partridge, the son of a former President of our Society, for the very beautiful frontispiece which he has designed for our programme this evening.

After dinner the President and Council received their guests in an adjoining room—and the guests and officers of the Society were presented to His Royal Highness by the President.

Order of Tables.

The Fellows and their Guests were seated at eleven tables.

The President, presiding at the High Table, was supported on his right by H.R.H. the Prince of Wales, and on his left by His Grace the Duke of Northumberland.

At the other tables the Vice-Chairmen were Dr. Buzzard, V.P., Mr. H. T. Butlin, V.P., Mr. Pearce Gould, V.P., Mr. Warrington Haward, *Hon. Treas.*, Mr. Godlee, *Hon. Libr.*, Dr. Newton Pitt, *Hon. Sec.*, Mr. Paget, *Hon. Sec.*, Dr. Donald Hood, *Hon. Sec. Dinner Committee*, Sir James Reid, Bart., G.C.V.O., K.C.B., and Mr. J. Y. W. MacAlister, *Secretary of the Society.*


NON EST VIVERE
SED VALERE VITA
CENTENARY
OF THE
ROYAL MEDICAL
&
CHIRVRGICAL
SOCIETY
M·DCCC·V·
M·CM·V·
Bernard Partridge.
ΥΓΙΕΙΑ
ΑΣΚΛΗΠΙΟΣ

The Royal Medical and Chirurgical Society, Centenary Banquet, Monday, May 22nd, 1905.

The President, SIR RICHARD DOUGLAS POWELL, Bart., K.C.V.O., in the Chair.

VINS

HOCK
Rupertsberger, 1889

—

CLARET
Branaire Duluc-Ducru, 1893

—

CHAMPAGNES
Krug & Co., 1898
Irroy, Carte d'Or, 1892

—

PORT
Martinez, Vintage 1887

—

COGNAC
Grande Marque, 1865

—

Liqueurs and Minerals

Menu

Croûte au Pôt

Truite Saumonée. Sauce Chambord
Salade de Concombres
Blanchailles

Poussin à la Windsor

Selle d'Agneau à la Broche
Pommes Nouvelles. Petits Pois à la Menthe

Caneton d'Aylesbury Rôti au Cresson
Salade de Laitues aux Œufs

Asperges Vertes. Sauce Mousseline

Jambon d'York au Champagne.

Gélée à l'Orange
Macedoine de Fruits

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"Bijou" Orchestra

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1. March ...	... "Pomp and Circumstance"	... *Elgar*
2. Valse	... "Adelen"	... *Strauss*
3. March	"Viscount Nelson"	... *Zehle*
4. Selection ...	... "Veronique"	... *Messager*
5. Morceau Mignon	"Salut d'Amour"	... *Elgar*
6. Two Step ...	... "Dixie Land"	... *Haines*
7. Valse	... "Gartenlaube"	... *Strauss*
8. Selection ...	... "La Bohême"	... *Puccini*
9. March ...	... "The Catch of the Season" ...	... *Haines Baker*
10. Valse	"An der Elbe"	*Strauss*
11. Selection ...	... "The Orchid"	... *Caryll*
12. Two Step ...	... "Nigger Alley"... ...	... *Andrews*
13. Selections from the Savoy Operas	... ...	... *Sullivan*

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Walter H. H. Jessop, M.B.	2	2	0	2	2	0	4	4	0
Alfred P. Hillier, M.D.	1	1	0	1	1	0	2	2	0
William Joseph Tyson, M.D.	2	2	0				2	2	0
Samuel Herbert Habershon, M.D.	2	2	0	1	1	0	3	3	0
Charles Barrett Lockwood	2	2	0	2	2	0	4	4	0
Charles Theodore Williams, M.D.	2	2	0	2	2	0	4	4	0
Vaughan Harley, M.D.	5	0	0				5	0	0
George Henry Savage, M.D.	2	2	0	2	2	0	4	4	0
Frederick Thomas Roberts, M.D.	2	2	0	2	2	0	4	4	0
The Right Hon. Lord Lister, P.C., O.M., D.C.L., LL.D., F.R.S.	5	0	0				5	0	0
Herbert T. Herring, M.B., B.S.	2	2	0	5	5	0	7	7	0
Alfred Pearce Gould, M.S.	5	0	0	5	5	0	10	5	0
J. B. Lawford				3	3	0	3	3	0
George Eastes, M.B.	2	2	0	3	3	0	5	5	0
Philip Henry Pye-Smith, M.D., F.R.S.	2	2	0				2	2	0
William McAdam Eccles, M.S.	1	1	0	2	2	0	3	3	0
E. Hurry Fenwick	2	2	0	3	3	0	5	5	0
Richard Lake	1	5	0	1	1	0	2	6	0
Herbert William Page, M.C.	2	2	0	1	1	0	3	3	0
Frederic Durham, M.B.	1	1	0				1	1	0
Francis de Havilland Hall, M.D.	2	2	0				2	2	0
Montagu Handfield-Jones, M.D.	2	2	0				2	2	0
George Cowell	2	2	0				2	2	0
Sir Thomas Lauder Brunton, M.D., D.Sc., LL.D., F.R.S.	2	2	0	5	5	0	7	7	0
Edmund Symes Thompson, M.D.	2	2	0				2	2	0

	Subscription.		Guarantee.		Total.	
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Francis Henry Champneys, M.D.	2	2 0			2	2 0
J. Hughlings Jackson, M.D., LL.D., F.R.S.	2	2 0			2	2 0
T. Henry Green, M.D.	2	2 0			2	2 0
Arthur Marmaduke Sheild, M.B., B.C.	1	1 0			1	1 0
Cuthbert Hilton Golding-Bird, M.B.	2	2 0			2	2 0
Frederick William Cock, M.D.	1	1 0			1	1 0
Alexander Haig, M.D.	2	2 0			2	2 0
Clement Dukes, M.D.	1	1 0			1	1 0
William Watson Cheyne, C.B., M.B., F.R.S.	2	2 0			2	2 0
George Henry Makins, C.B.	3	3 0			3	3 0
Edward Farquhar Buzzard, M.D.	1	1 0			1	1 0
G. F. Blacker, M.D.	1	1 0			1	1 0
Anthony Alfred Bowlby, C.M.G.	2	2 0			2	2 0
Charles James Cullingworth, M.D., D.C.L., LL.D.	1	1 0			1	1 0
Philip Frank, M.D.	2	2 0			2	2 0
Sir John Moore, M.D.	1	1 0			1	1 0
Sir Thomas Barlow, Bart., K.C.V.O., M.D., B.S.	5	0 0			5	0 0
William Adams Frost	2	2 0			2	2 0
Christopher Addison, M.D.	1	1 0			1	1 0
Fallon Percy Wightwick, M.D.	1	1 0			1	1 0
Thomas Oliver, M.D.	1	1 0			1	1 0
Sir William Henry Broadbent, Bart., K.C.V.O., M.D., F.R.S., LL.D.	5	0 0			5	0 0
Henry Albert Caley, M.D.	1	1 0			1	1 0
Hector William Gavin Mackenzie, M.D.	2	2 0			2	2 0
Emile Bossan, M.D.	1	1 0			1	1 0
Leonard A. Bidwell	1	1 0			1	1 0
Albert John Venn, M.D.	1	1 0			1	1 0
Arthur Pearson Luff, M.D.	2	2 0			2	2 0
Thomas Wakley, jun.	2	2 0			2	2 0
Professor F. Howard Marsh, M.C.	3	3 0			3	3 0
Robert Alexander Gibbons, M.D.	4	0 0	5	5 0	9	5 0
Clement Godson, M.D.	1	1 0			1	1 0
Sir William Richard Gowers, M.D., F.R.S.	2	2 0			2	2 0
Raymond Johnson, M.B., B.S.	1	1 0			1	1 0
Seymour John Sharkey, M.D.	2	2 0			2	2 0
Frederick Taylor, M.D.	2	2 0			2	2 0
Robert Boxall, M.D.	1	1 0			1	1 0
John Tweedy	3	3 0	3	3 0	6	6 0
Robert Leamon Bowles, M.D.	2	2 0			2	2 0
Lauriston Elgie Shaw, M.D.	2	2 0			2	2 0
Henry Hugh Clutton, M.B., M.C.	2	2 0			2	2 0
Sir Hugh Reeve Beevor, Bart., M.D.	2	2 0			2	2 0
William Ewart, M.D.	1	1 0			1	1 0
William Edward Lee, M.D.	1	1 0			1	1 0
Bernard Pitts, M.C.	1	1 0			1	1 0

	Subscription.			Guarantee.			Total.		
	£	s.	d.	£	s.	d.	£	s.	d.
Laurence Humphry, M.D.	1	1	0				1	1	0
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John Hammond Morgan, C.V.O.				2	2	0	2	2	0
Sir John Banks, K.C.B., M.D., LL.D., D.Sc.	2	2	0				2	2	0
William Hale White, M.D.	2	2	0				2	2	0
John Stephenson Selwyn-Harvey, M.D.	2	2	0				2	2	0
John David Malcolm, M.B., C.M.	1	1	0				1	1	0
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Sir Dyce Duckworth, M.D., LL.D.	2	2	0	2	2	0	4	4	0
David Ferrier, M.D., LL.D., F.R.S.	2	2	0	3	3	0	5	5	0
Sir John Williams, Bart., K.C.V.O., M.D.	2	2	0				2	2	0
Fletcher Beach, M.B.	1	1	0				1	1	0
Andrew Clark	1	1	0				1	1	0
Malcolm Alexander Morris	2	2	0				2	2	0
J. Lynn Thomas, C.B.	1	1	0				1	1	0
Richard William Brimacombe, M.D.	2	2	0				2	2	0
J. Inglis Parsons, M.D.	1	0	0				1	0	0
William Henry Allchin, M.D., F.R.S.	2	2	0				2	2	0
Paul Morgan Chapman, M.D.	1	1	0				1	1	0
Samuel West, M.D.				2	2	0	2	2	0
Edwin Clifford Beale, M.B.	2	2	0				2	2	0
John Mitchell Bruce, M.D.	2	2	0				2	2	0
Charles Buttar, M.D.	0	10	6				0	10	6
Professor Edward Albert Schäfer, LL.D., F.R.S.	1	1	0				1	1	0
Robert Marcus Gunn, M.B.	2	2	0				2	2	0
George Oliver, M.D.	2	2	0				2	2	0
Charles Slater, M.B.	1	1	0				1	1	0
Leonard Rogers, I.M.S.	1	1	0				1	1	0
Professor D. W. Finlay, M.D.	1	1	3				1	1	3
John Henry Dauber, M.B., B.Ch.				1	1	0	1	1	0
William Deane Butcher	1	1	0				1	1	0
James Frederic Goodhart, M.D.	2	2	0	2	2	0	4	4	0
Herbert Morley Fletcher, M.D.				1	1	0	1	1	0
Major C. E. Pollock	1	1	0				1	1	0
Joseph Frank Payne, M.D.	2	2	0				2	2	0
Theodore Dyke Acland, M.D.	1	1	0				1	1	0
Henry Langley Browne	1	1	0				1	1	0
Thomas Outterson Wood, M.D.	1	1	0	1	1	0	2	2	0
A. Elkin Cumberbatch, M.B.	2	2	0				2	2	0
James John Macwhirter Dunbar, M.D.	1	1	0				1	1	0
William Hern	2	2	0	2	2	0	4	4	0
Francis R. B. Bisshopp, M.D.	2	2	0				2	2	0
William Bezly Thorne, M.D.	1	1	0	1	1	0	2	2	0
German Sims Woodhead, M.D.	1	1	0				1	1	0
George Ogilvie, M.B., B.Sc.				3	3	0	3	3	0
Thomas Jessopp Bokenham	0	17	0				0	17	0
William Appleton Meredith, M.B., C.M.	1	1	0	1	1	0	2	2	0
Edward Arthur Saunders, M.B., B.Ch., D.P.H.	1	1	0				1	1	0

	Subscription.			Guarantee.			Total.		
	£	s.	d.	£	s.	d.	£	s.	d.
James Johnston, M.D.	1	1	0				1	1	0
Charles Coates, M.D.	3	3	0				3	3	0
Lionel Vernon Cargill	1	1	0				1	1	0
Frederick Swinford Edwards	1	1	0				1	1	0
Sidney Coupland, M.D.	2	2	0				2	2	0
Frederick Charles Wallis, M.B., B.C.	1	0	0				1	0	0
Reginald Edward Thompson, M.D.	2	2	0				2	2	0
Charles Edward Beevor, M.D.	2	2	0				2	2	0
Walter Spencer Anderson Griffith, M.D.	1	1	0				1	1	0
Joseph Sefton Sewill				1	1	0	1	1	0
Thomas Laurence Read	1	1	0				1	1	0
Wayland Charles Chaffey, M.D.				1	1	0	1	1	0
J. Walter Carr, M.D.				1	1	0	1	1	0
John Anderson, M.D., C.I.E.	2	2	0				2	2	0
William Pringle Morgan, M.B., B.Ch.	0	10	6				0	10	6
Sir Alan Reeve Manby, M.V.O., M.D.	1	1	0				1	1	0
Frank Joseph Wethered, M.D.	1	1	0				1	1	0
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Benjamin William Quartey-Papafio, M.D.	1	0	0	1	0	0	2	0	0
Henry Morris, M.B.				2	2	0	2	2	0
H. J. Waring, M.B., M.S., B.Sc.	1	1	0	1	1	0	2	2	0
Frederick E. Batten, M.D., B.C.	1	1	0				1	1	0
Edward Erskine Henderson, M.B., B.C.	0	17	0				0	17	0
Howard Henry Tooth, C.M.G., M.D.	1	0	0	2	0	0	3	0	0
J. S. Risien Russell, M.D.	1	1	0	1	1	0	2	2	0
Robert Percy Smith, M.D., B.S.	2	2	0				2	2	0
Richard Clement Lucas, B.S., M.B.	2	2	0				2	2	0
William Thorburn, M.B., B.S.	2	2	0				2	2	0
Joseph Arderne Ormerod, M.D.	1	1	0				1	1	0
Henry John Strong, M.D., J.P.	1	1	0				1	1	0
David Henry Goodsall	2	2	0				2	2	0
James Kingston Fowler, M.D.	1	18	0				1	18	0
William Ainslie Hollis, M.D.	1	1	0				1	1	0
Sir Hermann Weber, M.D.	3	3	0				3	3	0
John Alfred Masters, M.D.	1	1	0				1	1	0
William Sampson Handley, M.S.	1	1	0				1	1	0
Phineas S. Abraham, M.D.				1	1	0	1	1	0
Archibald Edward Garrod, M.D.	1	1	0	1	1	0	2	2	0
Professor Sheridan Delépine, B.Sc., M.B., C.M.	1	1	0				1	1	0
Surgeon-Major-General A. F. Bradshaw, C.B.	1	1	0				1	1	0
T. Horrocks Openshaw, C.M.G., M.B., M.S.				2	2	0	2	2	0
Reginald Harrison				2	2	0	2	2	0
Dawson Williams, M.D.	2	2	0				2	2	0
William Gordon, M.B., M.C.	2	2	0				2	2	0
John McFadyean	2	2	0				2	2	0
Reginald Langdon Langdon-Down, M.B., B.C.	2	2	0				2	2	0

	Subscription.			Guarantee.			Total.		
	£	s.	d.	£	s.	d.	£	s.	d.
George Lindsay Turnbull, M.D. .	1	1	0				1	1	0
Charters James Symonds, M.S., M.D. .				5	5	0	5	5	0
Professor William Osler, M.D. . .	2	2	0				2	2	0
William Cayley, M.D. . .	2	2	0				2	2	0
Charles Sissmore Tomes, F.R.S. .	2	2	0				2	2	0
Samuel William Carruthers, M.D., C.M. .	1	1	0				1	1	0
	£395	17	3	£304	11	0	£700	8	3

Checked and found correct,

NEWSON SMITH, LORD AND MUNDY,
Auditors.

20*th September*, 1905.

CENTENARY FUND, 1905

Statement of Receipts and Expenditure

Receipts.

	£	s.	d.
Subscriptions for Expenses	408	8	3
Guarantee Fund	293	17	0
	£702	5	3
Deficit paid by Treasurers of Society	11	2	1
	£713	7	4

Payments.

			£	s.	d.
Centenary Banquet, including—					
Society's Guests, etc.	530	6 3			
Less received for Tickets	462	18 0			
			67	8	3
Reception and Conversazione			328	0	8
Hire of Cases, etc., for Exhibition			18	2	3
Printing (*Catalogue of Exhibition*, Programmes, Menus, Table-plans for Dinner, Circulars, etc.), Stationery, Stamps, Telegrams, etc.			208	14	9
Temporary Clerical Assistance, etc.			66	7	1
Miscellaneous Disbursements			24	14	4
			£713	7	4

Clinton T. Dent,
Hon. Treasurer.

J. Y. W. Mac Alister,
Secretary.

Audited and approved,
Newson-Smith, Lord and Mundy,
Chartered Accountants,
37, Walbrook, E.C.

PROPOSED UNION OF MEDICAL SOCIETIES

At the Annual Meeting held on March 1st, 1905 (p. lxxxvii), the following resolution, arising out of the President's address, was moved by Sir Thomas Smith, seconded by Mr. Goodsall, and carried unanimously:

> That the Council of the Society be requested, with as little delay as possible, to invite the leading medical societies of London to arrange for a joint meeting for the purpose of considering the advisability of amalgamating, and to take the necessary preliminary steps for that purpose.

At the first meeting of the new Council the question was considered, and the following resolutions were passed.

> (1) That in the opinion of the Council it is desirable that the President of the Royal College of Physicians be asked to convene a general meeting at the Royal College of Physicians, to consider the proposal for the amalgamation of the chief Medical Societies of London.
>
> (2) That a Committee, consisting of the President, Mr. Warrington Haward, Sir William Broadbent, Mr. Dent, and the Hon. Secretaries, be appointed to consider and report upon questions relating to the meeting, which it has been suggested that the President of the Royal College of Physicians should be asked to summon.

The President of the Royal College of Physicians, Sir William Church, Bart., at once adopted the suggestion of the Council, and by means of letters sent to the

'Lancet' and 'British Medical Journal' convened a meeting to be held in the Royal College of Physicians on Monday, April 10th, 1905.

In order to place on record what has been done to forward the object of the resolution passed at the last Annual Meeting, the following reports are here printed:

REPORT OF MEETING AT ROYAL COLLEGE OF PHYSICIANS.

REPORT OF THE COMMITTEE OF REPRESENTATIVES OF THE SOCIETIES.

REPORT OF SPECIAL GENERAL MEETING OF THE ROYAL MEDICAL AND CHIRURGICAL SOCIETY TO CONSIDER THE FOREGOING REPORT.

UNION OF MEDICAL SOCIETIES

REPORT OF MEETING HELD AT THE ROYAL COLLEGE OF PHYSICIANS, PALL MALL EAST, LONDON, S.W., ON MONDAY, APRIL 10TH, 1905 AT 5 P.M.

CHAIRMAN: SIR WILLIAM CHURCH, BART., K.C.B., M.D.

A MEETING, convened by Sir William S. Church, Bart., President of the Royal College of Physicians, and composed of Fellows and Members of the Medical Societies of London, was held at the Royal College of Physicians, on Monday, April 10th 1905, at 5 p.m., Sir William Church in the chair.

The CHAIRMAN: Gentlemen, I do not think there is any need to waste your time in saying what are the objects of this meeting, for we are all acquainted with them; but I think it might, perhaps, be interesting, and also instructive, if I very briefly remind those who are here what has taken place in this direction in former years. It is in the recollection of many of us in this room that an attempt to unite the various medical societies of London has repeatedly been before the medical public, and has hitherto always failed to effect anything, but I certainly was not aware myself, until I looked it up, and was informed about it, that a movement of this sort had been before us so long. As early as the year 1808 the Medical Society approached the Royal Medical and Chirurgical Society, the only other Society then existing, and requested them to join the Medical Society, so that the two societies should form a fresh one, with a fresh name and with a fresh habitation, and that they should have joint property, which

should be the property of the united societies. That attempt failed owing to there being some doubt, apparently, whether a change of name and a change of place would not invalidate the title which they as societies had to certain properties. Then one can pass to the year 1850, when the Royal Medical and Chirurgical Society was approached by the Pathological Society, who asked if the Pathological Society might not be incorporated in the Royal Medical and Chirurgical Society. That proposal went off on various minor details. Next, in 1860, there was another serious attempt to join the societies together, which occupied the attention of the medical world of that day for some sixteen months, but in the end all their deliberations came to nothing. It is interesting to note that only a few years later, in 1867, the Medical Society, at the time when Dr. Hare was president, again approached the Royal Medical and Chirurgical Society, proposing that they should unite and form a single Society, but nothing came of it. Then, of course, it will be in the recollection of some of us in this room that in the year 1870, when Sir George Burrows was President of the Royal Medical and Chirurgical Society, there was a very serious attempt made that the societies should join. Communications between the different societies went on for nearly two years, but again nothing resulted. Still more of us recollect the attempt made by the late Sir Andrew Clarke, in which I do not think the societies got into communication, but Sir Andrew Clarke himself took great interest in the proceedings, and many and frequent meetings were held at his house, not official ones, but unofficial, by men who were interested in the various societies. I do not think, however, that at that time anything like official communication passed between the various societies. You will see that with the exception of the earliest they were all attempts made for one—generally the Royal Medical and Chirurgical Society—to absorb into it one or more of the other of the existing societies. Those who have been interested in the movement now taking place thought it would be wise that we should revert to what was evidently the idea when the first amalgamation was proposed in 1808—that is to say, that instead of any one society absorbing another in any way, a new central body, which I will call for convenience sake and speak of in the remarks which I shall have to make as the Royal Society of Medicine, should be established, and that all the existing societies should be regarded as subdivisions or sections of it, so that existing societies should not lose their personal individuality, but should remain as societies embraced in and covered by this larger Society, the Royal Society of Medicine. It will not be possible for me—and I would leave it for others who will address you presently—to say in what way I think this might be accomplished, and I will only put my ideas forward in the most sketchy manner. I think there can be no

doubt whatever that there would be very great advantage to medicine in London and to the profession, if some such society could be established which should embrace the whole of the medical art and the sciences which are affiliated to medical practice. At the present time, owing to the enormous advance in knowledge, and also to the necessity for now making use of special instruments to a very much greater extent than was ever the case before, medicine is necessarily split up into a large number of branches and, rightly enough, all those branches wish to have their own Society to further that portion of medicine in which the members of the Society are specially interested. In this way there does seem to me to be a certain amount of danger that those special subjects may come to be regarded almost as dissociated from what I might call general medicine, and anything that can tend to keep up a close connection such as there is, and certainly should be, between general medicine and the special branches is, I feel sure, in the interest of medicine itself. That is one of the advantages. Then I think another point is that all the component societies would gain in prestige by their being, not merely the Medical Society, the Royal Medical and Chirurgical Society, or Neurological Society, or Gynæcological Society or whatever little portion of medicine they represent, but a part of the larger body. The Fellows and Associates of the New Society would certainly enjoy, without increased expense, very much greater advantages than it is possible for them to have now. My idea is that there would have to be two grades, Fellows and Associates. Fellows would be those who joined the New Society upon certain financial terms which gave them the right of being present, at all events, at the meetings of all the included Societies, and Associates would be those who preferred merely to remain as members of some of the one or more sections, and who would have certain advantages and privileges in belonging to the New Society, such especially as the use of the library of the New Society, and the use of all the rooms. If any such scheme as is present in one's mind were ever carried out, it would require that there should be much larger accommodation than is at present afforded at Hanover Square under the Royal Medical and Chirurgical Society; but there is a capability in those premises for extension and enlargement; so that one could get a larger meeting-room and that there should be attached reading-rooms, smoking-rooms, and tea-rooms, and the premises would be, I think, much more comfortable than at the present moment. The Associates would have certain rights; but what those rights should be, of course, in an opening speech like this, I cannot dwell on, neither have I myself formed an opinion on them. Those are details which must be left to be worked out by any Committee which should

be appointed if we get so far as to appoint a Committee to-day, and they would be worked out by that Committee. Now what societies would be likely to join? We must have some idea as to that before us, and I think one might take as a working basis the societies which, at the present moment, meet at 20, Hanover Square, and 11, Chandos Street, not by any means implying that other societies would be excluded, but one must begin by taking those which occur to one as being likely to come in and join this plan. There is another reason which I might mention, and that is, that with the exception of what I might call the two parent societies, the Medical Society and the Royal Medical and Chirurgical Society, all the other bodies who meet at these two places are societies representing special branches of medicine. Then there are also two societies for which everybody has, I think, great regard, the Harveian and the Hunterian. I have not mentioned them before because they, like the Medical Society and the Royal Medical and Chirurgical Society, are general Societies—I mean by the term "general" they both embrace all medicine and the subjects that are allied to it. I think we may treat them in a different way. I do not, by any means, wish to imply that those two, the Harveian and the Hunterian, should be excluded, I only say that they are on a rather different footing than are the other societies which meet in the Medical Society's and the Royal Medical and Chirurgical Society's rooms. It is no use mincing matters, and I must say that it seems to me that it is essential if any such scheme as the one contemplated is to be carried out, the Medical Society, which is so large, powerful, and strong, must come in and join, and I say so for this reason, that the Medical Society and the Royal Medical and Chirurgical Society are the only two medical societies which own any considerable amount of property; and if such a combination as is suggested is made, one must look a good deal to the income which might be received from the property of the Medical Society to assist in maintaining the suggested Royal Society; the income of the Royal Medical and Chirurgical Society, which is now considerably in excess of that of the Medical Society, will not be increased in the same way as the income from the Medical Society's property will be. I mean by this, if those minor societies that now meet in the Medical Society's rooms join this new Society, and the Hanover Square premises are enlarged and made suitable for the larger Society, then the property of the Medical Society will be able to be let, and will, I hope, bring in a considerable sum of money; whereas the Royal Medical and Chirurgical Society would, on the other hand, have to give up some portion of their income; for they would have to turn out some, if not all, of their non-medical tenants, in order to get the room for the increase of accommodation which would be required there in consequence

of the junction of societies. Therefore, I take it, one should state to this meeting that unless we find that the Medical Society is willing to consider the matter, personally I do not see how you are to provide all the extra accommodation and advantages which I think there would be, and at the same time diminish, as I think can be done, the expense to individual members. I think many men wish to join a good many societies, and wish to have the advantages of a library, such as that of the Royal Medical and Chirurgical Society, and those who join, especially the younger men, would certainly be economically benefited, but without the Medical Society coming in I do not see how they could be. As I say, I only wish briefly to place this scheme before you—all the details and arrangements with regard to the subscriptions to the sections, to the parent Society, the arrangement as to the government of the Society, the Council, and how the Council should be appointed, are necessarily matters for a committee, but I will try to answer any question which may be asked me to-day. I am afraid that the details have not yet been considered sufficiently for it to be desirable to enter into them; they would, I think, have to be left to any committee which is appointed to-day to work out and formulate a scheme which could be laid before the profession at no distant time. Personally I think that there is another very great advantage, in addition to those I have already alluded to, in having a Royal Society with separate sections or subdivisions under its wing. It would be a very great advantage to British medicine that, whilst the separate sections would, of course, receive communications and read and discuss such as they thought proper (it is suggested that all the papers should be published monthly), the parent Society should have a strong Publications Committee, which would every year select from the communications that had been made to the various sections under its wing the most valuable ones. These, when published, would form a most valuable addition year by year to medicine, and be representative of British medicine. I hope I have not detained you too long, but I think I ought, before sitting down, to announce that I have received a great many communications, all of them from men of note. I have received communications from the Presidents of most of the societies that have been mentioned and all of them are favourable to the idea. They, of course, are not all unanimous as to the sort of scheme that should be appointed. There is one very important letter from Sir John Burdon Sanderson, President of the Pathological Society, who says that he was strongly in favour of such a combination of societies being made some years ago, and is still in favour of it, but he urges very strongly that we should go in for a very much larger scheme in which the two principal bodies should be the two Royal Colleges. I have told him that we have never considered his much larger proposal and that I was not prepared to

go into the questions proposed in his letter. I have heard from the President of the Obstetrical Society, also from Mr. Lockwood, President of the Harveian Society, from Dr. Whitehead, of the Epidemiological Society, Dr. Coupland, for a long time Treasurer of the Pathological Society, as well as from several other gentlemen who do not hold official positions in the societies, and all of them hope that something may come out of our meeting to-day. I have also got this telegram which I have not had time to read, which I find says, "Regret I cannot get to meeting to-day," from Mr. Tweedie, President of the Royal College of Surgeons.

Sir FREDERICK TREVES, Bart., G.C.V.O.: Sir William Church and Gentlemen,—In order, sir, that the matter you have so ably introduced to this meeting may be adequately discussed—and discussed, I hope, principally by the younger members of the societies concerned—I venture to move the resolution which appears first upon the agenda:

> "That in the opinion of this meeting, convened by the President of the Royal College of Physicians and composed of Fellows and Members of the medical societies of London, it is highly desirable that an effort should be made to unite the principal societies into a new body, to be known as the Royal Society of Medicine."

I will not, sir, take up the time of this meeting by discussing the purpose of this resolution at length, except to point out that such an amalgamation as has been suggested would have obvious advantages in many directions. In the first place, it would have the very great advantage of convenience. I take it to be evident that if all the branches of medicine could be disposed of, so far as the development of their scientific aspects is concerned, under one roof, it would be a convenience, even if the matter be regarded from no higher standard. In the second place, it must obviously be a matter of economy. Each society has its own organisation and management, its own home to provide, and, in some instances, its own library to maintain. The money that is expended on administration only, scattered as the outlay is through so many channels, could be greatly economised if this amalgamation should be carried out. Moreover, I am sure that everyone present will agree with me that if this union of societies should be made practical, it would be certainly to the advantage of medical science. It seems desirable that the science of medicine should be able to face the scientific world as a coherent whole, and that every phase of its work should remain under the general ægis of medicine. This would not be detrimental to any single speciality, and I am perfectly certain that there is no person, no matter what his particular branch of practice may be, who would not be benefited by occasionally attending a meeting on a special subject for which, perhaps, he has no direct concern, but with which he may well have a better

acquaintance. I feel sure that there would follow that closer union in the various elements of medicine which I think is exceedingly desirable, and that there would be given a much stronger position to the science as a science. I imagine, sir, that this amalgamated Society would in no way concern itself with medical politics or ethics, but would be simply and solely for the advancement of the art and science of medicine in the widest and most liberal sense of the term. I imagine that such a scheme as is now proposed is not in any way at variance with the project that filled the mind of the founders of the two oldest societies in this Metropolis, the Royal Medical and Chirurgical Society and the Medical Society. I imagine that such a scheme as is now being discussed is but an amplification of that which occupied the minds of those who instituted those old societies. They certainly were intended to embrace the whole of medicine and surgery, and they did embrace the whole of medicine and surgery so far as those sciences existed at that time. If we could get at the spirit which actuated the formation of these institutions, I feel confident that that spirit would be agreeable to such a proposal as is now before this meeting. That such an amalgamation as this must involve sacrifice is inevitable. It certainly involves sacrifice of sentiment. It is scarcely to be supposed that an ancient Society like the Medical Society of London should cede any of its old traditions with a perfectly equable mind. But, sir, we have always been celebrated as a profession for generosity and liberality of mind, and I think that this is an occasion when those two admirable qualities might very advantageously be exercised. I have the honour of proposing the resolution which I have read.

Sir THOMAS SMITH, BART. K.C.V.O. : Mr. President and Gentlemen,—As Sir Frederick Treves has so well said all that is necessary, it falls upon me merely formally to second this motion, and as it is very important that this thing should be thoroughly discussed—and I hope discussed by a large number of the younger members, as Sir Frederick Treves has said—I do not intend to occupy your time by any considerable remarks, but will only say that I honestly believe that an amalgamation such as is proposed would be very greatly to the benefit of all concerned and very greatly to the benefit of science at large. Having been occupied in the year 1870, and for two years with Sir George Burrows, in endeavouring to carry out an amalgamation, I know something of the difficulties and I am certain of this, that if this thing is to be carried through, as I trust it will be, it will require a very considerable, strong majority of the members of the various societies to give their adhesion to it, or really it is not worth going into. I beg formally to second the motion proposed by Sir Frederick Treves.

Mr. JOHN LANGTON : Mr. Chairman and Gentlemen,—I wish it

to be distinctly understood that I am simply going to speak my own views, as I am not in any way authorised by the Medical Society, of which I am the President, to attend here as a delegate.

The CHAIRMAN: I do not wish to interrupt, but I thought that I told the meeting that no official communications have passed between myself and any of the societies, therefore we are all speaking to-day our own individual opinions, and we are not in any way representing the opinions of our respective societies, and nothing said here to-day will be taken as representing the feeling of any society. Our discussion to-day is simply that of the profession, and not a discussion of the different views held by the different societies; those we hope to get if we get on further.

Mr. JOHN LANGTON: I wished to make that clear at the beginning, because I think you read letters or stated that you had received letters from the Presidents of other Societies.

The CHAIRMAN: I ought to mention that they wrote only in their individual capacities.

Mr. JOHN LANGTON: The Medical Society, as you have heard from our Chairman, has been on two previous occasions in favour of fusion or amalgamation with the Royal Medical and Chirurgical Society, and I hope that even now we shall be prepared to join in amalgamation with the other Societies. Although, as I have said, I have not taken my Council into consultation, and still less have I asked any of the Fellows of the Medical Society of London what their views on this subject might be, as the President of the Medical Society, but only speaking as a private member, I desire to say that I am in great measure in accord with what has fallen from Sir Frederick Treves and Sir Thomas Smith. The difficulties, I know, will be great; the fusion means a great deal of giving and very little taking; but, on the whole, I feel sure the amalgamation would be for the progress of medicine and surgery. It is not for each or any of the Societies to say "What shall we get, what shall we lose?" since if it is definitely for the advancement of science, then, I think, it must be for the good of all. I will not detain you any longer, except in so far as to say that as a Fellow of the Medical Society of London I shall do as much as I can to further the aim and the objects of this meeting. I do not know what power I have, but, at any rate, I will do my best, and I think that some of the Fellows, at any rate, will wish for the fusion of the different societies, for it will prevent in some measure the overlapping of work which occurs in the meetings of the various societies for the advancement of the science of surgery and medicine, and especially in those special subjects which have lately been developing and necessitating the formation of different societies, and thus preventing the incompleteness of the contributions and their subsequent dis-

cussion. I will therefore say that if this resolution should be carried I take it that we, as Presidents, shall be asked to present the scheme before our respective societies, when a further meeting will be held to take their decisions into consideration.

Dr. McCann: Sir William Church and Gentlemen,—You were good enough to say that you would be glad to hear the opinions of some of the junior members of the profession, and as I have been personally interested for some years in this question of the amalgamation of the medical societies, I should just like to say how cordially I am in agreement with the resolution which has been proposed to-day. Speaking as a junior member, I know very well that to join several societies, in addition to being a certain financial strain to some of the struggling members of the profession, is a difficult matter in this way, that it is almost impossible for any man to attend those societies unless he has practically nothing else to do, and this combination of the societies under one roof would not only diminish the expenses of each member, but would tend to economise his time. I hope, however, that at this meeting no attempt will be made to grapple with the details, because it seems to me that if a discussion is started on the details this very valuable proposal may be killed in its inception. I know that the medical mind, from its training, is apt to grapple with details in the first instance, and if it is done in this case I think this, which is an excellent proposal, will be killed at once. There is only one amendment that I should like to suggest to the resolution which has been proposed by Sir Frederick Treves to-day, and that is, that the title should be "The Royal Academy of Medicine," instead of "The Royal Society of Medicine." I suggest that for this reason it seems to me that the older societies could more gracefully acquiesce in such a proposal than if we invited them to form another society of medicine. I, therefore, have pleasure in proposing, if I can find a seconder in the room, that the words "Royal Academy of Medicine" be inserted instead of "Royal Society of Medicine."

The Chairman: If I might say so, it appears to me that it would be a pity to tie ourselves down at this meeting to any particular title. I would, therefore, ask the last speaker to withdraw the amendment and to leave it quite open. I merely for the sake of clearness made use of the term "Royal Society" and the name was put down in this resolution, but I think it would be better to leave the question perfectly open as to what should be the title of any parent, or what I might call mother society, to embrace all the others, so that it might be threshed out when we get a little further on.

Dr. McCann: If that is clearly understood, then I will withdraw my amendment.

Lt.-Col. A. B. R. Myers: As I was present when this matter was discussed in 1870, I should like to ask you if you would

kindly tell us why, after such a very interesting discussion as took place on the last occasion, when it was generally felt that it would be a great advantage to the medical profession, it suddenly collapsed. Everybody seemed in accordance with many points, although some were raised, undoubtedly, which caused friction; for instance, with regard to the Pathological Society there was a great question about the point of publication. Sir Thomas Smith will remember that another point cropped up in connection with the Royal Medical and Chirurgical Society, for they distinctly stipulated that the society should not be divided into a medical and chirurgical society, and they claimed that they should remain as they were—a medico-chirurgical. I cannot get hold of the records, but there seems to have been a sudden collapse, and I should imagine that the authorities at that time must have made up their minds, after discussing the details—we have just heard that if we discuss them the whole will collapse—I am afraid that on that occasion when they discussed them the scheme collapsed. It is quite clear taht the whole of those in this room are in accordance with this scheme, and hold the view that there should be one general society. Personally, I cannot follow the great advantage that it is said to be pecuniarily; I think there would be a distinct loss, because the younger men all feel, no doubt, that they would derive the great advantage of having to pay so much less subscription; actually they will, but I also think it will be found that one institution alone, such as we have now in Hanover Square, could not possibly manage, because there are only twelve days in a fortnight, and with a great many societies there would not be sufficient days available for each one to hold its meeting on a different day, and it would be found that we should have to have two places, or, at any rate, two meeting-halls, for the purpose. We must not go into details, but I certainly should like to ask you if you would kindly tell us why it was that, after such tremendous support that was given to this idea of combination in 1870, the thing suddenly collapsed.

Dr. C. Theodore Williams: I think I can answer that question. Although I did not take much part in it, I was present at the meeting which was held in Berners Street at which the scheme was brought forward, and it was defeated and out-voted. I do not know exactly why it was so, but it was whispered that two of the societies had thought better of it, and there were two leaders certainly who opposed it; one was Sir James Paget and the other Sir Richard Quain; both happened to be presidents of societies, one of the Clinical and the other of the Pathological. I remember the vote on the occasion, and the scheme was not beaten by a large majority, but by quite a small majority. With regard to what you and the others have said, I heartily agree with you as regards the high importance of forming this

society, and the only wonder to me is that it has never been formed before. I think the scheme which has been outlined to the meeting by you is one that would be of great advantage and convenience. From a financial point also it would certainly be a great thing if the junior members of the profession, instead of being mulcted in a large sum of something like ten or eleven guineas a year, could get the whole of the advantages for, I would say, three or four guineas a year, or something of that sort, with an entrance fee. That would save a great many pockets, and I know that a good many of the junior men are very anxious to attend the various Societies' meetings, but at present can only afford to be members of one. A compound subscription would be a great thing; but as I have not had the matter before me in detail I cannot say how it would be managed; but my friend Mr. MacAlister showed me some striking figures, and they would have to be carefully worked out to see whether they would answer financially. I was glad to hear the President of the Medical Society speak as he did, because, of course, that is the Society which one would naturally think would be really opposed to this, as it might be expected to have an idea that the proposal might injure its prestige, and no one would wish that. I am an old Fellow of the Medical Society, and I think that most of its Fellows would cordially come in and join the Royal Medical and Chirurgical Society in forming this new Society, because I think we should be brought more together and should work more together in consequence, and I have not the slightest doubt that the cause of medicine would greatly advance thereby. We should be placed more in the position of some of the foreign academies of medicine. We are told not to go into detail, but I can quite see that there may be some sort of difficulty in the matter. For instance, the societies have got various medals to award, all of which would henceforth have to be awarded by the Academy of Medicine, and not by each individual Society. I see that Sir Thomas Smith does not agree with me. All I can say is, that I most heartily welcome the scheme of amalgamation.

Dr. Glover Lyon: You have, sir, invited the opinions of some of the junior members of the profession, and although I am not young I am at least the youngest that has spoken up to now. I hope others younger than myself will pluck up courage to speak after me. I have just served as Secretary of the Hunterian Society, one of a number of more or less local Societies, which could not be replaced by the proposed Society. Some societies are maintained by local practitioners, and meet so far afield as West Ham. There are many reasons, however, in favour of an amalgamation of the two great central societies. I think the objection as to the size of the building goes a long way. I see Mr. MacAlister here, who for the first time has

put this amalgamation idea into practical shape, and he could tell us more about that than anyone else. There is one other point which I should like to raise for discussion. I see there are twenty-nine specialist societies, and I should like to know what number it is thought would join in this scheme.

Sir RICHARD DOUGLAS POWELL: I did not propose to join in this discussion because I felt that I had already said a good deal about it in another place, and I am only now rising to say that I do not think it has entered into the minds of those who propose this union of societies to do more than effect a sort of union of the central societies of London. In my opinion it would be a disaster to the medicine of London if all the outlying societies were to be merged into one central society. The Northern, Southern, South-Western, and other outlying societies of London are, I consider, invaluable in holding together the practitioners of those neighbourhoods and in encouraging them to work together, and they serve to feed the central societies in due time, when some of those men wish to come forward and to bring some of their work to some central society. I say that I think it would be disastrous, socially and professionally, for those societies to be merged into one single society in London. I think it would be a great pity if those central societies should not be able to combine. Speaking on the point of economy, I should think that roughly, very roughly of course, something like £16,000 a year is spent by medical men upon the central societies of London, and I do not profess to be a business man; but, on a very rough reckoning, I believe that the full, and even a better result might be obtained by an expenditure of something like £12,000 a year, so that if the whole sum has to be expended it seems to me that we might get a great deal more for it, and, on the other hand, we might get at least as much, and I think more, for a much smaller expenditure per annum. I am inclined to think that the reasons why these attempts to amalgamate societies have fallen through hitherto have been very much business reasons, because the matter has not been gone into sufficiently from a business side. I am very much inclined to think that, when the matter has been thoroughly gone into from the business side, it will be found to be quite workable. But there is one thing which is quite essential before we can make any scheme of this kind workable, and that is that we should have the mandate of the mass of those men who are members of the various societies. If they are not fairly universal in their opinion that this matter should be carried through, then it is not worth while our trying to carry it through. If they are of opinion that it would be of distinct advantage to medicine of the present and medicine of the future that this should be carried, then I do not think that there is anything to prevent its being carried through.

Mr. D. H. Goodsall: I should like to say that I think that the attempt to form a society in 1869 failed somewhat because it was an attempt at absorption. In order to carry out the present scheme I think the interests of all the societies should be given the greatest consideration, so that no society should feel that it had been snuffed out. Then as to the advantages, it seems to me that it cannot be questioned for a moment that a union of all the societies would increase the strength and increase the power with the authorities and increase the good results, I think, to the general public. Then, from an economical point of view, I do not think there is any doubt about it that the money now subscribed will not all be required to do the present quantity of work. There would be one secretary and one place of meeting, and I think that the more it is considered the more we shall see what a great saving in the present expenditure would be gained by an amalgamation, and it would also certainly enable the junior members to join several societies instead of only one.

Dr. Hingston Fox: May I make a remark with regard to the Harveian and Hunterian Societies, following on what my friend Dr. Glover Lyon has said. I admit to a certain extent the local element enters into the Harveian and Hunterian Societies, and, in a sense, into the central societies. I hope, therefore, that upon any committee that is formed Fellows of those two societies will be put upon it, in order that their interests and their probable advantages in any amalgamation that may be agreed to will be duly considered.

The Chairman: I think perhaps I ought in my opening remarks to have told the members present what are the societies which I thought would be first of all considered—those which hold their meetings at Hanover Square and Chandos Street. At Hanover Square there are the Royal Medical and Chirurgical, the Pathological, the Clinical, the Obstetrical, the Balneological and Climatological, the Gynæcological, Anæsthetists, and the Laryngological Societies. Then at the Medical Society in Chandos Street there are the Medical, the Epidemiological, the Ophthalmological, the Otological, the Dermatological, the Neurological Societies. As regards space, there is not sufficient space at the present moment in Hanover Square, but that is one of the things which would have to be considered; there is ample room for a considerable increase of the present accommodation, and it would be a matter of funds and arrangements; probably, if necessary, it would be possible for two societies to meet on the same day. I also said in my opening remarks—I thought I had made it clear, for I endeavoured at all events to make it clear—that those who were interested in this scheme recognised that the Harveian and Hunterian Societies were on rather a different plane to the societies which treated special subjects, and I guarded myself by saying that I trusted that, although I

did not think they were primarily concerned to-day, they would not be excluded from joining or having their claims represented on the Committee.

The resolution was carried unanimously.

Sir WILLIAM BROADBENT, Bart., K.C.V.O.: Sir William Church and gentlemen,—The resolution that I have been asked to propose is:

> "That the Councils of the Medical Society, Royal Medical and Chirurgical Society, Pathological Society, and Clinical Society be severally invited to appoint two delegates to form, with the President of the Royal College of Physicians, the President of the Royal College of Surgeons, and five other gentlemen, a committee which shall (1) make such inquiries as may be necessary, (2) submit a scheme of union to the various societies for their consideration."

With your permission, sir, I will suggest a slight modification of this resolution, and it is to this effect, that while the Medical Society and the Royal Medical and Chirurgical Society as the oldest of the general societies, and as having funds, property, and libraries, should have two representatives on this original committee, instead of the Pathological Society and Clinical Society each having two, they should each have one, and that the Obstetrical Society and the Epidemiological Society should each have one, making the original number of official representatives of societies the same, but working on a representation somewhat larger. That is subject, of course, to the opinion of the meeting. I think it is extremely important that this idea of amalgamating the societies should have had such a send-off as it has had to-day. The difficulties to be encountered are mainly those of detail, the adjustment of the interests and of the sentiment of the different societies, and it will require a great wave of general opinion to carry some of the societies over the difficulties which have proved, hitherto, fatal to success in the carrying out of this idea. Some of those difficulties have already been suggested, and this is not the time or place to go into them, but a great thing, I think, would be to have this committee, which shall make inquiries, and which shall suggest the lines upon which the amalgamation may take place. This is, of course, imperative, and I should hope that the labours of the committee will bring about a scheme which, submitted first to individual societies and then to a general meeting, will bring about the result which we all have so much at heart. It is something more than a mere amalgamation which is proposed; it is, so to speak, a new creation, or evolution, raising the ideal of medical science and of its central organisation to a higher plane. What takes place now is not merely overlapping when the same subjects are discussed at different societies, but that these discussions are from that very overlapping imperfect. When

we have all these different branches co-operating for the same end, when some great subject comes up it would be discussed at a meeting of the great central body, and there would then be elicited the opinion, not merely of those immediately and specially interested in the point in question, but of representatives from every department of medical science. If I might give an illustration, I would say that supposing, for instance, we were discussing puerperal convulsions, which interests primarily, of course, those engaged in obstetrical work, if we had a discussion on a large subject like that brought up, we should have the pathologist, the neurologist, the experimental physiologist, the pathological chemist, and the general physician, and should bring to bear on such a discussion the whole range of medical science and medical practice. And we should thus not merely prevent overlapping, but we should get more efficient and thorough discussion of some of those large general questions. If I might say so, I should myself prefer the words "Royal Academy of Medicine" to "Royal Society." I think that when a young man enters the profession to take up any special subject he would like still to be able to say that he was a member of, say, the Ophthalmological or the Laryngological, or some other special society. It would carry more weight to be a member of a society than to be a member of a branch, and I think that the idea would be to have all societies within a great royal academy. I think in that way we should also be able to invite the co-operation of those local societies of which Sir Douglas Powell spoke so forcibly. It would be the greatest possible misfortune to extinguish or interfere with local societies, and I think that a large central royal academy might even benefit them by bringing them to a better position through their relations with it, and they might send up subjects for discussion to the great central authority. It is of the greatest importance that this committee, when it shall be set up, shall command general confidence, and be thoroughly businesslike, and have a thorough acquaintance with all the difficulties that are likely to beset the path of this great project. The importance of this committee cannot be overlooked. I hope that the constitution suggested—two representatives of the Medical Society, two of the Royal Medical and Chirurgical Society, and one each of the Pathological, Clinical, Obstetrical, and Epidemiological Societies—will form the nucleus, and in addition to those there would be the presidents of the two colleges and five representatives to be chosen for their special qualification for work of this kind. I beg to move that this committee be elected, and that it have the composition intimated in the resolution.

Mr. HERBERT F. WATERHOUSE: I have very great pleasure in seconding the resolution proposed by Sir William Broadbent. I think the unanimity which has been shown by this meeting

as regards the principles of this scheme is most striking. There is practically no dissension; everyone of us here understands that the creation of this great new Society will be for the benefit of scientific medicine in London; and it is very obvious that, as we are all united on the general principle, the only points of difference can be those of detail, and those points of detail will, of course, have to be worked out by the committee. It is, therefore, very obvious that the committee must be carefully chosen, because the members of the committee will have a very great deal of arduous work. There will be a great deal of sentiment to be got over, but when the committee feel that they practically have the whole of this meeting at their back and that this meeting has definitely, and with no uncertain voice, resolved that we are going to have one great new Society created in London, and that no one of the old societies is going to be absorbed or, as a gentleman said, snuffed out, but that we are going to have one new central Institute to further the study of medicine, surgery, and their branches, I am certain that all difficulties will very rapidly melt away. As one of the younger members I can see nothing but benefit that can result. We shall be able to have a library, such a medical library, in this Academy or Institute, as we certainly do not possess at the present moment in London. It will do good in that it will bring members of the profession more into contact, and will bring the members who study different specialities more into touch one with another, and I am confident that the establishment of this Society will be of extraordinary and unlimited benefit to medical study in London, therefore, I most cordially beg to second the resolution proposed by Sir W. Broadbent.

Dr. C. THEODORE WILLIAMS: It occurred to me that those societies who are left out of representation on the committee will not be in very good humour to encourage the scheme. I have been through the twenty-two on the list, and I think it is very properly proposed that the Royal Medical and Chirurgical Society and the Medical Society should each have two representatives on the committee, but might we not have one from each of the other societies? Otherwise how can they be represented? How can anyone speak for the Laryngological Society and the Ophthalmological Society if there is no member of these societies present? I know it would be a large committee, but as we are asking a large number of societies to join, the committee must be a large one; I do not see any way out of it. I think it would give rise to very unpleasant feelings if we do not start the matter fairly. I am sure that if I were a member of one of the smaller societies, I should not like to be left out, for I should like to have my say as well as other people. I know it will be a long list, but still it is worth while in order to get perfect harmony.

Mr. LANGTON: I think, as a matter of sentiment, we should endeavour to obtain a representative from each of the other societies. This resolution must go to each of the societies, and I think they should determine by a general meeting of each society the line of action they decide to adopt.

Sir THOMAS SMITH: Might I suggest that if the five other gentlemen mentioned in this resolution were omitted it would reduce the size of the committee somewhat. I see you have put down, besides the other representatives, five other gentlemen. If they were left out you could take in five other societies.

Dr. ARCHIBALD GARROD: Might I point out that there are two societies in this list which stand on quite a different footing to the others in the fact that they possess considerable property. You have said that without their agreement this scheme is impossible. Would it not be better if the Medical Society and the Royal Medical and Chirurgical Society were asked to appoint delegates and to invite members of the other societies and come to agreement without introducing any invidious distinction by our inviting others?

Sir FELIX SEMON: Would it not be simpler, sir, for the Medical Society and the Royal Medical and Chirurgical Society each to be asked to send two or three delegates, and for the rest of the committee to be formed by the Presidents of all those societies which it is proposed to invite to join the scheme?

Dr. ARCHIBALD GARROD: I think there is a strong feeling that it is necessary that the Medical Society and the Royal Medical and Chirurgical Society should first come to an understanding if the scheme is to come to anything. In order to ascertain the feeling of the meeting I will move as an amendment:

> "That the Medical Society and the Royal Medical and Chirurgical Society be invited to appoint members."

Sir WILLIAM BROADBENT, Bart., K.C.V.O.: Might I point out that we have already got one amendment before the meeting, and we cannot discuss two at the same time.

Dr. W. EWART: I should be very happy to second this amendment.

The CHAIRMAN: I do not know that I am strictly in order myself, but I will throw out a suggestion that may get over the difficulty of amendment. I think it would be very desirable that we should get a committee that really would work, and would it not be well to appoint, perhaps, a larger committee than was imagined when the agenda were drawn up, and appoint a committee consisting of representatives of all the societies wishing to come in, giving them power to, among themselves, elect a working committee to draw up a scheme? I think that would be the wisest thing we could do to-day, because, at the present moment, we hardly know, although there is such a

general consent in the room, which societies might wish to come in. I think it will be best to appoint a committee consisting of two representatives of the Medical Society, two of the Royal Medical and Chirurgical Society, for they are really the ones that bring in the basis of everything—the money—and one representative to each society that wishes to come in.

Sir WILLIAM BROADBENT, Bart., K.C.V.O.: As the mover of the original resolution I should like to accept that. If I had had the drawing up of the resolution that would have been the direction of my ideas. As you will remember, sir, I suggested a little extension of the one already down.

Mr. H. F. WATERHOUSE: I accept that.

Sir WILLIAM BROADBENT, Bart., K.C.V.O.: While this resolution is being written out might I add one other suggestion, that this committee when appointed should be empowered to invite the co-operation of others not representing societies. It might be desirable to get the co-operation of some thoroughly business men, and it would, I think, add to the efficiency of this committee if they had that power.

The CHAIRMAN: I think that would be in your power, although it would be better, perhaps, that it should be mentioned. There is other work in connection with committee that we might go on to whilst the resolution is being drawn up. It will be necessary that there shall be a secretary appointed, although, of course, it is rather out of order to appoint a secretary before we have passed a resolution appointing a committee, but I just wanted to bring it to your notice, although we can hardly pass it. I am happy to be able to inform you that Dr. Latham has consented, if it is the wish of the meeting, to act as secretary. It is open to anyone to propose any other name, but I thought I would just take the opportunity whilst the resolution is being drawn up to mention Dr. Latham's name.

Sir RICHARD DOUGLAS POWELL: I shall be glad to second that proposition.

The CHAIRMAN: I have now got the resolution placed in my hands. It is—

> "That the Council of the Medical Society and the Royal Medical and Chirurgical Society be invited to send two delegates, and that the Council of the societies wishing to join the union be invited to form with the Presidents of the two Royal Colleges a committee, with power to add to their number."

I think that if you would not mind letting me word the resolution after the meeting it would be best.

The resolution was carried unanimously.

The CHAIRMAN: I will now propose that Dr. Latham be asked to act as Secretary. He has already assisted me very

greatly in this matter, and I am quite sure the Committee could not have a better Secretary than he would make.

Mr. HERBERT S. PENDLEBURY: I second it.

The resolution was carried unanimously.

Dr. JOHN FAWCETT: I will not occupy the time of the meeting for many minutes, but the appointment of a committee which has just been agreed to would seem to render it desirable that some date should approximately be fixed at which the Report of that committee could be laid before a General Meeting. With that object in view I venture to suggest that a meeting be called during the early part of July, at which the Report of the committee be submitted for consideration. I have named July because it would give an opportunity to members to discuss the Report before the holiday season commences. I do not know whether I am optimistic, but I venture to think that three months would perhaps allow the committee sufficient time to carry out their investigations. Should a later date be decided upon, it would mean some time during the winter session, and that would delay the whole matter. With regard to the special subject about which I have been talking I have no more to say; but I should like, sir, as one of the junior members, to say that, although I can only speak for myself and one or two others with whom I have discussed the subject, we should heartily welcome a change such as has been suggested. I believe it would be a desirable thing in every way, both from the standpoint of economy and efficiency, and in enhancing the position of the medical societies in London; I hope it may be possible to find means to consolidate and co-ordinate the various interests of the societies concerned, and that this meeting may be the first official step of the present century in the promotion of that object. The resolution I have to propose is, "That a General Meeting of the societies concerned be convened, if possible, in July next, to receive the Report of the committee."

Mr. HERBERT S. PENDLEBURY: Sir, I beg to second the resolution which has just been proposed by Dr. John Fawcett. It seems to me essential that as definite a time as possible should be assigned for the consideration of the report of the committee that we have just appointed, and I am only sorry that the actual date is not put down. Might I suggest that I hope it will be possible to have some little sketch of the committee's results sent round before the actual General Meeting occurs, so that we may be able to criticise it.

The CHAIRMAN: Certainly a Report would be sent round.

Mr. HERBERT S. PENDLEBURY: Speaking for myself as a junior and unofficial member of several societies, I can only say that I am in complete accord with the idea of amalgamation. We have heard doubts expressed as to the financial feasibility of the scheme, and doubts as to the possibility of ultimate

agreement between societies of conflicting interests, but I believe that:

> "... Our doubts are traitors,
> And make us lose the good we oft might win,
> By fearing to attempt."

The CHAIRMAN: I wish we could have fixed a definite date in July, but it seems almost impossible. I trust by that time, as Mr. Pendlebury has said, that we shall be in a position to circulate a Report amongst the members of the societies which will add very much to the chances of a successful meeting in July.

The resolution was carried unanimously.

Mr. ALFRED WILLETT: I should like to move a vote of thanks to you, Sir William, for your admirable conduct in the chair.

Sir FELIX SEMON: I have the greatest pleasure in seconding that.

The resolution was carried unanimously.

The CHAIRMAN: I thank you for your kind expressions, and am particularly pleased this meeting has been so well attended. I hope the interest will be kept up.

The meeting then ended.

UNION OF MEDICAL SOCIETIES

Report of the Executive Committee as amended and adopted at the Meeting of the General Committee of Representatives of the Societies, held on July 19th, 1905.*

Your Committee beg to report that in accordance with their powers they co-opted Mr. Makins, and appointed Dr. Arthur Latham and Mr. Herbert S. Pendlebury to act as Honorary Secretaries.

We instructed Messrs. Newson-Smith, Lord and Mundy, Chartered Accountants, to investigate the rolls of membership and the accounts of the following societies:

Medical; Royal Medical and Chirurgical; Pathological; Clinical; Obstetrical; Gynæcological; Neurological; Epidemiological; Medico-Psychological; Opthalmological; Laryngological; Laryngological-Otological-Rhinological Association; Otological; Dermatological of London; Dermatological of Great Britain and Ireland; Society for the Study of Diseases of Children; Odontological; Association of Medical Officers of Insurance Companies; Balneological; Society of Anæsthetists; Electro-Therapeutic; Therapeutical.

The Accountants inform us that the twenty-two societies whose membership lists were collated, have a total membership of 7229, constituted by 4997 persons. Of these, 3725 are members of one society only, and the balance, 1272, are members of more than one society.

The Accountants, upon the figures placed before them by the societies, further inform us that—

The balance of assets (including certain bequests and

* N.B.—This is a reprint of the Report as issued by the Committee, but it also shows the additions and alterations suggested by the Council of the Royal Medical and Chirurgical Society, which are indicated as follows:—Words added are underlined; words deleted are enclosed in square brackets.

trust funds) over liabilities of these societies is more than £40,000.

If 3800 members of societies other than the Royal Medical and Chirurgical Society become members at a subscription of £1 1*s.* per annum, the New Society will have a small balance of income over expenditure. If any members of these societies become Fellows at a subscription of £3 3*s.* per annum, the balance will be proportionately increased.

This estimate is based on the present income and expenditure of the Royal Medical and Chirurgical Society together with the present income and expenditure of seventeen other societies as given in subjoined table, and makes no allowance for any economies which may be effected by the Union.

Estimate of INCOME and EXPENDITURE of the proposed Royal Society if housed at 20, Hanover Square, on basis of Income and Expenditure of existing eighteen Societies for the year ending December 31st 1904:

EXPENDITURE.		INCOME.	
Royal Medical and Chirurgical Society	£4307	Royal Medical and Chirurgical Society	£4911
Loss of rent by Union ...	770		
Of other seventeen Societies.		*Of other seventeen Societies.*	
'Transactions,' less sales, but without allowance for income by advertisements	2545	Rent of Medical Society ...	940
Staff	650	Investments	350
Library and Museum ...	166	Interest from investment of present cash balance	50
Interest on obligation ...	121	Entrance fees	186
Stationery, miscellaneous and exceptional expenditure	1670	3800 subscriptions at £1 1s.	3990*
Balance	198		
	£10,427		£10,427

* The actual amount received last year from subscriptions by these seventeen Societies was £5033.

N.B.—It is to be remembered that the Royal Medical and Chirurgical Society has a Debenture Debt of £33,000 which must be paid off or provided for in 1939. On the other hand, its assets are valued at upwards of £60,000.

In view of the facts that 3525 persons already subscribe £1 1*s.* to these eighteen societies per annum, that 1272 pay from £2 2*s.* upwards, and that under the proposed scheme many individuals who are not Fellows of the Royal Medical and Chirurgical Society would become Fellows of the Royal Society of Medicine at an annual subscription of £3 3*s.*, your Committee consider that the New Society would have a sufficient balance of income over expenditure to provide for improvements and for any sinking fund rendered necessary by extensions to 20, Hanover Square, supposing that all the scheduled societies joined.

This is shown more clearly from the following estimate of subscriptions which may be received from members of the various societies who are not Fellows of the Royal Medical and Chirurgical Society:

*600 members of societies as Fellows at £3 3*s.*	£1890
3200 members at £1 1*s.* . . .	3360
Library subscriptions at £1 1*s.*, say . .	525
	£5775

If this £5775 replaces the conservative estimate of

* The estimate of 600 is arrived at as follows:—The latest figures supplied by the accountants show that 1300 individuals pay £2 2*s.* upwards in annual subscriptions.

[751 individuals belong to 2 societies.
300 „ „ 3 „
134 „ „ 4 „
72 „ „ 5 „
31 „ „ 6 „
5 „ „ 7 „
6 „ „ 8 „
1 „ „ 9 „]

The number of Fellows of the Royal Medical and Chirurgical Society who pay £3 3*s.* per annum is 440; of these, 390 are members of one or more other societies. If we deduct 390 from 1300 we have left 910 as the number of individuals—apart from Fellows of the R.M.C.S. who pay £3 3*s.*—who belong to more than one society. The Committee suggest that probably 600 of these would become Fellows of the New Society at an annual subscription of £3 3*s.*

£3990 from subscriptions in the foregoing estimate of income and expenditure, the estimated annual balance of £198 becomes £1983.

We cannot obtain further information as to the financial practicability of the scheme until we know exactly which societies have definitely decided to join the proposed Royal Society.

Your Committee suggest that every member of the societies which amalgamate shall have the option of joining the New Society, subject to the rules of the said Society, without entrance fee, and that as far as possible members of these societies shall retain their present privileges in the New Society. For example, any Fellow of the Royal Medical and Chirurgical Society who has compounded for his subscription will be entitled to join the Royal Society of Medicine as a Fellow without further payment of any kind, and a member of any society who has compounded for his subscription will be entitled to join the corresponding section of the Royal Society of Medicine without further payment.

Your Committee consider that the following suggestions might be embodied in a scheme for the formation of the New Society, namely, that:

1. The New Society shall be called the Royal Society of Medicine [or the Royal Academy of Medicine, according to the vote of the societies concerned.]

2. All monies, books, premises, and other properties belonging to any of the societies which join the proposed union shall become the property of the New Society, and shall be vested in the Fellows.

3. The Society shall be housed at 20, Hanover Square.

Increased accommodation, if required, can be obtained by extending the premises, and by the absorption of certain rooms now occupied by tenants of the Royal Medical and Chirurgical Society.

4. [The Society shall meet four or more times a year to discuss subjects of medical and scientific interest.]

The Fellows of the Society shall meet annually for the election of Officers and other business: and four or more times a year for the election of Fellows and for such other business as may be desired by the Council.

*5. The Society shall consist of the following sections with power to add new sections, or to modify existing ones with the consent of the members:

1. Anæsthetic.
2. Clinical.
3. Dermatological.
4. Diseases of Children.
5. Epidemiological.
6. Laryngological, Otological, and Rhinological.
7. Medical.
8. Mental Medicine (Psychiatry).
9. Neurological.
10. Obstetrical and Gynæcological.
11. Odontological.
12. Ophthalmological.
13. Pathological.
14. State Medicine.
15. Surgical.
16. Therapeutical, including General Therapeutics, Pharmacology, Electro-therapeutics, Balneology, and Climatology.

It is hoped that in the early future an Anatomical and Physiological Section may be formed.

6. The Society shall consist of Fellows, Members, Honorary Fellows, and Honorary Members.

7. The Fellowship of the Society shall be restricted to men who are registered medical practitioners of the British

* N.B.—The Council of the Royal Medical and Chirurgical Society are of opinion that it would be premature at the present time to discuss the list of Sections of the new Society.

Empire, and to men whose scientific attainments are satisfactory to the General Council.

7*a*. The Election of Fellows shall be on the same lines as are at present followed in the Royal Medical and Chirurgical Society.

8. A Fellow shall pay, subject to any future bye-law, an annual subscription of £3 3*s*., and shall have the right to attend all meetings of the Society or of any of its sections, and shall have full use of the Library. After the Society is constituted (*i. e.* after a date to be subsequently determined) a new Fellow shall pay an Entrance Fee of £2 2*s*.

9. Each section shall have the power to elect as members of the Society such persons as the Council of the Section may think fit, provided that the names of the candidates proposed for election be previously submitted [by the Sectional Council to the Council of the Society for their approval] to the Council of the Society and approved by them.

10. A member of any particular Section shall pay, subject to any future bye-law, £1 1*s*. per annum, and shall have the right to attend all the meetings of his Section, but must pay an additional £1 1*s*. per annum if he wishes to have the use of the Library, and a further subscription of 10*s*. 6*d*. per annum for each additional Section.

11. The general management of the Society shall be under the control of a General Council, consisting of the President, the Presidents of the various Sections, two Treasurers, two Librarians, two Secretaries, and eight other Fellows. The Members of Council, with the exception of the Presidents of the Sections (who are to be *ex-officio* members) shall be elected by the Fellows.

12. The entire management of the funds of the Society shall be in the hands of a Finance Committee, subject to and acting as a Sub-Committee of the General Council.

13. In order to enable existing societies to retain their individuality as far as possible, each Section shall have

power to make the necessary laws and regulations for the conduct of its business.

14. The Presidents of Sections shall be *ex officio* Vice-Presidents of the Society, and shall serve on the General Council of the Society.

15. In the estimate on page ccviii provision has been made for a monthly publication (July, August, and September excepted), to be called 'The Proceedings of the Royal Society of Medicine.' This publication will be under the control of an Editorial Committee, and will contain a record of the work of each Section during the previous month. By such prompt publication the value of the records will be greatly increased.

In addition, the Committee will select, subject to the approval of the General Council, such papers as they may consider worthy of publication in the 'Transactions' of the Society.

The Committee further *suggest* that it would be to the advantage of the Royal Society if a Committee were formed of representatives of the various sections, charged with the duty of receiving all papers to be read before the Society or its Sections. This Committee should have power to decide before which Section any given paper should be read, and to decide whether other Sections or the whole Society should be asked to join in the discussion. Authors of papers should have the privilege of suggesting the Section or Sections before which they wished to read their papers.

It is hoped greatly to increase the value of the present Library at 20 Hanover Square, and to provide an additional reading-room for the use of Fellows and Members.

Your Committee ask the various Societies concerned to consider the scheme submitted *at an early date* and to inform the Honorary Secretaries of their views. As soon

as the Committee is in possession of these views further steps can be taken.

In conclusion your Committee beg to express the unanimous opinion that the Union of Medical Societies will do much to enhance the prestige of British Medicine, that it will increase the value of the work at present carried out by existing societies, and that it will be to the great convenience of the Medical Profession.

(Signed) W. S. CHURCH, *Chairman.*

R. DOUGLAS POWELL.
HENRY HEAD.
JOHN LANGTON.
DAVID H. GOODSALL.
G. H. MAKINS.
CHARTERS J. SYMONDS.

ARTHUR LATHAM, } *Honorary*
HERBERT S. PENDLEBURY, } *Secretaries*

UNION OF MEDICAL SOCIETIES

SPECIAL GENERAL MEETING

HELD AT

THE SOCIETY'S HOUSE, 20, HANOVER SQUARE, LONDON, W.,
ON THURSDAY, NOVEMBER 23RD, 1905, AT 5 O'CLOCK, P.M.

SIR RICHARD DOUGLAS POWELL, Bart., K.C.V.O., M.D.,
President, in the Chair.

G. NEWTON PITT, M.D., }
STEPHEN PAGET, } Hon. Secs.

Present—159 Fellows, and the Secretary.

The PRESIDENT called upon Dr. Newton Pitt (Hon. Secretary) to read the notice convening the meeting, which was as follows:

20, HANOVER SQUARE;
November 14th, 1905.

NOTICE IS HEREBY GIVEN that a SPECIAL GENERAL MEETING of the Royal Medical and Chirurgical Society of London will be held at 20, Hanover Square, on Thursday, the Twenty-third day of November instant, at Five o'clock in the afternoon, for the following purpose, viz. to consider the accompanying "*Report on the Union of Medical Societies as amended and adopted at the Meeting of the General Committee of Representatives of the Societies held on July 19, 1905*," and, if approved, to pass the following resolutions:

1, That the "*Report on the Union of Medical Societies as amended and adopted at the Meeting of the General Committee of Representatives of the Societies held on July 19, 1905*," be and is hereby approved and

adopted, subject to the modifications recommended by the Council of the Royal Medical and Chirurgical Society, and that the Council be and is hereby instructed to endeavour, in negotiating with the General Committee of Representatives of the Societies, to secure the adoption of all those modifications.

2, That the Council be and is hereby authorised to take all such steps and proceedings and to do such acts and things as the Council, in its discretion, shall consider necessary or expedient for carrying into effect the amalgamation of the Royal Medical and Chirurgical Society with all or any sufficient number (at the discretion of the Council) of the Societies named in the "*Report on the Union of Medical Societies as amended and adopted at the Meeting of the General Committee of Representatives of the Societies held on July 19, 1905*," or with any other Society or Societies, either in accordance with the scheme set forth in the Report (subject or not as the Council shall think fit to the modifications recommended by the Council or to such (if any) other modifications, amendments, or alterations as the Council in its discretion shall consider necessary or expedient), or in accordance with such other scheme as the Council in its discretion shall think advisable.

A copy of the Report referred to above is enclosed herewith. The print as it stands in black ink is the Report originally approved by the General Committee of Representatives; the modifications and amendments shown in red ink[1] are those recommended by the Council of the Royal Medical and Chirurgical Society.

(Signed) RICHARD DOUGLAS POWELL, *President.*
G. NEWTON PITT, }
STEPHEN PAGET, } *Hon. Secretaries.*
J. Y. W. MACALISTER, *Secretary.*

The PRESIDENT.—Gentlemen, you all have in your hands, I think, the Report which we have met to discuss this evening. The object of our meeting is to consider this Report, with certain emendations, which you will observe are marked in red ink,[1] and to approve of it or otherwise. If approved

[1] In the Report, as printed in this volume, underlining and square brackets are used instead of red ink to indicate alterations.

you are asked to instruct the Council of the Society to proceed further in the matter. Perhaps I may be allowed very briefly to recapitulate the chief points with regard to the history of this movement, so that we may know exactly where we are; and I would first of all point out, as I think it is important we should bear this in mind, that this Society now occupies a stronger position, both as regards its finance and the number of its Fellows, than it has at any time during the 100 years which have elapsed since its foundation. It has, as the centenary volume records, taken a large share in advancing the science of medicine and in extending and testing new methods of medical and surgical practice. It has seemed to many Fellows, however, that a larger scheme is needed than that on which the Society was founded 100 years ago, in order fully to embrace the whole domain of medicine, and to advance the special branches which have developed in the growth of medical science, whilst at the same time keeping them in firm and wholesome union with the great principles of medicine. A greater adaptability in former years might perhaps have met the needs of new development and progress, and have avoided the necessity for the creation of so many new societies. All these societies are engaged in the output of admirable work. But it is certain that the expense of doing so is very great, that several societies are engaged in discussing practically the same questions at meetings sometimes simultaneously and often scantily attended. With a view to remedy this it was unanimously resolved at the last annual meeting of this Society,

> "That the Council of the Society be requested, with as little delay as possible, to invite the leading medical societies of London to arrange for a joint meeting for the purpose of considering the advisability of amalgamating, and to take the necessary preliminary steps for that purpose."

At the next meeting of your Council a resolution was adopted requesting the President of the Royal College of

Physicians to convene a general meeting of the members of the London medical societies for the purpose of considering the question of union. At this meeting the proposal to amalgamate the central societies was cordially accepted, and a committee of delegates from the various societies was formed, which, after many sittings, has drawn up the Report which is now before you. This Report has been under the careful consideration of your Council, and with certain modifications of variable importance it has been approved. A copy of the Report has been circulated amongst the Fellows of the Society showing the modifications of the Council printed in red. The object of the present general meeting of the Society is not so much to criticise this scheme clause by clause as to express general approval of it or otherwise, as a basis upon which this Society would desire the process of amalgamation to go forward. Possibly each society will send in to the committee of delegates a similarly annotated report and express its approval or otherwise of the general scheme, and its willingness or otherwise to go forward with it. There must then be a final meeting of all the societies. It is impossible, and it would be unprofitable, to discuss minute details. But I would point out that the financial side of the scheme has been pretty fully set out under expert direction, and on the moderate basis of success assumed it would appear to be very sound. It must be here noted that this Society is indebted to certain debenture-holders among its Fellows, and that this debt forms a first mortgage charge upon the property of the Society, and this liability must be fully safeguarded in any change that may occur. This being fully seen to, the debenture-holders will probably be satisfied to remain in possession of debentures which they originally took from a loyal feeling towards the Society. Other liabilities of other societies will of course have to be equally secured. It is to be hoped that a large proportion of the constituents of the new Society will join as Fellows. This will be so if the matter is taken up with that degree of warmth

and enthusiasm that has hitherto been shown with regard to it. The question as to the number of sections into which the subject of medicine should be divided must in great measure depend upon the number of societies that join. The Council has only ventured to express a general opinion on this point, for it is felt that it must stand over for discussion and decision by the delegates from all the societies who agree to join. It is obvious that the combined library of the new Society would be much larger and more valuable than our present library, large and valuable as that library is, and that its accommodation and convenience would be enhanced, and this is a point which will be jealously watched by this Society, "the formation of a library" being one of the primary objects of its original foundation, and a main object of its solicitude during the 100 years of its existence. Another main feature of the new Society will be, it is hoped, the full and punctual publication monthly of the transactions of each section. This will require careful organisation, and, in the opinion of your Council, will need the services of a paid editorial staff. Finally, it is to be hoped that in the new Society steps will be taken to secure a larger amount of social or conversational interchange of ideas amongst its Fellows and Members, and thus carry out one of the ideas in the minds of the founders of our own Society, "the affording an opportunity for an easy and agreeable exchange of practical knowledge." With those preliminary remarks I would point out that, if the first resolution be accepted, the second will follow almost as a matter of course, as it is merely defining in technical and somewhat involved phraseology the powers required by the Council to carry your wishes into effect. I have no doubt the main discussion will arise upon the first resolution, and I will ask Sir William Church if he will commence the discussion by moving it. Before doing so I think the Hon. Secretaries have to mention the names of some gentlemen who would have been here but have been prevented from coming.

Dr. NEWTON PITT (Hon. Secretary) read a number of letters from Fellows who expressed their regret that they could not be present, all of them expressing their approval of the Council's proposals.

Sir WILLIAM CHURCH.—Mr. President and Gentlemen,—In obedience to your wishes I rise to move the first resolution, although I cannot but feel that it would have been better if this resolution had been allocated to one who was not so closely identified with the matter in hand as I am myself. That being the case, the remarks I shall make this evening will be very few, and I would like to reserve any further observations that I may make, as I think it is possible I may be asked some questions in the course of the discussion which will follow. Therefore I will now very briefly move the first resolution. Before doing so formally I would like to say that I have gathered, both from what I heard an afternoon or two ago at the Clinical Society and also from what I hear generally, that there is some misapprehension with regard to this scheme which is placed before you. Some have thought that it was a scheme for amalgamating the Societies. Of course that was the business of the Executive Committee appointed at the meeting of the General Committee of Representatives. What the Committee had to do was to investigate and see whether it was possible, from financial and other reasons, for amalgamation to take place. That is treated of in the first part of this scheme which is laid before you, and a very great deal of time and labour was gone through by that Committee, and I think I may say that they were satisfied that it was feasible somewhat on the lines laid down. We endeavoured to give such information as we could to show what were the resources of the existing Societies who were likely to join. Until we know what Societies are going to join together it is impossible to go into financial details more closely than they have been gone into in this Report. Another misapprehension which I found was in the minds of some was the idea that the scheme was that all medical societies in the metropolitan

district, and even in the provinces, should be affiliated to this new amalgamated body. Now, those of us who initiated this movement had never that idea in our minds. We only thought that what you might call the home societies, that is to say those that meet in this house and those which meet in the Medical Society's premises might be amalgamated together, and, if so, it would be a very great step gained. Any of you who have at all followed the Report must have seen the great difficulty that there has been in arriving at any satisfactory conclusions as to the feasibility of that scheme. It was impossible to go into the larger scheme, which was never intended, that some medical societies on the periphery of London, and also provincial ones, should in any sense be affiliated with this central body. That may come in time, but, at all events, the time is not yet ripe for anything of the sort. The latter part of the Report, as is definitely stated in it, contains suggestions which the Committee thought might be embodied in the scheme for the formation of a new Society. They were specially put in, in order that when the various Societies had this Report in their hands and considered it they might offer suggestions as to how far they would be prepared to accept these suggestions, or to make further suggestions which might appear to them to be preferable. At present I do not know what will be the course of procedure in the future, whether the same Executive Committee which was appointed at the meeting of the General Committee of Representatives of July 19th will be called together again, or whether a new committee of representatives of the Societies will have to be called together to consider all the answers that we get from various Societies, and the suggestions which they make. It will then perhaps be possible to draw up a more or less definite scheme to place before these societies. Our President has reminded you of the very important position which this Society holds. It does not claim to be the oldest, but I think, without any undue pride, we may lay claim to be the most powerful—we are certainly the richest

—of these medical societies. Therefore this Society, I think, is doing more perhaps than any other Society in the way of endeavouring to promote the general welfare of the profession and the welfare of medicine in coming forward and taking, as it has done, a very leading part in endeavouring to obtain this desirable amalgamation. For that it is desirable I think is admitted by all, or admitted by almost all. Therefore I will not say anything more upon that ground. But certainly it is a very serious matter for this Society, and, as Hon. Treasurer, I would like to tell the Society that, during the more than ten years I have been Treasurer, it has never been in a sounder financial position than it is now. We have managed to wipe out our floating debt, which was some years ago very considerable, and, as you have seen in the accounts laid before you from year to year, during the last few years our annual expenditure has been within our annual income. So that we are in a perfectly sound position, and in that way we have nothing to gain by amalgamation. I would therefore now formally move the resolution, and I shall be happy, so far as I am able, to answer any questions which may be put with regard to the matter contained in this Report. The resolution which I have to put is as follows:

> "That the '*Report on the Union of Medical Societies as amended and adopted at the Meeting of the General Committee of Representatives of the Societies held on July 19th, 1905*,' be and is hereby approved and adopted, subject to the modifications recommended by the Council of the Royal Medical and Chirurgical Society, and that the Council be and is hereby instructed to endeavour, in negotiating with the General Committee of Representatives of the Societies, to secure the adoption of all those modifications."

Dr. ROBERT HUTCHISON.—I rise, sir, I confess, with some diffidence, to second the proposal which has been moved by Sir William Church. On an occasion like this it may be thought, and quite naturally, that the initiative should be left to senior Fellows in the Society, but I venture to

think, sir, that the junior Fellows are concerned in this matter in a way more immediate, if possible, than the seniors, and that on more than one ground. In the first place, this proposal which lies before us, if it be adopted, will tend, we believe, to great pecuniary economy, and I need hardly say that anything which makes for pecuniary economy appeals with special force to the junior Fellows of the Society. We hope, also, that it will make for greater ease and efficiency in the working of the different societies which at present exist, and that, I need hardly say, will also cause satisfaction to the juniors; for in the organisation of the present special societies, in arranging for their meetings, and in their secretarial work, the juniors have necessarily to play a leading part. On those two grounds I am sure that the junior Fellows of the Society will heartily support this proposal. But further, sir, I would say that, looking to the future, our support is again most essential to the success of any such amalgamation as is projected. After all, it is we, the junior Fellows, who in future will bear the burden and heat of the day, and upon us will fall the responsibility of seeing that this proposal, if it be carried out, is one that tends to success and prosperity. And so much is that the case, that I will go so far as to say that if there were any strong feeling against it felt by the junior Fellows, and if it were passed without their approval, such a proposal would be foredoomed to fail. But, sir, I venture to think that our support will be amply forthcoming. In these days, when even the most junior of us is necessarily more or less a specialist, I cannot help thinking that such a proposal as this, which will give expression to the essential solidarity of the profession, which will break down the walls which separate us, and unite us all, as it undoubtedly will, and make it easier for us to advance medical science in London, I cannot help thinking that such a proposal is bound to succeed, and it is in that belief that I commend it, not only to this meeting, but more especially to my colleagues in the junior ranks of the profession.

The PRESIDENT.—The resolution has now been moved and seconded, and we shall be very glad indeed to hear the views of any Fellow present who wishes to speak on the subject.

LIEUT.-COLONEL MYERS.—Mr. President and Fellows, I wish to rise to move an amendment, which I hope someone will second:

> "That these resolutions be postponed for six months, so that before any decision is come to by the Society we may be informed what are the responsibilities attaching to amalgamation, and what are the debts, if any, which may be owing by other societies."

In all probability there are some debts. Are we going to accept the debts of these various societies? We have had no information about that whatever. As a very old Fellow of this Society—I may say I am in my forty-third year of Fellowship—I wish to ask you to bear with me whilst I make a few remarks about this subject, feeling very strongly in opposition to the present movement. What are our gains and what are our losses in such a very grave movement, far graver, it may be, than we can possibly at the present moment realise? With regard to our gains—prestige. It is the prestige of a very great Society, we are told, and that we must lose something for the body politic; but after all, it only means the conglomeration of various societies affiliated to the parent societies. What good will come of it? There may be many other societies formed, and I do not see how the new Society could well refuse to take them in. Who knows but that in the course of time we might have a Chiropodistical Society, and before the last word is said even an Umbilicological Society! Now as to economy. We all agree that it will be very economical for other societies. I was told at the first meeting of the General Committee by a friend that he would certainly vote for the amalgamation, because he subscribes twenty-two guineas, and in future he will only have to pay three guineas. That is of course very practical, and I cannot help thinking that this movement is

greatly dominated by economy. But is economy everything? I think not. Now what are our losses? It is only a few months ago, Mr. President, that we celebrated the centenary of this Society. And why did we celebrate it? To honour it, and to keep green the memories of those great men who have developed the Society into what Sir William Church stated had become the first medical society in the kingdom, and I may also add a society of world-wide celebrity. Well, now, what do you propose to do? You are proposing to commit suicide, that is what it comes to. ("No, no.") You say "no," but it is suicide, because the Society will entirely lose its identity, and those of us who are now old will pass away, and in the course of time the Council will not consist chiefly of Medico-Chirurgical Fellows, but of fellows of all societies. In fact, we might not have one representative of this Society on the Council of the new Society, when there are so many sections to be represented. I cannot understand how we can agree to such a change. Now, sir, here we stand with assets of about £64,000, with a balance in our bank of £1000, and a magnificent library of which we all cannot help being proud, and what is to become of us? We are going to wipe the whole thing out, and be absorbed. I can understand that the smaller societies will accept this movement as a very great boon, but even for them, as I may yet point out, it will not be all gain. Now the last matter I want to refer to—and I am sorry to have kept you so long—has reference to the question of the publication of the 'Transactions.' Looking over the volumes of this Society, will anyone present, especially the authors, say that there are any papers in these volumes that ought to be expunged? I think not. Would the Medical, the Clinical, or the Pathological Society, say they would like to see any of the papers which they have selected deleted from their volumes? Certainly not. And why should not the other societies which have carefully studied their papers, feel that their volumes are equally valuable and deserving of publication? What is the proposal? An Editorial Committee, with power to select what contribu-

tions shall appear in the volume of 'Transactions' of this new society. It must be a fairly large Committee, because the subjects to be dealt with will spread over such a large range. Let us take an instance. Supposing a Fellow of the Balneological Society were called upon to form an opinion upon a contribution from the "Laryngological, Otological, and Rhinological Association." I do not think he would be quite fitted for it. Therefore the Editorial Committee should have a representative from every section upon it. Well, sir, I must say I pity, from the bottom of my heart, that Editorial Committee. And not only will that Committee have to select the papers for the Annual volume, but they will have to go through, monthly, all the reports of sections in order to produce a monthly journal. Sir, I shall be surprised if there are enough Solomons in the profession to undertake so grave a task, and I question whether their mental strength would stand the strain. Their position will also be a difficult one from this point of view, that they will probably have to produce a volume the size of the Post Office Directory. That is what it would have to be if every Society had what they wanted published. I hope I have not detained the Society too long, and I appeal to you to think the matter over very carefully before you allow your Society to commit suicide.

The PRESIDENT.—Is this amendment seconded?

These was no response.

The PRESIDENT.—Unless there is a seconder, as there does not appear to be, the amendment necessarily falls. I would like, however, to say to Colonel Myers that the Editorial Committee is only a detail which has yet to be considered by the representatives of all the combining societies, if they arrive at that stage. It is not a necessary feature of the new scheme.

Dr. BATTEN.—May I ask if it is proposed to adopt all the amendments of the Council *en bloc*, or is it proposed to discuss each individual one? There is one amendment put down which requires consideration, and that is apropos of paragraph 5, in regard to which it is proposed by the

Council, "The Council of the Royal Medical and Chirurgical Society are of opinion that it would be premature at the present time to discuss the list of sections of the new Society." With regard to that, I should have thought that the list of Societies which is put down there should be stated as the minimum. I think it should be clear that the new Society may add to these, but they shall not take away from them. I say that because there are individual societies, for instance the Neurological, which, if the section of Neurology were cut out, could not join the new Society. It seems to me, therefore, that that amendment, if it is down as an amendment, is a mistake, and that those sections should exist as the minimum of sections, to which the new Society might add other sections if it so desired.

Dr. EDGAR WILLETT.—May I say one or two words, Sir? I represent a small Society—that which happens for alphabetical reasons to be put first, namely, the Anæsthetic Section. The question has come up before the Society of Anæsthetists—I mention this quite informally—and it has been decided that the Society could not join the amalgamation unless women were admitted, and that view I know is held by some other societies as well, including the Obstetrical Society. If it can be modified in this way on page 5, section 7, and made to read, "The Fellowship of the Society shall be restricted to registered medical practitioners of the British Empire, and those whose scientific attainments are satisfactory to the General Council," it would meet the objection.

The PRESIDENT.—Of course, we shall hear later on the views of the Society of Anæsthetists, the views of the Obstetrical Society, and the views of the Gynæcological Society, but we are concerned at the present moment with the views of this Society. It is perfectly open to you to move an amendment if you desire to do so.

Dr. EDGAR WILLETT.—If the resolution which Sir William Church has moved prevents eventually the other Societies joining on the conditions which they wish to adopt, my amendment is what I have read. If the whole of this

amended scheme as indicated in red ink were passed that would definitely cut out women.

A FELLOW.—The word "man" has before now been interpreted to include "woman."

Dr. SEYMOUR TAYLOR.—I have been carefully perusing this document which has been sent to the Fellows, and have devoted to it a good deal of time and attention, and it was one of the difficulties that I foresaw would come before us when I read paragraph 7, page 5. I read certain things there which I said to myself would be a stumbling-block for our scheme. I would draw attention to the last line of paragraph 7: "and to men whose scientific attainments are satisfactory to the General Council." I would ask information whether it is the intention of the Committee who drew up this Report that laymen should be included as Honorary Fellows in the Society. If so, I would ask whether it should not pass a resolution or instructions to the Committee that they should establish a law that they shall have no management whatever, and no voice in the management of the Society. Secondly, there is the question of sex which I have annotated, and I was prepared to see some member of the Society rise—Dr. Edgar Willett has raised the point—on that question. I thought there would be some difficulty in certain societies joining under this scheme. I will tell you at once that I see more than difficulty, I can see that the scheme may even be wrecked, if you do allow women to come into the Society, and I think it should be an instruction to the Committee who drew up these further Rules, that the word "man" shall not include "woman." There is another point, namely, about the finance of this coming institution, which it is necessary for us to know about. First of all, we have many tenants in this building, and it would be necessary that the Fellows of the Society should be told what notice it is necessary to give to these tenants, so that we should not get them out without undue compensation.

Sir WILLIAM CHURCH.—Perhaps I may be allowed to say, in answer to one or two questions, that the points that have

been raised have been present to the minds of the Committee, and I thought I made it clear in my remarks that this scheme is not a scheme for the Amalgamated Society; it is a scheme to be sent round to the societies, of which we are one, showing in what way the Committee thought amalgamation might be made. These points will have to be considered when answers from the societies come in. Do I understand Dr. Seymour Taylor to say that this Society is here to pass a resolution to-night that it will have nothing to do with this scheme if women are admitted Fellows? It was suggested that ladies, or women, should not be admitted Fellows, but it was felt that several very important societies would not wish to lose their lady members. There is a difference between a Fellow and an Associate or Member of the Society. What this scheme proposes is, that women should not be Fellows of this Society, but that they are members of its various branches or sections. Then with regard to Colonel Myers, I might mention, although he did not get any support, that what he asks us to wait six months for, has long long ago been examined and gone into. The Committee are aware of what liabilities all the other societies may have, and I am happy to be able to tell you that they have practically no liabilities, with the exception of the Medical Society, and that is such a sound concern that it is perfectly able to meet its liabilities at any moment. I will not impose longer on the meeting, but I thought it was worth while answering those points.

The PRESIDENT.—If no other Fellow wishes to address the meeting, I will now ask you to vote on the first resolution, and I ask those who are in favour of it to hold up their hands.

The resolution was carried, with only two dissentients, amid applause.

Dr. P. H. PYE-SMITH, F.R.S.—You have asked me, Sir, to move the second resolution. It is a somewhat long one, and you are probably all familiar with it after having read it through. But I hope other Fellows will agree with me that it is not unnecessarily long, and if I may take it as

read I think it will shorten the time of my speech. I would only congratulate the Society on having advanced thus far, and while we all must have felt great sympathy with one of our esteemed senior Fellows in his attachment to the old Society and its unchanged glories, I think we must feel also that the mere fact that this Society has done such admirable work, and has attained such a high position during so many past years, gives rise to the suspicion, considering how the world around us changes, that the time may have come when a forward step should be taken; in other words, that reform is much better than perseverance in methods which admit of no change. And this is particularly the case, I think, with regard to medical and other societies, and I think we owe a great deal, Sir, to yourself and to Sir William Church and other members of the Royal Medical and Chirurgical Society, who conducted the Association in the way that they have, and that they have seen the direction in which the course of events is trending. Those of us who attend medical and other societies must have seen that the time for reading papers is less than it was once, that discussions are less formal than they were, and that on the whole the less which is read and the more which is argued the better. That is the case with the Royal Society and other societies, and it is certainly the case in the Pathological Society and in the Physiological Society, and in both the Societies which cultivate Dermatology. And that is the direction in which, I take it, reform is required, and which I trust we are taking a step in the direction of cultivating. I hope the Committee will find it an advantage to make a change in that respect. Moreover, we have in this Society, two, I may say three, advantages which will be perfectly intact under the new scheme, and which are, to my mind, more valuable than anything which we can possibly lose. One is our magnificent library, the other is our admirable 'Transactions,' and the other is the memories which surround the Society, and which are not the least precious among our possessions. Therefore I move, with great

confidence, this second resolution, which gives power to the Council of this Society to act in this matter, and it is not until action has been taken, backed up by the decision we have made, that the Committee will be able to negotiate advantageously with the other Societies. That these negotiations will end satisfactorily, I am sure, is to the advantage not only of this Society, but of the other Societies which are interested in it. One word only before I sit down, and that is in regard to the proposed name; nothing has been settled in regard to that at present. I would ask the Council to carefully consider, before approaching other Societies, whether the proposal to use the words "Royal Academy of Medicine" is not in every way better than the word "Society." I think we all feel it might savour of arrogation of the claim of another Society, that of Fellow of the Royal Society, and I think the terms scarcely cover the suggestion. It would be a mistake to have any possibility of confusion, and I cannot help thinking that this is an argument which should weigh very strongly. It may be said that the term "Royal Academy" in itself might give rise to confusion, but that is not the case, I think, because we are a scientific Society like the Royal Society, all the earlier members of which cultivated medicine. But the Royal Academy is so very different from our sphere, even in the beautifully drawn Pathological Proceedings, that there should be no possible confusion. Moreover there is another advantage, that our sister kingdom of Ireland—where the same amalgamation was carried out very successfully, and is a moral to us Englishmen, who cannot believe that Irishmen can compose differences, and see great objects—invented and applied the term "Royal Academy of Medicine in Ireland." With that recommendation I beg to move the resolution, which is as follows:

> "That the Council be and is hereby authorised to take all such steps and proceedings, and to do all such acts and things as the Council, in its discretion, shall consider necessary or expedient for carrying into effect the amalgamation of the Royal Medical and Chirurgical

Society with all or any sufficient number (at the discretion of the Council) of the Societies named in the '*Report on the Union of Medical Societies as amended and adopted at the Meeting of the General Committee of Representatives of the Societies held on July 19th, 1905*,' or with any other Society or Societies, either in accordance with the scheme set forth in the Report (subject or not as the Council shall think fit to the modifications recommended by the Couucil, or to such (if any) other modifications, amendments, or alterations as the Council in its discretion shall consider necessary or expedient), or in accordance with such other scheme as the Council, in its discretion, shall think advisable."

Mr. D'ARCY POWER.—Sir, I rise with great pleasure to second the resolution which has been spoken to by Dr. Pye-Smith. It is nothing but a vote of confidence, I think, in our Council, whom we elect annually, and it is therefore with very great pleasure that we leave these further details in their hands. They will, I have no doubt, carry out in every possible way to our satisfaction the details of the scheme which we have just voted upon. I suppose it rests with ourselves to make ourselves *primus inter pares*. It is quite possible. We are the chief Society of England, and have been so for many years, and it remains for the Fellows to continue to place ourselves in that position, even amongst the other societies, when we are amalgamated with them. Therefore very few words are required from me in seconding the proposition.

The PRESIDENT.—This is the second proposition. I think it will be much easier to each gentleman to read it himself than for me to read it out. It is practically only to carry into effect the first. I do not suppose there will be very much discussion upon it, but if there is any Fellow present who would like to speak upon it before we put it to the vote, I am sure we shall be very pleased to hear what he has to say.

Dr. BUZZARD.—I should like to point out to Dr. Pye-Smith, in reference to the remark which fell from him concerning the projected name of the new Society, that we have just round the corner here, in Tenterden Street, the

Royal Academy of Music, which has been established there for a great number of years. Members of that Academy constantly use the letters R.A.M., and those letters are consequently recognised as indicating the Royal Academy of Music. There is a convenience, no doubt, in being able to abbreviate a designation, and we should, to abbreviate our designation, naturally use the letters R.A.M. I am inclined to think that it is very possible some difficulties may arise in regard to that.

Dr. PYE-SMITH.—But the letter F could come before it, which would surely make a difference?

Dr. BUZZARD.—But there are also Fellows of the Royal Academy of Music.

The PRESIDENT.—This question must arise when we have got a little further on, and I think we have reached a further stage to-day, but when the whole scheme comes before the representatives of all the Societies we shall finally have to discuss the question of the names. It is a very difficult question to decide, but, after all, if we get the Society, I am sure we can find a name for it. If no other Fellow wishes to address the meeting, I will put this second resolution.

The resolution was then put and carried unanimously.

The PRESIDENT.—That concludes our business, gentlemen, and I feel deeply gratified, and I congratulate the Society on having so unanimously come to these decisions. I think we all feel that the objects of this Society are greater even than the Society itself, great as that Society has been during the last 100 years.

THE PATHOLOGY AND PREVENTION OF SECONDARY PAROTITIS

BY

RUPERT BUCKNALL, M.S., F.R.C.S.

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SECONDARY parotitis, though a comparatively rare affection, has always attracted a certain amount of attention clinically, on account of the varying circumstances under which it arises, the special symptoms which it presents, and the serious consequences which it may occasion.

Its pathology, however, has proved of even greater interest, on account of its obscurity: much discussion has centred round it, and various different theories have been advanced to explain its origin, but it still remains a subject for speculation.

Thus in the current text-books various different theories of origin are mentioned side by side, on a more or less equal footing; and if any choice is made in English works on the subject, the theory advocated is the one put forward by Paget, who regarded the affection as a "sympathetic inflammation."

On the continent, however, the theory of Hanau and Pilliet, who regarded secondary parotitis as due to the infection of Stenson's duct with micro-organisms from the mouth, is more commonly accepted.

The object of the following paper is to collect all the evidence that has been published in favour of Hanau and Pilliet's theory, and also to bring forward additional evidence in support of it.

It is hoped that by doing so it will be possible to show that secondary parotitis is invariably due to the direct infection of Stenson's duct with micro-organisms from the mouth; a conclusion of some importance, since it leads to the suggestion that secondary parotitis may invariably be prevented from occurring for the following reasons:— (1) It has been shown by experiment that the parotid duct in animals can only be infected if certain conditions predisposing to infection are present; (2) it can be shown that the same predisposing conditions are present in all cases of secondary parotitis in man, and that they may be avoided. It is probable, therefore, that by preventing these conditions, in those cases in which secondary parotitis is liable to occur, its onset may be invariably avoided.

The importance of such a possibility is more evident when it is borne in mind that secondary parotitis is occasionally a fatal disease, on its own account; and that it even more frequently leads to death in an indirect manner by adding to the grave symptoms of the primary disorder already present.

As, however, the theory of origin now advocated is not the most generally accepted one in this country, it will render the subject more complete if the claims of the various other theories are submitted to criticism before the evidence in favour of duct infection is examined.

Moreover, for purposes of reference it will be more convenient, in dealing with the various theories, to begin by giving a brief summary of the chief features of secondary parotitis and the circumstances under which it arises.

The brief account now given is based on a personal examination of twelve cases, and a study of the published records of over two hundred others.

The subsequent critical survey of the pathology of the affection is based on an examination of evidence derived from three sources: (1) a study of all the actual pathological facts previously published; (2) an examination of the morbid anatomy and bacteriology of six successive cases; (3) a study of certain facts in connection with the causes which lead to infection of the parotid duct, which have been established by experiment.

I. Features of Secondary Parotitis.

Secondary parotitis is an acute inflammatory affection of the parotid gland which is characterised, and distinguished from primary parotitis or mumps in three ways: —(1) It invariably occurs as a complication during the course of some other affection; (2) it is not contagious; (3) it not infrequently suppurates, and gives rise to a parotid abscess.

(1) *The Circumstances under which Secondary Parotitis Arises.*

A study of a large number of cases shows that secondary parotitis has been met with during the course of a large number of different affections, which may be grouped under three headings, as follows:

(A) Acute diseases.
(B) Chronic diseases.
(C) Post-operative states.

(A) The acute diseases during the course of which parotitis has occurred may be arranged as follows:— (1) Typhus fever; (2) enteric fever; (3) scarlet fever; (4) diphtheria; (5) pneumonia; (6) appendicitis, salpingitis, and acute pelvic, and peritoneal inflammations of all kinds; (7) cystitis and pyelonephritis. Of these, typhus

fever, when it occurs, is most frequently complicated by parotitis, Keen, in the Toner Lecture, having collected nearly two hundred examples.

At the present time, however, parotitis is usually met with during the course of enteric fever, pneumonia, and the different varieties of peritonitis.

(B) The chronic diseases are far less frequently followed by parotitis than the acute ones. They include:—(1) Diabetes; (2) mercurial stomatitis; (3) iodism and plumbism; (4) general paralysis of the insane; (5) any form of grave asthenia; (6) disorders of menstruation.

(C) The operations followed by parotitis have almost invariably been operations on the abdomen, and particularly operations on the pelvic organs. Many of them have been undertaken for septic disorders, and others have become septic during the performance of the operation. A certain proportion, on the other hand, have run an aseptic course from start to finish. Together they include:—(1) Exploratory laparotomy; (2) gastrotomy, gastrostomy, gastro-enterostomy and other operations on the stomach, and pylorus; (3) enterectomy; (4) colotomy; (5) for radical cure, and strangulation of all kinds; (6) for appendicitis, and other forms of peritonitis; (7) ovariotomy, and operations on the tubes and uterus; (8) renal operations; (9) operations on the liver and bile passages.

Of these operations, those in which sepsis has been present have been far more frequently followed by secondary parotitis than the aseptic ones; in fact, the only aseptic operation at all frequently followed by parotitis is ovariotomy as practised in former times. This special incidence will be fully explained later, when dealing with the theory of origin now advocated.

Time of Onset of Secondary Parotitis.

When occurring during the course of an acute or chronic disease, the day of the disease on which secondary parotitis arises is too variable to admit of any classifica-

tion, for in some instances it has arisen during the first few days, while in others it has not appeared until the period of convalescence has been established.

When following operations the date of onset has varied from the second to the thirtieth day, but as a rule parotitis has appeared from the sixth to the eighth day after operation.

(2) *Symptoms and Course of Secondary Parotitis.*

The symptoms of secondary parotitis may be divided into *local* and *general.*

The disease begins locally with pain and swelling in the parotid region.

The pain is aching or shooting in character, at first strictly localised to the parotid, and later spreading to other areas supplied by the fifth nerve, such as the side of the head and ear, and often being most acutely felt in the last-named situation. Movement of the jaw becomes painful and impossible, and attempts at movement are liable to cause sudden exacerbations of pain.

At first the swelling is usually situated near the centre of the parotid gland. It is firm and tender, and in most cases soon extends to the other parts of the gland, so as to give rise to a typical parotid swelling.

Very soon the swelling extends to the overlying cheek and adjacent parts, the whole cheek becoming brawny, and the eyelids puffy and closed, about the fourth day.

A little later the lips become swollen and œdematous, and the whole side of the face presents a brawny appearance, and feels tense. The skin may become hot and red if suppuration is about to occur.

THE GENERAL SYMPTOMS come on almost immediately. They consist of fever, malaise, depression, and thirst. Dryness of the mouth is also a source of discomfort, though it has often been complained of before the parotitis arises. The rise of temperature may be ushered in by a rigor, but this is rare, and as a rule a rise of from one to three degrees

occurs and persists. If fever was previously present such a rise may be less evident.

Giddiness, tinnitus, and headache due to obstructive congestion of the head and scalp may now be complained of; all the symptoms usually reach their acme about the fourth or fifth day.

The swelling may now subside or suppuration may occur.

In the former instance the swelling, pain, and discomfort rapidly become less marked, the temperature gradually falls, and complete recovery ensues in about a fortnight.

In the event of suppuration occurring, as it does in exactly half the number of recorded cases (sixty-five, sixty-four) the symptoms persist and become worse.

The swelling becomes more brawny, red, and œdematous, and softening may occur about its centre. The temperature becomes irregular, the pain lancinating, and rigors may occasionally occur.

In a considerable number of instances, however, the actual presence of pus is not easy to determine, for the skin may remain pale, and fluctuation may be absent on account of the fact that the pus lies deeply beneath the tense parotid fascia.

Should pus form, the case may pursue one of several different courses.

1. In the first place the pus may slowly become absorbed, convalescence being prolonged, and recovery slow.

2. Secondly, the pus may find its way into the parotid duct and drain away into the mouth.

3. Thirdly, the abscess may be incised and efficiently opened, in which case gradual healing of the wound usually follows, if operative interference has been sufficiently timely.

4. If left too long the pus may burst into the mouth or less often the external auditory meatus. In either instance it may drain efficiently, but such fortunate results

after improper treatment are however rare, and much more commonly one or other of the following accidents supervenes: (1) The patient may die of septic poisoning without the abscess bursting at all; (2) many sinuses may be formed, and prolonged suppuration, with or without the formation of a parotid fistula, may occur; (3) the pus may burst into the joint of the jaw and destroy it; (4) it may strip up the periosteum and lead to necrosis of the jaw; (5) it may broach the jugular vein, as recorded by Smith and Bloxam, the facial artery as mentioned by Bichet, or any other large vessel, giving rise to secondary hæmorrhage; (6) thrombosis of the facial vein, or less frequently the jugular, may lead to embolic pyæmia; (7) the facial nerve may become involved with consequent facial paralysis, which may be temporary or permanent; (8) the abscess may burrow into the middle ear or extend down the neck to the mediastinum; (9) dyspnœa and dysphagia may be present if the abscess should fail to burst, owing to pressure on the larynx or pharynx.

Course and Termination.

The mild cases all end in complete recovery, provided that the primary disease is not fatal, the most frequent sequel being facial paralysis.

If pus form, the result depends on the treatment adopted, for if the abscess be opened early and thoroughly, closure without the formation of a fistula almost invariably occurs in a few days.

The frequency of death in neglected cases is difficult to estimate, as some of the fatal symptoms may be due to the primary disease. There is no doubt, however, that a neglected parotitis is a serious and not infrequently a fatal complication, either of itself, or by increasing the gravity of the symptoms already present, and so preventing recovery.

It is probable that in actual practice the milder, non-suppurating cases are far more frequent than published

statistics tend to show, for there is a greater tendency, for various reasons, for the suppurative cases to be recorded. Hence the statement that half the recorded cases are of the suppurative type must only be accepted provisionally.

II. The Pathology of Secondary Parotitis.

Secondary parotitis was first of all regarded as a variety of primary parotitis or mumps. It was very soon noticed, however, that the former was a secondary and non-contagious affection, prone to lead to suppuration; while the latter was a primary disease of a contagious nature, which never suppurated. It was consequently recognised that secondary parotitis must have a separate pathology of its own; and various different theories were put forward to explain its origin. These will now be considered in order.

(1) *The Pyæmic or Embolic Theory.*

When secondary parotitis was first recognised it was thought that it always occurred during the course of septic affections, and it was consequently regarded as a pyæmic phenomenon due to embolism of the parotid vessels, with septic clots derived from the primary focus of disease.

It was soon shown, however, there were two objections to this theory: (*a*) Secondary parotitis was found to occur in cases in which no septic element was present, and in which septic thrombosis and embolism could not be supposed to occur. (*b*) Even in those cases in which the primary disease was of a septic nature, post-mortem examination failed to demonstrate septic thrombi in the area of primary disease; and evidence of septic embolism in other parts was also absent.

In the rare instances of true embolic pyæmia, on the other hand, in which the parotid becomes the seat of an

embolic abscess, many other organs are similarly affected, and the parotid is one of the last to be attacked.

More recently the question of embolic origin has been definitely proved to be incorrect, for it has been shown by microscopic examination that the conditions present are different in secondary parotitis, and in parotitis of pyæmic origin.

In the former, the process of inflammation begins around the ducts, in the centres of the lobules; and moreover many lobules are simultaneously affected.

In the latter the inflammation begins around the arteries which run in the perilobular tissue; and the inflamed mass is a single one, involving the area of gland-tissue supplied by the particular vessel which has become blocked with septic clot.

(2) *Heat Degeneration Theory.*

Liebermeister regarded secondary parotitis as a parenchymatous degeneration due to hyperpyrexia; but the want of relationship between the occurrence of parotitis and the presence of a really high temperature, or indeed any fever at all in some cases, soon led to this theory being abandoned.

(3) *Toxin Excretion Theory.*

Bouchard next advanced the view that the parotid gland-tissue became affected during an ineffectual attempt on the part of the gland to excrete the toxins manufactured by the organisms giving rise to the primary disease.

He quoted a "critical flow" of saliva as an example of the successful excretion of toxins; but his theory gained very little acceptance, for it was found that secondary parotitis was prone to occur during the course of diseases in which toxæmia was absent.

(4) *The Sympathetic Theory.*

The next theory brought forward is apparently the most widely accepted one in this country at the present day.

It was suggested by Stephen Paget after a study of the clinical features of a large number of cases.

The theory is based on the fact that many of the cases of parotitis, and especially of the older ones, occurred during the course of diseases, or after operations on the ovaries, prostate, testicles, and generative organs of the male and female generally. In view of this special incidence Paget suggested that the inflammation of the parotid was due to the existence of a "sympathetic relationship" between the parotid on the one hand and the generative organs, and possibly the peritoneum, on the other.

In support of this suggestion he drew attention to the existence of a similar kind of "sympathy" between the same organs, when the testicles and ovaries become affected during the course of "mumps."

Against this theory it may be advanced: (1) That all evidence of such "sympathetic relationships" and "sympathetic inflammations" in other parts of the body have been gradually proved to be infective; as, for instance, the occurrence of orchitis during gonorrhœa or after septic catheterisation. (2) That in many of the cases of parotitis more recently recorded, other parts and organs, and not the generative organs, have been primarily at fault. (3) That the great frequency of secondary parotitis after ovariotomy can be more adequately accounted for by the theory of duct infection, as will be shown later.

(5) *The Duct Infection Theory.*

The suggestion that secondary parotitis might be due to the direct infection of Stenson's duct with micro-organisms from the mouth was first made by Hanau and Pilliet in 1889 (Ref. 1 and 2).

They found on microscopical examination of sections of

the parotid (1) that the ducts were choked with *débris* containing micro-organisms; (2) that the inflammatory processes present, invariably began around the ducts in the centre of each lobule, and only spread later to the periphery of the lobule and the perilobular connective-tissue in which the blood-vessels are situated.

They concluded, therefore, that secondary parotitis could not be of embolic origin, or else the inflammation would have originated around the vessels, and that the fact that inflammation began simultaneously in the centres of many lobules at once, pointed to an ascending infection of the ducts as the real source of the affection—a conclusion which was further borne out by the actual presence of micro-organisms in the ducts themselves in such cases.

Additional evidence of different kinds has been subsequently brought forward in support of this view.

(1) *Microscopic sections* serve to show that in all cases of secondary parotitis the disease pursues the following course:—(*a*) The ducts become blocked with *débris* containing micro-organisms. (*b*) Inflammation first begins in the centre of each lobule around the ducts, and at a point farthest away from the vessels. (*c*) Many lobules are simultaneously affected; they each become centrally necrosed, and then finally by extension they fuse to form a multilocular abscess cavity.

(2) *Bacteriological examination* first carried out by Girode, (Ref. 3) has served to prove (*a*) that several different kinds of organisms may give rise to secondary parotitis, the commonest being the staphylococcus, the next frequent the pneumococcus; and after that the pneumobacillus, the typhoid bacillus, the colon bacillus, and the streptococcus; in order of frequency. (*b*) That on taking cultivations from the gland itself, the pus contained in it, the orifice of Stenson's duct, and the oral cavity, respectively; the organisms found at each spot are invariably identical in each individual case—a fact which supports the conclusion that the infection of the gland spreads from the mouth *via* the duct. (*c*) That the organism giving rise

to secondary parotitis is by no means invariably the same as that giving rise to the primary disease during the course of which the attack of parotitis has arisen. Thus in the cases complicating pneumonia, the organism causing parotitis may be the pneumococcus, or the staphylococcus; in typhoid, it may occasionally be the typhoid bacillus, but it is much more commonly the staphylococcus or pneumococcus.

In *embolic parotitis* due to pyæmia on the other hand, the organism in the parotid is invariably identical with that giving rise to the primary disease, and the abscesses in other parts of the body.

Microscopical or bacteriological evidence of direct, or ascending infection, has been brought forward by the following observers:—Girode (3), twelve cases; Prantois (4), two cases; Diaz (5), Swann (6), Bosquier (7), Morley (8), Subkovski (9).

Toupet (10), Testa (11), Subkovski (9), and Fischel (12) found the pneumococcus in the gland in cases of lobar pneumonia; and Janowski (13) and Lehmann (14) found the typhoid bacillus in cases of enteric fever.

Girode on the other hand, found the staphylococcus to be the cause of the parotid infection, in a case of pneumococcus pneumonia; and the pneumobacillus to be present in the gland in a case of puerperal septicæmia, of streptococcus origin.

Various observers moreover have shown that parotitis complicating enteric fever is far more frequently due to the staphylococcus than the typhoid bacillus.

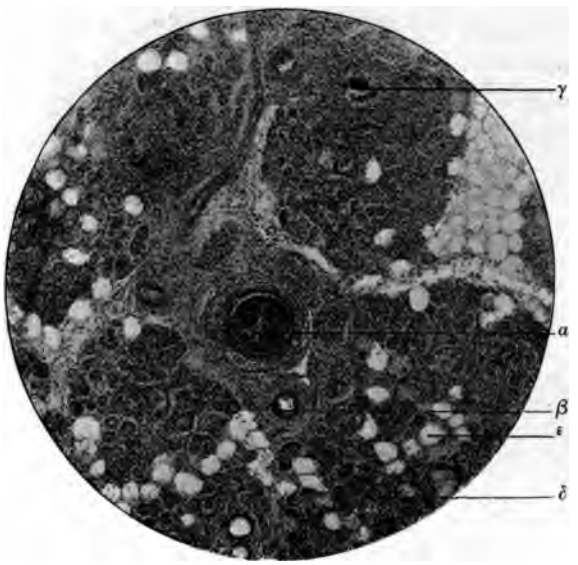
As additional evidence in support of the theory of duct infection, a brief account of the following six cases of secondary parotitis may be given.

The cases form a successive series met with in the

course of hospital practice, and in each instance a bacteriological or microscopical examination afforded ample evidence that the affection of the parotid was due to an ascending infection of Stenson's duct from the mouth.

CASE 1.—A man aged 25 who was operated on for an ulcerative perforation of the stomach seven hours

FIG. 1.



CASE 1.—Secondary Parotitis (early stage).
(α) Large duct (interlobular) distended with infected secretion.
(β) Interlobular artery, unaffected.
(γ) Small intra-lobular duct distended with infected secretion, showing inflammatory infiltration of adjacent central portions of lobule around it.
(δ) Acini of parotid.
(ε) Fat cells.

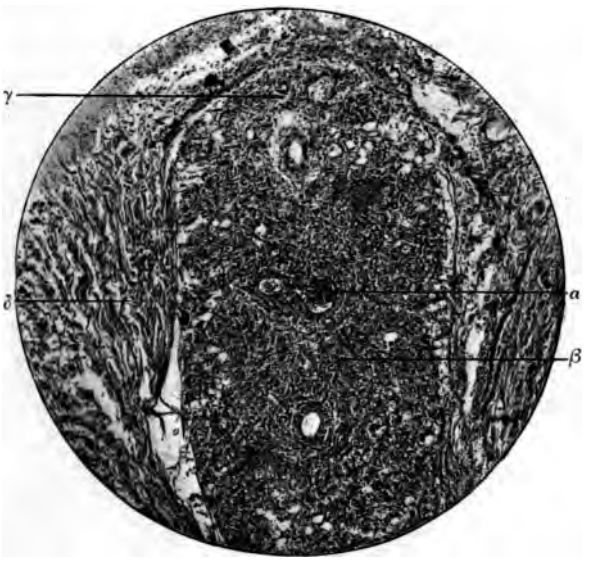
after perforation had occurred. In spite of suturing the aperture, and cleansing, and drainage of the peritoneal cavity, peritonitis persisted, and he died with a temperature of 107° seven days after operation.

No food by mouth until the third day, when it was tried, but not persisted in, as it led to vomiting. Rectal feeding only until the end.

The mouth was very foul from the beginning of the illness. On the sixth day the left parotid became painful and swollen to twice its natural size. It was even larger on the ensuing day, when death occurred.

Post-mortem.—Death from peritoneal septicæmia; no signs of pyæmia.

FIG. 2.



CASE 1.—Secondary Parotitis.

Lobule under low power showing more advanced changes.

(α) Duct distended and blocked with *débris*.
(β) Central portion of lobule inflamed and infiltrated with leucocytes and inflammatory cells, which have replaced original gland tissue.
(γ) Less marked inflammatory processes at periphery of lobule, in which lies a vessel with patent lumen.
(δ) Interlobular tissue, almost unaffected.

On the sixth day, when parotitis first began, cultivations were taken from the mouth, and from the fluid squeezed from the parotid duct. After death cultivations were taken from the cut surface of the affected gland. They all produced a growth of *Staphylococcus aureus*.

On section, to the naked eye the parotid was inflamed, but no pus was visible.

On microscopic section, the ducts of some lobules were blocked with granular *débris* containing micro-organisms.

The central portions of the lobules around the ducts were evidently inflamed, the epithelial cells being cloudy and degenerated, and the parts invaded with leucocytes. The periphery of each lobule was practically normal; and the interlobular vessels healthy. In some lobules, however, the inflammation had reached the periphery, but here it was much less marked than in the centre around the ducts.

Case 2.—A woman of 23 with history of dyspepsia, sustained a perforation of the stomach on March 26th, and was brought to hospital on March 29th, when a subdiaphragmatic abscess was opened and drained.

Before admission she had vomited continuously, and she continued to do so, all food by the mouth being stopped in consequence.

For four days after operation the temperature varied between 99° and 101°, and the pulse 90 to 108 per minute.

During the evening of the fourth day the temperature rose to 102° and the pulse to 120. A second subphrenic abscess was found posteriorly and opened trans-pleurally.

Diarrhœa now set in, and was treated with opium.

On the fifth day the right parotid became painful, and a swelling began to form just over the angle of the jaw, which was firm and tender. Cultivations were at once taken from the oral cavity and the fluid squeezed from the duct.

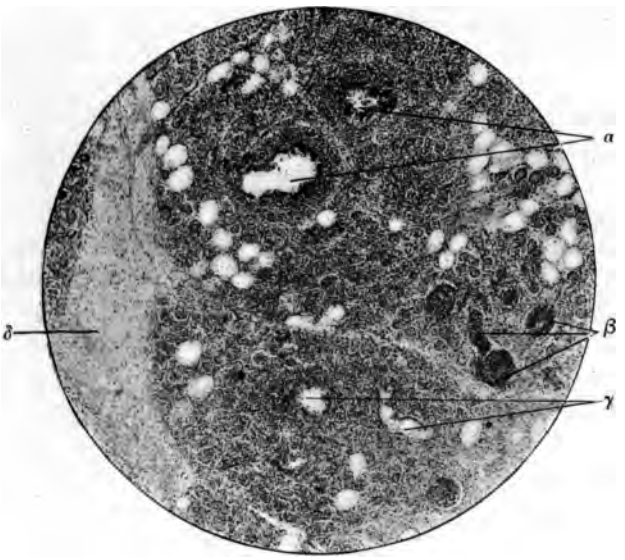
Next day the swelling was more marked, and the temperature 102·6°.

The next day, the third of its onset, the swelling was less marked and softer, efforts to cleanse the mouth having been apparently partially effectual in preventing further infection.

Two days later death occurred from septic absorption, other abscesses being found in the pelvis and around the cæcum; but no signs of pyæmia or thrombosis being detected anywhere.

During the two days before death—the fourth and fifth of its existence—the parotid swelling subsided consider-

FIG. 3.



CASE 2.—Secondary Parotitis (later stage than Fig. 2).

(α) Inflammation and necrosis around ducts in central portion of lobule.
(β) Smaller ducts, showing earlier stage of distension.
(γ) Distended ducts in adjacent lobule, with inflammatory changes beginning around them.
(δ) Interlobular tissue very slightly infiltrated. (The vessels in this tissue near by were unaffected.)

ably and became much softer, almost vanishing just before death occurred.

On section, to the naked eye the portion of the parotid affected lay around and behind the bifurcation of the duct of Stenson.

Minute cheesy masses could be squeezed out of the

centre of each lobule, the parts generally looking inflamed to the naked eye.

Microscopic sections of Stenson's duct showed it to be choked with granular *débris* containing micrococci.

The lobules affected were breaking down centrally so as to form an indefinite mass of necrosed cells and leucocytes, the periphery of each lobule being either slightly inflamed or healthy, and the vessels practically normal.

Many lobules were similarly affected, and in different parts of the gland the different stages could be traced as follows: (1) Blocking of duct and slight inflammation of central portion of lobule around it; (2) more severe central inflammation of lobule, with signs of recovery in the peripheral parts. The gland bore evidence of the fact that central suppuration may occur and then subside, the pus being either absorbed or being excreted *via* the duct. The cultivations from the mouth and duct during life, and those from the cut gland after death, grew abundant staphylococci, mixed aureus, and albus.

Case 3.—A man of 63, who had long been in the habit of using a catheter on account of an enlarged prostate. He had previously suffered from septic cystitis, and was admitted for such an attack with uræmic symptoms.

In spite of all care he remained drowsy, lying with his mouth open, and his tongue became very foul. Temperature about 100°, urine foul, kidneys large and tender.

Before admission he had had none but liquid food for a fortnight, and he remained on milk diet in the hospital.

On November 16th, ten days after admission, he complained of pain over the right parotid region, and a swelling formed posteriorly which soon involved the whole parotid and led to a brawny condition of the cheek and œdema of the lips and eyelids.

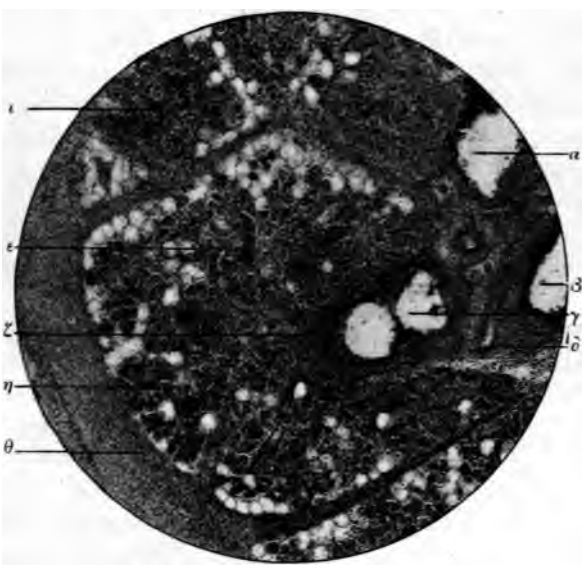
On November 17th the temperature reached 101°.

On November 19th the whole side of the face was swollen in a typical manner, the jaw fixed, and the swelling very tense.

On November 20th the skin was red and hot. Temperature 102°, but no fluctuation. An incision was made and some pus evacuated.

From November 20th to the 27th the parotid slowly became smaller and the discharge more serous.

FIG. 4.



CASE 3.—Secondary Parotitis (more advanced stage than Fig. 3) under low power.

(α) Necrosed area extending into field from adjacent lobule more severely affected.
(β) Large interlobular duct, with distended lumen and shed epithelial lining.
(γ) Ducts of lobule shown, with necrosis of surrounding portions of lobule.
(δ) Artery with lumen patent.
(ε) Small duct distended and blocked.
(ζ) Necrosing tissue in main duct region.
(η) Periphery of lobule, very slightly inflamed.
(θ) Interlobular septum, only slightly inflamed.
(ι) Inflamed gland tissue of adjacent lobule.

On December 3rd a fresh abscess burst into the original wound, and more pus began to flow. The gland was nearly normal in size, and the mouth was easily opened.

On December 6th, after an access of uræmic symptoms, the patient died, the parotid being nearly normal in size and the sinus healing.

Post-mortem.—The usual picture of back pressure and septic pyelo-nephritis was presented, but there were no signs of thrombosis or pyæmia.

Cultivations during life from the mouth, the orifice of Stenson's duct, and the pus evacuated from the parotid grew abundant *Staphylococcus aureus.*

On section the shrunken remains of a loculated abscess cavity occupied the centre of the gland.

Microscopically the lobules around presented all phases of infection and inflammation, from simple blocking of the ducts with granular material and micro-organisms, to complete necrosis of adjacent lobules; the inflammation being most marked in the central portions of the lobules. Except where necrosis was severe the interstitial tissue was practically unaffected, and the vessels were normal.

Cases 4 and 5.—Both occurred during the course of enteric fever.

Case 4.—A girl of 26, whose left parotid became affected during a relapse six weeks after the onset of the disease.

Two days later the right parotid became swollen.

The left parotid suppurated, and was opened on the sixth day, healing on the twentieth.

The right parotid subsided in the course of eight days.

Five days before the onset of parotitis patient had received an opium enema for hæmorrhage, which was repeated next day. The mouth was very foul.

Cultivations from the mouth, duct, and pus each grew *Staphylococcus aureus.*

Case 5.—A man of 32, whose right parotid became swollen on the twentieth day of an attack of enteric.

Four days later the whole face on that side was brawny

and œdematous, and next day fluctuation could be detected.

An incision reached pus rather superficially seated, and after draining for nine days the wound healed.

The patient invariably lay with the mouth open, adenoids being present, and the oral cavity was very dry and foul.

Cultivations from the mouth, duct-orifice, and pus each showed growths of *Staphylococcus aureus.*

In neither case could the colon bacillus or typhoid bacillus be found, though special cultivations and tests were made to detect them.

Case 6.—The sixth case is an example of secondary parotitis occurring during diabetic coma.

The patient was brought to hospital in a comatose state; the condition of the mouth was very foul, and the affection of the parotid was not observed before admission.

The right parotid was swollen, and the skin over it tense and brawny, and a sense of deep fluctuation could be detected.

Cultivations were taken from the mouth and duct contents, and others from the pus which drained away from the gland after an incision had been made into it. They all showed a growth of *Staphylococcus albus.*

Death occurring three days later, the parotid was excised through the mouth, no other examination of the body being permitted.

Under the microscope the gland showed very distinctly the various phases of duct infection. In the least affected lobules the ducts were simply distended with *débris* containing cocci.

In those more advanced the central parts of each lobule around the duct were inflamed and invaded with micro-organisms. In others the whole lobule, except its most peripheral layer of cells, had broken down and undergone necrosis.

Conclusions.

On studying the evidence of duct infection afforded by these six cases and the others previously mentioned, there can be no doubt that they all without exception afford proof of having arisen in consequence of an ascending infection of the parotid duct; and as the same evidence has been forthcoming in all the cases of secondary parotitis that have been submitted to a definite pathological examination, it is more than probable that all cases of secondary parotitis arise in this way.

This suggestion is still further supported by a study of some of the clinical features of secondary parotitis, as will be shown later. In the meantime, however, the experimental evidence of Claisse and Duplay (15), which goes to prove that infection of the parotid duct can only occur in the presence of certain abnormal predisposing conditions, will be referred to.

These observers showed by means of experiments on animals :

(1) That the healthy parotid duct cannot be infected, either by smearing its orifice with micro-organisms, or by introducing organisms into its lumen, *via* the normal orifice of the duct, or an artificial fistula.

(2) That an ascending infection of the duct, leading to the production of parotitis, showing all the appearances which have been observed in secondary parotitis in the human subject, can easily be produced by either of these methods under the following circumstances :—(1) If the micro-organisms introduced were excessively numerous or virulent. (2) If the general vitality of the animal had been previously depressed by starvation or other methods. (3) If the normal secretion of the parotid had been altered in quantity or quality, in one or other of the following ways: (*a*) By ligaturing the duct and preventing the flow of saliva, after infection had been attempted. (*b*) By administering drugs such as opium which check and alter the salivary secretion.

It is evident, therefore, that for ascending infection to occur in the case of the parotid duct, as has also been more or less proved in the cases of the kidney and bile passages, one or other of the following abnormal conditions must previously be present :—(1) Micro-organisms must be present at the orifice of the parotid duct, in larger numbers, or of more virulent type, than usual. (2) The general vitality of the subject must be reduced so as to render him more liable to succumb to microbic infection. (3) The quantity of secrètion passing down the duct and protectively draining it, must be diminished. (4) The quality, and more particularly the normal bactericidal properties of the saliva secreted must be lowered.

Turning now to the question of secondary parotitis in man, and referring to the list of disorders which have been complicated by this affection, it is evident that they all present one common feature, viz. that they are all particularly likely to be accompanied by those conditions which Claisse and Duplay have shown to be necessarily present, before duct infection can be produced in animals.

Moreover, the comparative frequency with which parotitis occurs in connection with each individual disorder, varies directly with the degree and frequency with which these predisposing conditions are met with in each instance :

Thus on referring to the diseases which are most prone to be complicated by parotitis, the following facts are evident.

I. *That in typhus and enteric fever,* the diseases most frequently complicated in this way, all four of Claisse and Duplay's predisposing causes are present in a severe degree. Thus—

(1) More micro-organisms than usual are present in the mouth, and they are more virulent than normal for several reasons. (*a*) The normal drainage of the mouth by swallowing is interfered with, owing to the diminution in the amount of saliva secreted when solid food is withheld

on the one hand, and the increased quantity evaporated in fever cases in which the mouth is held open on the other. Mechanical cleansing of the mouth by chewing is also absent, and in the presence of an excess of dead material and a lack of drainage the oral micro-organisms become more numerous and virulent. (*b*) In enteric fever the special organisms of the disease may also be present in the mouth, and so infect the duct.

(2) Lowering of the vitality of the patient, and increased liability to microbic infection generally are both well-marked in these diseases.

(3) The quantity of secretion passing down and protectively draining the parotid duct in these cases is diminished, as the result of fever and absence of solid food, and consequently organisms can more easily ascend the duct than usual. The quantity of secretion passing down the duct may also be further diminished in some cases owing to the administration of opium.

(4) The quality and bactericidal value of the secretion of the parotid cannot be definitely proved to be diminished in these cases. It is well known, however, that the chemical constitution of the salivary secretion is profoundly altered in fevers, and probably therefore its bactericidal power is also affected.

Exactly similar predisposing causes of duct infection are also specially liable to be present in all the other diseases with which secondary parotitis is prone to occur.

Thus in *scarlet fever* special organisms are present in the oral cavity, which is particularly likely to become septic, as is shown by the state of the throat and the glandular complications which so frequently occur.

In *pneumonia* there is a special organism present in the mouth, and the rapid mouth breathing and the fever dry up the cavity, and prevent proper drainage.

In all forms of *peritonitis*, fever, mouth breathing,

vomiting, constipation and the administration of opium, lead to dryness and sepsis of the mouth, and moreover all solid food by the mouth is strictly withheld.

In *pyelo-nephritis* the foul and dry condition of the mouth and liability to microbic infection are proverbial, as is also the case in diabetes.

In *mercurial stomatitis, iodism and plumbism*, the foul condition of the mouth is a special feature, and as these drugs are secreted by the saliva they probably alter its qualities, and so lead to duct infection.

In *old age, dementia, and grave asthenia* mouth breathing is common, and the condition of the mouth is frequently neglected.

In all cases of *abnormal menstruation* the affection of the parotid may be regarded as an exaggeration of those conditions of altered salivary secretion, which have been noted in cases of ordinary menstruation. In some such instances salivation is increased, in others diminished.

On turning to the operations liable to be followed by secondary parotitis, the presence of causes predisposing to duct infection can be similarly detected.

Thus in abdominal operations generally, the dorsal decubitus and tight binder lead to mouth breathing, and the mouth is frequently inflamed or damaged, owing to the prolonged administration of an anæsthetic, and the use of gags, etc.

Food by the mouth is withheld, or liquids only are given; vomiting is not infrequent owing to the long administration of chloroform, and constipation and the employment of opium also help to render the mouth more septic and to diminish the quality and quantity of the saliva secreted.

The greater frequency of parotitis after septic cases is due to two factors:—(1) The patients have usually been ill for some time before the operation, and the mouth is consequently foul to start with, in consequence of fever,

constipation, vomiting, mouth breathing, the administration of opium, and lack of food. (2) The presence of fever after operation and the greater amount of general depression, coupled with more strict absence of food, more frequent vomiting, and the more frequent presence of constipation in these cases, leads to a drier and more septic condition of the mouth than occurs in aseptic cases.

The comparative frequency of secondary parotitis after ovariotomy as compared with other aseptic abdominal operations can be similarly explained.

Thus in early days ovariotomy was practically the only aseptic abdominal operation frequently performed, and it was carried out with a routine which specially predisposed to a septic and dry condition of the mouth.

Thus in the first place the importance of cleansing the mouth and sterilising anæsthetic apparatus before operations had not been recognised. In the next place great care was taken to adopt a strict dorsal decubitus, with the head low, and to apply a very tight abdominal binder, both of which lead to mouth breathing.

Opium was given as a routine treatment, and the bowels were kept locked whilst all food by the mouth was withheld for several days.

The influence of these measures in provoking the onset of secondary parotitis by causing foulness of the mouth is evident, for since they have been abandoned in more recent times instances of parotitis after ovariotomy have become extremely rare, and indeed no more frequent than after other aseptic abdominal operations.

Summary.

From a study of these facts it seems highly probable that secondary parotitis is invariably due to an infection of Stenson's duct, dependent on a septic condition of the mouth, and that its onset may be prevented by attention to the following details:

(1) The patient's mouth should be carefully cleaned and

rendered aseptic before operations, and at the commencement of long febrile illnesses. If necessary causes of nasal obstruction leading to mouth breathing, such as adenoids, should be removed.

(2) The anæsthetic apparatus should be sterile.

(3) The mouth should be periodically cleansed afterwards, especially after every attack of vomiting.

(4) The bowels should be opened early, and food by the mouth, and especially solid food, given as soon as possible.

(5) Opium should not be given unless absolutely necessary.

(6) The head should not be placed too low nor the binder fixed too tightly, and the dorsal decubitus should be given up as soon as possible.

Treatment.

Should parotitis threaten to appear every effort should be made to cleanse the mouth and prevent continued reinfection of the duct, as it is probable that resolution of the inflammation is then far more likely to occur. The administration of a sialogogue and an aperient should also be entertained.

If the disease progresses it is most important not to wait for fluctuation before incising the region of the swelling, for in the most dangerous cases in which the pus is most likely to burrow and lead to serious trouble it lies so deeply beneath the tense parotid fascia that fluctuation may not appear until it is too late.

If symptoms progress, therefore, for four days, and the swelling increases and becomes œdematous, an incision should be made, as also in cases in which the temperature becomes high and intermittent, whether fluctuation can be detected or not.

A transverse incision should be made over the point of greatest prominence, having due regard to the situation of the branches of the facial nerve and Stenson's duct; the

parotid fascia should be freely incised, and not opened by Hilton's method, as usually recommended.

The finger should be introduced into the abscess cavity as in an abscess of the breast, in order to break down the interlobular septa and convert the various loculi of the abscess into one large cavity, which should then be drained.

Otherwise undrained loculi will continue to grow and burrow, and a further operation may be called for, or burrowing may occur and lead to a fistula or other troubles.

ADDENDUM.

To render this paper more complete, attention may be directed to the following facts :

(1) The submaxillary and sublingual glands may be affected in an exactly similar manner to the parotid during the course of exactly similar maladies. At times they have been affected with the parotid and at times apart from it.

Microscopic sections have been made, and they show that the changes present are exactly similar to those found in the parotid under similar circumstances, thus indicating a similar mode of origin by duct infection.

(2) Sections have been examined under the microscope from cases of parotitis and submaxillary gland suppuration due to the retention of a calculus in the main duct. They have shown exactly similar changes to those seen in suppurative cases of secondary parotitis, and since they are obviously due to duct infection, they bear out the theory of origin now advocated for secondary parotitis.

(3) In two cases of chronic parotitis due to thickening of the parotid secretion and blocking of the duct I have found the thickened secretion squeezed from the duct to contain staphylococci, and it is significant that in each instance a septic focus existed in the mouth. Such cases may be regarded, therefore, as examples of *chronic ascending parotitis*.

(4) In a case of fibrous hypertrophy of the submaxillary gland, due to the involvement of Wharton's duct in the scar left after removal of the tongue for cancer, I was led to remove the gland in consequence of its stony hardness, fearing it might enclose infected lymphatic nodules.

None were present, however, and the gland on section showed dilatation and destruction of the ducts and chronic inflammatory changes, spreading from the centre of each lobule to its periphery and leading to the formation of an excess of fibrous tissue and to atrophy of the proper glandular elements.

That the inflammatory changes present were in part due to duct infection was shown by the fact that cultivations from the ducts afforded a growth of staphylococci.

(5) Evidence has been brought forward by various observers to show that epidemic parotitis is due to the infection of Stenson's duct with a specific micro-organism.

When considered together, therefore, these facts tend to show that a process of "duct infection" is probably responsible for the origin of all the varieties of simple parotitis, including epidemic parotitis, secondary parotitis, and parotitis due to duct obstruction of any kind by calculi, foreign bodies, scars, or thickened secretion. Parotitis due to tubercle may also arise in a similar manner, and the only exceptions are those varieties due to syphilis, to pyæmia, and to the direct spread of inflammation from adjacent parts, such as the cheek or jaw.

There is also evidence to show that the process of duct infection responsible for ascending parotitis is due to one or other of the following causes :

1. The presence of specific micro-organisms in the buccal cavity, such as the special organisms of mumps, typhoid fever, and probably typhus and pneumonia also.

2. An increase in the number or virulence of the normal buccal organisms and especially of the staphylococci.

3. Anything interfering with the quantity or quality

of the saliva protectively draining down the duct. Either an actual obstruction, such as a calculus, or an alteration in the saliva secreted, resulting from general ill-health or the administration of drugs, may produce this effect.

The changes which result from "duct infection" of the parotid vary in degree, and the following varieties of parotitis may be distinguished:

1. *Hyperacute parotitis.*—A rare variety, in which sudden necrosis of a large part of the gland occurs.

2. *Acute suppurative parotitis,* in which the broken-down lobules coalesce to form a lobulated abscess which may either (*a*) rapidly extend and point; (*b*) extend slowly, and remain for a time latent; (*c*) Drain *viâ* Stenson's duct, and so end in recovery.

3. *Acute parotitis,* in which suppuration fails to occur, as is usual in epidemic parotitis and about half the cases of the secondary variety.

4. *Chronic parotitis,* usually due to some definite obstruction in the duct, and leading to dilatation of the ducts, atrophy of the glandular elements, and periacinar sclerosis, and hypertrophy.

Both the *submaxillary* and the *sublingual* salivary glands may become the seat of duct infection leading to a series of changes identical with those just described for the parotid. In the case of the *kidney, biliary ducts, male genital tract,* and the *breast* similar circumstances are known to lead to duct infection, and varying degrees of inflammatory change are met with in consequence.

It is interesting also to compare pancreatitis with parotitis and to see what evidence exists to show that the different varieties of pancreatitis are due to "duct infection."

In the first place, it is known that the pancreas may become affected during "mumps," like the parotid.

Secondly, Mayo-Robson's latest classification of pancreatitis in which hæmorrhage is regarded as an epiphenomenon, is practically identical with the classification of parotitis just given. Thus he recognises—

1. *Hyperacute pancreatitis* with rapid and extensive gangrene of the glandular tissues.

2. *Acute suppurative pancreatitis,* which begins in the centres of the lobules like parotitis, and forms a multilocular abscess which may similarly pursue one of three courses: (1) Rapidly grow and point. (2) Grow slowly, remain latent, and form a "cyst." (3) ? Discharge *per* the duct.

3. *Acute pancreatitis,* resulting in resolution, if death does not occur.

4. *Chronic pancreatitis,* in which there is almost invariably some definite duct obstruction, as in chronic parotitis, and in which exactly similar changes are present in the gland, consisting of duct dilatation, parenchymatous atrophy, and interacinar sclerosis.

Thirdly, in all cases of pancreatitis there is evidence of the presence of the same causes that have just been shown to lead to duct infection in cases of secondary parotitis, viz. abnormally numerous or virulent organisms in the vicinity of the duct orifice, or some fault of secretion tending to render infection of the duct more likely to occur.

Thus, ulcers in the duodenum and stomach, or catarrhal states due to alcoholism, have been present in many cases, and infected biliary passages in others.

The flow of pancreatic juice has been definitely impeded in some cases owing to the presence of calculi in the pancreatic or common bile-duct, or to scars, strictures, adhesions, and adjacent tumours pressing on the duct. In other instances it is possible that a thickening of the secretion or some alteration in its quantity or quality may have led to infection occurring.

Microscopic examinations show that the inflammation in pancreatitis probably begins, and is most marked, around the ducts, in the centres of the lobules, as in cases of parotitis. Further evidence in favour of this view, and therefore in favour of duct infection as a mode of origin, is afforded by the fact that after severe pancreatitis the

islands of Langerhans, which lie in the interstitial tissue, remote from the ducts, are still intact, and diabetes is absent. In inflammation by direct extension from adjacent parts, on the other hand, if a large part of the pancreas is destroyed *en masse*, the islands disappear also, and diabetes ensues.

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For Discussion *see end of following paper* (*Dr. Tebbs*).

SYMPTOMATIC PAROTITIS

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SYMPTOMATIC, secondary, or metastatic parotitis is a subject round which much interest and discussion have centred. A complication of other diseases, rather than a disease *sui generis*, it is best known, perhaps, as a sequela of abdominal operations and disorders, and of certain of the specific fevers, but there are many other conditions of which it is a recognised, if less common, complication. The only systematic study of the subject in this country is contained in the admirable papers by Mr. Stephen Paget (1) published in the years 1886 and 1887, which constitute the chief source of our knowledge of this disorder. Series of cases have also been published in America by Goodell and Morley (2), and in France by Benoit.

The present paper is founded on a collection of 77 hitherto unpublished cases of this disorder, nearly all of which are taken from the records of St. George's Hospital. I must express my indebtedness to the members of the staff of that hospital for their kind permission to use these cases, and to the other gentlemen who have kindly communicated cases to me. I have referred to other published cases but have not included them in the series. An analysis of the 77 cases will be found at the end of this paper.

Previous observers have, I think, been rather inclined to

look on the disorder as more or less of a specific disease, and to endeavour to adopt one explanation that will cover all the cases. Believing symptomatic parotitis to be a disease of rather heterogeneous pathogeny, I have attempted to classify the cases provisionally as follows:

A. Acute parotitis following or in connection with (1) abdominal operations and diseases of the alimentary canal and its appendages; (2) operation on and disturbances of the generative organs; (3) operation on or injury of portions of the body other than the abdominal and pelvic viscera; (4) certain diseases of metabolism and chronic intoxications; (5) the specific fevers; (6) inflammatory conditions of neighbouring parts.

B. Recurrent parotitis and chronic enlargement of the gland.

The acute cases seem to fall naturally into two main groups, one of which is undoubtedly of septic and the other probably of toxic origin. Group 4, and probably some cases in group 3, represent the toxic parotitis, and the other groups the septic variety. Some of the recurrent cases appear to depend largely, if not wholly, on vaso-motor disturbances, and would constitute a separate group.

Believing that in the majority of the septic cases the infection of the gland arises not by a spread along the duct from the mouth but by the blood stream, I have paid special attention in the cases to the evidence of a primary septic focus, and to the co-existence of other septic complications in the patients that develop parotitis.

I propose to treat these groups one by one, and finally to take up the consideration of certain general points in connection with the disorder.

In the case of post-operative parotitis I have endeavoured to arrive at some idea of its relative frequency after various operations, by taking the total number of such operations performed during a period of fourteen years, and working out the percentage of cases followed by this complication. Cases fatal within forty-eight hours of operation may be excluded in reckoning percentages.

Acute Parotitis.

1. Parotitis following Operation on and Diseases of the Abdominal Viscera.

Gastric Operations and Diseases.

Of the various primary disorders that may be followed by this complication lesions of the stomach would appear to furnish the greatest proportion of cases. I find that the complication reaches its highest percentage in connection with operations for gastric ulcer. During the fourteen years 1890 to 1903, 49 operations were performed in St. George's Hospital for gastric ulcer, 42 of which were for perforated ulcer and 7 for non-perforated, 5 of the latter involving opening of the stomach. Among the 38 patients who survived the operation for forty-eight hours there were 7 cases of parotitis, which gives a percentage of rather over 18 among surviving cases.

The cases of perforated gastric ulcer formed the subject of a paper read before this Society last year by Mr. T. C. English (3). Parotitis formed only one of the numerous septic complications that arose in the surviving cases. Among 42 patients with perforated ulcer, of whom 32 survived operation for forty-eight hours, 15 developed pleurisy, 2 empyema, 3 pneumonia, 1 pulmonary abscess, 3 thrombosis, and 1 acute nephritis. Nearly all the patients that developed parotitis had some other septic complication. In case No. 1 of my series suppurative parotitis was followed by pulmonary abscess, empyema, and pericarditis. Cases Nos. 2 and 4 developed pleurisy, and No. 3 pneumonia, in addition to parotitis. These complications are to be ascribed to the septic soiling of the peritoneum that occurs with the perforation of the ulcer. In cases of operation for non-perforated ulcer, other septic complications were, as might have been

anticipated, less frequent. A number of instances of parotitis after operation for gastric ulcer will be found in the literature. Dr. Phillips and Mr. Silcock (4) and Dr. Aitken (5) have recorded cases. In the former case the patient also developed a sub-diaphragmatic abscess and in the latter septic pneumonia. Dr. Blumer (6) records a case following excision of a gastric ulcer; and Mr. Mansell Moullin, (7) in his report on thirteen cases of operation for recent gastric ulcer, mentions parotitis as occurring in two of the patients. One case in Morley's series followed operation for gastric ulcer.

The relative frequency of parotitis after other gastric operations performed at St. George's Hospital is shown by the following table, which summarises the operations performed during a period of fourteen years:

Operation.	No. of cases.	No. of cases surviving 48 hours.	No. of cases of parotitis
Gastro-enterostomy	53	43	3
Gastrostomy	35	31	1
Division of adhesions	7	7	1
Gastrotomy	5	5	0
Gastroplication	2	2	0
Pyloroplasty	5	5	0
Loreta's operation	2	2	0
	109	95	5

That is to say, parotitis occurred in rather over 5 per cent. of the patients that survived operation forty-eight hours. If we add to these the gastric ulcer cases, making in all 158 cases of operation on the stomach, with 133 patients surviving forty-eight hours, and 12 cases of parotitis, we find a percentage of 9 for all gastric operations. It will be noticed that the incidence of parotitis is considerably less in the cases contained in the above table (5 per cent.) than in cases of perforated gastric ulcer (18 per cent.), which is suggestive in view of the lesser degree of septic soiling of the peritoneum in the former. Other septic complications are also less common than in cases of perforation. Case No. 12 in my series developed

thrombosis in addition to parotitis after the operation of gastro-enterostomy. Cases of parotitis following operations on the stomach will be found in Mr. Paget's and in Morley's series, and a case following gastrotomy for the removal of a hair-ball is recorded by the late Mr. Knowsley Thornton (8).

These figures would show that parotitis occurs as a well-marked complication of gastric operations, and that its frequency increases in proportion to the amount of peritoneal sepsis.

In Cases of Gastric Ulcer not treated by Operation.

Although much less frequent than in the operative cases, parotitis forms a well-marked complication of cases of gastric ulcer treated by medical measures. It occurs with such regularity that it seems remarkable that it has been so little recognised as a complication of this disease. Taking 652 consecutive cases, extending over a period of thirteen years, I found that it occurred thirteen times, or in exactly 2 per cent. of the patients. There was never any suspicion of an epidemic among the patients. The fact that the average period of starvation or rectal alimentation in these cases was so much longer than in the operative cases is certainly suggestive of the inadequacy of the usually accepted explanation that parotitis is due to the cessation of mouth feeding, especially when we consider the relative frequency of the complication in the cases treated surgically and those treated medically. Moreover, the infection was of a much milder type in these medical cases, for in two only out of fifteen was there any suppuration, and in one of these it involved only the lymphatic glands over the mastoid process, and not the main parotid. In one case (No. 44) thrombosis occurred in addition to parotitis.

A peculiar case is recorded by Dr. Sydney Phillips (9). A girl, aged 19, came under observation with symptoms strongly suggestive of perforated gastric ulcer, but appar-

ently due to thrombosis of the inferior vena cava, which proved fatal. The patient developed parotitis, and a few days later the thrombosis, spreading downwards, appeared in the veins of the leg. Extensive thrombosis, extending right through the iliacs and inferior cava, was found at the autopsy. The patient was on rectal feeding for one day only. A possible explanation would seem to be that the parotid was predisposed to infection—the patient had had dyspeptic symptoms for some time—and that the organisms which were responsible for the thrombosis, reaching the gland by the blood stream, set up the inflammatory process.

Lesions of the Intestinal Tract.

Parotitis may occur as an after-complication of operations on the intestinal tract, but less commonly than after gastric operations. Its frequency seems to diminish as the site of operation passes down the gut. It is almost unknown after operations on the rectum; but cases following division of a rectal stricture and the excision of hæmorrhoids have been recorded by Paget and Auld. A possible source of fallacy is to refer this complication to the operation rather than to the underlying condition which the operation is intended to relieve. For instance, two of the cases in my series followed the operation of colotomy in patients with acute intestinal obstruction; but inasmuch as I find that parotitis is a common complication of acute intestinal obstruction, while we have had no instances of its following colotomy performed for other conditions, I prefer to assign the parotitis to the intestinal obstruction rather than to the operation.

One of my cases (No. 49) occurred in a patient with duodenal ulcer. The symptoms closely resembled those of enteric fever, and the patient was treated accordingly, and fed by the mouth. A large chronic non-perforated ulcer of the duodenum was discovered at autopsy.

Mr. Paget has recorded a case of parotitis in connection with perforated duodenal ulcer going on to general peritonitis and ending fatally. One would expect this complication to be fairly frequent in cases of perforated duodenal ulcer. I have not met with any instances of it, but our records only contain eight cases in fourteen years, and of these three only survived operation for forty-eight hours.

I have not been able to find any instances of this complication after operation on the small or large intestine in our records, except, as I have mentioned, after colotomy performed for acute obstruction. These operations are summarised in the following table:

Operation.	No. of cases.	No. of cases surviving 48 hours.
Intestinal resection, anastomosis, etc., mostly for injury	30	16
Intussusception	22	10
Colotomy for other conditions than acute obstruction	140	133
	192	159

Intestinal Obstruction.

Three cases of parotitis in connection with intestinal obstruction will be found in Mr. Paget's series published in the 'Lancet' in 1886, in two of which enterostomy was performed. Morley also records cases after enterostomy and operation for obstruction. I have found parotitis quite a common complication of acute intestinal obstruction, as the following table shows:

Disease.	No. of cases.	Surviving 48 hours.	Cases of parotitis.
Acute obstruction due to bands, volvulus, impacted foreign bodies, etc.	50	31	3
Acute supervening on chronic obstruction due to malignant disease, tuberculous peritonitis, etc.	30	21	2
	80	52	5

This gives a percentage of nearly ten among surviving cases.

In one of the ovariotomy cases in my series (No. 28) the removal of the cyst was followed by acute intestinal obstruction, due to kinking of the gut, about the time that the parotitis developed.

These cases of intestinal obstruction (Nos. 15 to 19 in my series) mostly bear clear evidence of intra-peritoneal sepsis. In No. 15, a case of volvulus, more than a foot of intestine was found intensely congested, and in places gangrenous; there was also much infiltration of the mesentery. In No. 16 the obstruction was due to a band, and the gut was almost ulcerated through at the site of constriction. At the autopsy the injured gut was found to have given way, and general peritonitis was present. No. 17 was also a case of obstruction by a band; the patient recovered, but with free suppuration of the wound. In No. 19, where the obstruction was due to carcinoma of the sigmoid flexure, the gut was also found at the autopsy to have given way, and general peritonitis was present. In addition to parotitis the patient also developed a left-sided pleurisy.

The frequency of parotitis as a complication of these cases of acute obstruction is quite explicable if we consider that we have not only the enormous amount of gastro-intestinal disturbance which accompanies these cases to dispose the gland to infection, but also the presence of intra-peritoneal sepsis to act as a primary focus of infection for the parotitis and other septic complications.

Strangulation of external herniæ is, of course, a variety of intestinal obstruction, and is occasionally followed by parotitis, but less frequently than the internal variety. The reason for this would appear to be that the condition is sooner recognised and treated, and also because it does not involve an abdominal section, with its necessary damage to the peritoneum and abdominal contents, which apparently acts as a strongly disposing cause to the development of this complication. I found four cases of parotitis among 346 cases of strangulated hernia, 305

patients surviving operation for forty-eight hours; that is to say, it occurred in rather over 1 per cent. of cases. These four cases (Nos. 20 to 23) also illustrate the association of parotitis with other septic complications. No. 20 developed thrombosis, No. 21 pleurisy with effusion. In No. 22 the parotitis was accompanied by pneumonia, and four days after the onset of these complications the patient suddenly collapsed and died, apparently from embolism. In No. 23 there was a very mild attack of symmetrical parotitis lasting only thirty-six hours, and no other complications.

Mr. Paget collected several cases of parotitis following herniotomy and Morley's series also includes a case. Mr. F. B. Jefferies (10) records a case where it followed operation for strangulated umbilical hernia. The parotitis was associated with bronchitis, and the tissues of the neck became so swollen and infiltrated that there was marked dyspnœa and dysphagia, which apparently largely determined the fatal result.

I have found only one case of parotitis after operation for non-strangulated hernia (Case No. 24). In this case there was a large ventral hernia, and the operation was practically an abdominal section. Operation was followed by much vomiting, and the wound suppurated freely. In addition to suppurative parotitis there was an abscess of the soft palate.

There have been no instances of parotitis among 855 cases of simple "radical cure."

Ulceration of Intestine.

Just as parotitis may complicate gastric or duodenal ulcer, so it may occur with ulceration lower down the gut. Case No. 51 was an instance of ulcerative colitis, the ulceration being found after death to be confined to the cæcum and beginning of the ascending colon. The patient was admitted with hæmatemesis and melæna and was fed for some time by rectum. In Case No. 52 the patient

had extensive follicular ulceration of the large, and to a less extent, of the small intestine. The symptoms in this case were intractable diarrhœa and vomiting. An early stage of peritonitis was present at the time of death. The patient was fed entirely by mouth. Case No. 57 occurred in connection with a severe attack of gastro-enteritis in an infant of four months. Four days after the onset of symptoms the left parotid became involved, and two days later again the right. Both parotids suppurated, as well as some of the cervical glands. A brother of this patient was said to have had an exactly similar association of suppurative parotitis with gastro-enteritis which proved fatal.

In this connection the comparatively frequent association of parotitis with enteric fever is interesting.

Parotitis in Appendicitis.

Parotitis may occur in appendicitis, but is almost always associated with intra-peritoneal sepsis, either in the form of abscess or peritonitis. It is apparently not particularly common, for there was no instance among the 515 cases in our records during the years 1890 to 1903. Quite recently, however, a case of parotitis has occurred in a patient in the hospital, following appendicectomy (Case No. 27). The patient, a girl aged 16, had suffered for some considerable time from pelvic pain. Laparotomy being performed, the pelvic organs were found to be apparently normal, as was also the appendix, which was removed. Left-sided parotitis came on five days after operation, with dryness of mouth and some febrile disturbance. It subsided within a week; the right parotid was not affected. The occurrence of parotitis, without appendix abscess and the affection of the left parotid alone, are somewhat unusual features.

The first recorded case of parotitis complicating appendicitis is apparently Barlow's, which was published in 1886. Of Morley's cases, two followed "removal of

the appendix." Mr. Paget also records cases; a reference to his series in the 'Lancet' of 1886 will illustrate the class of case in which this complication occurs. Thus, in Case No. 14 of his series there was general peritonitis; in No. 15 the autopsy revealed peritoneal abscesses, some of great size, perforation of the appendix, and signs of old general peritonitis; in No. 19 general peritonitis and appendix abscess were present. Other cases are recorded by Stanley Thomas (11), Elder (12), Barjon (13), and Fiske Jones (14). Appendix abscess is definitely stated to have been present in all except the first of these. Fiske Jones' case is interesting, because the parotitis recurred with recurrent attacks of appendix abscess—there were three attacks in all. The attacks of parotitis always followed exactly the same sequence. Exactly forty-eight hours after each operation the right parotid became swollen, and forty-eight hours later again the left. The parotitis took six or seven days to subside.

A very interesting case has recently been published by Mr. Brennan Dyball (15), in which the parotid infection was extraordinarily severe, and is considered by the author to have been the immediate cause of death. The appendicitis was of the fulminating type, and at the operation, performed shortly after the onset of symptoms, the appendix was found to be gangrenous, with acute inflammation of the meso-appendix and free pus in the peritoneal cavity. The parotid sloughed in this case, although early incisions were made. The teeth in this patient were in a state of advanced caries, and the author attributes the parotid infection to the septic oral cavity. The patient, however, exhibited symptoms of profound toxæmia or septicæmia very shortly after the implication of the parotid and long before the parotitis had reached its height. The association of a virulent infection of the appendix, going on to actual gangrene in a couple of days, with an acute infection of the parotid, which also ended in gangrene, is at least suggestive of the origin of the parotitis in the abdominal infection.

F. E. Bunts (38) has recently published a paper on "Parotitis complicating Appendicitis." After a reference to some of the above cases, he describes three cases that came under his own observation, and a fourth under the care of Hamann. All four were presumably cases of abscess, although two only came to operation. The parotids suppurated in all four cases. In two of the cases the pus both from the appendix and parotid abscesses was examined bacteriologically. The organism isolated from the appendix abscess was the *Bacillus coli communis* in both cases—the parotid abscess in one case furnished the coli communis and the *Staphylococcus pyogenes aureus*; in the other, the staphylococcus only. Bunts lays much stress on the examination in this latter case—where different organisms were found in the two abscesses—as tending to show that the parotid infection could not have arisen from the abdominal abscess. It is conceivable, however, that a staphylococcus may have been present at an earlier stage in the appendicular lesion, and have been subsequently outgrown by the coli communis.

It is interesting to notice that in cases of appendicitis there is a greater tendency for the right parotid to be affected than the left. If the affection is bilateral, it usually starts in the right parotid.

Parotitis following Abdominal Operations on the Pelvic Organs.

I shall consider this class of operation at the present time, because a careful consideration of the cases would seem to point to the damage to the intestines and peritoneum, involved in these operations, and peritoneal sepsis as being more important factors in the causation of parotitis than some obscure sympathetic connection between the organs of generation and the salivary glands. The cases of parotitis following operations on the generative organs in which the peritoneal cavity is not directly involved I shall consider later.

In Mr. Paget's series of 101 cases, somewhat over one half followed interference with the pelvic organs, twenty-seven cases alone following ovariotomy. Of fifty-one cases of post-operative parotitis collected by Morley in America, twenty-six followed ovariotomy and eight more abdominal operations on the uterus and its appendages. Benoit's thirty-five cases, forming the subject of a Paris thesis, were all collected from the records of ovariotomy. Mœricke asserted that he had never seen it except after this operation. It is apparently the preponderance of cases following the operation of ovariotomy in these series that has led to the statement, found in most text-books, that ovariotomy is the operation which is most prone to be followed by the complication of parotitis. This is perhaps to be explained by their collection from the records of a time when ovariotomy was the only abdominal operation extensively practised. But an examination of series of consecutive cases will show that the incidence of parotitis after abdominal operations on the pelvic organs is relatively low. Our obstetric in-patient records for twenty-one consecutive years show that among 3065 in-patients, with 400 laparotomies, there were six cases of parotitis only.

The cases are distributed as follows:

Operation.	No. of Cases.	Cases of Parotitis.
Ovariotomy	225	4
Operations on Fallopian tubes	37	1
Rupt. ectopic gestation	33	1
Other abdominal sections	105	0
	400	6

Parotitis, then, would seem to occur in from one or two per cent. of cases only; it is about as frequent as in cases of exploratory laparotomy, and much less common than after gastric operations. This complication was relatively more frequent in earlier days. In the records of 300 cases of ovariotomy published by Knowsley Thornton (16) parotitis is mentioned as occurring in three

cases. Mœricke recorded five cases in 200 ovariotomies, Goodell four in 153.

Of the cases in my series (Nos. 28 to 34) four followed removal of ovarian cysts, one exploratory laparotomy and tapping of a malignant cyst, one an operation for ruptured ectopic gestation, and one removal of the appendages for hydrosalpinx. The points that these cases emphasise are —the accompanying damage to the abdominal contents involved in these operations, and the presence of peritoneal sepsis.

In the case of ruptured ectopic gestation (No. 28), the placenta was left *in situ*, the umbilical cord brought out of the abdomen, and the placental site drained. Peritonitis supervened a month after operation, and three weeks later, again, parotitis. Subsequently symptoms of obstruction came on, and the patient died. At the autopsy general peritonitis was found, with an abscess at the placental site. The parotitis in this case is clearly to be referred to the peritonitis, and not to the original operation.

A similar case after operation for ruptured ectopic gestation is recorded by Lewers (17). In his case the abdominal wound reopened spontaneously and a purulent discharge set in.

The same points are shown in the ovariotomy cases. In Case No. 29, shortly after the onset of parotitis, the patient exhibited symptoms of acute intestinal obstruction. On reopening the abdomen a coil of intestine was found kinked by a band; there was also some peritonitis present. In case No. 30 the cyst was adherent to the intestines, and the adhesions had to be broken down. The patient died of general peritonitis, which came on at the time the parotitis developed. In No. 31 the cyst was so adherent that it was left and drained. The case was also complicated by tuberculous salpingitis and peritonitis. The discharge from the wound rapidly became purulent, and the symptoms were generally septic. Parotitis developed a day or two before the patient's death, three weeks after the original operation; it is clearly to be assigned to the

peritonitis and not the operation. In No. 32, again, there were many adhesions, the division of which necessitated much damage to the intestines. The patient in this case, in addition to parotitis, developed not only pelvic peritonitis, but also symptoms of general pyæmia. In No. 33, where the appendages were removed for hydrosalpinx, there were many adhesions. In No. 34 a malignant ovarian cyst was found at operation and tapped; there were many secondary growths about the peritoneum. Parotitis developed three weeks after the operation, shortly before the patient's death, and probably other factors than the operation were responsible for it.

The other recorded cases of parotitis after operation on the pelvic organs also bear clear evidence of sepsis. Of the three cases in Knowsley Thornton's series, one died on the tenth day of pyæmia, and another on the ninth day of septicæmia. An almost similar frequency of the terms "pyæmia" and "peritonitis" will be found in the ovariotomy cases recorded in Mr. Paget's series in the 'Lancet' of 1886.

Of the seven cases in my series following operations on the pelvic organs the parotitis followed the operation sufficiently closely to be referred to it in four cases only. In two cases it developed only with the onset of septic peritonitis. In one case it was associated with acute intestinal obstruction. In most of the cases there is evidence of damage to the peritoneum and intestines at the time of operation, and in all but the sixth and seventh of intra-peritoneal sepsis. The proportion of cases, then, that can be referred unequivocally to damage to the pelvic organs dwindles still more, and one is justified in saying that on the whole parotitis is not a very common complication of operations on these organs. Since writing this I have come across an observation of Brunn's to the effect that cases of parotitis after ovariotomy were most frequent when multiple adhesions between the viscera were encountered, which bears out my own observation.

One case of parotitis (No. 51) occurred in connection with pelvic peritonitis, treated without operation.

Parotitis after other Abdominal Operations.

There is practically no operation which involves the opening of the peritoneal cavity which may not be followed by parotitis. Among 265 cases of abdominal section, including operations on the liver and gall-bladder, spleen, pancreas, and kidney—operations for tuberculous peritonitis, injuries, and new growths—exploratory laparotomies, etc., I have found two cases of parotitis. In both cases (Nos. 25, 26), one of cholelithiasis, and the other of tuberculous peritonitis, the operation was simply exploratory.

Parotitis in connection with Diseases of the Liver and Gall-bladder.

Three of the cases in my series occurred in connection with cholelithiasis—in one case (No. 26) following an exploratory operation. In all three patients there was a great deal of gastro-intestinal disturbance in the form of vomiting, and in one case, in addition, diarrhœa. One patient (Case No. 26) was admitted with symptoms suggestive of a mild degree of intestinal obstruction. The other two cases (Nos. 54 and 55), which were treated without operation, had had violent attacks of vomiting and colicky pain. The mouth in all three patients was extremely dry on admission. In case No. 55, in which the parotitis went on to suppuration, pyæmic abscesses subsequently developed elsewhere. The feeding in these patients was by the mouth.

The gastro-intestinal disturbance in these cases is probably the cause that disposes the gland to infection. In all three cases the onset of parotitis was preceded for some considerable time by suppression of secretion. In these cases there is probably a primary septic focus within the abdomen. It is well known that in cholelithiasis the infection of the gall-bladder may spread outwards, giving

rise to a localised peritonitis, which, as Mayo Robson has pointed out, may be sufficiently severe to cause intestinal obstruction, either by giving rise to adhesions or by paralysis of the intestine from peritonitis. A certain degree of intestinal obstruction was exhibited by the first of my cases. In one of the patients the parotid abscess was merely one of several pyæmic abscesses; in such a case the path of infection is almost certainly the blood-stream, and the primary focus the septic gall-bladder or the infected tissues in its neighbourhood.

The Connection between Parotitis and Diseases of the Pancreas.

The pancreas and the salivary glands are so similar structurally and physiologically that coincident involvement of the two organs would not be at all surprising. It has been observed that in certain diseased conditions of the pancreas the salivary glands may be affected, mostly in the direction of alteration of their secretion—as evidenced by the term "pancreatic salivation." Mayo Robson (18) has recently recorded a case of parotitis in a patient with suppurative catarrh of the pancreatic ducts associated with suppurative cholangitis. Right-sided parotitis was present, and the parotid was incised at the time of operation. According to Robson, suppurative catarrh of the pancreatic ducts is generally, if not always, combined with suppurative cholangitis, and inasmuch as parotitis is not an infrequent complication of cholelithiasis it is difficult to say which condition was responsible for this complication. One would imagine that the pancreatic condition would more strongly dispose to the development of parotitis than the hepatic, and it is possible that the pancreas might be involved in these long-standing cases of cholelithiasis that develop parotitis. I know of no published cases of parotitis in uncomplicated pancreatic disease, but the study of these diseases is comparatively recent. Apparently the opposite condition of secondary

involvement of the pancreas in inflammatory conditions of the parotid does obtain—at all events in the case of epidemic mumps. Cuche (19), Priestley (20), Jacob (21), and Simonin (22) have all described pancreatitis as a complication of ordinary mumps. Simonin found symptoms of acute pancreatitis in ten cases out of 652 patients in an epidemic of mumps, or in 1·3 per cent. Whether the pancreas is affected in cases of symptomatic parotitis or not I have no knowledge. I have examined the post-mortem records of cases dying after the development of parotitis, with especial reference to the condition of the pancreas, but these are not sufficiently extensive to throw much light on the subject. The condition of the pancreas is mentioned in seven of these cases. In four it is described as being normal, but in three of these the parotitis had commenced fifteen, thirty-two, and forty-one days respectively before death. In three cases there was evident naked-eye alteration in the gland. In case No. 53, a patient who died with follicular ulceration of the intestine and who developed parotitis a fortnight before death, the pancreas was found at autopsy to be congested; in case No. 61, a case of lobar pneumonia, in which parotitis developed five days before death, in conjunction with multiple abscesses—a condition of interstitial pancreatitis was found at the autopsy; and in case No. 67, a patient who died of erysipelas and in whom all the salivary glands were inflamed and swollen, the pancreas was found to show fatty infiltration. These results are rather heterogeneous, but I think that it is quite possible that with increasing recognition of pancreatic symptoms the pancreas may be found to be involved in a certain proportion of these cases—as it is in epidemic mumps.

I shall have to refer later on to parotitis in connection with gout. It is interesting to note that Portal in 1804 recorded a case of acute suppurative pancreatitis following an attack of gout in the feet.

Dr. Peacocke has described a case of parotitis in a patient suffering from diabetes (23). The patient was admitted to

hospital in a condition almost of coma, and died a month later. The parotitis developed about a fortnight after admission and was accompanied by slight fever, which continued till death. It appears possible that the parotitis in such a case as this might be due to some underlying disease of the pancreas, at any rate as a predisposing cause.

Parotitis in connection with Peritonitis.

The association of parotitis with peritonitis in abdominal cases is so close that one would have no hesitation in saying that peritonitis is the commonest immediate cause of parotitis. Although it is convenient to classify the cases according to the primary condition present, such as perforated gastric ulcer, appendicitis, and so on, it must be remembered that the majority of cases are to be referred to peritoneal infection in the first instance. In many of my cases the development of parotitis coincided in point of time, not with the operation, but with the onset of peritonitis. In series of cases taken from earlier days, when exploratory laparotomy was less common, many instances are described in connection with acute peritonitis. Mr. Paget's series contains eight instances. Addenbrooke (37) has described a case in connection with acute suppurative peritonitis in a boy; Hutchinson (40) a case in connection with acute peritonitis following rupture of the duodenum. Case No. 51 in my series occurred in connection with pelvic peritonitis. In many of the earlier cases the acute peritonitis was secondary to appendicitis.

On the Causation of Parotitis following Operations on and Diseases of the Abdominal Viscera.

Several theories have been advanced from time to time to account for this complication. In his paper, published in 1887, Mr. Paget discusses the various hypotheses that had been formulated up to that time. He discarded firstly the idea that it was a pyæmic or septicæmic manifestation, because in 93 per cent. of the cases in his series parotitis

was a solitary complication. The fact of a complication being a solitary one does not, however, preclude the possibility of hæmic infection. Secondly, he concluded that it was not due to "inflammation extending from a parched and sordid mouth, up to the duct of the gland," because on this view the socia parotidis should be the first part affected, which did not appear to be the case. He concluded that the reflex action of the nervous system played a large part in its causation, instancing the physiological experiment that interference with the alimentary canal of the dog hinders or arrests salivary secretion. An attack of acute parotitis is, however, something more than a mere suppression or retention of the secretion.

The view most generally held is that in the absence of food from the mouth the salivary flow is not stimulated; the mouth consequently becomes dry and septic, and an opportunity is afforded for a spread of infection to the gland by the duct.

Martin has endeavoured to attribute the dryness of mouth to the effect of anæsthetics. The fact that parotitis is by no means confined to patients who have undergone anæsthetisation shows this view to be inadequate.

Of the German observers, Pfannensteil traces it to infection from the mouth, whilst Mœricke, arguing from gynæcological cases, puts it down to a sympathetic relation between the ovaries and salivary glands. Such a relation, if it exists, cannot do more than dispose the gland to infection. There would appear to be a better marked nervous relation between the salivary glands and the gastro-intestinal tract.

Benoit, in his thesis, concludes that parotitis following ovariotomy is usually due to a spread of infection from the mouth, but the infection may occur by the blood-stream. Condamin, who criticises Benoit's conclusions, is very averse to the idea of a hæmic infection.

Morley, whose series is taken largely from gynæcological cases, lays stress on the dryness of mouth following abdominal operations on the pelvic organs, which he

ascribes to a sympathetic relation between the generative organs and the salivary glands. The actual infection he considers occurs from the mouth.

Pozzi considers that the onset of parotitis after cœliotomy is evidence of a certain degree of septicæmia. Osler, like Benoit, thinks that infection may occur both by the duct and the blood-stream.

The method of infection is discussed at length by Bunts in his recent paper (*loc. cit.*); he argues for infection by the duct, stating that inflammation of the duct has in some cases been found to precede inflammation of the gland. Injury to the gland or duct during operation or by the anæsthetic, which he suggests as a contributory cause, must play a very minor part, because there is no reason why this should occur especially in abdominal operations, to which the complication is practically confined. Nor, as I have shown, is this form of parotitis necessarily post-operative. After arguing strongly in favour of infection by the duct, Bunts nevertheless, at the end of his paper, admits the possibility of a hæmic infection. To quote his words: "Duct infection, in the light of modern pathology, and from the clinical study of many cases, would seem the only tenable theory; and while no doubt a large percentage of these infections occur primarily in Stenson's duct, the known fact that many pathogenic bacteria are cast off through the salivary glands makes it not improbable that, the *locus minoris resistentiæ* having been prepared by the dryness of the mouth, the stagnation of the saliva, the inactivity of the masticatory muscles from the enforced liquid diet or from possible injury to the duct or gland in operation, the bacteria may develop directly in the parotid, even while the duct is the site of an inflammation derived from bacteria in the buccal cavity."

The various observers, then, for the most part recognise a predisposing cause—the suppression of secretion, which is variously attributed to reflex nervous effects and to the absence of food from the mouth, and an exciting cause, infection. The actual inflammation is always preceded by

suppression of secretion—this is constant; the parotitis itself is inflammatory and septic. The process presents all the appearances of an acute inflammation; in severe cases the attack may even go on to extensive cellulitis of the surrounding structures, involving in some cases the whole of the side of the face and neck. It is almost invariably accompanied by febrile disturbance—there was a rise of at least 2° in temperature in 34 out of 44 cases in my series that followed abdominal lesions. The frequent termination of the process in suppuration is again conclusive of its nature. The older view that the swelling of the gland might be merely passive and due to a retention of secretion is quite inadequate.

We have, therefore, to deal with two stages in the process. The first stage consists of the suppression of the secretion, a process which throws the gland out of its normal condition, and renders it more liable to infection, and the causes that operate to produce this suppression of secretion may be called the predisposing causes. The second stage consists of the actual inflammation, and the cause at work in this process—the exciting cause—is infection. These may be considered separately.

A. *The Predisposing Causes leading to Suppression of Secretion.*

That the onset of symptomatic parotitis is preceded by suppression of the salivary secretion is an old observation. This suppression has been ascribed to the absence of food from the mouth; it is suggested that in default of their natural stimuli the salivary glands are not excited and cease to secrete. But, although the absence of food may assist in the suppression of the secretion, it is probably not the main cause. In my series of cases not only had many of the patients been receiving solid food by the mouth for considerable periods before the onset of parotitis, but in many cases—in fact, in over one-third—mouth-feeding had never been interrupted. Even among

the fifteen non-operative cases of gastric ulcer, six only were on rectal alimentation at the time of the onset of parotitis, and two of the cases had been on mouth-feeding for ten and eleven days respectively. We must therefore look to other causes for the suppression of the secretion. These causes are to be found in injury or disease of the alimentary canal. Apart from the direct experiment quoted in Mr. Paget's paper that interference with the alimentary canal of the dog leads to a suppression of salivary secretion, we have abundant evidence clinically of the effect of abdominal operations on the secretion. Thirst and dryness of mouth are always more marked after abdominal than other operations, and I have always found this to be especially manifested after operations on the stomach. Dryness of mouth is also especially noticeable in cases of peritonitis. Injury or disease of the gastro-intestinal tract seems to have the effect in some way of inhibiting salivary secretion. This same inhibition may also be marked after operations on the pelvic organs, and I would suggest that it may be due not so much to the interference with the pelvic organs themselves as to accompanying damage to the gastro-intestinal tract or peritoneum. The probable cause of this suppression of secretion is a reflex nervous inhibition, set up by reflex nervous impulses from the injured alimentary canal. There is a possibility that another cause may be at work as well. Bayliss and Starling have pointed out the *rôle* played by the body "secretin" as an excitant of pancreatic secretion. Enriquez and Hallion (24) state that Lambert and Meyer find that the presence of this body in the circulation accelerates the salivary secretion, although this is denied by Bayliss and Starling (25). It is obvious that grave lesions of the stomach, leading to a temporary paralysis of that organ, or such a condition as peritonitis, would largely interfere with the formation of this body, and might in that way lead to a diminution of the salivary secretion. In the absence, however, of more exact knowledge on this point, we must allot the chief

share in the suppression of the salivary secretion to reflex nervous influences. The fact, too, that, with a unilateral primary lesion, the parotid of the same side seems more prone to become affected also points in the same direction.

Suppression of the secretion of the gland, in whatever way caused, by throwing the gland out of play, would seem to lower its resistance and leave it prone to infection. It remains, therefore, to consider the method in which the gland becomes infected.

The exciting cause, infection.—The direct or exciting cause of parotitis is undoubtedly infection; the only question is the path by which it reaches the gland. There are three possible ways in which infection might gain access to the gland—firstly, by the blood-stream, secondly, from ti · mouth along the duct, and thirdly, by direct spread of an inflammatory process from surrounding structures. The more generally accepted explanation is that infection takes place by the duct; Osler, Benoit, Pozzi and Bunts, however, consider that it may occur by the blood-stream. My own opinion is decidedly in favour of the latter view, and I will endeavour to state as briefly as possible my reasons for this view.

1. In the first place, if we examine carefully the cases of parotitis that occur after abdominal operations, we shall find abundant evidence of a primary septic focus within the abdomen. In the thirty-four post-operative cases in my series there was actual peritoneal sepsis in nineteen and suspicion of it in others. I have drawn especial attention to this in the preceding part of this paper.

An analysis of the sixty cases collected by Mr. Paget and published in the 'Lancet' for 1886 demonstrates clearly the importance of this septic element.

Cases.	Evidences of sepsis.
Five cases following use of catheter or sound	Chronic cystitis; operation followed by rigors, pyrexia, etc.
Case following introduction of pessary	Operation followed by rigor and temperature of 104·5°.

Cases—*cont.*	Evidences of sepsis—*cont.*
Seven cases after delivery or abortion	Pyæmia, two cases; peritonitis, two cases; continued fever, one case.
Three cases of appendicitis	General peritonitis in all.
Pyo-salpinx	Pyæmia.
Bullet wound, abdomen	Pyæmia.
Perforated duodenal ulcer	General peritonitis.
Gastric ulcer	Vaginitis; bed-sores.
Herniotomy	Peritonitis.
Removal of omentum	Pyæmia.
Gonorrhœa	Pyæmia.
Fifteen cases of ovariotomy	Peritonitis, two cases; perimetritis, one case; pyæmia, one case; death with septic symptoms, one case.
Pelvic cellulitis	
Pelvic abscess and peritonitis	

So that there is direct evidence of sepsis in about one half of the sixty cases.

If we turn now to the operations and diseases which I have found to be especially liable to this complication, the influence of intra-abdominal sepsis cannot be overlooked. I find that parotitis follows operation for gastric ulcer in 18 per cent. of cases; whereas in cases of gastric ulcer treated without operation it occurs in but 2 per cent. And the operative cases are on the average confined to rectal alimentation for a shorter period than the non-operative. One cannot but conclude that the septic soiling of the peritoneum in the case of a patient with perforated gastric ulcer has much to do with the production of this complication. After other gastric operations, such as gastrostomy or gastro-enterostomy, the percentage of parotitis is much lower, because there is less peritoneal contamination; but it is probable that in a certain proportion of these cases some slight contamination of the peritoneum does occur. I have pointed out that in practically all the cases of appendicitis in which parotitis occurred, which are recorded in detail, either abscess or general peritonitis was present. In cases of acute intestinal obstruction and strangulated

hernia, where damaged gut is returned into the abdomen, there is certain to be some amount of—it may be localised—peritonitis. In some of the cases in my series the damaged gut was found to have given way, and a condition of general peritonitis to be present. In the gynæcological cases, again, there was abundant evidence of peritoneal sepsis.

2. The second point is the frequent association of parotitis with other septic complications. I have already mentioned the large proportion of septic complications that Mr. English has found to complicate the after course of patients with perforated gastric ulcer. All our cases of perforated ulcer that developed parotitis developed some other complication. Apart from peritonitis and intra-abdominal complications, and suppuration of the wound, I find that 11 of the 34 post-operative cases in my series developed other complications, such as thrombosis, pleurisy, secondary pneumonia, and metastatic abscesses. Other metastatic complications are, moreover, especially prone to occur in patients who develop parotitis during the height of the specific fevers. When parotitis forms one of many secondary infections, it seems hardly reasonable to suppose that the infection in the case of the other complications occurs by the blood, while in the case of parotitis it occurs by the duct. Nor does it seem justifiable to separate out the cases of parotitis which occur with other septic complications and label them pyæmic and adopt a different explanation for the parotitis that occurs as a solitary event.

3. A careful study of the cases, moreover, seems to show that the severity of the parotid infection varies with the severity of the primary condition and with that of the other complications, suggesting that the same organisms are at work in both processes. Where parotitis occurred with other complications in the cases in my series, if the other complication resolved, the parotitis usually did the same, while if the other complication were a suppurative one, the parotitis usually went on to suppuration. To

take an example, a resolving parotitis would be associated with a simple pleurisy, a suppurative parotitis with an empyema. The following table shows an analysis of the cases in which parotitis was associated with other complications.

Cases where Parotitis resolved.

Case No.	Disease or operation.	Other complications.
2	Perforated gastric ulcer	Pleurisy.
4	„	„
3	„	Pneumonia.
12	Gastro-enterostomy	Thrombosis.
44	Gastric ulcer	„
19	Intestinal obstruction	Pleurisy.
20	Strangulated hernia	Thrombosis.
21	„	Pleurisy.
22	„	Pneumonia.

Cases where Parotitis went on to Abscess.

Case No.	Disease or operation.	Other complications.
1	Perforated gastric ulcer	Empyema, multiple pulmonary abscesses.
17	Intestinal obstruction	Much suppuration of abdominal wound.
24	Ventral hernia	Abscess of palate, sepsis of wound.
32	Ovariotomy	Pyæmia.
54	Cholelithiasis	Multiple abscesses.
76	Removal of exostosis	„

The same point will usually be found to obtain in cases of parotitis occurring in the specific fevers—that a

suppurative parotitis is associated with other metastatic abscesses.

Moreover there is a general, though not absolute, relation between the severity of the parotitis and that of the primary condition. In fifteen cases of gastric ulcer treated without operation, the gland itself suppurated in only one instance, whereas in cases where there was well-marked intra-peritoneal sepsis suppuration was much more frequent. Moreover in Mr. Dyball's case, that I have already quoted, of fulminating appendicitis ending in gangrene of the appendix within a couple of days, and associated with symptoms of profound toxæmia, the infection of the parotid was so intense that the gland actually sloughed.

4. The complication of parotitis was, relatively to the number of abdominal operations performed, far more frequent fifteen or twenty years ago than at the present time. I find that there were almost as many cases of parotitis during the years 1890 to 1897 as there were during the years 1897 to 1904, although three or four times the number of abdominal sections were performed during the latter period. There appears to be some connection between the relative decrease in the number of cases of parotitis, and improvements in antiseptic surgery. If this complication were due to a spread of infection from the mouth, improvements in antiseptic surgery should have no effect on it. McDonald (39) has drawn attention to its relative frequency in the early history of abdominal surgery.

5. As Mr. Paget pointed out, if the infection were an ascending one by the duct, the first portion of the gland to be involved should be the socia parotidis. I have always looked for this involvement of the socia, and have found it in three cases, but it was always subsequent to the involvement of the main gland. The same process occurred in Mr. Jefferies' case (*loc. cit.*).

6. With a view to testing the influence of oral sepsis, I have tried to observe as far as possible the condition of

the mouth in cases that developed or seemed likely to develop this complication. I could not establish any relation between this complication and oral sepsis. Parotitis often occurred in patients in whom there was no indication of a septic condition of the mouth or teeth, while patients with the foulest mouths escaped it. Moreover with a view to testing the efficiency of oral antiseptics, I analysed 300 consecutive cases of gastric ulcer. Among 68 cases in which antiseptic mouth-washes were systematically employed, there were four cases of parotitis, while among 232 cases treated without them there were only 2 cases; and in order to exclude other factors, taking the cases that were being fed by rectum only, parotitis was still four times as frequent in those in whom mouth-washes were used. Nor can it be objected that mouth-washes were used in patients with particularly septic mouths, for it is the practice of some to use them as a matter of routine in all cases, while others do not. The only conclusion is that oral antisepsis has little effect in preventing the complication.

To summarise the argument, then, with regard to the path of infection. The tendency to the development of parotitis after abdominal operations seems to bear a marked relation to the amount of intra-abdominal sepsis. Further, the parotid infection, although it may be a solitary event, is frequently associated with other metastatic complications of hæmic origin, and its severity varies with the severity of these and of the intra-abdominal sepsis. Like other septic complications, its frequency has diminished with improvements in antiseptic surgery. Lastly, the tendency to parotitis does not vary with the degree of oral sepsis, and measures adapted to combat this have not the slightest effect in preventing the complication.

We may explain the process as follows: In these patients that develop parotitis there is a species of mild septicæmia. The organisms may be absorbed from the peritoneal cavity, or, in the case of gastric operations or ulceration of the gastro-intestinal tract, through some

abrasion of the mucous surface, and reach the parotid by the blood-stream. In the case of a healthy functional gland, they might pass through it, as they pass through the kidney, and be thrown off in the secretion. But in these cases the secretion has been, reflexly or otherwise, suppressed, and the organisms, instead of passing through the gland, are retained within it and start an inflammatory process. The suppression of secretion acts, not only by lowering the resistance of the gland, but also by allowing the organisms to obtain a footing. In fact, in this production of parotitis after an abdominal operation, we have an exact parallel in those cases of acute nephritis following operation on the lower urinary passages, where the distribution of the renal lesions shows the path of infection to have been by the blood-stream. In each case the effect of the operation is to depress the vital activity of the gland, and allow the organisms which would otherwise pass through it and be excreted to obtain a footing and start an inflammatory process. The only direct evidence for the theory of a spread of infection by the duct is an inflammation of the duct preceding that of the gland, which is said to have been observed in some cases. Finally, if infection occurred by the duct, parotitis should be a common and not a rare phenomenon, since the necessary factors for such an infection are constantly present both in post-operative and in febrile conditions.

II. Parotitis following Operation on and Disturbances of the Uro-genital Organs.

I would divide these cases into the following groups: Parotitis in connection with—(1) Abdominal operations on the pelvic organs; (2) other operations on and diseases of the uro-genital organs; (3) parturition and abortion; (4) pregnancy and menstrual disorders.

There is no doubt that disturbances of the generative organs may be accompanied by disturbances of the salivary glands, and even by parotitis. In the camel the salivary

glands are said to enlarge during the breeding season. Clinically we are familiar with the metastatic orchitis, and the less common mastitis, vulvo-vaginitis, and ovaritis in the female, that may accompany ordinary epidemic mumps. But a parotitis following a lesion of the genital organs is not common, and, except in Group 4, is confined practically to septic cases, and is probably of a pyæmic nature. The nearest parallel, perhaps, to the metastatic orchitis of mumps are the cases of parotitis following injury to the testicle. Billroth recorded such a case following a blow on the testicle, and Hutchinson (40) states that Mackenzie communicated to him two similar cases, both in young men.

Group (1) I have already considered, and I have shown that most of such cases bear evidence of intra-peritoneal sepsis, and that the accompanying damage to the alimentary canal and peritoneum may possibly act as the disposing cause of the parotitis.

With regard to Group (2), illustrative cases are not very common, and I have found no instances in the records I have consulted. The best collection of such cases will be found in Mr. Paget's series in the 'Lancet' of 1886. This collection contains five cases of parotitis following the introduction of instruments into the bladder in patients with chronic cystitis, and one following the introduction of a pessary. The co-existence of rigors and high temperature and the fatal result in three of the cases are in favour of the complication being pyæmic. Two cases in Mr. Paget's series followed operation on the cervix uteri. Dr. Goodell's paper read at a meeting of the American Gynæcological Society and quoted by Mr. Paget, also contained cases of parotitis following operation for lacerated cervix, vesico-vaginal fistula, Tait's operation (two cases) and hysterectomy. When we find it recorded that both cases of Tait's operation and the one for vesico-vaginal fistula ended fatally, there is at least a suspicion of post-operative sepsis to account for the result.

Group (3): the cases in connection with parturition and abortion all appear to be essentially septic. My

series contains two cases. Of the first case (No 71) I have few details. Unilateral parotitis developed during the puerperium and subsided without giving much trouble. In the second case (No 72) the patient, in addition to parotitis, developed parametritis and septic pneumonia. The temperature was of a hectic character and the case ended fatally.

In Mr. Paget's series ('Lancet', 1886) will be found seven cases after delivery and abortion. Two cases developed peritonitis, two pyæmia, and one had continued fever from uterine sepsis. The origin of the infection would seem to be as clear in these as in the post-operative cases.

Condamin (26) has recently published an interesting case of parotitis after abortion of a molar pregnancy. It was found necessary to curette the uterus for retained products of conception, and parotitis developed five days after operation. The attack was preceded for four or five days by suppression of the salivary secretion.

Mr. Paget found that puerperal parotitis usually developed some days later than post-operative. The reason for this, I should suggest, is that uterine infection frequently occurs some days after delivery, whilst in the post-operative cases peritoneal infection is usually either present prior to or occurs at the time of operation.

Group (4): the parotitis that occurs in connection with pregnancy and menstrual disorders is of a different order to the above and exhibits certain special characteristics. There are fewer of the attendant phenomena of inflammation with it, it never apparently goes on to suppuration, and it tends to be recurrent and periodic, recurring with pregnancy or the menstrual epochs. The phenomena suggest a strong nervous element in its causation.

The salivary glands are frequently affected during pregnancy, less commonly in connection with menstruation; but the affection is usually in the direction of increase or diminution in their secretory activity. Like the vomiting of pregnancy, it is to be regarded as a neurosis. In a case mentioned by Mr. Paget, in which abortion had to be

induced for continuous vomiting, salivation was a marked feature. Dr. Goodell has recorded two cases—one of hypersecretion of the salivary glands at each menstrual period, and the other of a patient with a painful left ovary, in whom at every period the secretion of the left parotid was suppressed.

The sympathetic connection between the generative organs and the salivary glands is well illustrated by the above cases, but actual parotitis is rare under these conditions.

A case of recurrent parotitis which first appeared in the second pregnancy and recurred regularly in the five succeeding ones is recorded by Dr. Harkin (27). The left parotid was affected; it steadily enlarged during the pregnancy, and subsided after delivery, leaving a little permanent enlargement. One of my cases of parotitis (No. 68) occurred in connection with pregnancy. The pregnancy was, however, complicated by chorea. The parotitis was certainly of the symptomatic, and not of the epidemic, variety.

With regard to parotitis in connection with menstrual disorders Professor Peter's case is classical (28). It occurred in a young woman with amenorrhœa. At what should have been the periods there were firstly for two consecutive months attacks of parotitis, the next month swelling of the labia, then two more attacks of parotitis, and in the following month menstruation.

Mr. Paget records that he found two instances of parotitis at the menopause. A very interesting case has been published recently by Dalché (29). The menopause in his patient was accompanied by much nervous disturbance. For a whole year the patient suffered from recurring attacks of parotitis, mostly at intervals of about a month, although the periods between them might be slightly longer or shorter. Both parotids were affected, with little or no fever, and without noticeable dryness of mouth. There is a case of Mr. Battle's that I shall quote later, where persistent

xerostomia, with subsequent chronic enlargement of the parotids, followed the menopause.

These cases are difficult to explain. The nervous system would appear to play an important *rôle* in determining the attack, but it is difficult to see how nervous influences could do more than influence the secretion. There seems no reason to look on the underlying cause as being septic, for the definite regularity of the attacks would be against an infective origin. Possibly, as Dalché suggests, the actual swelling of the gland may be of vaso-motor origin, and we may adopt his view that the process is lighted up by a genital auto-intoxication, and provisionally classify this group as toxic.

III. Parotitis following Operation or Injury of Portions of the Body other than the Abdominal and Pelvic Viscera.

Examples of this are exceedingly rare, and the parotitis is apparently always pyæmic. Mr. Paget collected thirteen such cases, and in all of them the parotitis was part of a general pyæmia. McDonald records a case after amputation of the leg. Cases No. 75 and 76 in my series belong to this group. In case No. 76 the patient, a boy aged 13, had an exostosis of the humerus removed. In addition to a suppurative parotitis he developed multiple abscesses. The case ended fatally. For the notes of Case No. 75 I am indebted to the kindness of Mr. Edgecumbe Venning. The patient, an old gentleman aged 82, fell, striking the left side of the chest against a fender and fracturing two ribs. He recovered from the accident, but eleven days later developed left-sided parotitis, with elevation of temperature. Although at first threatening to suppurate, the swelling subsided within the course of a few days. Eighteen days after the appearance of the parotitis a tender swelling, the size of a pigeon's egg, developed

over the right sterno-mastoid, also accompanied by febrile disturbance. This also subsided, but the patient gradually sank and died.

IV. Parotitis in connection with Diseases of Metabolism and Chronic Intoxication.

There is a variety of parotitis that occurs in connection with certain chronic metabolic diseases, such as gout and diabetes. Parotitis may also occur in cases of chronic poisoning with lead, copper and potassium iodide. I have grouped these together as cases of toxic parotitis. The cases are undoubtedly rare and have been described mostly by French observers.

Parotitis in Gout.

Four cases of this rare complication of acute articular gout have been collected by Dr. Debout D'Estrées (30) in a paper read before this Society. One patient was under the care of Sir Archibald Garrod; the other cases were observed in France. In three of these cases there was a very remarkable sequence of events. The attacks began with swelling of one parotid, on the subsidence of which there was an acute gouty attack in a joint on the opposite side of the body; then in succession the opposite parotid and the corresponding joint to that first affected became involved. In one of the cases this sequence of events recurred five times in succession at intervals of one to two years. The fourth case differed from the others in that an attack of gout in the right wrist was followed by a painful swelling of the right parotid. The sequence of events in these patients would apparently leave no doubt that the same cause is at work in the case of the parotitis and the articular attacks, namely, irritation by urates. One of the patients experienced a saline taste in the mouth before meals, which the

author considers to be due to the excretion of urates in the saliva.

I have recently been fortunate enough to observe one of these rare cases, in private practice. The patient (No 77 of series), an alcoholic man of 52, had had six or seven attacks of gout. The present attack commenced with involvement of the left ankle, the opposite ankle being attacked on the following day, and subsequently the left knee. Three days after the onset of the gout the left parotid became swollen and painful, and two days later the right parotid became involved. The parotitis was associated with difficulty in swallowing and dryness of mouth. The skin over the parotids was inflamed and œdematous, resembling somewhat the skin over a gouty joint. The duct papillæ were not inflamed and there was no saline taste in the mouth. The submaxillary glands were slightly enlarged and tender, and there was some swelling of the tissues beneath the chin. The parotitis was not accompanied by any elevation of the temperature. The swelling of the glands lasted about nine days and then subsided. As it developed the articular attack subsided, and on the resolution of the parotids there was a fresh outbreak in the feet. The parotitis was associated with peculiar attacks of paroxysmal pain, which, starting in the neck, extended upwards to the vertex and forwards to the parotid region and external auditory meatus along the course of the second cervical nerve. These attacks would recur once or twice an hour and were particularly distressing to the patient.

One of the cases in my series (No. 66) is interesting in this connection, in that a septic suppurative arthritis in a gouty subject aged 75 was followed by suppurative parotitis of the opposite side as the sole secondary pyæmic complication. It suggests that there was some lowering of resistance on the part of the parotid, which determined its selection by the micro-organisms.

Another of my cases (No. 56) illustrates the occurrence of parotitis in a gouty patient aged 40, during the

terminal uræmia of chronic nephritis. There were no other infective complications. Flexner has shown that terminal infections are common in granular kidney and morbus cordis, and the parotitis in this case appears to be of that nature. The selection of the parotid for the terminal infection is interesting.

I have also found parotitis occurring in patients with gonorrhœal arthritis, but in this case it is probably part of a general pyæmia.

Parotitis occurring in cases of chronic lead-poisoning (saturnine parotitis) is probably closely allied to gouty parotitis. It usually occurs in conjunction with attacks of colic, and possibly the intestinal disturbance may predispose the gland to attack. A paper on the subject has been published by Petit and Hudelo (31) in connection with two cases that they exhibited before the *Société Médicale des Hôpitaux,* in Paris, in 1899. The attacks may be recurrent, and may leave some permanent enlargement of the gland. In one of the above cases lead was detected in the saliva. The authors suggest that the parotitis may be due either to irritation of the gland by the lead during its excretion, or to a superadded infection of buccal origin. The first view would seem the more probable, especially in view of the fact that the parotid affection tends in these cases to be recurrent or chronic. Chronic enlargement of the glands in plumbism was described by Comby in 1882. A study of the subject will be found in 'La Presse Médicale' for December, 1897.

Parotitis has also been described by Chauffard as occurring in connection with poisoning by copper, and by several observers as following the administration of potassium iodide.

V. Parotitis in the Specific Fevers.

Various authors have drawn attention to the occurrence of parotitis as a complication of the following specific

fevers: typhus, enteric, pneumonia, cholera, yellow and relapsing fevers, secondary syphilis, pyæmia, and, rarely, smallpox. The primary diseases in which the cases in my series occurred as a complication were enteric, pneumonia, influenza, scarlet fever, facial erysipelas, gonorrhœa, and measles.

In Enteric Fever.

Parotitis is by no means a rare complication of this disease, its frequency varying somewhat in different epidemics. Mr. Paget, quoting the reports of the London Fever Hospital for the years 1870 to 1885, states that it occurred in 13 out of 2000 enteric patients, but only once amongst 7000 cases of scarlet fever. According to Osler, there were 45 instances among the 2000 Munich cases of enteric. Keen collected 75 cases of this complication in enteric, and 353 in typhus, but the total number of cases among which these occurred is not stated. Osler states that it is less common in enteric than in typhus.

Two cases in my series, both fatal, occurred in patients during the course of enteric fever. In the first case (No. 58), in addition to the parotitis there were other terminal infections—pericarditis and pleurisy. Post mortem, the *Bacillus coli communis* was isolated from the parotid, while pure cultures of the *Bacillus typhosus* were grown from the spleen, liver, and bile. Although the presence of the *Bacillus coli communis* does not exclude infection from the mouth, still, the well-known association of this organism with secondary typhoidal infections is suggestive.

In the second case (No. 59) the parotitis was also a terminal infection. There were no other metastatic complications.

The comparative frequency of parotitis in enteric fever compared with that in the other common infectious diseases is interesting as showing the part played by lesions of the alimentary canal in disposing the gland to infection. In cholera, moreover, another disease with its local mani-

festations in the intestine, parotitis is, according to Osler, by no means uncommon. The fact, too, that parotitis is so much commoner in enteric than in scarlet fever, a disease associated with virulent oral sepsis, would again point to a metastatic origin of the infection rather than to a spread of infection by the duct.

In Pneumonia.

Three of the cases in my series occurred in patients with lobar pneumonia, in one during the height of the disease, in the other two during convalescence.

In the case in which it occurred during the height of the disease (No. 61) it was accompanied by pericarditis and multiple abscesses, being distinctly pyæmic. The case was fatal, and the parotitis was again a terminal infection.

In case No. 60 it developed on the eighth day of convalescence, and although its onset was accompanied by a rapid rise of temperature from 98° to 104°, it resolved without suppuration and did not hinder the patient's progress. In case No. 62 it also developed on the eighth day of convalescence, and was accompanied by tonsillitis; there was no febrile disturbance with the attack, and it resolved without suppuration.

In Influenza.

Case No. 65 occurred in a patient with influenza, just at the height of the primary disease. It was unilateral and resolved rapidly.

In Scarlet Fever.

Although this disease is one rich in metastatic complications, parotitis is very uncommon, and there would seem to be no reason for regarding it, when it does occur, as other than metastatic. If infection occurred by the

duct, it should be more common. One case in my series (No. 64) occurred in a patient with this disease. It developed about the sixth day of the disease, was unilateral, and resolved, without suppuration. The patient recovered.

One case (No. 63) followed measles in a debilitated infant, but was simply part of a general pyæmia, and was, in fact, the first indication of it. The parotitis proceeded to suppuration, and was followed by multiple abscesses and septic broncho-pneumonia, the case terminating fatally.

The cases of parotitis occurring in facial erysipelas and gonorrhœal pyæmia I shall refer to in the succeeding section.

Parotitis, then, as a complication of the specific fevers, may occur either during the height of the fever or during convalescence. As a rule it is of very grave significance if occurring at the height of the disease, and is almost always pyæmic, pointing to a generalisation of the infection. It usually appears earlier than the other metastatic infections, and may be valuable from the point of view of prognosis. It is practically always suppurative under these conditions. During convalescence it is not a grave complication, and does not affect the course of the case. Cases occurring during convalescence should be distinguished from superinfection with ordinary mumps, and the most valuable diagnostic sign is the suppression of salivary secretion that accompanies and usually precedes the symptomatic form. The absence of history of possible infection, and the fact of the parotitis remaining confined to the patient, will help to exclude the epidemic variety.

VI. Parotitis in connection with Local Inflammatory Conditions of the Face and Neck.

Four of the cases of parotitis in my series occurred in connection with local inflammatory conditions, and I have ventured to draw attention to them because I cannot find that the process has been noticed to any extent. The parotid involvement would seem to occur by direct

spread of infection, and the cases have consequently been grouped by themselves.

Case No. 68 occurred in connection with erysipelas of the face in a patient suffering from gonorrhœa. He had had eight or nine gonorrhœal attacks and the last two had been complicated by arthritis. The subsidence of the second attack of arthritis was followed by the appearance of an erysipelatous patch which gradually spread over the face. Unilateral parotitis developed a week after the subsidence of the erysipelas, and lasted for about a fortnight. A similar case is recorded by the late Dr. S. O. Habershon (32) and quoted by Mr. Paget. The patient exhibited symptoms of general septicæmia and the parotid suppurated. In both these cases the parotitis followed an erysipelatous attack in the neighbourhood, and may be due to a direct spread of infection. On the other hand, it might be a pyæmic manifestation. Parotitis occurred as part of a general pyæmic infection in one of the cases collected by Thayer and Lazear (33) and published in their paper on "Gonorrhœal Endocarditis and Septicæmia."

Case No. 67 of my series is an example of the occurrence of parotitis in connection with facial erysipelas. The patient was an elderly alcoholic man, and the erysipelas followed a wound above the orbit. There was enormous swelling and inflammation of all the salivary glands.

Case No. 68 is a case of parotitis in connection with Ludwig's angina. In addition to involvement of the fauces and larynx, the patient presented on admission much inflammatory œdema of the superficial tissues beneath the chin and behind the ramus of the jaw on the left side. The left parotid was much enlarged, and the right was involved to a slighter extent. Although the primary condition yielded rapidly to treatment, the parotid enlargement was still present a month later.

Case No 69 is a very interesting case, in which parotitis developed in connection with a peculiar skin eruption of the face. I am indebted for the details to Dr. Des Vœux. The patient, a woman of 29, had suffered for seven years

from a peculiar skin eruption, recurring annually in autumn or winter and lasting for about six months. She had occasional attacks of joint pains. In the winter of 1901, with the rash, a painful swelling of the parotid developed on the right side, accompanied by some febrile disturbance. In the following winter a corresponding swelling of the left parotid appeared with the appearance of the rash on the left side of the face. The attacks left the parotids permanently enlarged. The case was exhibited before the Dermatological Society last February (34). The diagnosis of the skin eruption rested between Erythema multiforme and Lupus erythematosus.

In these cases it seems most reasonable to suppose that the parotitis is due to a direct spread of infection to the gland from neighbouring structures. It must be remembered that the salivary glands have lymphatic glands embedded in them, which drain the superficial parts of the face and neck. In the case of erysipelas or cellulitis of the facial structures, these glands may become infected, and it is conceivable that the infection might spread from them to the parenchyma of the salivary glands.

B. Chronic and Recurrent Parotitis.

Most of the recurrent and chronic cases of parotitis belong to the toxic group. The best example of recurrence is exhibited by the group that occurs in connection with menstrual disturbances. In gouty and saturnine parotitis there is a similar tendency to recurrence, and in the latter case the glands may become chronically enlarged, with occasionally acute exacerbations following exacerbations of the primary disease. Mr. Battle (35) has recorded an interesting case, in which persistent xerostomia followed the menopause. After three years the parotids slowly enlarged and remained permanently enlarged, with the occasional supervention of acute attacks.

In one of my cases, where parotitis occurred in connec-

tion with a curious eruption of the face, the glandular enlargement became chronic. Occasionally chronic enlargement occurs without assignable cause. Case 75 in my series illustrates this. It was a case of slow but progressive enlargement of the glands in a man of 24, without obstruction of the duct, and with no suspicion of new growth. The enlargement was associated with xerostomia, but saliva was secreted on the introduction of food into the mouth.

A similar case of chronic enlargement associated with xerostomia is recorded by Dr. Herringham (36). In this case the enlargement followed an attack of coryza. Another case of chronic enlargement in a man of 18 with muscular atrophy was exhibited by Dr. Mitchell Clarke at the Neurological Society in February, 1903.

Two cases of recurrent parotitis with xerostomia will be found in Hutchinson's 'Archives of Surgery' (40). In the first case the parotid secretion was very easily arrested and seemed to be influenced by various kinds of food; the patient had suffered for twenty years from attacks of swelling of the gland, recurring every few months. The attacks were associated with pain in the fingers, and there was a gouty family history. In the second case the attacks coincided with attacks of polyuria. Both cases occurred in female patients, and Hutchinson remarks that this disease seems to be almost confined to the female sex. The phenomena of the attacks suggest some interference with the vaso-motor mechanism of the gland, and there is a possibility that the exciting cause in these cases might be purely nervous in origin.

Some General Points in connection with Symptomatic Parotitis.

Symptomatic parotitis is a complication which may arise under such very varying conditions, and is indeed a disorder of such complex pathogeny, that it is impossible to regard it as a specific disease. Consequently it would hardly be profitable to discuss such considerations as an

incubation period, for even in the septic cases we can rarely be certain at what period the infection of the glands occurs. In the majority of post-operative cases it occurs as a rule within a few days of operation: the greatest number in my series occurred on the third day, the same day as the greatest number in Morley's cases. I have not found it to appear earlier than 48 hours after operation. If the onset is delayed for a considerable time after operation some other determining cause, such as septic peritonitis, is to be looked for. In the cases of gastric ulcer, treated medically, its onset did not seem to be connected with any recognisable factor. When other complications occur in addition to parotitis, parotitis as a rule precedes them, and should suggest a watch being kept for such complications. It should also suggest the possibility of sepsis within the operation area.

The duration of the attack is very variable and depends on its severity. The swelling may be over and gone within 36 hours, or may persist for a month or more. It tends to persist longer, and sometimes to become chronic in the toxic cases.

The severity of the attack is variable. The attack may be mild, with little febrile disturbance, and subside quickly, or it may be more severe and go on to suppuration. Occasionally the inflammatory process may extend beyond the gland itself, involving the surrounding structures in a condition of spreading cellulitis, even down as far as the clavicle. Very rarely the inflammation may be so intense that the gland actually sloughs. This occurred in Mr. Dyball's case (*loc. cit.*) and he has collected two or three other instances of it. It is confined to debilitated individuals, or to conditions of the severest type of infection, usually associated with septicæmia.

Febrile disturbance occurs in the majority of cases. It occurred in 21 out of 27 post-operative cases, in which the temperature was recorded. The attack may be attended by very high temperature and yet resolve, and the temperature is of little assistance in determining the question of

possible suppuration. In one case the temperature ran up 6° with the attack, but there was no suppuration.

The question of suppuration is an important but difficult one. In my series the proportion of cases in which parotid abscess occurred was very low—15 only out of 76 acute cases, or about 20 per cent. Among the cases following abdominal operations 7 out of 34 suppurated, among cases of gastric ulcer treated medically only 2 out of 15. If the complication occurs during the height of a specific fever, it almost invariably suppurates. The tendency to suppuration varies with the primary condition: the gland is more likely to suppurate in a patient with suppurative peritonitis or pyæmia than in a patient with simple gastric ulcer. In Mr. Paget's series 57 per cent. of the cases suppurated, but this series was a much earlier one than mine, and the cases exhibited a much higher proportion of sepsis. The proportion of suppuration in Morley's cases, 40 per cent., is intermediate between Mr. Paget's and mine. The fact that in large series of cases the percentage of cases suppurating varies with the amount of primary sepsis among the cases is in favour of the view that infection occurs by the bloodstream and not from the mouth.

The occurrence of suppuration is of slightly graver prognostic import, but as Mr. Paget has remarked, the cases do not die because the parotid suppurates. The occurrence of parotitis affects the prognosis only in so far as it is an induration of some primary septic focus, or as a forerunner of other septic complications.

It is worth recording that in three or four of the cases suppuration involved the lymphatic glands in the neighbourhood only, and not the main parotid.

Side affected.—The actual figures for the side affected in the cases in my series in which it is recorded are: Right side only, 21; left side only, 22; bilateral, 26. When bilateral, the involvement is rarely synchronous, one side usually being affected a day or two earlier than the other. With a unilateral primary lesion there is a slightly greater tendency for the gland of the same side

to be affected. Of gynæcological cases, in Nos 29, 31, and 33 of my series and in several published cases the parotitis was on the same side as the diseased uterine appendages. In the published cases of parotitis complicating appendix abscess the parotitis most commonly was either right-sided or commenced on the right side. There is also apparently a greater tendency for the left parotid to be affected after lesions of the anterior surface of the stomach, and the right after lesions of the posterior surface. It must be understood that this merely expresses the general tendency, and is far from being constant.

Sex and age.—Sex has apparently no influence in disposing to this complication. The same remark applies to age, except that the complication is not common in children, even after abdominal operations.

Organisms present.—The literature of the subject contains comparatively little on the bacteriology of symptomatic parotitis. In Bunts' and Morley's cases the organism present was *Staphylococcus pyogenes aureus*. The same organism was present in three cases in my series—one following ovariotomy, one after suture of a gastric ulcer, and the third in a case of gastric ulcer treated medically. As already mentioned, in a case dying of enteric the *Bacillus coli communis* was isolated from the gland. In all the cases the organisms were present in pure culture. In the cases of parotitis complicating appendix abscess published by Bunts the *Staphylococcus pyogenes* was isolated in pure culture in one case, while in another there was a mixed infection with staphylococcus and the *B. coli communis*.

Morbid anatomy.—The gland was examined microscopically in the case of a patient dying of enteric fever (No 58) by Dr. Salusbury Trevor. There was a patchy small-celled infiltration between the acini, with much fatty change in the cells of the parenchyma. The changes corresponded closely to those in the case described by Mr. Paget. The inflammatory changes seem to be chiefly interstitial in character.

The differential diagnosis of symptomatic and epidemic parotitis.—The question of the parotitis being unilateral or bilateral is of no value in the diagnosis. The most important point is the condition of the secretion. It is usually little affected in ordinary mumps (Fagge and Pye-Smith), while in the symptomatic variety the gland swelling is constantly accompanied and usually preceded by suppression of secretion. The outlying structures are more commonly involved in the accompanying inflammation in all but the mildest cases of symptomatic parotitis. Epidemic mumps is common in children, the symptomatic variety is uncommon. Genital metastasis does not occur in the symptomatic form, and the occurrence of suppuration is distinctive. The symptomatic variety is not infectious from one patient to another. Dr. Addenbrooke (37) has recorded a case in which parotitis following an operation for peritonitis in a boy of 13 was followed by an outbreak of mumps in other members of the family. Such cases are open to suspicion of being instances of the epidemic variety, because, after all, there is no reason why epidemic parotitis should not occur in a patient after operation as well as at any other time.

Prophylactic treatment.—Condamin suggests the employment of injections of pilocarpine, when operation is followed by marked suppression of secretion. The use of antiseptic mouth-washes, with a view to combating oral sepsis, has in our experience had no effect whatever in the prevention of parotitis. At the same time, they do no harm.

When parotitis has appeared, the ordinary lead and opium lotion is useful as a local application. To employ belladonna is most irrational, because it tends to still further paralyse secretion and increase the dryness of mouth which is one of the most distressing features of this complication. The question of incising the swelling needs careful consideration. The large proportion of cases ending in suppuration in the earlier series has led to a statement as to the necessity for early incision of the

gland. The results of my series do not support this line of treatment, and I would suggest that the surgeon wait for evidence of suppuration. More particularly is expectant treatment indicated when there are no signs of sepsis elsewhere. It has been remarked, and not without justice, that the surest way to encourage suppuration is to incise the gland. At the same time, in post-operative cases the swelling should be watched carefully, as it is not possible to tell which cases will end in abscess formation. If an abscess form and an exit be not given for the pus, it sometimes burrows through into the external ear. Even when this has occurred, however, it has apparently been followed by no permanent ill effects. It should be remembered that over and over again the most threatening cases resolve completely.

Conclusions.

1. That cases of acute symptomatic parotitis fall into two main groups—septic and toxic. The septic group includes post-operative cases, cases occurring in connection with lesions of the alimentary canal and its appendages, the infective fevers and general pyæmia, the puerperium, and cases arising by direct spread of infection in inflammatory conditions of the head and neck. The toxic group consists of cases occurring in connection with diseases of metabolism, such as gout and certain chronic intoxications, as well as, in all probability, cases in connection with disturbances of menstruation. There is another group of cases, mostly recurrent, which appear to be largely determined by vaso-motor disturbances.

2. That post-operative parotitis is almost entirely confined, as a complication, to abdominal operations, being commonest after operations on the stomach and conditions of profound gastro-intestinal disturbance, such as acute intestinal obstruction.

3. That it is a well-marked complication of gastric ulcer.

4. That it is not particularly common after operations on the pelvic organs.

5. That damage to the alimentary canal disposes the gland to infection by suppressing its secretion; and that the disposing cause after operations on the pelvic organs is probably the accompanying damage to the alimentary canal and peritoneum.

6. That with a unilateral primary lesion the gland of the same side is on the whole more disposed to infection.

7. That parotitis as a complication of abdominal lesions is almost always associated with some degree of intra-abdominal sepsis, more particularly peritonitis.

8. That it may be a solitary complication, but is frequently accompanied by other septic complications, and its severity varies with the severity of the latter, when present.

9. That the majority of cases tend to subside without abscess formation, and the tendency to suppuration depends on the degree of general sepsis.

10. That it does not depend on the absence of food from the mouth, and that the employment of oral antiseptics is of no value in preventing this complication.

11. That the path of infection is in the majority of cases by the blood-stream and not by the duct from the mouth.

12. That suppuration sometimes affects the lymphatic glands in the neighbourhood of the parotid, without involving the gland itself.

13. That the organism usually present in the post-operative cases is the *Staphylococcus pyogenes aureus.*

14. That except in so far as it is an indication of sepsis it does not affect the prognosis of the case.

15. That it may occur in connection with the specific fevers, either during the height of the disease or during convalescence. If occurring during the height of the disease, it is a sign of a generalised infection, and is of grave import; during convalescence it is usually of slight importance.

16. That in cases occurring during pregnancy, or in connection with menstrual irregularities there is a strong nervous element, but the exciting cause is possibly toxic.

17. That the toxic cases tend to recurrence and to chronic enlargement of the gland.

18. That sex and age have no influence on its incidence, except that it is uncommon in young children.

19. That the chief sign that distinguishes it from epidemic parotitis is a preceding and accompanying suppression of secretion.

20. That early incision of the gland in cases of parotitis is not indicated as a routine treatment.

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Post-operative Cases.

Case.	Sex.	Age.	Disease.	Operation.	Feeding at onset.	Date of onset.
1	F.	26	Perforating gastric ulcer, anterior surface	Suture	None	3rd day
2	F.	26	„	„	? rectal	7th day
3	F.	24	„	„	Rectal	3rd day
4	F.	30	Perforating gastric ulcer, posterior surface	„	Mouth for 4 days	6th day
5	F.	29	Perforating gastric ulcer, anterior surface	„	Rectal	3rd day
6	F.	25	Non-perforating ulcer	„	By mouth 8 days before onset	9th day
7	F.	42	Non-perforating ulcer, lesser curvature	„	Rectal	2nd day
8	F	38	Non-perforating ulcer, posterior surface	Excision	„	„
9	F	41	Old gastric ulcer	Division of adhesions	By mouth 2 days	3rd day
10	F	42	Carcinoma of œsophagus	Gastrostomy	By stomach	4th day
11	M.	?	„	„	„	10th day
12	F	40	Carcinoma pylori	Posterior gastro-jejunostomy	Mouth	?
13	M.	55	„	Anterior gastro-jejunostomy	Mouth and rectum	13th day
14	M.	34	„	Posterior gastro-jejunostomy	Mouth 3 days	6th day
15	M.	36	Volvulus	Reduction	?	?
16	F.	49	Obstruction by band	Division of band	Rectal	4th day
17	F.	44	„	„	Mouth 2 days	3rd day
18	F.	56	Malignant obstruction	Colotomy	Mouth	13th day
19	F.	—	„	„	Mouth and rectum	5th day
20	M.	38	Strangulated hernia	Herniotomy, omentum removed	Mouth 8 days before onset	12th day
21	F.	34	„	„	Mouth 3 days	4th day
22	F.	70	„	„	Mouth 5 days	5th day
23	M.	49	„	Herniotomy	Mouth	„

Post-operative Cases.

Duration.	Side.	Temperature raised.	Suppuration.	Other complications.	Result.
1 month +	R., then L.	2°	Yes	Pneumonia, pulmonary abscess, empyema	Death.
3 days	L.	nil	No	Pleurisy	Recovery.
6 days	R., then L.	To 102°	„	Pneumonia	„
3 days each side	„	nil	„	Pleurisy	„
?	„	?	„	?	„
20 days	R.	2°	„	—	„
Till death	„	3°	Yes	—	Death 8th day after operation.
4 days	„	2·3°	No	—	Recovery.
?	L.	3°	„	—	„
3 days	„	nil	„	—	„
Till death	R.	?	„	—	Death.
?	?	?	„	Thrombosis	Recovery.
9–10 days	L., then R.	2°	„	—	„
1 month +	L.	Temperature irregular	Yes	—	„
?	?	?	„	—	„
Till death	L.	3°	No	General peritonitis	Death 5 days after operation.
1 month	R.	3°	Yes	Suppuration of wound	Recovery.
A few days	„	nil	No	—	„
Till death	?	raised	„	Pleurisy	Death.
?	L.	?	„	Thrombosis	Recovery.
7 days	R.	1°	„	Pleural effusion	„
Till death	R., then L.	To 102°	„	Pneumonia	Death 10 days after operation.
36 hours	Symmetrical	nil	„	—	Recovery.

Post-operative Cases—continued.

Case.	Sex.	Age.	Disease.	Operation.	Feeding at onset.	Date of onset.
24	F.	44	Ventral hernia	Sac excised	Mouth	?
25	F.	27	Tuberculous peritonitis	Laparotomy	,,	2nd day
26	F.	37	Cholelithiasis	,,	,,	6th day
27	F.	16	Pelvic neurosis	Appendicectomy	Fluids by mouth	5th day
28	F.	42	Ectopic gestation	Laparotomy, placenta left	Mouth	7 weeks after operation
29	F.	29	Ovarian cyst	Ovariotomy	Mouth and rectum	3rd day
30	F.	—	,,	,,	Mouth	2nd day
31	F.	—	,,	,,	,,	20th day
32	F.	35	,,	,,	Mouth 2 days	3rd day
33	F.	31	Hydrosalpinx	Removal of appendages	Mouth and rectum	,,
34	F.	—	Malignant ovarian cyst	Exploratory laparotomy, tapping of cyst	Mouth	3 weeks after operation

Analysis of 34 post-operative cases.
23 recovered, the gland suppurating in 5 and resolving in eighteen cases.
11 died, ,, ,, 2.

Non-operative Abdominal Cases.

Case.	Sex.	Age.	Disease.	Feeding at onset.	Date of onset.
35	F.	48	Gastric ulcer	Mouth 10 days	22nd day
36	F.	27	,,	Rectum	5th day
37	F.	23	,,	,,	15th day
38	F.	21	,,	,,	14th day
39	F.	16	,,	,,	8th day
40	F.	22	,,	Mouth	Before admission
41	F.	33	,,	Mouth 3 days	15th day
42	F.	34	,,	Rectum	3rd day
43	F.	41	,,	,,	9th day
44	F.	19	,,	Mouth and rectum	14th day
45	F.	29	,,	Mouth 3 days	15th day
46	F.	24	,,	Mouth 1 day	9th day

Post-operative Cases—continued.

Duration.	Side.	Temperature raised.	Suppuration.	Other complications.	Result.
?	?	?	Yes	Abscess of palate, sepsis of wound	Recovery
2 days	Double	102–4°	No	—	„
?	L.	1°	„	—	„
7 days	„	To 102°	„	—	„
1 month	R., then L.	?	„	Peritonitis (before parotitis)	Death.
Till death	R.	2°	„	Intestinal obstruction	Death 10 days after operation.
„	Double	3°	„	Pelvic peritonitis	Death 6 days after operation.
„	L.	Irregular	„	Peritonitis	Death 24 days after operation.
3 weeks +	L., then R.	2°	Yes	Pelvic sepsis, signs of hepatic abscess	Recovery.
1 month +	R.	1°	No	—	„
Till death	„	nil	„	—	Death.

Feeding by mouth, 23 cases; by mouth and rectum, 4 cases; by rectum, 6 cases; starved, 1 case.

Non-operative Abdominal Cases.

Duration.	Side.	Temperature raised.	Suppuration.	Other complications.	Result.
?	L.	?	No	—	Recovery.
19 days	L., then R.	To 102°	„	—	„
11 days	R.	?	„	—	Death.
6 days	R.	?	„	—	Recovery.
Till death	L., then R.	1°	„	—	Death.
2 to 3 days	L.	?	„	—	Recovery.
11 days	R.	2°	„	Urticaria	„
22 days	R.	2°	Yes	—	„
?	L.	2°	?	—	„
6 days	?	2°	No	—	„
4 days	L., then R.	3°	„	—	„
?	L., then R.	2°	„	Thrombosis	„

Non-operative Abdominal Cases—continued.

Case.	Sex.	Age.	Disease.	Feeding at onset.	Date of onset.
47	F.	24	Gastric ulcer	Mouth 6 days	7th day
48	F.	26	„	Mouth 11 days	20th day
49	F.	24	„	Mouth 2 days	6th day
50	M.	?	Duodenal ulcer	Mouth	7 days
51	F.	43	Pelvic peritonitis	„	7 days
52	F.	29	Ulcerative colitis	Rectum	3 days
53	F.	48	Ulceration of intestine	Mouth	10 days
54	F.	58	Cholelithiasis	„	8 days
55	F.	51	„	„	6 days
56	M.	46	Uræmia and hæmorrhage	„	6 days
57	F.	4 mos.	Gastro-enteritis	„	4 days
			Specific Fevers, etc.		
58	M.	28	Enteric	Mouth	28th day of disease
59	M.	17	„	„	20th day of disease
60	M.	—	Pneumonia	„	16 days after admission during convalescence
61	M.	36	„	„	8th day of disease
62	M.	22	„	„	9 days after fall of temperature in convalescence
63	F.	6 mos.	Measles	„	After attack, pyæmic
64	F.	21	Scarlet fever	„	5th day of disease
65	M.	20	Influenza	„	At height of disease
66	M.	75	Pyæmia	„	6th day after admission
67	M.	—	Erysipelas	„	?
68	M.	34	Erysipelas, gonorrhœal arthritis	„	13 days after onset of erysipelas
69	M.	—	Angina ludov.	„	With angina

Non-operative Abdominal Cases—continued.

Duration.	Side.	Temperature raised.	Suppuration.	Other complications.	Result.
2 weeks	L.	To 103·6	Yes, of lymph glands	—	Recovery.
5 days	R.	None	No	—	„
1 week	L.	1°	„	—	„
Till death	R.	3°	Yes	—	Death.
1 month +	R.	1°	No	—	Recovery.
12 days	Double	3°	„	—	Death.
8 days	R., then L.	2°	„	—	„
?	R.	None	„	—	„
5 weeks +	L.	To 103°	Yes	Multiple abscesses	Recovery.
Till death	L.	2°	No	—	Death.
—	L., then R.	Irregular	Yes	Bronchitis	Recovery.

Specific Fevers, etc.

Till death	R.	Slight rise	No	Pericarditis, pleural effusion, acute nephritis	Death next day.
	Double	„	„	—	Death 2 days after onset.
5 days	L.	Nil	„	Acute nephritis	Recovery.
Till death	,	?	Yes	Multiple abscesses, pericarditis	Death 5 days after.
4–5 days	Double	6°	No	—	Recovery.
?	L.	?	Yes	Multiple abscesses	Death.
?	„	?	No	—	Recovery.
2–3 days	?	—	„	—	„
Till death	L.	—	Yes	Pyæmic arthritis	Death.
„	Double	—	No	—	„
13 days	R.	—	„	—	Recovery.
1 month	Double	—	„	—	„

Various Diseases.

Case.	Sex.	Age.	Disease.	Feeding at onset.	Date of onset.
70	F.	29	Pregnancy and chorea	Mouth	13 days after admission
71	F.	?	Puerperium	„	?
72	F.	32	„	„	3 days after delivery
73	F.	30	Erythema multiforme	„	With rash
74	M.	24	Chronic enlargement of parotids	„	—
75	M.	82	Fracture of ribs, left side	„	11 days after injury
76	M.	13	Following excision of exostosis	„	?
77	M.	52	Gout	„	3 days after articular attack

Various Diseases.

Duration.	Side.	Temperature raised.	Suppuration.	Other complications.	Result.
14 days	Double	?	No	Herpes	Recovery.
A few days	?	?	„	—	„
Subsided after 4–5 days	R.	Septic	„	Parametritis, pneumonia	Death.
Recurrent and chronic	Double	Raised	„	Occasional articular attacks	Recovery.
—	„	—	„	—	„
A few days	L	Raised	„	Swelling over right sterno-mastoid	Death.
?	?	Raised	Yes	Multiple pyæmic abscesses	„
9 days	Double	Unaffected	No	—	Recovery.

DISCUSSION

The PRESIDENT, in thanking the authors of the papers for their interesting communications, remarked that they were at one in holding the view of the secondary infection in symptomatic parotitis, yet held contrary views as to the path of infection, the one holding that it was through Stenson's duct, and the other through the blood-stream, the conditions for infection being found in the functional inactivity of the duct. He hoped they should hear the experiences of those present as to how far one or the other of these views seemed to be valid, and whether in different cases one or the other might not be applicable.

Mr. STEPHEN PAGET alluded to the series of cases published by him many years ago, and referred to in the papers. On one point he desired to take exception to statements in one or other of the papers; in regard to treatment he considered that a timely incision was indicated in the suspected presence of pus formation, and short of this he believed that leeching and belladonna fomentations were valuable. The cause of all these cases of parotitis was clearly infection, and the view previously held as to the possibility of a sympathetic parotitis was now untenable. The sections which Mr. Bucknall had exhibited seemed to him to prove almost conclusively that infection took place through the duct, but at the same time he had to admit that Dr. Tebbs in his paper had brought forward considerable evidence pointing to an infection by means of the blood-stream, a reflex inhibition of the secretion of the gland being a predisposing cause. If the parotitis were held to be only due to infection by the duct in a debilitated subject, the question arose, Why was it not more common, seeing that all the necessary conditions for the occurrence of duct infection were so frequent?

Mr. J. D. MALCOLM also thought that the microscopic sections shown by Mr. Bucknall indicated clearly that, in the cases from which these sections were obtained, infection of the gland took place through the duct. He thought that the importance of septic mischief in connection with an operation, as a cause of this complication, had been exaggerated. His experience was all associated with abdominal operations, and he had seen parotitis when there was no other cause of trouble after a simple ovariotomy, sometimes as late as three weeks after the operation. He had pointed out ('Brit. Med. Journ.,' 1899, vol. ii, p. 1673) that, after an abdominal section, the dorsal position, the liquid food, and consequent absence of pressure of the muscles of the gland and duct during masticating, the febrile condition, and in former times the free use of opium, all tended to dry the mouth and so

favoured infection through the duct. In recent years he had seen this complication less frequently, but modern treatment, in addition to diminishing the risk of sepsis, allowed the patients to take liquids freely and to take food which required chewing early, and it did not produce dryness of the mouth by the administration of opiates. He referred to two cases of ovariotomy in which the opening of Stenson's duct was found inflamed as soon as there was the slightest evidence of mischief in the parotid gland.

Mr. W. McAdam Eccles thought that both the views advanced as to the path of infection in cases of secondary parotitis were very likely true, and he compared the infection of the parotid with that of the testicle in urethral inflammation, when the secondary epididymitis might be caused, he believed, either by direct extension or by the blood-stream. He had seen cases of parotitis from both causes. He alluded to two cases of abdominal disease that had been under his observation, in which, with no operation, parotitis had supervened after severe hæmorrhage into the intestinal tract. In both cases the temperature had been raised before the onset of the attack, and suppuration occurred in the gland.

Dr. C. O. Hawthorne said that he considered the occurrence of parotitis in cases of gastric ulcer was very rare, and he was surprised at the number of cases in which it had occurred in Dr. Tebbs' series. He mentioned two cases of parotitis following hæmatemesis which he had recorded, and he could only find reference to one other case, and that was one included in Mr. Stephen Paget's paper. He had been of opinion that infection came by the blood-stream, but the specimens which had been shown by Mr. Bucknall seemed to prove that the infection in some cases, at all events, took place along the duct. He could not, however, understand why these cases were not of greater frequency if infection was dependent on the condition of the mouth. He suggested that the condition might be due to some toxin, and compared it to the swelling of the gland which was sometimes produced by the administration of potassium iodide.

Mr. Bucknall, in reply, said that the cases advanced in favour of the view of infection by the blood-stream, as in septic peritonitis, might equally well be quoted in support of duct infection, as in them the mouth became very septic. In typhoid the mouth was usually much more affected in the way of ulceration than in diphtheria, hence the greater frequency of parotitis in that disease. In some cases where there might be a very septic state of the mouth, the disease might directly increase the secretions from the gland, and thus tend to prevent duct infection and parotitis. If infection were by the capillaries, it would be expected that they would be thrombosed and prominent in the sections, which was not so.

Dr. TEBBS thought: (1) The fact that the same organism was present in the gland, the duct, and the mouth, might be explained by the elimination of the organisms by the gland; (2) the preponderance of organisms in the inspissated secretion blocking the ducts would be accounted for by their being situated in a favourable culture medium, so that they would accumulate here rather than in the peripheral parts of the lobules, where they would be exposed to the bactericidal action of living gland cells; (3) that the argument deduced from the organism found in the gland being sometimes different to that of the primary disease might be subverted by the fact that when an infection became generalised the organisms of the secondary lesions were frequently different to those of the primary disease.

TWO CASES OF FUNGATING ENDOCARDITIS FOLLOWED BY CONVALESCENCE

BY

G. NEWTON PITT, M.D.

Received October 26th—Read November 8th, 1904

CASE 1.—*Acute fungating endocarditis of septic type with hemiplegia.**

R. Walker, aged 13, was admitted into Guy's Hospital under my care on June 24th, 1903. He had chorea commencing on May 13th, but not severely; six days later a systolic apical bruit, not traceable to the back, was detected. On June 5th there was a trace of albumen in his urine; for three days he had headache and vomiting, and on the 7th it was noticed he had an extensor plantar reflex on the right side. The bruit became louder, and could be heard both back and front.

June 8th.—A rigor with abdominal pain, and the urine contains a considerable amount of albumen, some casts, blood and pus cells.

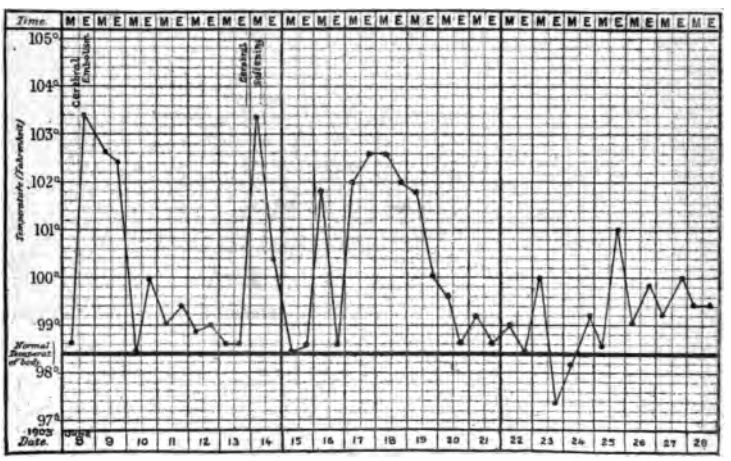
June 13th.—At 11 p.m., after a quiet day, he complained suddenly of severe pain in the head and all over the body. In a few minutes complete right hemiplegia developed, and later his power of speech was lost.

On admission the boy was very emaciated; the right hemiplegia was complete. He is drowsy, but can be roused with difficulty, and has occasional incontinence of

* The notes are by Messrs. F. D. Crew, L. T. Patterson Clavier, and J. H. Mayston.

urine. The right abdominal and cremasteric reflexes are absent; the left are present. On the right, the plantar reflex is extensor, on the left flexor, the knee-jerks are present. Reactions of pupils normal. No ocular paralysis. He is unable to speak and can only utter one sort of grunt, but can understand much of what is said. He is dull, but smiles when roused. The ulnar, triceps, and biceps jerks are only present on the right side. *Heart:* A diffused heaving impulse can be felt in the fourth, fifth, and sixth spaces for three inches to the left of the sternum. Apical,

CHART I.

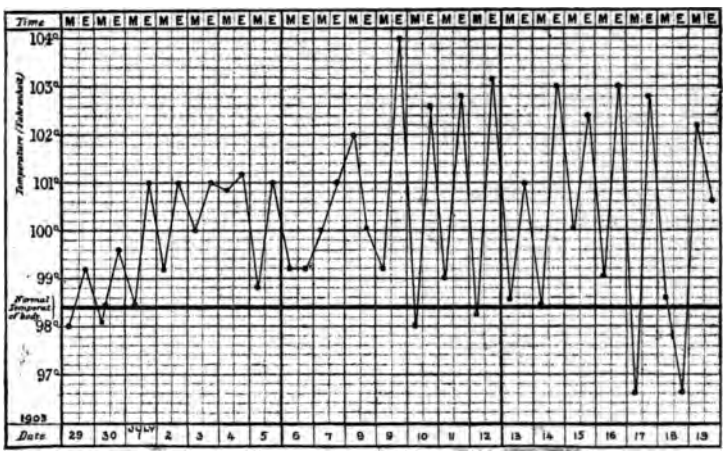


systolic, blowing bruit traceable two inches into axilla; at the base a systolic bruit is audible, loudest on the right side. Short systolic bruit in tricuspid area; pulse, 128; lungs and abdomen normal; spleen not palpable. The urine contains less than 1 per 1000 of albumen; and of urea, 2·8 per cent. Diagnosis: chorea, mitral incompetence, with fungating vegetations. Right hemiplegia and aphasia due to embolism. He was put on a mixture containing digitalis and nux vomica.

June 30th.—He is unable to close his lips and scarcely

separates his teeth in speaking. He has recovered the power of speech considerably, but is also difficult to understand on account of defective movements. He could say the alphabet consecutively with defective pronunciation of some letters, but when stopped could not go on again. He can use a considerable number of words, but is often at a loss. He can copy letters with his left hand, "mirror writing," can multiply figures by two or three, but not by a higher figure. He names coins, but does not know the number of pence in a half-crown.

CHART II.



July 7th.—His speech is practically limited to the use of a few nouns. He reads fairly accurately. He cannot recite from memory nor after two minutes' delay repeat a sentence.

July 12th.—Embolism of left femoral artery.

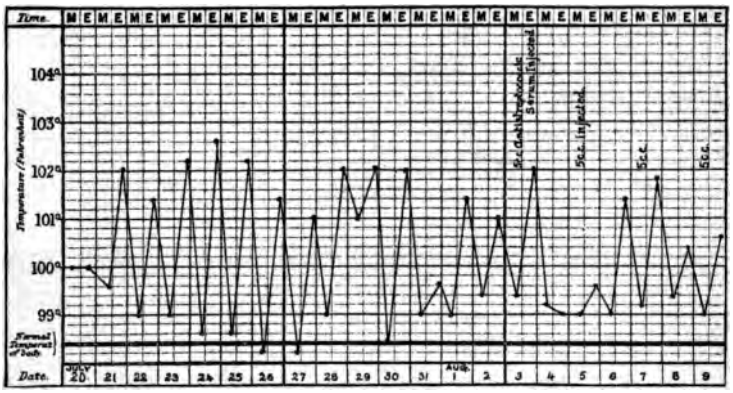
July 19th.—The temperature has varied from 103 to normal daily for the past fortnight. The amount of albumen in the urine has increased, and there is some blood.

July 24th.—He is able to move the toes of the right

foot, and there is some return of power in the right hand. The incontinence is very occasional now.

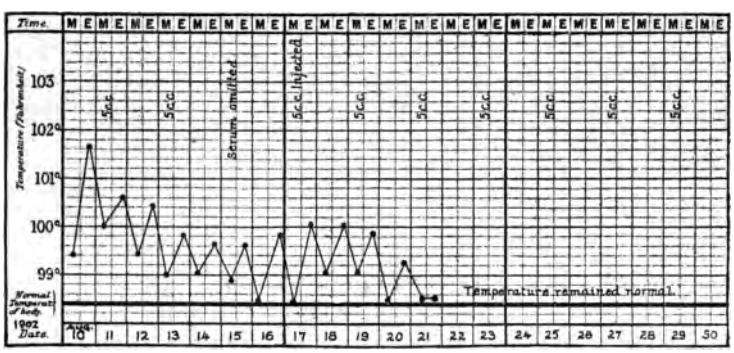
August 3rd.—The pulse rate has varied from 110 to 140.

CHART III.



The boy has been extremely anæmic and toxic for the past month, and has deteriorated greatly in health; we there-

CHART IV.



fore have decided to give him injections of antistreptococcic serum, 5 c.c. on alternate days.

August 7th.—He moves the fingers of the right hand

slowly, and power is returning in the leg. There is marked ankle clonus on right side.

August 11th.—Frontal headache; spleen palpable.

August 14th.—One dose of serum omitted on account of general erythema.

August 18th.—Spleen 2 inches beyond the ribs.

August 21st.—The improvement in the boy's condition is most remarkable. He is bright and cheerful. The toxic look has gone; his speech and the power in his right limbs has markedly improved.

August 31st.—A systolic bruit still audible at apex, and traceable outwards. Impulse less diffused and internal to nipple. He can now chat freely, and uses his right hand. There is still albumen, 1–2 parts per 1000, and a trace of blood in the urine and the spleen is still palpable.

September 7th.—The antistreptococcic injections are now to be given every four days; they were discontinued on the 15th. The temperature has been normal since August 21st.

September 28th.—Speech normal to a casual observer, writing still defective. He gets up daily.

October 25th, 1903.—Walks with slight dragging of leg. Went home looking the picture of health.

His doctor, Mr. G. McGregor, writes on November 2nd, 1904, to say the boy is in good health, and has been at light work since March last. He is a little short of breath when he runs. There is a long, loud, systolic, apical bruit. The grasp of the right hand is not quite so good as that of the left.

CASE 2.—*Acute right apical pneumonia; pneumococcal fungating endocarditis of the tricuspid valve; convalescence.*

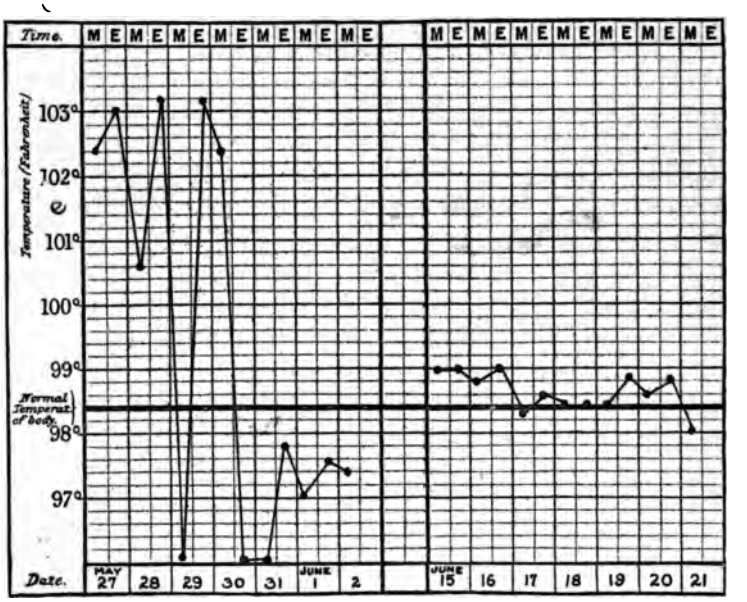
A boy, aged 11, was admitted under my care on May 27th, 1901, for acute pneumonia at right apex. (The notes of the case are by Messrs. T. C. Lucas and J. Goss.) On May 25th he had vomiting and a headache. At night he was delirious. He vomited twice.

On admission, pulse, 124; temperature, 102·4°; respirations, 38.

Deficient entry of air at the right apex. Gradually the apex became solid, and on the 31st, four days later, there was a patch of tubular breathing with bronchophony at the apex, and some distant tubular breathing at the upper part of lower lobe.

Ice poultices were applied to the right upper chest on the

CHART V.



29th. The temperature had fallen to normal on the night of the 30th, and next day the boy was comfortable. The boy would have got up on June 4th, apparently well, except that on June 1st a reduplication of the first sound was noticed at the fifth left space, pulse 68, and on the 4th a systolic blowing bruit was audible in the same region.

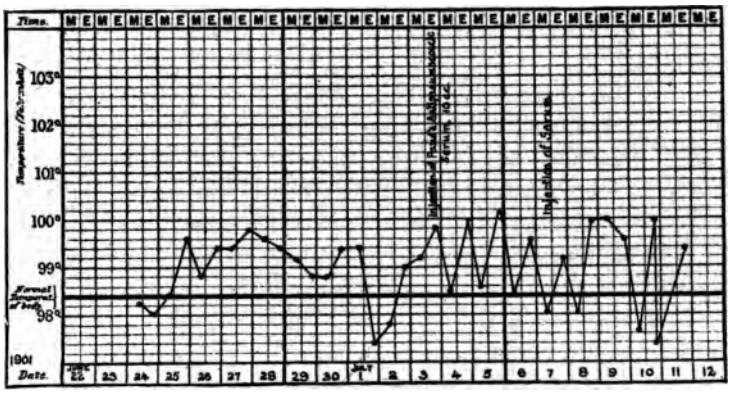
June 7th.—The bruit is now audible over the whole

cardiac area, with a maximum in the 4th and 5th left spaces. Temperature normal. Leucocyte count 10,420. The breath sounds at the right apex are diminished. No evidence of any empyema.

June 8th.—A systolic musical bruit is audible in 3rd and 4th left spaces, and an extremely short dubious diastolic bruit in the pulmonary area. The cardiac dulness has increased upwards. The right ventricle is dilated. Apex beat, 4th space, ½ inch internal to mamma.

June 13th.—Temperature above normal, but below 99·2° for the past week.

CHART VI.



June 15th.—The bruit less loud. Breath-sounds at right apex are coarser than those at left. There is tricuspid incompetence. The second pulmonary sound is not accentuated.

June 17.—The temperature is as low as 98·4° for first time since June 7th; patient looks better. Leucocytes 9140. Urine normal. The bruit is distinctly musical over only a small area, but a bruit can be heard as far as the apex.

June 22nd.—The evidence is in favour of a subacute fungating endocarditis of the tricuspid valve.

July 3rd.—As the temperature does not become normal, the boy is not well, and there is no evidence of any lesion

of the pulmonary, but only of the tricuspid valve, it was decided to inject him with Pané's antipneumococcic serum; 10 c.c. were given.

July 7th.—Antipneumococcic serum given again.

July 20.—The bruit is less musical, and it has a soft-blowing character. Boy feels well. Temperature not normal.

July 24.—Bruit again musical. Boy to get up after dinner.

July 27.—Leucocytes 8500. The temperature became normal.

August 9th.—Boy well and noisy. Bruit still audible, though less musical in character. The boy returned home. When seen some time later he was very well, but the bruit persisted.

Remarks.—Case 1.—The evidence that the first case was one of a malignant fungating endocarditis is conclusive. The presence of cerebral embolism, albuminuria, and hæmaturia, with a pyæmic temperature, and a dull, drowsy condition of stupor, followed by extreme emaciation, formed a typical picture. The boy steadily, during July, became more and more septic, with a temperature oscillating from 4 to 6 degrees in the 24 hours; and it looked as if his illness would rapidly prove fatal.

On August 3rd we commenced injections of 5 c.c. of antistreptococcic serum, and he had nineteen between that date and September 15th. The effect within ten days was most striking; he lost his toxic look, and became brighter. Within eighteen days his temperature became normal and never rose again. He regained power in his paralysed limbs, and gradually recovered his speech. He left the hospital six weeks later the picture of health, his speech almost normal, and able to walk about very fairly well.

An examination of his blood in August had given an impure culture of staphylo- and streptococci, but the variety of the streptococcus was not determined.

As the illness commenced with chorea, which had passed off before he came under observation, there can

be little doubt that the organism was that of acute rheumatism, and was the cause of all the boy's illness. It is almost universally assumed when cocci which will grow in strings are found in the blood of a case of malignant endocarditis that there has been a secondary infection by streptococcus pyogenes of valves previously damaged by rheumatism; the probability is that in such cases the whole of the process is due to rheumatism, and that the streptococci are those specific to rheumatism.

It will be very important in future in all cases of malignant endocarditis to ascertain whether formic acid is to be found in the urine in excess, as this seems to be the most striking characteristic of the growth of the rheumatic coccus. Any quantity much above ·0363 grams of formic acid per 1000 c.c. of urine would be abnormal.

The statement is generally made that a cerebral abscess may result from an embolism detached from fungating vegetations. It is doubtful if there is any post-mortem evidence to support this view, while there is abundant evidence that softening without suppuration occurs, as has taken place in this case apparently. The group of cases in which fungating vegetations are secondary to a local suppurating focus is a very small one; in these infarcts may suppurate, but they do not in the ordinary cases due to rheumatism. It is a very striking fact that embolism of the middle cerebral artery may often give rise to no symptoms at the time, and that the first evidence may be due to the rupture of a cerebral aneurism many days later, or to the later development of a patch of softening. An examination of the history during life of a case of cerebral aneurism often shows that there was nothing noted which would indicate when the embolism took place.

In this boy an extensor plantar reflex on the right side was noted on June 7th, with headache and vomiting; yet it was not till June 13th, six days later, that he complained of severe pain in his head, and within a few minutes he developed right hemiplegia. There is little doubt that

he had an embolus on the 7th, and it will be noted that the temperature was 103·4° next day, while the complete blockage of the vessel and the local softening was on the 13th, and caused a sudden rise of temperature from 98·2° to 103·4° the next morning.

Case 2.—The attack of pneumonia was typical, and after five days the boy appeared well; his heart sounds were normal. Five days later he developed a well-marked tricuspid incompetence; his temperature, which had been normal, again rose, and each day was a degree above the normal, and the boy was not well.

Nothing abnormal could be found in the lungs. The temperature was not normal for over six weeks. He had two injections of antipneumococcic serum, after which for a few days the temperature was less stable, oscillating between 98° and 100°, but gradually became normal three weeks later.

The boy completely regained his health and spirits, but the valve lesion persisted.

The development of marked incompetence of a valve in five days showed that we had a fungating endocarditis to deal with, but the type was not a virulent one. The recovery of these two cases appears to be worthy of being put on record, as the general impression is that the illness is necessarily a fatal one. This is not so; Dr. Dreschfeld refers to two cases of recovery, and a few others have been reported.

(Sir R. Douglas Powell has drawn my attention to three cases of recovery to which he referred in the 'Lumleian Lectures' for 1899, on p. 105.)

DISCUSSION.

THE PRESIDENT referred to the question of the supposed origin of these cases from the rheumatic poison, and expressed the opinion from clinical evidence that in most, if not all, of the cases there was some other poison concerned in addition to the rheumatic poison. In almost all the cases that had come within his experience in addition to the rheumatic lesion there had been engrafted some other poison effect, *e.g.* pneumococcal, typhoid, drain poison, or transferred sepsis. Only one case had he seen which followed directly on acute rheumatism. The cases recorded in the paper, which had been taken in an early stage, were very encouraging, but the good effect of the antistreptococcus serum was rather against the view that the endocarditis was due to the rheumatic poison alone.

Dr. F. J. POYNTON congratulated Dr. Pitt on the good results obtained. The evidence that the organism of acute rheumatism might be one of the causes of malignant endocarditis was almost conclusive. Recovery from this disease was probably not so infrequent as had been considered. There was, he believed, no sharp line of demarcation between simple and malignant endocarditis, but that they merged gradually into each other.

Dr. WILLIAM EWART, in connection with the views expressed by Sir Richard Douglas Powell as to the probably septic nature of the affection even in those who had suffered from rheumatism, referred to a patient then under his care (but since then dead) in whom the cardiac symptoms evolved after parturition, apparently from septic causes. When admitted into St. George's Hospital a few weeks later, she developed acute rheumatic pelzarthritis; and streptococci having been found in the blood, she was treated for both affections, viz. with antistreptococcus serum injections and with salicylates, and subsequently with tincture of the perchloride of iron, with temporary benefit. Although it was generally undesirable to assume double infections, the facts of this case suggested that a septic infection had been grafted upon rheumatic changes, the patient having previously suffered from acute rheumatism.

Dr. JOHN F. H. BROADBENT asked what the serum had to do with recovery. If it were of the rheumatic type, or if two organisms were present, its apparently good result was difficult to understand. He thought that a process of vaccination with sterilised cultures of organisms obtained from the blood might stimulate the production of protective substances in the less acute cases of malignant endocarditis. Antistreptococcic sera were not to be relied on. There was no guarantee that they contained any antitoxin or protective substances—they might even

contain toxins. Too much importance must not be attached to the apparent results of injections in which the presence of an antitoxin could not be determined. Recovery from the hemiplegia probably depended on shrinking of the embolus, of which he had seen one instance in which the embolus was situated in the middle cerebral artery, where it divided into its four main branches opposite the island of Reil.

Dr. O. K. WILLIAMSON asked if a leucocyte count had been made in Dr. Pitt's cases.

Dr. NEWTON PITT, in reply, said that in many cases the use of antistreptococcic serum had not given good results. He believed that most cases of fungating endocarditis were rheumatic in origin. One test which promised to be useful was whether the cultivation of the organism obtained from the blood yielded formic acid. The note of the blood-count in his first case could not be found, but leucocytosis was not necessarily present in malignant endocarditis.

THE SEVENTH CERVICAL RIB AND ITS EFFECTS UPON THE BRACHIAL PLEXUS

BY

WILLIAM THORBURN

(COMMUNICATED BY DR. G. NEWTON PITT)

Received April 28th—Read November 8th, 1904

GENTLEMEN,—Although cases of supernumerary cervical ribs are not very rare and are tolerably well recognised, my excuse for bringing the matter before you to-night lies in the fact that I have been unable to find any complete description of the clinical symptoms which they produce and especially in the slight notice which they have attracted from neurologists. As illustrating the latter fact we may note that in Duval and Guillain's Monograph upon the Brachial Plexus the seventh cervical rib is not even mentioned, and that several cases of radicular paralysis of the plexus have been recently published in which this condition, although a probable cause of the symptoms met with, is not considered as an ætiological factor.

With regard to the anatomical condition to which we have to refer I need only summarise the account given by

Sir William Turner in the 'Journal of Anatomy and Physiology' for 1870 (vol. iv, p. 131) an account which embodies the extensive statistics of Professor Wenzel Gruber.

More or less well developed ribs are liable to be met with in connection with the seventh cervical vertebra, the condition being so frequently bilateral that seventy-six examples were found in forty-five bodies. Occasionally the "rib" is a mere epiphysis articulating only with the transverse process of the vertebra, but more commonly it is a developed anterior transverse process and consists of a defined head, neck and tubercle, with or without a body. If no "body" be present the rudimentary rib does not project beyond the transverse process of the vertebra and has no clinical importance, but in the more fully developed cases it extends forwards and outwards into the posterior triangle terminating in a free end, or joining the first dorsal rib, the first costal cartilage or the sternum. Posteriorly this rib articulates in the usual manner with the body and transverse process of the seventh cervical vertebra. When it terminates in a free end it is bony throughout; to the first dorsal rib it may be united by fibrous tissue or by a joint with articular cartilage and a synovial membrane; junction with the sternum is by cartilage which unites with the first dorsal costal cartilage. The first dorsal rib often bears a well-defined bony tubercle with which is articulated the distal end of its cervical companion. The latter condition is well shown in the radiograms of Cases 1 and 2.

Between the cervical and the first dorsal ribs there is usually an external and sometimes an internal intercostal muscle, while in well-developed cases the anterior and posterior scalene muscles may be attached wholly or in part to the anterior end of the abnormal bone which then presents a groove between the insertion of the two muscles on which rest the subclavian artery and the lower cords of the brachial plexus.

With regard to the morphological interest of these

cases I need only refer further to papers published by Mr. Lane in the 'Guy's Hospital Reports' for 1883–4.

Clinically the supernumerary rib becomes of importance when it attains a size sufficient to project well into the posterior triangle of the neck, producing symptoms either as a deformity or as a source of pressure upon the subclavian artery or the brachial plexus.

It is of interest to note that such complications are generally met with in young women and on the right side of the neck. Of four cases which I have seen all were women, the ages being nineteen, twenty-one, twenty-two, and fifty-three, and the last having had symptoms for many years. In two of these deformity and symptoms of pain, etc., were present on the right side only, in a third they were much more marked on the right side, and in the fourth, although deformity was detected only on the left side, subjective symptoms were equally present on both. This selection of age and sex, although noted by others with regard to clinical symptoms, does not appear to govern the distribution of cases among dissecting-room specimens and is not quite readily explained. The age at which symptoms appear is probably determined by the completion of skeletal growth and the presence of deformity might be more readily noted in the female, but there is no apparent reason why the latter should suffer more from pressure symptoms, or why these symptoms should select the right side of the body.

We may now proceed to the consideration of the symptoms liable to be produced, illustrating them by two cases of minor degree, in which local troubles and vascular peculiarities were alone present, and by two far more interesting examples of serious injury to the brachial plexus.

Case 1, sent to me by Dr. Kant, of Romiley, is that of a girl, aged twenty-two, who had noticed a "small hard lump" in the right side of the neck for more than a year, the lump having slowly increased in size. The neck pre-

sented obvious fulness in the right posterior triangle and a bony mass could be clearly felt extending from the seventh cervical vertebra downwards, outwards, and then forwards. The rib is very clearly shown in the radiogram

FIG. 1.

CASE 1.—Radiogram from behind, showing cervical rib on right side.

(Fig. 1) and appears to unite with the scalene tubercle on the first dorsal rib. The left side of the neck presents no deformity.

There are no pressure symptoms in this case. The subclavian artery is readily felt immediately to the inner end of the bony mass, the brachial and radial pulses being of equal volume on both sides. The only evidence of any

affection of the brachial plexus is the statement that the right upper limb becomes tired very quickly.

Case 2, shown to me by Dr. Melland, is a woman fifty-

Fig. 2.

Case 2.—Radiogram from behind, showing cervical rib on left side.

three years of age, who had noticed a lump on the left side of the neck for many years. Through the somewhat fat tissues is clearly felt a bony prominence occupying the usual position in the left posterior triangle. The radiogram shows admirably the supernumerary rib and its articulation with the tubercle on the first dorsal rib (Fig. 2).

The left subclavian artery is distinctly higher in the

neck than that of the right side and presents visible pulsation, the left radial pulse being slightly smaller than the right; a systolic murmur is also audible above the left clavicle only. A vaguely described numbness and tingling is frequently felt in the arms and hands, but is equally present on both sides.

These two cases illustrate sufficiently the usual local appearances, and the second presents symptoms of vascular disturbance. It is conceivable that a greater degree of pressure upon the subclavian artery might cause severe anæmia and atrophy or even gangrene, but I have not met with any such condition. It was also noted by Sir James Paget in a letter quoted by Sir William Turner that the prominent artery coursing over the extra rib might be mistaken for an aneurism, a possible error in diagnosis to which reference is found in various text-books but which I do not know to have been actually made.

Of much greater interest than these minor cases are two examples of severe pressure upon the brachial plexus which present several points of neurological interest.

Case 3, sent by Dr. Matthews, of Levenshulme, is that of a lady, twenty-one years of age, who had suffered from pain in the right upper limb from her twelfth year, the pain being much increased about a year before I saw her, when loss of power was also noticed in the right hand.

On each side of the neck was felt a bony prominence with the characters already described, that upon the right being distinctly the larger. These swellings were so inconspicuous as to have previously escaped the observation of the patient. The right radial pulse was distinctly weaker than the left. No radiogram was obtained.

The nervous symptoms were described with great precision by the very intelligent patient. The "grip" of the right hand was much weakened; the thumb could not be properly used, abduction, adduction, and opposition being very weak; abduction of the index finger was very

feeble, adduction less markedly so; the lateral movements of the middle finger were weakened to a less extent; the ring and little fingers presented no marked weakness but were very liable to cramp, while the middle finger also became cramped on lifting small objects. Cramp was never noted in the more paralysed thumb and index finger.

Atrophy was well marked in the thenar eminence and over the abductor indicis, while there was hollowing between the index and middle fingers, but no wasting could be detected in the intermetacarpal spaces of the ulnar side of the hand or in the hypothenar eminence. The flexor carpi ulnaris appeared to be slightly wasted, and there was a little hollowness in front of the wrist.

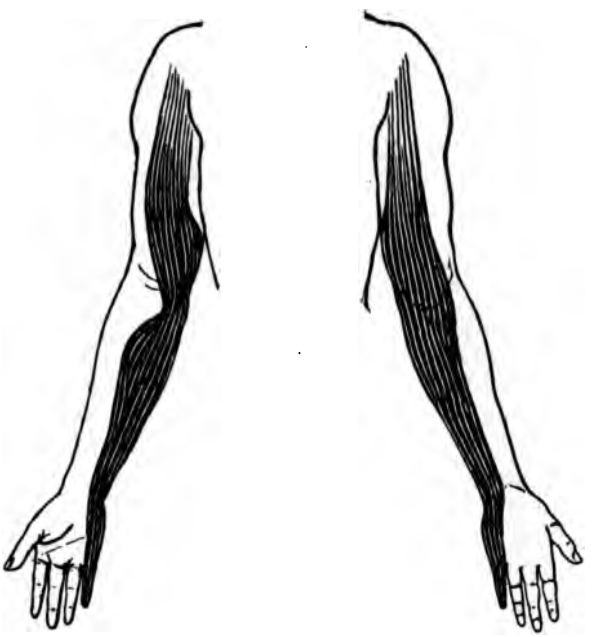
The location of pain was described with much accuracy, and the patient painted for me the painful area of which I was then able to take photographs, from which the accompanying diagrams have been prepared (Fig. 3). Except on the inner aspect of the upper arm, which is supplied by the second dorsal nerve, this pain was felt internally to two lines running from the shoulder to the cleft between the little and ring fingers. Of these lines the anterior started from the tip of the acromion and ran down the centre of the biceps until within a few inches of the elbow; veering inwards it then passed internal to the tendon of the biceps, returned once more almost to the centre of the forearm and again worked gradually inwards, crossing the wrist at the line of the ulnar artery and extending to the innermost interdigital cleft. The posterior line ran from the angle of the acromion slightly inwards to the olecranon and thence almost straight down over the lower end of the ulna to meet its fellow in the web between the little and ring fingers.

Sensation was said to be slightly defective in the whole of the painful area and in the hand but no objective estimation of anæsthesia was possible. The sensory defect appeared to be mainly thermal, and was noted by the patient especially in washing. The right hand also felt

cold subjectively and it presented very slight œdema, which may have been due to pressure upon the subclavian vein.

In November of 1898 I removed the right cervical rib, and four and a half years later the patient wrote to me: "I have not felt the pain in my right arm at all since the

FIG. 3.



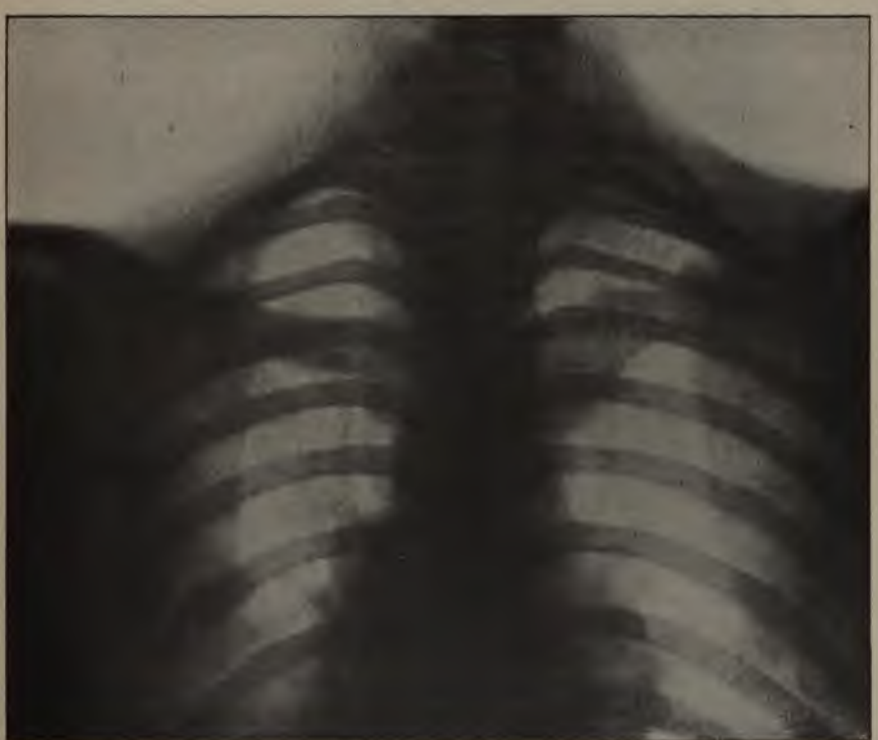
Painful areas on front and back of right upper limb, from photographs of areas painted by the patient.

operation. Sensation and strength in the fingers have returned slowly, and, though I am still very awkward, I am glad to say I can use my fingers much better than I could. A little while ago I could not use my first finger at the first joint, but I can do so now quite readily. With regard to the left side, I sometimes have the same dull aching pain in my arm, and the fingers of my left hand are very weak; also the muscle at the ball of the

thumb is almost flat. That muscle on the right side has developed again to a great extent."

Case 4 was transferred to my wards by Dr. Dreschfeld. The patient is a girl aged 19, working in a waterproof manufactory, who had been troubled for two years with

Fig. 4.



Case 4.—Radiogram from behind, showing cervical rib on right side.

pain in the right upper limb. When first felt this extended from immediately above the elbow down the inner side of the forearm to the hand, but at a later date it became more diffused. Cramp and weakness of the right hand gradually supervened.

The neck presented no visible swelling, but the cervical ribs could be readily felt upon both sides—the right being the larger. The radiogram, taken, as in other cases, from behind, is by no means clear, but indicates beyond question an excess of bone upon the right side (Fig. 4). The right subclavian artery was prominent, and the right radial pulse distinctly weaker than the left.

The right hand was very weak, the fingers being held close together and in a position of partial flexion, most marked at the interphalangeal joints. Abduction, adduction, and opposition of the thumb were all very weak, and lateral movements of the fingers were almost lost, except that slight abduction remained to the little finger. The position of flexion was more marked in the ring and little fingers than in the index and middle. Cramp was especially marked when the hand became cold, causing increased flexion, which affected principally the ring finger, and to a less extent the little and middle fingers, sparing the thumb and index. Clonic twitching also occasionally attacked the ring finger only. The constant spastic flexion of the inner fingers had given rise to a habit of straightening them out by means of the other hand. Atrophy was very marked in the thenar eminence, abductor indicis and interosseous spaces, but did not involve the hypothenar eminence. The right arm and forearm and the left upper limb presented no motor changes.

Pain and blunting of sensation were well marked, but of ill-defined outline, in an area extending from about an inch or two above the internal condyle of the humerus down the inner side of the forearm to the wrist, and thence onwards to the two inner fingers. At times this region would feel subjectively hot and burning, at other times cold and clammy.

I removed the right cervical rib, which extended well forwards into the neck, and was attached by a synovial joint to the enlarged scalene tubercle of the first dorsal rib. At the end of a year the symptoms of paralysis,

spasm, and atrophy had passed away, the hand being very slightly weaker and less developed than its fellow, but slight pain was still said to be felt inside the forearm.

The symptoms in the last two cases are remarkably similar, and consist essentially in paralysis of the intrinsic muscles of the hand, with pain and some sensory loss about the territory of the first dorsal root.

With regard to the motor troubles, we note especially that paralysis is most marked in the muscles of the thumb, and diminishes as we pass inwards to the little finger. Spasm, on the other hand, is more marked on the ulnar side of the hand, and is absent on the radial side. As in other lesions of the brachial plexus, atrophy is great, and, when contrasted with ordinary lesions of peripheral nerves, out of proportion to the amount of paralysis. In Case 3 there appeared to be some weakness of the flexors of the fingers, and possibly also atrophy of the flexor carpi ulnaris. In Case 4 there was spasm of the long flexors of the fingers, and especially of their inner or ulnar portions.

It is tolerably certain that the pressure upon the brachial plexus impinges upon its inner cord, which is derived from the lower trunk, and again from the eighth cervical and first dorsal roots, and it is also probable that the fibres derived from the first dorsal root lie lowest and in closest proximity to the bone, so that the clinical results will be similar to those of an affection of that root alone, although the eighth cervical may also be involved. In the scheme of distribution of the brachial plexus, which I first published in 1887, I assigned to the first dorsal root the principal innervation of the intrinsic muscles of the hand, but later observers have also attributed to it the long flexors of the fingers, which are slightly involved in these cases, and which were certainly involved in Charcot's classical case of a bullet wound of the root itself. The indications that the nerves for the radial side of the hand lie below or behind those for the

ulnar side at the point of pressure, and that paralysis and atrophy thus predominate on the outer side of the hand while spasm attacks its inner side is of considerable interest, and may prove of diagnostic value.

The pain in Case 4 is limited to an area which in my first scheme of the distribution of the brachial sensory roots would have involved the first dorsal and eighth and seventh cervical, while according to the later work of Kocher it would be covered almost entirely by the first dorsal. In Case 3 the painful area would be covered by my original first dorsal and eighth cervical areas, or again by Kocher's first dorsal area. The anæsthetic areas in affection of the plexus are, however, generally smaller, and the painful areas larger than the area of distribution of the affected roots, and these observations cannot be regarded as having any great value from the point of view of localisation, but, so far as they go, they tend rather to support the opinion that the first dorsal root extends to the inner one or two fingers instead of stopping at or near the level of the wrist as originally maintained by Head and myself.

It is perhaps worth noting at this point that the oculo-pupillary fibres of the first dorsal root are never affected in these cases inasmuch as these fibres leave the root considerably above the point of pressure.

The above cases present points of the closest similarity to certain interesting examples of "uni-radicular paralysis" of the brachial plexus recently published by Farquhar Buzzard and Edwin Bramwell, in none of which was the pathology fully explained, and in none of which was the possible presence of a cervical rib specifically excluded. For purposes of comparison we may briefly recapitulate these cases.

Buzzard ('Brain,' 1902, p. 299) described five cases as follows:

Case 1.—Woman aged 40; attack during convalescence from Malta fever; pain (soon passing away) shooting down right arm; atrophy of right thenar eminence, partial

atrophy and paralysis of interossei and long flexors of fingers; partial anæsthesia on inner side of forearm from just above internal condyle to just above wrist; eye not affected; condition stationary.

CASE 2.—Girl aged 19; gradual development after scarlatina and chorea some years before: right side; great atrophy of abductor and opponens pollicis; less marked in adductor and flexor brevis pollicis, marked in interossei, hypothenar muscles, and long flexors of fingers; partial anæsthesia as in Case 1; no eye symptoms.

CASE 3.—Woman aged 33; development after anæmia at eighteenth year; wasting of most of intrinsic muscles of right hand; feeling of coldness in hand; partial anæsthesia as in Cases 1 and 2; no eye symptoms.

CASE 4.—Woman aged 28; gradual development after influenza and gastric ailments; wasting and loss of power in abductor, opponens, and adductor pollicis; less marked in interossei, lumbricals, and hypothenar muscles, the abductor minimi digiti escaping; weakness of deep and superficial flexors of fingers, and possibly of pronator quadratus; partial anæsthesia as in Cases 2 and 3; no eye symptoms.

CASE 5.—Man aged 28; gradual development after influenza; much atrophy of abductor and opponens pollicis, less of flexor brevis and adductor; partial anæsthesia on inner side of forearm with "a narrow strip extending into the axilla;" no eye symptoms.

Bramwell's case is that of a man aged 18 with pain on the inner side of the forearm, coldness of the hand, flattening of the thenar and hypothenar eminences, prominence of the flexor tendons at the wrist, and a slightly claw-like hand; weakness affected the deep and superficial flexors of the fingers and all intrinsic muscles of the hand;

sensation of all kinds was impaired on the inner side of the forearm, from above the internal condyle of the humerus to the styloid process of the ulna; the orbital fibres were not involved.

Buzzard regards his cases as being probably vascular lesions, while Bramwell suggests that his may have been due to pressure against "the sharp internal border of the first rib," but whatever the explanation of the pathology these carefully recorded cases fall most closely into line with mine and illustrate a definite grouping of symptoms of injury to the first dorsal root or its continuation below the origin of its orbital fibres, the essentials of the clinical picture being the distribution of pain or anæsthesia on the inner side of the forearm, paralysis of the intrinsic muscles of the hand with occasional involvement of the long flexors, and especially a very strong tendency for paralysis and atrophy to be more marked on the radial side of the hand, while spastic symptoms may involve the ulnar side or the long flexors of the fingers. It is also noteworthy that four out of the six cases are women, and all are young adults.

I should like in conclusion to say a few words as to the diagnosis and treatment of the nervous symptoms due to the cervical rib.

With regard to diagnosis there can be little doubt that the real source of mischief is liable to be overlooked, as it had been in some of my cases. More especially would this danger arise if, as in my third case, the symptoms were bilateral. If, however, the condition be borne in mind it will probably be recognised. The escape of all descending fibres of the spinal cord should suggest a single or double lesion of the plexus. The escape of the orbital fibres will place this lesion fairly low down. Local examination of the neck will probably reveal a definite swelling even if this has not previously attracted attention, and a radiogram will, of course, furnish convincing evidence.

The treatment obviously consists in removal of the rib

whenever serious symptoms are produced, and it is equally clear that removal must be complete and must include the periosteum, which would otherwise form a mass of new bone. For this purpose I have used a vertical incision in the posterior triangle, with its centre over the most prominent part of the bone. The latter is readily identified and cleared by blunt dissection and retraction, when it can be cut away with bone pliers. The operation appears to be regarded by some writers as difficult and dangerous, but I have not had any trouble with it, nor required to do a subperiosteal excision. When the anterior end of the rib is attached to bone it may be a little difficult to reach its termination, in which case it can be cut cleanly away so far as is readily practicable, and the anterior end, with the large scalene tubercle, can then be nibbled away with gouge forceps.

DISCUSSION.

Mr. C. B. KEETLEY showed a patient (female, aged about 32) with a cervical rib. He had attended her for some years with Dr. Charles Simpson, of Towcester, and described the case. There was in this patient occasional neuralgia of the neck and arm, and great pulsation of the subclavian artery. The pain, which was first noticed fifteen years before, was chiefly referred to the outer side of the upper arm, and when it was at all marked her voice became altered. There was no muscular wasting, and he believed that motor paralyses were rare in these cases. The rib seemed to be about two thirds of the length of the first dorsal rib. The case had been mistaken for aneurysm. Genuine aneurysm had complicated such cases. In his case the radial pulses were different on the two sides. In most cases of cervical rib there were no painful symptoms produced, but in a minority disturbances of sensation, such as neuralgia, "pins and needles," and occasional numbness did occur. Œdema, thrombosis, and even gangrene, had been recorded from pressure on the subclavian vessels. Of course, in some of the cases the rib was not long enough to reach forward to and pass under those vessels. In the operation the pleura had been opened on several occasions, but not, so far as he knew, with any bad results. Dr. Charles Simpson kept the patient under observation with a view to operation should palliative measures at any time prove insufficient. Care should be taken to prevent external pressure by the clothing.

Mr. A. CARLESS thought the difficulty of diagnosis in these cases lay in the likelihood of the cervical rib being overlooked; if thought of, its presence was easily detected, if not by palpation, at least by skiagraphy. Pushing forward of the subclavian artery had not infrequently been mistaken for aneurysm, and only recently a case of this type had been sent up to him at hospital from the West of England for supposed subclavian aneurysm. The preponderance of the condition among females did not appear to be universal, and he referred to statistics on this point from the German army. The operation for removing the rib was probably less serious than many operations for the removal of tuberculous glands in that neighbourhood.

Dr. FARQUHAR BUZZARD believed that the cases of uniradicular brachial plexus palsy which he had described, and which Mr. Thorburn had referred to as being possibly similar to his own, were not of the same origin for the following reasons:—(1) Some had been due to a lesion of the fifth cervical root, some to one of the first dorsal root, and one case had presented the

symptoms of both these lesions; (2) in three cases the onset was acute, the atrophy rapid, and the pain present only at the beginning; (3) herpes had been a prominent feature of one of the fifth cervical cases; and (4) the sensory loss in all his cases had been dissociative in character, unaccompanied by numbness, and typical of a single-root lesion in its distribution.

Dr. PURVES STEWART had seen two cases of cervical ribs among out-patients without symptoms, and in those cases in which the ribs were bilateral the pain might be unilateral. There must be some other exciting cause of the symptoms. If exertion were an exciting cause of the pain it should be more common in men, which was not the case. It was hard to understand why a seventh cervical rib caused a first dorsal pain; on this principle a first dorsal rib should give a second dorsal pain. A seventh cervical rib, although an anatomical peculiarity, was not necessarily pathological.

The PRESIDENT asked if in the operation the sheath of the artery was likely to be damaged, or to suffer from loss of support on removal of the rib, so as to lead to subsequent dilatation of the vessel.

Mr. THORBURN, in reply, said he had had no experience of any such damage to the artery. The prevalence in females only applies to cases in which symptoms are produced, instances met with clinically, and not to necropsies. The relation of the second dorsal root to the first dorsal rib is very different from that of the brachial plexus to the seventh cervical rib, there being no doubt that the plexus is lifted up and stretched by the supernumerary rib, while removal of the rib cures the symptoms.

THE INFLUENCE OF STIGMATA OF DEGENERATION UPON THE PROGNOSIS OF EPILEPSY

BY

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In two previous communications to this Society ('Med.-Chir. Trans.,' vols. lxxxvi and lxxxvii) attention was directed, in the first, to the general prognosis and curability of epilepsy, and, in the second, to the influence of the mental condition upon the prognosis of the disease.

The object of the present paper is more particularly to ascertain whether "stigmata of degeneration," indicating the existence of a strong hereditary degenerative predisposition, play a part in prognosis; and if so, how far such stigmata may be used as guides in estimating the general prognosis of the disease in any given case of epilepsy.

Stigmata of degeneration may be defined, primarily, as structural deviations from the normal, arising during the course of development, in those who are the subjects of a neuropathic heredity. Although primarily anatomical, there have also been described physiological and psychical stigmata, but these will not receive consideration in the present research.

Structural stigmata have formed the subject-matter of considerable investigation since attention was directed to their importance by the writings of Morel and Moreau (of Tours). In the most pronounced forms of mental deficiency, such as are seen in imbeciles and idiots, anatomical variations from the normal are not uncommon. Such abnormalities are exemplified in microcephaly and other cranial deformities; in prognathism and facial asymmetry; in defective development of the hard palate; in irregularities and displacements of the teeth; in malformed limbs, badly designed ears, and numerous other malformations.

In the slighter forms of neuroses, such as neurasthenia, migraine, and simple melancholia, the neuropathic stigmata are less frequent and less pronounced than are seen in the more exaggerated conditions above mentioned.

In epilepsy, as in the other degenerative neuroses, stigmata of degeneration are present, and have received considerable attention, more especially at the hands of Ch. Féré.[1]

In addition to the cranial asymmetry which Lasègue[2] considered to be constant in true epilepsy, Féré has called attention to the frequency of cranio-facial asymmetry amongst epileptics. It is not absolutely characteristic of the disease, as it is found in a certain small percentage of healthy individuals.

Among other features of a degenerative character, the configuration of the hard palate deserves special attention. This has been the subject of much investigation and discussion, and its study and relation to the several degenerative neuroses is associated with the names, more especially in this country, of Langdon-Down and Clouston.[3]

Deformities of the ears have for long been regarded as important structural stigmata of degeneration. It is

[1] Féré, 'Les Epilepsies et les Epileptiques,' Paris, 1890.

[2] Lasègue, quoted by Féré, op. cit.

[3] Clouston, 'The Neuroses of Development,' Edinb., 1891.

scarcely necessary to do more in this connection than mention the names of Morel, Darwin, Wildermuth, Stahl, Binder, and others.

Abnormal implantation of the teeth in connection with epilepsy and the developmental neuroses has recently been the subject of careful investigation at the hands of Talbot.[1]

In addition to the above-mentioned stigmata, Féré has also drawn attention to, and described in detail, various asymmetries of the thorax and pelvis, unequal length and size of the limbs and syndactylism, found in epileptics.

Personal Observations.

In whatever form they are met, it has to be borne in mind that stigmata of degeneration are deviations from the normal, occurring during the period of growth, and, if anatomical in character, have to be distinguished from developmental defects arising before birth, such as hare-lip and cleft palate; or deformities acquired after birth, as are seen in cicatricial adhesions and scars, fractures, and so on.

My investigations into this subject have been confined to stigmata of a structural character, and these have been limited to the developmental deformities appertaining mainly to the head and face.

The cases upon which the observations have been made were taken from amongst a number of epileptics in the Colony of Epileptics, Chalfont St. Peter. No selection was made, but 100 consecutive cases of epilepsy, comprising both males and females, were examined.

It will be noticed that all were confirmed epileptics, the duration of the disease varying considerably. Many of them also were young people, and it has not been possible to give any data with reference to the presence or absence of stigmata in cases of epilepsy which have

[1] Talbot, 'Irregularities of the Teeth,' Philad., 1901.

been cured, or in those in whom the disease had commenced late in life.

In the subsequent pages allusion will be made to the following as structural evidences of neuropathic degeneration:

1. Facial (including nasal) deformity and asymmetry.
2. Deformities of the hard palate.
3. Dental anomalies and displacements.
4. Deformities of the external ears.

Stammering has been referred to as a stigma, as it was observed in four cases, but it would more correctly be included under physiological abnormalities. So also have high *errors of refraction*, as they were found in three cases. Although the exact value of errors of refraction as stigmata of degeneration, when occurring alone, is not clear, reference has been made to them here, as Féré has emphasised the frequent existence of astigmatism in epileptics. According to this authority it was found in 72 per cent. of his cases.

The not infrequent occurrence of large tuberculous-looking upper lips in many of the epileptics has not been regarded as evidence of degeneration; but it is a fact which seems to accentuate the close relationship which exists between epilepsy and tuberculosis.

1. *Facial deformity and asymmetry.*—Under this heading are included inequalities of the two sides of the face, in whole or in part; irregularities of the nose not arising from traumatic causes; prognathism and feeble or arrested development of the lower jaw. Arrested development of the upper jaw is indicated in the state of the hard palate.

According to the investigations herein detailed, facial asymmetry is more common amongst female than male epileptics; moreover it rarely occurs alone (in only 14 per cent.), being associated with other well-marked structural stigmata. 42 per cent. of the cases showed facial asymmetry, nasal deformity, and defective development of the lower jaw.

2. *Deformities of the hard palate.*—Abnormal development of the hard palate has long been recognised as one of the most trustworthy of the degenerative stigmata. Various types of abnormal palate have been described by different observers, but I have adopted Clouston's[1] classification, as being at once simple and comprehensive. This authority has described three varieties of hard palate: (*a*) the normal or "typical" palate, which is an arch-shaped structure with a low, regular, and wide dome; (*b*) the "neurotic" palate, which is higher than the normal, somewhat narrower, but has a fair, dome-shaped arch. It is the palate commonly seen in persons of a nervous temperament, and is not infrequently met with in neurasthenics, sufferers from migraine, depression, and dipsomania; (*c*) the "deformed" palate, which presents various abnormal shapes, is very high, narrow, keel-shaped, and somewhat irregular, and its roof exhibits a V-shaped or saddle-shaped appearance.

The *torus palatinus* is a mesial prominence along the palatal suture and occupies the whole, or a part, of the palatal roof. Cases presenting this abnormality have been placed in a fourth subdivision.

As in the case of facial asymmetry, palatal deformity, as a neuropathic stigma, does not usually exist alone, only 17 per cent. of the present series showing this deformity, unassociated with other evidence of structural degeneration.

The subjoined table shows the percentage frequency of the four types of palate in 100 epileptics:

Normal . . .	54 per cent.
"Neurotic" . .	33 „
Deformed . . .	11 „
Torus palatinus . .	2 „

This gives a total of 46 per cent. with palatal stigmata of degeneration.

[1] Clouston, op. cit.

3. *Dental anomalies.* — The dental anomalies consist chiefly in displacements of the teeth, one of the most frequent being displacement of the lateral incisors on one or both sides. Abnormalities in the eruption of the teeth, more especially when retarded, may also be regarded as neuropathic stigmata. Striation and faulty enamelling may also be included.

4. *Deformities of the ears.*—Although the relationship between the shape of the hard palate, the formation of the base of the skull and brain development would appear to be fairly clear and generally recognised, it is less easy to understand the connection between the ears and the degree of brain growth. But yet the external ear offers a constant and fruitful field for the study of neuropathic stigmata. Abnormalities of the ears have been extensively studied by numerous observers, as many as twenty-two varieties of aural deformity having been described (Peterson).[1]

For the present research the following subdivisions have been adopted:

(*a*) Normal ears.

(*b*) Abnormally shaped ears. This includes ugly ears; ears without, or with adherent, lobules; excessive conchoidal development; abnormal thinning of the margin; deformities of the antihelix; the Morel ear; and other abnormal structural appearances.

(*c*) Abnormal size. Ears that are too large, too small, too long, or too prominent.

(*d*) Asymmetrical ears; the Blainville ear.

As with the other stigmata, an abnormal development of the ears is rarely seen alone, only 9 per cent. of the cases showing such as a solitary stigma.

The subjoined table gives the percentage frequency of the different forms of ear in 100 epileptics:

[1] Peterson, 'Nervous and Mental Diseases,' Church and Peterson, New York, 1904.

Normal ears . .	65 per cent.
Abnormal shape . .	17 „
Abnormal size . .	11 „
Asymmetrical ears .	5 „

giving a total of 33 per cent. with aural stigmata.

It does not seem possible to attach any particular degree of mental impairment to the different forms of aural abnormality. It may, however, be stated in general terms that the more pronounced the deformity, such as is seen in the Morel ear, in unusual conchoidal development of the auricle, and in abnormally large and prominent ears, the greater is the degree of mental enfeeblement.

Stammering was present in only 4 out of the 100 cases, all being males.

High errors of *refraction* were found in three cases, two of myopia, and one of hypermetropia, but it is doubtful how far they should be regarded as stigmata.

The following table has been constructed to show at a glance the relative frequency and percentage of the three chief forms of stigmata described above in 100 epileptics.

	Facial asymmetry.	Abnormal palates.	Abnormal ears.
In 100 epileptics . .	42 % ...	46 % ...	33 %

Statistical Facts bearing upon the Frequency of Stigmata of Degeneration.

The relation between the several forms of neuropathic stigmata, their frequency, and their bearing upon the prognosis of epilepsy will be described under the following headings:

1. Sex.
2. Hereditary disposition to epilepsy and insanity.
3. Age at onset of the convulsions.
4. Duration of the disease from the onset of the seizures.
5. Type of the seizures.
6. The mental condition.

1. *Sex.*—The subjoined Table A has been constructed to show the proportion of males and females who presented evidence of neuropathic stigmata.

Of 100 confirmed epileptics, 62 were males and 38 were females.

Sex.	Single stigmata.	Combined stigmata.	None.	With stigmata.	Without stigmata.
Males	29	23	10	52 %	10 %
Females	15	8	15	23 %	15 %
				75 %	25 %

By "single stigmata" are meant abnormal development, or deformity of the hard palate or external ears, facial asymmetry or displacement of the teeth, while under "combined stigmata" are included a combination of two or more of the above.

From this table it is apparent that 75 per cent. of the cases presented neuropathic stigmata, while only 25 per cent. were without those obvious structural defects upon which observations were especially made.

It is also seen that the proportion of males exhibiting neuropathic stigmata is considerably greater than the females, while, on the other hand, the percentage of females apparently free from such stigmata is larger than the males.

If one inquires more fully into the different varieties of stigmata and the proportion of each found in the sexes, it is noticeable that the hard palate is more often deformed in males, while facial asymmetry is more commonly observed in women. In like manner the auricle more frequently exhibits deformity, either of size or of shape, in males than in females. This is more clearly seen from a study of the following Table B, which shows the total number of cases of each sex and their relation to the several forms of stigmata.

Sex.	Abnormal palate.	Facial asymmetry.	Abnormal ears.	Stammering.	Combined stigmata.	No obvious stigmata.	Totals.
Males	13	4	8	4	23	10	62
Females	4	10	1	0	8	15	38

The greater freedom from neuropathic stigmata exhibited by female epileptics, appears to stand in close association with a corresponding exemption from mental deterioration, which is also seen in this sex ('Med.-Chir. Trans.,' vol. lxxxvii, p. 356). How far this exemption would point to a lessened hereditary degenerative influence upon the female sex, is a point which is open to argument.

2. *Hereditary disposition to epilepsy and insanity.*—Owing to the difficulty in obtaining precise and satisfactory information upon the hereditary history in the institutional cases, little can be said as to the influence of a predisposition to epilepsy or insanity in the production of neuropathic stigmata.

It was generally observed, however, that amongst those in whom an hereditary tendency was transmitted directly from one or other parent, the structural abnormality was more pronounced than in those who gave merely a collateral hereditary history. There were, on the other hand, a few cases in which direct parental heredity was unaccompanied by any obvious anatomical evidence of developmental deformity.

The subjoined Table C shows the percentage frequency of a family history so far as it was obtained.

"No family predisposition known" in 74 per cent. Epilepsy in 19 per cent.; of these 13 per cent. showed, 6 per cent. did not show, stigmata. Insanity in 9 per cent.; of these 8 per cent. showed, 1 per cent. did not show, stigmata.

These figures are two small to base any general conclusions upon them, but it is not unreasonable to argue that if the family history could have been more fully obtained, a larger percentage of stigmata would have been found in those epileptics with a hereditary degenerative tendency. The fact that a small percentage (7 per cent.) did not exhibit any stigmata, even though the family history was positive, does not invalidate the above conclusion, as it is scarcely to be expected that structural evidence of degeneracy is to be found in every epileptic.

(3) *Age at onset of the convulsions.*—The following table, D, was constructed with a view to ascertain whether the presence of stigmata of degeneration stood in any relationship to the age at onset of the disease. Its object is to show the percentage frequency of stigmata according to the age at onset of the disease, measured in quinquennial periods up to twenty years. The number of cases at the Epileptic Colony in which the disease commenced after this age was so small as to prevent any generalisations being made.

Table D shows the age at onset of the convulsions in quinquennial periods up to twenty years of age, and the percentage frequency of stigmata in each group.

Age.	Single stigmata.	Combined stigmata.	None.	Percentages.	
				With.	Without.
Birth—5	14	7	3	21%	3%
6—10	6	3	4	9%	4%
11—15	21	10	8	31%	8%
16—20	7	4	6	11%	6%

The general conclusion may be drawn that the major proportion of the cases of confirmed epilepsy commencing under twenty years of age show stigmata of degeneration, either singly or combined, viz. 72 per cent. with, and 21 per cent. without. The discrepancy which exists between the percentages given here and those in Table A is due to the elimination from Table D of the few cases in which the disease commenced after twenty years of age. Of the cases which commenced under five years of age, seven times as many exhibited neuropathic stigmata as were without; while in the quinquennial period, eleven to fifteen, only about four times as many presented degenerative features.

From this it is apparent that of the two quinquennial periods in which the onset of epilepsy is most common, that from birth to five years is the most fruitful in the production of neuropathic stigmata. It is during this period that the growth of the brain is most active; it is during this period also that numerous other hereditary

degenerative affections appear, such as stammering, backwardness in walking and talking, delayed dentition, deaf mutism, and some forms of idiocy and imbecility.

I have elsewhere shown[1] that amongst the cases of epilepsy commencing during the first quinquennium is found the highest percentage of those who show profound mental impairment, and that epilepsy commencing in infancy and childhood is least favourable for arrest of the fits, and most favourable for the production of confirmed cases. From every point of view, therefore, *epilepsy commencing under five years of age must be regarded as the most unfavourable variety of the disease.*

(4) *The duration of the disease.*—Does the presence of neuropathic stigmata afford any clue as to the likelihood of the disease becoming confirmed?

The following Table E was constructed with a view to ascertain whether the existence of stigmata afforded any conclusive evidence as to the chronicity of the disease. The term duration signifies the course of the malady from the commencement of the fits.

Duration.	Single stigmata.	Combined stigmata.	None.	Percentages. With.	Percentages. Without.
1— 5 years	3	3	6	6%	6%
6—10 „	13	3	10	16%	10%
11—15 „	14	5	4	19%	4%
16—20 „	6	9	5	15%	1%
Over 20 „	12	5	5	17%	5%
Of 53 „	0	0	1		

The shortest duration was three years and the longest was forty-six, with the exception of the unusual case in which the disease had lasted for fifty-three years.

The majority of the cases which had lasted from six to over forty years showed stigmata of degeneration, but in the exceptional case which had lasted for fifty-three years no obvious stigmata were found.

The general conclusion, however, is obvious, that all cases of epilepsy do not show neuropathic stigmata, and

[1] 'Med.-Chir. Trans.,' vol. lxxxvii.

that *the absence of stigmata does not necessarily imply an early or satisfactory termination of the disease.*

5. *Type of the seizures.*—Does the presence or absence of neuropathic stigmata afford any constant evidence as to the type of seizure likely to be observed?

Table F shows the various types of seizure and the percentage frequency of neuropathic stigmata.

Type.	Single stigmata.	Combined stigmata.	None.	Percentages. With.	Percentages. Without.
Grand mal.	26	11	17	37%	17%
Petit mal	3	3	4	6%	4%
Combined grand and petit mal.	12	5	2	17%	2%
Serial fits	3	4	3	7%	3%
Serial and single fits	2	1	1	3%	1%
Nocturnal fits	0	2	0	2%	0

It will be observed at the outset that cases with convulsive (grand mal) seizures as the type of the disease are the most frequent and numerous, and that of these rather more than twice as many have degenerative stigmata. Of the remainder it will be seen that there is a considerably higher percentage of cases with stigmata amongst those of combined grand and petit mal than of the other types of the disease. In this combination also there is a much higher percentage of cases with stigmata, viz. about eight times. This is in conformity with the view elsewhere expressed that such a combination of seizures is an expression of the disease in a severe form, for it has been shown that the greatest frequency and highest degrees of dementia are found in those cases which are characterised by a combination of the grand and petit mal attacks, only a small percentage of such cases presenting no obvious mental deterioration.

The petit mal type of the disease is not one characterised by dominating evidence of a hereditary degenerative disposition, there being only a small excess of cases with stigmata over those without. To this observation may be

added the further one, that the mind is affected to only a slight extent, chiefly in the form of impairment of memory for recent events, in those cases in which the petit mal seizures occur alone.

In like manner cases of serial epilepsy, either occurring as such or in combination with single fits, do not exhibit so marked an excess of stigmata as the combined grand and petit mal cases. The large percentage of cases of serial epilepsy, which show mental deterioration of a pronounced type, in contrast to the relatively small percentage of such cases which show stigmata, might at first sight appear contradictory to what has just been stated. The explanation is probably to be found in the debilitating influence of serial fits directly upon the mental state rather than the dementia being an expression of a strong hereditary degenerative taint. Support to this view will be found in the fact that serial fits are more commonly followed by temporary dementia and delusional states than are single convulsive or petit mal attacks.

6. *The mental condition.*—Does the degree of mental impairment in epilepsy stand in any relationship to the presence or absence of neuropathic stigmata?

It would appear from the subjoined table as if this were one of the most important points determined by the investigation.

The mental condition in epilepsy has been divided into four classes, which have been elsewhere defined,[1] but a brief recapitulation may here be made. Class A contains those which may be regarded as of normal mental capacity. Class B contains those whose chief mental defect is impairment of memory; Class C, those who combine defective power of initiation and capacity for work, with slow comprehension and irritability; while Class D contains mainly epileptic dements.

Table G gives the frequency and percentage of neuropathic stigmata in the four mental classes.

[1] 'Med.-Chir. Trans.,' vol. lxxxvii, p. 351.

Mental state.	Single stigmata.	Combined stigmata.	None.	Percentages.	
				With.	Without.
A. . .	2 ...	0 ...	7 ...	2% ...	7%
B. . .	18 ...	1 ...	12 ...	19% ...	12%
C. . .	15 ...	11 ...	5 ...	26% ...	5%
D. . .	16 ...	11 ...	2 ...	27% ...	2%

From the above it is seen that in Class A there are only two cases in one hundred presenting stigmata of degeneration, while there are 7 per cent. without any; and in Class D there are 29 per cent. with stigmata, and only 2 per cent. without.

If groups A and B, which contain the slightest degrees of mental impairment be classified together, and also C and D containing the most marked forms of mental enfeeblement, it will be evident that the former contains an almost equal percentage of cases with and without stigmata (21 per cent. with, 19 per cent. without), while the latter contains 53 per cent. with, and only 7 per cent. without, stigmata.

From this it is apparent that there exists a very close relation between the presence of neuropathic stigmata and the degree of mental impairment; *the more pronounced the mental enfeeblement the more frequent the evidence of structural degeneration.*

This is one of the most important arguments in favour of the view that the interparoxysmal mental state in epilepsy is an integral part of the disease, and as such, is a prominent symptom of the malady.

It has been elsewhere argued that the mental condition is not a direct consequence of the fits; and although it may be to some extent influenced by co-existing factors, such as the character-combination, the frequency or the duration of the attacks, it is an expression of the same hereditary degenerative condition, which gives rise to the seizures.

That the underlying nervous state is a degenerative neurosis is shown by the presence in the majority of cases of confirmed epilepsy, of those stigmata which have been

generally regarded as structural evidence of a neuropathic constitution.

Summary and Conclusions.

The purpose of the communication was to ascertain, by means of a statistical investigation, whether the presence of structural stigmata of degeneration exercised any influence, and if so, to what extent, upon the prognosis of epilepsy.

Stigmata of degeneration are defined as structural deviations from the normal, arising during the periods of development and brain growth, in those who are the subjects of a hereditary degenerative predisposition.

One hundred consecutive cases of confirmed epilepsy from amongst the patients resident at the Colony for Epileptics, Chalfont St. Peter, were examined with this object.

The stigmata to which attention was especially directed were—(1) facial, including nasal deformity and asymmetry. This was found to be more common in females than in males. Although present in 42 per cent. of the total number of cases, it was only found in 14 per cent. as a single stigma; (2) deformities of the hard palate. These form some of the most frequent and trustworthy of degenerative phenomena. Abnormal palates were found in 42 per cent. of the cases, but only in 17 per cent. as a solitary stigma; (3) deformities of the external ears indicated by abnormal size, shape, and symmetry, were present in 33 per cent., but in only 9 per cent. as a solitary stigma; (4) dental anomalies and displacements.

Brief reference is also made to stammering and high errors of refraction as physiological stigmata.

The statistical facts bearing upon prognosis are considered under the following headings:

1. *Sex.*—Of 100 epileptics, 62 were males and 38 were

females. Males exhibit neuropathic stigmata to a much greater extent than females, in the proportion of 52 males to 23 females. Of the total number of cases, males and females, 75 per cent. presented stigmata, and 25 per cent. were free. It is pointed out that the greater freedom of females from stigmata is in relationship to the greater exemption from mental deterioration seen in the female sex.

2. *Hereditary history.*—Owing to the difficulty in obtaining precise and trustworthy information on this point it was not possible to deduce any general conclusions, but it was observed that direct parental heredity to epilepsy or insanity was usually associated with more pronounced stigmata than collateral heredity.

3. *Age at onset of the convulsions.*—Those cases in which the disease commenced between birth and five years of age showed a larger percentage of stigmata in comparison to those without any than at other ages. This is in harmony with what has been elsewhere shown, that epilepsy commencing during the first quinquennium is characterised by the highest percentage of cases showing profound mental impairment, and is most favourable for the development of the confirmed malady.

4. It was apparent from a study of the *duration of the convulsions* that the absence of stigmata did not necessarily imply an early or favourable termination of the disease.

5. *The type of the seizures.*—The greatest difference in the proportion of cases, with and without stigmata, was observed in those which presented the combined grand and petit mal type. Such combination has also been shown to be associated with the highest grades of dementia. The purely petit mal type did not exhibit marked evidence of a hereditary degenerative disposition. Cases of serial epilepsy do not show marked evidence of stigmata.

6. The relation between the presence of stigmata and *the mental state* was especially obvious. Those epileptics, who only showed the slighter degrees of mental impairment, presented a nearly equal proportion with and with-

out stigmata (21 per cent. with and 19 per cent. without). On the other hand, of those in whom there was marked mental enfeeblement, 53 per cent. exhibited and only 7 per cent. were free from them. Hence there would appear to be a close association between degrees of mental impairment and the presence of neuropathic stigmata, a fact which is proffered as an argument in favour of the view that the interparoxysmal mental condition in epilepsy is an integral part of the disease.

DISCUSSION.

Dr. C. HUBERT BOND pointed out that as regards the age-at-onset of the epilepsy there was a remarkable similitude between Dr. Turner's percentages and those based on an analysis of the cases at the Ewell Colony for Insane Epileptics, tending to confirm the speaker's view that any distinction between the sane and not insane epileptics is largely one of degree and artificial. In order to firmly establish any conclusions, he considered it to be highly important that the cases taken as a basis should be sufficiently numerous to admit of division into groups according to age-periods, and that comparisons should only be made between cases considered at similar ages; otherwise a variety of statistical fallacies become possible. He would also like to see in all tables dealing with epilepsy a sharp distinction made between cases in which there was congenital mental deficiency (or mental defect developing in early childhood) and cases free from such defect. He attaches great importance to the study of stigmata of degeneration in epilepsy, particularly to their presence in combination as opposed to the existence of one singly; he believed them, however, to have a more direct relation to the congenital mental defect than to the epilepsy, and thought that they might be used as a basis for distinguishing spurious from true mental defect. In that case their study should prove of immense utility when the public begin to press—as they almost inevitably will—for the segregation of these cases.

Mr. S. PAGET asked if the neuropathic degeneration with which the stigmata were associated related to epilepsy alone or to some more general neuropathic state. He could not understand the difference between ante-natal and post-natal causes in relation to stigmata. He could not see that there was any difference between the causes of such malformations and the ante-natal factors producing variations in stature. Facial asymmetry was practically universal; the late Lord Leighton had said that only some stupid society beauties had really symmetrical faces. The proportion of stigmata in the epileptic cases brought forward probably corresponded closely with their incidence in the London poor in general; they were the lot of the majority. He asked if there were one-sided deformities in regard to the ears.

Dr. G. E. SHUTTLEWORTH questioned if the existence of a single stigma formed a sufficient basis for prognosis; he thought not, while the coexistence of several stigmata might be so regarded. Evidence obtained from hatters showed that marked

asymmetry of head was especially prevalent among distinguished people. The percentage of developmental defects was almost twice as frequent in males as females, as Dr. Warner had shown in his statistics of school children, but when they did occur in females they were usually more pronounced and significant. Might not the mental enfeeblement noted by Dr. Aldren Turner in cases showing decided structural stigmata be regarded as essentially of congenital origin rather than a consequence of the epilepsy?

Dr. FLETCHER BEACH asked if there was much cranial deformity found in the cases of epilepsy, as, for instance, microcephaly and hydrocephalus, also the ratio of the deformed palate to the neurotic palate in such cases. He asked if webbed fingers had been found often in epilepsy; if asymmetry was found between the two sides of the body associated with paralysis, on which side did it occur?

Dr. TURNER, in reply, said he had not himself looked for stigmata amongst hospital out-patients suffering from other than nervous maladies, but he had been told that such were not common. Hare-lip and cleft palate, being due to arrest in the closure of a branchial cleft, could not be fitly compared with narrow or deformed palates, or with malformed ears. He had not observed webbed fingers in epileptics, but he has seen asymmetrical hands and limbs. He considered that single stigmata were of some clinical importance, although less valuable than combinations of stigmata. A study of the stigmata of degeneration would indicate that epileptics occupied a position midway between the insane and the imbecile.

AN ANALYSIS OF NINETY-NINE CASES OF INOPERABLE CARCINOMA OF THE BREAST TREATED BY OOPHORECTOMY

BY

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Eight years have now elapsed since Beatson published a paper in the 'Lancet' (1) advocating double oophorectomy combined with the administration of thyroid extract in the treatment of inoperable carcinoma of the breast.

His reasons for adopting this line of treatment were as follows:

Firstly, he knew that in cows lactation can be kept up permanently when once begun by "getting rid of ovarian influence," by spaying the cow after calving.

Secondly, he considered how the lactating breast resembled carcinoma in its earliest stages in proliferation of the alveolar epithelium, but differed from it in the fact that in the lactating breast the cells became vacuolated, broke down and formed milk, whereas in carcinoma the cells passed through the basement membrane of the alveoli and invaded the stroma.

Basing his line of argument on these facts, he hoped that removal of the ovaries might produce fatty degeneration of the malignant cells in carcinoma of the breast.

Great interest was aroused by his paper, and the brilliant immediate results in his two first patients, together with the marked benefit obtained at the hands of one or two others in similar cases, induced a considerable number of surgeons to give the new and original line of treatment a trial. Later reports of Beatson's cases, however, showed that though there was marked temporary improvement, the disease ultimately reappeared, and also a certain number of other patients who were treated on the same lines received absolutely no benefit from the operation; so that the interest has gradually waned, and at the present time, with a few exceptions, the prevailing opinion is that this line of treatment is hardly justified by its results, and consequently it is but seldom followed. The literature on the subject is not extensive; and apart from able papers by Stanley Boyd (2) and Alexis Thomson (3), and eight cases published by Dr. Herman (4), it is largely made up of accounts of isolated instances of the efficacy or otherwise of the treatment.

Many of the cases in the present series have already been published by Stanley Boyd and Alexis Thomson; but, thanks to the courtesy of several surgeons, I am able to give later details of a considerable number of the most successful ones, so that out of the twenty-three cases in Table A, in twenty cases the progress of the case from oophorectomy to the date of death or reappearance of the disease is recorded, and in one the condition of the patient, who is still alive and well; the two remaining patients were lost sight of, but the last note in the one case says the masses in the supra-clavicular region were increasing in size, and in the other that the growth was ulcerating. In Table B there are thirteen cases. In two of these six months after the operation there was no evidence of recrudescence and the patients were enjoying good health; of the remaining ten, seven were followed up to the date of

death, and in three the disease was again active when the last note was made.

Thus, this series of cases is of some importance as a means of estimating the value of the treatment under discussion, not only from the weight of numbers, but also from the fact that in nearly all cases where benefit was derived the subsequent history of the patient is given up to the time of her death or the reappearance of the disease.

For convenient reference I have followed the classification adopted by Alexis Thomson. In Table A very material benefit was derived and the patient lived for more than a year after the operation. In Table B the benefit was definite, but not so great as in Table A. In Table C there was little or no improvement in the general or local condition.

Some of the cases in Table A are worthy of special attention, Nos. 3, 5, 7, 9, and 12 in particular. It is interesting to note that in No. 4 all obvious signs of the disease had so completely disappeared twelve months after oophorectomy, that on examining the patient at that time one surgeon inclined to the belief that she had not suffered from carcinoma at all. Microscopical examination, however, and subsequent reappearance of the growth proved the original diagnosis to have been correct.

The fact that the catamenia have ceased does not appear to contra-indicate operation, though better results are obtained if the patient is still regular.

In Table A three patients had ceased to menstruate—viz. Nos. 2, 15, and 20. No. 7 had been irregular for three years, had seen nothing for three months, and her age was 47, so that it may fairly be assumed that she had reached the menopause. In her case oophorectomy was performed for extensive carcinoma in the neck and axilla, with the remarkable result that the lumps disappeared and she was free from obvious disease for four years and a half.

In Table B menstruation had ceased in five cases: Nos. 24, 25, 27, 32, and 34.

In Table C menstruation is said to have ceased in eight cases; in nine others, who were 50 years old or more, no reference is made to the catamenia, but as the average age at which menstruation ceases is 47 years and 8 months, according to Leith Napier (5), it is highly probable that they had reached the menopause. Of the other cases where information is lacking on the point, one patient was 48, but she died on the third day, and therefore can be neglected. Two others were 44, and the remainder 42 or less.

Thus, out of a total of twenty-six cases operated upon after the menopause there was improvement in nine cases, or 34·6 per cent.; in four of these, 15·4 per cent., the benefit was very definite. These figures compare well with the percentage of results in all cases here reported.

Percentage in the Different Tables.

	No. of cases.	Table A.	Tables A & B.	Table C.
Menses ceased	26	15·4	34·6	65·4
All cases	99	23·2	36·4	63·6

From this table it will be seen that though the percentage of menopause cases in Table A is below the average of all cases, the total percentage of cases in Tables A and B is roughly the same, whether all cases are taken or only those in which the menses have ceased.

Although the numbers are small, it may be concluded that the menopause does not contra-indicate oophorectomy.

There was a good deal of discussion at one time as to whether oophorectomy should be combined with the subsequent administration of thyroid extract or not. Beatson gave it in his cases, but was of the opinion that it had little effect when given alone. Cheyne and Burghard (6) say "any good that follows oophorectomy and administration of thyroid extract seems to us to be attributable to the operation alone." In a few cases of carcinoma, im-

provement, coincident with the administration of thyroid extract without previous oophorectomy, has been reported.

Page and Bishop (7) record the case of a woman, aged 61, suffering from recurrent nodules of carcinoma, who was treated by thyroid extract alone. She had previously been operated upon twice, once for removal of the breast, and later for removal of recurrent nodules. After taking the extract for eighteen months, she was quite free from obvious carcinoma; during these eighteen months, however, at a time of anxiety and depression of general health, a growth developed, grew to the size of a walnut, and then disappeared.

Sheild (8), in a case of recurrent nodules in the skin of the chest and abdomen, with enlarged glands in the neck five months after removal of the breast for carcinoma, mentions the fact that the patient was alive two months later, and "getting great relief from thyroid extract."

Beaver (9) records a case of inoperable carcinoma of the uterus with a large fixed mass in the pelvis. Thyroid extract was given. In two months the mass was much smaller, and the patient was free from pain; in eleven months nothing abnormal could be felt in the pelvis.

Bell (10) claims a good result from the exhibition of thyroid extract in three cases of carcinoma of the cervix.

Treatment in the first case was complicated by local application of fuming nitric acid, and later by administration of elixir of mammary gland.

In the second case the growth was curetted and zinc chloride applied. Eight months later this patient was free from pain, and there was no trace of disease.

The third case was treated by ichthyol tampons and thyroid for two months; all trace of malignancy is said to have disappeared at the end of that time.

With regard to the above cases of carcinoma of the uterus, their value would, of course, be increased had a microscopical examination been made, though there can be little doubt that the diagnosis was correct.

Page and Bishop's case is very interesting, but the mere fact that during the treatment a fresh nodule appeared and remained for more than nine months suggests that it may possibly have been one of the cases of spontaneous disappearance of carcinoma that are met with from time to time, although it is just conceivable that the reappearance of the growth was due to the temporary depression of the general health of the patient, with possibly omission of the thyroid extract for a time, as Page and Bishop suggest.

Out of the 99 cases collected here, thyroid is said to have been given in 43 and withheld in 26. Of the cases appearing in Table A, thyroid was given in 15 cases and withheld in 7; in Table B 6 cases were given thyroid and 6 were not.

Percentage in the Different Tables.

	No. of Cases.	Table A.	Tables A and B.	Table C.
Thyroid	43	34·9	49	51
No thyroid	26	26·9	50	50

To sum up, firstly, on comparing the distribution of cases treated with and without the extract in the different tables, we find that the percentage of thyroid cases in Table A is somewhat larger than the percentage of non-thyroid cases; secondly, from a general consideration of Table A the benefit is of longer duration in thyroid than in non-thyroid cases, though No. 9 is a notable exception; thirdly, cases of carcinoma in various parts of the body have been recorded which improved very markedly while the extract was being administered.

From these facts it would appear that thyroid extract is not a necessary factor in the treatment, though evidence is slightly in favour of its administration.

It seems to be impossible at present to say which patients will be benefited by the operation, but from the examples here given deductions can be made which will be of some help in forming a prognosis.

Secondary deposits in the viscera contra-indicate the

operation. With the exception of Nos. 2 and 11 in Table A, no improvement has been found when there have been secondary deposits within the thorax or abdomen.

The left breast was removed from Edmunds' patient (11) (No. 2) five years previously, and at the time of oophorectomy she had carcinoma of the right breast, enlarged glands in the axilla, etc., and definite evidence of intra-thoracic growth, viz. dyspnœa on exertion, and frequent asthmatic attacks, evident stridor, and diminished vocal resonance over the left base; the chest otherwise was fairly resonant. A year later the tumour in the breast and axillary glands had almost disappeared, there was no longer any stridor, and the dyspnœa, which caused great distress, had nearly gone. Nine months later, however, the tumour in the breast had increased and the dyspnœa was worse. When the post mortem (12) was made, a mass of scirrhous carcinoma was found at the bifurcation of the trachea invading the roots of the lungs; there was also recent infiltration of the lumbar glands. In this case the fact that there was intra-thoracic growth at the time of oophorectomy cannot be doubted, nor can the fact that after the operation there was very marked and definite diminution in the size of the growth in the breast and axillary glands, and presumably in the size of the intra-thoracic growth, evidenced by the disappearance of the stridor, and the great decrease in the dyspnœa for eighteen months.

In D'Arcy Power's (13) case, secondary deposits were found in the ovaries when they were removed, but, in spite of this, the condition of the patient was much improved; this case therefore is an exception to the general rule. There was no improvement in seven other patients who were known to have secondary deposits within the thorax or abdomen at the time of operation. In two there were deposits in the liver, in two there were deposits in the omentum and peritoneum, in two the pleura was involved, and in one there was a nodule of carcinoma in one ovary.

Edmunds' case is of particular interest, for though there was intra-thoracic growth, it was not an example of a secondary deposit in the lungs, but of involvement of the bronchial glands, the growth, in all probability, extending from the breast *viâ* the lymphatics; this is confirmed by the character of the symptoms and physical signs before oophorectomy, and by the result of the post-mortem examination. Although this is a solitary case, it is sufficiently striking to afford a hope of relief from oophorectomy in inoperable carcinoma of the breast, even though there is evidence of intra-thoracic growth, if the clinical signs suggest involvement of the lymphatic glands rather than secondary deposits in the lungs; definite evidence of secondary deposits in the lungs contra-indicates the operation.

As in the case of the "complete operation" for carcinoma of the breast, the smaller and more localised the growth, the brighter the prospect for the patient; the larger the tumour, the longer it takes to go. A more favourable result is to be anticipated in slowly than in rapidly growing carcinomata, and an early recurrence after removal of the primary growth is unfavourable.

In the early days of this operation it was hoped that it would be especially effective in the case of young subjects; this, however, has not proved to be the case, and at the present time it is generally held that the age at which most benefit is likely to result is when the patient is approaching the menopause.

The following table includes all cases in the series with the exception of one in Table A and of one in Table C whose ages are not given. The cases have been grouped according to their ages, and the percentage in each table recorded. Thus, thirteen cases were under thirty-five, four, or 30·8 per cent., appear in Table A, five, or 38·5 per cent., in Tables A and B taken together, and eight, or 61·5 per cent., in Table C.

Percentage in Different Tables.

	No. of Cases.	Table A.	Tables A and B.	Table C.
Under 35	13	30·8	38·5	61·5
35 to 40	16	31·25	43·75	56·25
40 to 45	30	23·3	36·6	63·3
45 to 50	16	37·5	50	50
Over 50	19	None	21·1	78·9

On looking at this table one is immediately struck by the very unsatisfactory results of the operation on patients over 50. Of the nineteen cases, fifteen, or 78·9 per cent., are placed in Table C, which either means that they showed no improvement at all after the operation or that it was so slight as to be of no value to the patient; none of them improved sufficiently to be placed in Table A. On the other hand, considerably better results have been obtained between 45 and 50 than during any other five years; 37·5 per cent. of the cases appear in Table A, and 50 per cent.—that is to say, one half of the cases—were distinctly benefited by the operation.

From these figures we may conclude that the most favourable age is between 45 and 50—that is to say, shortly before, during, or shortly after the menopause; that after 50 it is hardly worth while to perform the operation, and that there is certainly evidence in favour of a further trial in patients who are relatively young.

The site of recurrence, whether it be in the breast, axillary, or supra-clavicular glands, or take the form of nodules in the skin, does not seem to affect the prognosis. Local changes are first noticed from seven to ten days after the operation, the growths becoming smaller and softer. The length of time they take to entirely disappear varies, but depends to some extent on their size; the smaller the growth the more rapidly does it go. In Case 17 the recurrent nodules round the scar of the previous amputation of the breast disappeared in one month; a small inoperable axillary growth (Case 23) went in the same time; in three cases the time taken was three months, and in two others six and eight months

respectively. In case 9 oophorectomy was performed for extensive infiltration of the skin and fat of the chest wall; "in five months the disease had disappeared, except a mass in the pectoral, which went by fits and starts, with long stationary intervals between, in three years."

In four cases special mention is made of pain in the breast two to four days after the operation. Two of these were under the care of Mr. D'Arcy Power (14). Case 11 had a great deal of pain in both breasts. Case 90 had pain in the affected breast for two weeks from the second day after removal of the ovaries. Case 47 complained four days after operation that the breast and arm hurt her more than ever. Case 34 had had pain for some time, but four days after operation a note was made that she was complaining of pain in the breast and axilla; five days later, however, it was distinctly less.

The mortality of the operation is certainly higher than is generally anticipated, though Treves and Hutchinson (16) say that the operation is occasionally followed by considerable shock, and should only be resorted to when the patient's age is favourable to the chance of benefit resulting from it.

Of the ninety-nine cases tabulated here six died soon after the operation. Nos. 87 and 88 died from exhaustion; No. 86 died on the third day; Nos. 47 and 62 died suddenly when all was apparently going well, three and three and a half weeks after the operation respectively; No. 96 became maniacal and died in two weeks.

At first sight this mortality of 6 per cent. is discouraging, and makes one hesitate to advise an operation which is of real benefit in only 36 per cent. of cases, but a closer investigation of the various causes of death is somewhat reassuring.

No. 88 was obviously in a very bad condition before operation, and at the present time, in the light of our more extended knowledge of the subject, such a patient, very anæmic and cachectic, with extensive local recurrences and deposits in the liver, would not be operated upon.

Nos. 47 and 62 probably died from pulmonary embolism. The former did very well after the operation, but died suddenly at home five days after her discharge from the hospital; the latter died suddenly from dyspnœa.

Three cases, Nos. 18, 58, and 96, raise a point that afforded a good deal of discussion at one time—whether removal of the ovaries is a cause of insanity or not. In a considerable number of cases insanity has occurred after removal of the ovaries, but this is by no means the only operation with this unfortunate sequel. Gastro-enterostomy, radical cure of hernia, excision of the head of the humerus, and many others have been followed by insanity. Kelly (15), from an analysis of a series of cases of insanity following gynæcological operations, says: "Insanity is more frequent after simple than after grave operations. The removal of ovaries and tubes, and with their removal the ablation of their functions, does not appear to stand in any causative relation." The previous mental history of the patient and the hereditary tendency must also be taken into consideration; if unfavourable, they of course predispose to post-operative insanity. The following case may be mentioned as one out of many. A woman was recently operated upon by Dr. Russell Andrews for double ovarian cyst. She had been in Bethlem twice and consequently post-operative insanity was anticipated, but the indications for operation were imperative. Physically she made an excellent recovery, but mania developed seven days after the operation.

The benefit received from oophorectomy has been great in a considerable number of cases, and some of the results are very remarkable. The most important features are the improved health, increase in weight, alleviation of pain, healing of ulcers, temporary diminution in size or even disappearance of the growth, and prolongation of life.

Temporary improvement occurred in some cases in Table C, but was not great enough to allow them to be placed in Tables A or B.

The increase in weight, when noted, varies from seven pounds to two stone seven and a half pounds. Alleviation and ultimate cessation of pain is a striking feature, while in most cases an allusion is made to the improved health of the patient. The average duration of life after oophorectomy in Table A—that is, in 23 per cent. of the cases—is 35·2 months; and, when it is remembered that, with possibly one exception, these patients were suffering from carcinoma which was too far advanced for removal, it must be admitted that the prolongation of life was real and considerable.

It has been stated that the improvement lasts as a rule from six to twelve months, but it is often of longer duration. Information on this point is, unfortunately, lacking in many cases; but in Table A one case was benefited for six months, eight for varying periods from twelve to eighteen months, three for twenty, twenty-four, and thirty months respectively, while four enjoyed good health for four and a half years or more.

In Table B one case was benefited for seven months, another for nine months, and two for six months. I have, unfortunately, been unable to obtain further details of the two last cases, but at the end of six months one patient was in excellent health, and in the other the local condition was improving.

In three patients a fixed, inoperable growth became much softer and smaller after oophorectomy, and was subsequently removed.

In No. 10, Table A, the left breast was transformed into a hard, fixed mass that extended to the axilla; after oophorectomy the breast became smaller and more movable, and the axillary mass disappeared. The breast, pectoralis major, and axillary glands, were removed in November, 1900. Recurrent nodules near the scar were excised in April, 1901. Later still masses appeared in the supra-clavicular region. Dr. Donald tells me that she was relieved for about two years and then lost sight of.

In the second case, No. 19, Table A, there was extensive

ulceration, and the mass was fixed to the pectorals. Oophorectomy was performed, the ulcer healed, the growth became smaller and movable, and the complete operation was performed four months after removal of the ovaries. This patient lived for sixteen months after oophorectomy, and died from intra-thoracic growth.

In the third case, No. 27, Table B, the ovaries were removed in January, 1902, for inoperable cancer of the breast with ulceration. The complete operation was performed in March, recurrent nodules appeared round the scar in April, and the patient died in September in the same year.

These three cases are instructive, though few in number, and point to the advisability of removing the growth when it becomes operable; though it is as well to wait until the improvement resulting from oophorectomy ceases to be definite; for the smaller the growth at the time of removal the greater the probability of freedom from local recurrence, and the greater the amount of apparently healthy tissue that can be removed with the remaining growth.

It is exceedingly important that the growth should be excised very freely: both pectoral muscles should be removed, with as much skin as possible, having regard to the situation and extent of the disease at the time of oophorectomy. The edges of the wound should be approximated as far as possible in all cases, if necessary by means of undercutting or sliding flaps, methods to be preferred to skin-grafting as being less conducive to ulceration in case of local recurrences. It goes without saying that the axilla should be thoroughly cleared out whether the glands are palpable or not; and further, if at any time hard or enlarged glands have been felt above the clavicle, the supra-clavicular triangle should be cleared of fat and lymphatic tissue. It is now generally recognised, thanks to the work of Halsted and others, that in cases of carcinoma of the breast absence of local recurrence is to a great extent directly dependent on very free removal of

the disease at the first operation, so that now "in cases really favourable for operation" recurrence in the area of the operation wound—that is to say, in the breast or the axilla—is quite a rarity (17), whereas a short time ago it was relatively common.

An additional reason for free removal is that when the disease has completely disappeared after oophorectomy it has reappeared in the same situation in a large number of cases. In the event, therefore, of an inoperable growth becoming smaller and sufficiently movable for excision, although clinically the surrounding structures are quite free from disease, it is probable that a certain number of cancer-cells still remain in the immediate neighbourhood of the obvious growth, in the tissues which were at one time definitely involved. In this connection it may be noted that in two of the above cases recurrence took place in the neighbourhood of the scar, although the growth was freely excised.

Removal of the breast and ovaries together, or with an interval of a few days between the operations, has been performed in six cases.

In No. 21, Table A, there was extensive carcinoma of the left breast; it was adherent to the skin and deep structures, though not absolutely fixed; there were small, hard, painless glands in the axilla and supra-clavicular regions. The breast and ovaries were removed, but the glands were not touched. Two and a half years later she was in excellent health, there was no recurrence in the scar, but there was one very small hard gland above the clavicle. Two or three small glands could be felt in the axilla, but they were soft and not particularly suggestive of carcinoma. Ten months later, however, there was a small nodule in the scar and the supra-clavicular glands were considerably enlarged; there was no alteration in the axillary condition.

In Nos. 49 and 61 removal of the ovaries possibly prevented local recurrence; in No. 54 it probably had no effect; in Nos. 39 and 83 the result was doubtful. This

method of procedure may be adopted when it is possible to excise the growth in the breast, but the axillary or supra-clavicular glands are too extensively involved to admit of removal. At a later date should the axillary and supra-clavicular glands diminish in size, and become operable, they may be removed as soon as the improvement ceases.

The explanation of the changes that have followed oophorectomy in the successful cases is still unknown. Mr. Stanley Boyd suggests that the solution of the problem lies in an internal secretion of the ovary which varies from time to time, thus accounting for the varying success of the operation. I would go still further, and suggest that a morbid variation of the secretion of the ovary may be one of the predisposing causes of cancer of the breast, and that when the malignant changes have once begun, their further development may be favoured by this secretion.[1] If the ovaries are removed the malign influence exerted by them is also removed, and, provided that the new growth has not been disseminated by the blood and obtained too strong a hold of the patient, the removal of this influence gives the patient an opportunity of retaliation, with the result that the growth becomes smaller and may even disappear. But as the secretion of the ovary is not the cause of carcinoma of the breast, but only predisposes to its commencement and favours its course, the removal of the ovary will not cure the disease, but will only remove one of the factors making for its progress, and hence the fact that the improvement as a rule is temporary. What the nature of this secretion is, and how its effect is produced, we do not know; nor do we recognise any clinical evidence which will tell us when it is at work, so that at present it is not possible to

[1] Since writing the above I find that I have overlooked a paper by Mr. Stanley Boyd, in the 'British Medical Journal' of February 4th, 1899, in which he says, "Such evidence as we have tends to show that in cancer the ovary is not an exciting cause, but only a predisposing influence, of greater or less strength in different cases."

forecast the effect oophorectomy will produce on the growth in any given case. Carcinoma, as is well known, not infrequently develops in a breast which is already the seat of chronic mastitis, and it may be that there is a connection between the secretion of the ovary, one or more of the varieties of chronic mastitis, and mammary carcinoma.

That oophorectomy does not cause the disappearance of the growth by diminishing its blood supply is obvious; since the same changes apparently take place whether the disease presents as a growth in a breast that has never been operated upon, as deposits in the axillary or supraclavicular glands, or as recurrent nodules in the skin; so that the changes which follow removal of the ovaries must be the result of some influence acting or ceasing to act upon the mammary epithelium. This influence is probably of the nature of an internal secretion.

In a recent paper Dr. Russell Andrews (18) has given an excellent summary of our present knowledge of the internal secretion of the ovary.

That there is a close relationship between ovaries and breasts there can be no doubt; the development of the breasts at puberty, their atrophy at the menopause, the fact that if the ovaries of a guinea-pig are removed soon after birth the breasts do not develop, and the fact that in cows lactation when once begun can be prolonged indefinitely by removing the ovaries, are all important as showing the relationship. Further, the most favourable age for oophorectomy in inoperable cancer of the breast is between 45 and 50—that is to say, about the time of the menopause—and it is not difficult to suppose that normally there are variations in the ovarian secretion at this time.

With regard to the connection between the ovaries and carcinoma of the breast, it is of great interest that in chorioepithelioma an excessive production of lutein cells in the ovaries has been described, though, of course, this analogy must not be strained.

I have only been able to find one account of the microscopical appearances of a growth which was becoming smaller after oophorectomy. Dr. R. M. Buchanan examined a nodule from Dr. Beatson's second case and reported a great increase in the stroma, and that the epithelial cells were undergoing marked fatty degeneration (19).

To come to the main point at issue, is the operation worth doing or not? If all patients more than 50 years old are omitted, there are seventy-five left; of these twenty-two, or 29·3 per cent., are in Table A, thirty-one, or 41·3 per cent., in Tables A and B, and forty-four, or 58·7 per cent., in Table C; that is to say, that 41·3 per cent. of all the cases under fifty years of age which have been recorded were distinctly benefited. The different ways in which the improvement is manifested have already been alluded to in sufficient detail.

What are the disadvantages? Firstly, an operation which will keep the patient in bed for two or three weeks; secondly, removal of the ovaries and consequent loss of certain functions connected with them; thirdly, the unpleasant symptoms sometimes associated with the artificial menopause may supervene; and fourthly, the operation may not affect the disease. As Dr. Herman has suggested, the best way is to lay the facts before the patient and let her decide whether she will undergo the operation or not; if the surgeon is asked for his opinion, he should advise in favour of the operation. It may have no effect, but 41·3 per cent. of cases under 50 have improved considerably after the operation.

The danger of the operation is slight, and with regard to the other disadvantages they can have but little weight when the probable duration of life and condition of a patient suffering from inoperable carcinoma of the breast are taken into consideration; besides, there is a possibility, though a remote one, that the patient's life may be prolonged for several years by the operation.

The patient should be warned that the operation offers a prospect of temporary relief only, not of a cure.

Although No. 3 has been free from all signs of carcinoma for five years, it is too soon to consider her cured, for No. 9 had excellent health for seven years before there was any evidence of recurrence. At the end of 1903, however, she began to suffer from a steadily increasing dyspnœa and last March some blood-stained fluid was drawn off from the chest; this gave her great relief, and she has had less dyspnœa ever since, but it is very highly probable that there are secondary growths in the lungs.

I am deeply indebted to the staff of the London Hospital for permission to use their cases, and to Messrs. Stanley Boyd, Watson Cheyne, Donald, Edge, Edmunds, McGavin, D'Arcy Power, Alexis Thomson and Waterhouse for very kindly giving me later details of cases already published.

Summary.

1. Of ninety-nine cases of inoperable carcinoma mammæ treated by oophorectomy there was a very marked improvement in 23·2 per cent., and distinct, though less marked, improvement in thirteen other cases; that is, 36·4 per cent. of all cases operated upon were materially benefited by the operation. If the patients who were more than 50 years old are omitted, of the remaining seventy-five cases 29·3 per cent. showed very marked improvement, and nine others showed distinct improvement; that is, 41·3 per cent. were benefited by the operation.

2. In successful cases the benefit has been great, and is mainly shown in relief from pain, marked improvement in health, diminution or even disappearance of the growth, healing of ulcers, and prolongation of life.

3. The duration of the improvement is not very often stated, but in fifteen cases the improved condition of the patient was maintained for more than twelve months, and four other patients had good health for four and a quarter years or more.

4. Oophorectomy does not cure the disease, for in all the cases in which the growth has disappeared after the operation it has subsequently reappeared, locally or elsewhere, with the exception of one patient, who is alive and free from recognisable cancer at the present time, five years after oophorectomy.

5. The most favourable age for operation is from 45 to 50; in relatively young patients it should be given a further trial, but after 50 it is rarely worth doing. The fact that the patient has passed the menopause does not contra-indicate the operation.

6. Thyroid extract is not a necessary factor in the treatment, though the results have been slightly better when it has been given.

7. Secondary growths in the viscera contra-indicate the operation; rapidity of growth, or an early recurrence after the primary operation, makes the prognosis unfavourable.

8. The mortality in this series of cases is high—a little over 6 per cent. It should be noted, however, that the actual cause of death in several of these cases may be regarded as accidental; in two the fatal issue was due to pulmonary embolism, and in one to acute mania.

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TABLE OF CASES.

TABLE A.

No.	Surgeon.	Age.	Menses.	Previous operations.	Nature of disease for which operation was performed.	Date of oophorectomy.
1	Eddowes	48	Regular	Breast removed September, 1898	Involvement of skin and axilla	June, 1899
2	Walter Edmunds	36	Ceased October, 1899	Left breast and glands removed 1894; recurrence near the scar excised fifteen months later; one or two small recurrences excised subsequently	Carcinoma right breast and axillary glands; stridor; dyspnœa; evidence of intra-thoracic growth	March 11th, 1901
3	Waterhouse	37	Regular	Complete operation May, 1897	Mass behind pectoral and above clavicle, swelling of arm	August, 1899
4	Edge	36	Regular	Left breast removed June 13th, 1900; glands removed from axilla October 17th, 1900	Enlarged glands in both axillæ and left supra-clavicular region, also lumps in the right breast and right axilla	March, 1901
5	Watson Cheyne	48	Regular	No notes	Recurrence in cervical glands and swelling of arm	July, 1899
6	Watson Cheyne	?	Excessive large fibroid	Several operations previously	Extensive local recurrence	May, 1899; incomplete removal of recurrence
7	Herman	47	Irregular for three years; nil for three months	Breast removed October, 1890	Extensive recurrence in neck and axilla	March, 1897

TABLE A.

Thyroid.	Result.	Duration of life after oophorectomy.	Remarks.
Given steadily	Disappearance of disease and marked gain in health	32 months, +	Extensive recurrence February, 1902.
Given	Nine weeks after operation stridor had disappeared. One year after operation nearly free from dyspnœa, and had put on flesh. There was a small tumour in the right breast, which patient said had disappeared completely at one time. No enlarged glands in axilla. Death, April 4th, 1903	25 months	Twenty-one months after oophorectomy the dyspnœa was worse. Growth in the breast growing slightly and commencing to ulcerate. There were also some glands in the axilla. Removal of breast proposed three months later, but patient died under chloroform before the operation was commenced. P.M.—Some hard scirrhus found at the roots of the lungs. Decidedly benefited for eighteen months.
Given four months	Disappearance of swellings; free from any trace of recurrence July, 1904	59 months, still alive	The swellings completely disappeared within four months of oophorectomy.
None	December 18th, gained over two stone in weight, better than for years. No pain, though it was severe before oophorectomy. March 25th, 1902, no lumps to be felt	20 months	In the summer, 1902, she began to go downhill, and died October 25th, 1902. Hard masses in both axillæ. Carcinoma of the liver, etc. Very great and marked improvement for at least one year.
3 months	Gradual disappearance of the glands. In excellent health until autumn, 1903	67 months	In autumn, 1903, supra-clavicular glands began to enlarge. Died from progress of disease February 28th, 1905.
Given steadily	All signs of cancer disappeared in three months. Fourteen months after operation in better health than for some time past. Fibroid almost gone. Thirty months after operation in good health, but there were fresh nodules on the chest. Died in May, 1902	36 months	—
Given steadily	Six months later no growth could be felt. Had gained eleven pounds in weight. December, 1901, free from recognisable cancer. Died June 3rd, 1903, from deposits in viscera	74 months	Four and a half years' good health.

The sign + signifies that the patient was alive when the last note was made.

TABLE A—

No.	Surgeon.	Age.	Menses.	Previous operations.	Nature of disease for which operation was performed.	Date of oophorectomy.
8	Eve	43	Regular	Complete operation 1895	Three small recurrent nodules, one just above the scar, and two in the scar itself; supra-clavicular glands enlarged; right arm swollen one month	July 26th, 1898
9	S. Boyd	44	Regular	Excision of tumour September, 1892 breast removed June, 1893; attempted removal of recurrence June, 1896	Large infiltration of skin and fat of chest wall	December, 1896
10	Donald	46	Regular	None	Left breast transformed into a hard mass fixed to the chest wall and skin; the growth extended to the axilla	—
11	D'Arcy Power	42	Regular	None	Carcinoma of right breast fixed to deep structures; secondary deposits in skin and axillary and supra-clavicular glands; suspicious lump in left breast	April 24th, 1902; ovaries cystic and contained nodules of spheroidal-celled carcinoma
12	Beatson	33	Regular	Complete operation January 25th, 1895	Multiple recurrent nodules along scar and into axilla	June, 1895

continued.

Thyroid.	Result.	Duration of life after oophorectomy.	Remarks.
12 months	Recurrence in scar and gland disappeared in three months. Recurrence in scar and supra-clavicular removed November 2nd, 1899. Extensive growth in skin and chest March, 1902. Died June 2nd, 1902	47 months	—
Not given	In five months the disease had disappeared except a mass in the pectoral, which went by fits and starts with long stationary periods between in three years. Summer of 1903 quite free from obvious disease	100 months, still alive April, 1905	She suffered from gradually increasing dyspnœa during the winter of 1903. In March, 1904, some blood-stained fluid was withdrawn from the chest; since then her general condition has improved, and there has been less dyspnœa.
Given	November 12th, 1900, mass in breast much smaller. Axillary mass could not be felt. Removal of breast, pectoralis major and axillary glands. Recurrent nodules near the scar were excised in April, 1901; later masses appeared in the supra-clavicular region. Last note at the end of 1901 was that the masses were increasing in size, but she was better in health than before the operation	2 years, +	Relieved for about two years, then lost sight of.
Not given	No pain when discharged, one month after operation. Had gained 7 lbs. in weight. August 28th: left breast free from cancer. Right breast much softer and more movable. Glands in axilla imperceptible, but there was pain and swelling in the right arm down to the internal condyle. June, 1903: all symptoms improved; weighed 1 st. 2½ lbs. more than at date of oophorectomy. Cancer remaining in abeyance. She was paralysed for six weeks before her death, which took place October 30th, 1903	18 months	After the operation had a great deal of pain in both breasts.
Given steadily	Disappearance of nodules within eight months	46 months	Fresh nodules. Disease of spine. Died April, 1899.

TABLE A—

No.	Surgeon.	Age.	Menses.	Previous operations.	Nature of disease for which operation was performed.	Date of oophorectomy.
13	Stanley Boyd	37	Regular	Complete operation March, 1896	Multiple nodules on chest-wall	February, 1897
14	Stanley Boyd	31	Regular	Complete operation May, 1898	Nodules on chest-wall, glands above clavicle	August, 1900
15	Herman	49	Ceased	Right breast, 1895	Ulcerating recurrence in scar; large mass in the left breast and enlarged glands in left axilla	July, 1898
16	Herman	44	Regular	Breast removed June, 1895	Nodules in scar; glands above clavicle, etc.	May, 1899
17	Herman	43	Regular	Breast removed August, 1897; recurrence removed April, 1898	Nodules around scar	June, 1899
18	Hopkins Walters	41	Regular	Complete operation November, 1890	Nodules in scar with extensive ulcer, etc.	October, 1896
19	J. W. Smith	43	Regular	—	Extensive ulceration; mass infiltrating pectorals	July, 1899
20	Herman	46	Ceased	—	Inoperable breast, nodules in skin; pain in the breast one month	June 22nd, 1900
21	P. Furnivall	32	Regular	—	Extensive carcinoma left breast, adherent to skin and deeper structures, not absolutely fixed; small, hard, painless glands in left axilla and supra-clavicular region	May 16th, 1901 Removal of breast at the same time. Axillary glands not removed

continued.

Thyroid.	Result.	Duration of life after oophorectomy.	Remarks.
Given one month.	In six months most of nodules disappeared	17 months	Local recurrence. Growths in viscera. Died July, 1898.
—	Rapid disappearance of recurrence. Marked gain in strength and weight	14 months	Relapse of growth. Cancer of liver. Died October, 1901.
Given steadily	Ulcer healed, and there was no distinct lump in the left breast three months after operation. Ten months after operation had gained 2 st. 7½ lbs. Eighteen months' good health	29 months	Recurrence of ulceration 18 months after operation. Died December, 1900.
Given steadily	Shrinkage of nodules in ten days. Gained 17 lbs. in weight. Eighteen months' good health. Nodules appeared in axilla four months after operation	26 months	Paralysis of leg. Died December, 1901.
Given steadily	Disappearance of nodules in a month. Cessation of pain. Gained 12 lbs. in weight in nine months. Twelve months' good health. Recurrent nodules nine months after operation	23 months	Growths in stomach and liver. Died May, 1901.
None	Healing of ulcer. Shrinkage of nodules. Gain in weight. Three weeks' dementia after oophorectomy	16 months	Intra-thoracic growths. Died February, 1898.
None	Healing of ulcer. Shrinkage of growth, which became movable. Complete operation November, 1899	16 months	Secondary growths in pleura. Died October, 1900.
Given two weeks	Five months after operation there was a small lump only, adherent to the chest-wall, which could be covered by a sixpence; a small area of skin was red and adherent to it. She gained 1 st. in weight in two months. Eighteen months after operation condition the same	24 months, +	Ulceration of the growth. Patient lost sight of 1902.
Not given	December 2nd, 1903: two or three small glands in left axilla; very small hard gland in left supra-clavicular region, no pain, and no recurrence in scar. September 28th, 1904: small nodule in the scar; considerable increase in size in the gland in left supra-clavicular region	40 months, still alive	—

TABLE A—

No.	Surgeon.	Age.	Menses.	Previous operations.	Nature of disease for which operation was performed.	Date of oophorectomy.
22	Watson Cheyne	34	Regular	—	Nodules in the skin and a mass in the axilla	October, 1896
23	S. Boyd	39	Regular	Breast and glands removed October, 1895; two subcutaneous nodules removed from chest-wall May, 1898	Small inoperable axillary growth	September, 1899

TABLE B.

24	Abbe	70	Ceased	Breast removed 1899	Ulceration and multiple nodules	April, 1901
25	Abbe	70	Ceased	—	Inoperable disease of breast and glands	May, 1901
26	Abbe	42	Regular	Complete operation May, 1900	Nodules in skin; pleura involved; glands	March, 1901
27	Abbe	70	Ceased	Breast removed September, 1899	Recurrence in scar and glands	May, 1901
28	Annandale	39	Regular	—	Inoperable cancer of breast and ulcer	January, 1902
29	J. M. Cotterill	27	Regular	Breast removed June, 1900	Multiple nodules in skin; cancer in other breast and glands	July, 1901
30	Alexis Thomson	36	Regular	Breast removed July, 1901	—	Attempted removal of supra-clavicular glands and oophorectomy; November, 1901
31	Pearce Gould	41	Regular	None	Extensive disease of breast glands and nodules in skin	January, 1900

continued.

Thyroid.	Result.	Duration of life after oophorectomy.	Remarks.
Given	Diminution in the growths for six months. Seventeen months after operation she was alive, but there were fresh nodules and ulceration. Believed to have died a few months afterwards	? 20 months	—
Not given	Complete disappearance in 1 month. Recurrence of nodules March, 1900. Death Sept., 1902	36 months	She had only a few recurrences on the chest-wall. Ultimately suffered great pain, apparently from infiltration about the brachial nerves. Finally she had effusion into the pleural cavity. She died worn out chiefly by pain and loss of sleep. She had bad heart disease.
TABLE B.			
None	Improved general health. Ulcer healed. 3½ months after oophorectomy nodules very slowly wasting in thickness, ulcer remained healed	7 months, +	Fresh nodules November, 1901.
None	Diminution in size of growths. Improved health	6 months, +	In excellent health November, 1901.
Given	All signs of cancer disappeared in 8 weeks, apart from the fluid in the chest; in 4 months the fluid was diminishing and the patient appeared to be perfectly well	8 months, +	Loss of weight; extension in pleura.
None	Diminution of growths	6 months, +	Gained in weight. Local disease improving November, 1901.
Given steadily	Improved general health. Complete operation, March, 1902	8 months	Recurrence in nodules round cicatrix April 15th, 1902. Death September, 1902.
Given	Partial disappearance of nodules	12 months	Generalised carcinoma July, 1902.
Given	In improved health August, 1902, but at the end of September the disease was assuming a more acute form. There was swelling of the face and neck, and acute pain. Died at the end of December, 1902	13 months, +	Operation certainly prolonged her life, but there was great suffering.
Given steadily	Marked diminution in severity of local disease	3 months	Died April, 1900, intestinal obstruction apart from cancer. Only one nodule of cancer found in chest at necropsy.

TABLE B—

No.	Surgeon.	Age.	Menses.	Previous operations.	Nature of disease for which operation was performed.	Date of oophorectomy.
32	Hopkins Walters	51	Ceased	None	Inoperable cancer of breast, skin, and glands	March, 1897
33	Beatson	44	Regular	None	Inoperable cancer of breast and glands	March, 1897
34	Herman	49	Irregular and recently ceased	None	Inoperable breast	February, 1901; ovaries atrophic
35	Waterhouse	45	Regular	Removal of left breast, etc., November 6th, 1902, for large soft carcinoma; axillary glands markedly involved	Several recurrent nodules round the scar, and an enlarged supra-clavicular gland five months after the first operation	March 26th, 1903; double oophorectomy with free excision of recurrent nodules and supra-clavicular glands
36	J. Hutchinson	43	Regular until quite recently; now goes a few days over	Large scirrhus left breast adherent to skin and pectoralis major; enlarged axillary glands; complete operation May, 1901	Small nodules of recurrence in the scar, enlarged glands in left axilla and above the clavicle	May 27th, 1902

TABLE C.

37	Stanley Boyd	49	Ceased	Removal of breast August, 1891	Growth in liver, etc.	March, 1897
38	Stanley Boyd	64	Ceased	Removal of breast April, 1896	Mass in chest wall	March, 1897
39	Stanley Boyd	43	Ceased	Removal of breast January, 1900	Extensive disease in breast and axilla	January, 1900
40	Hopkins, Walter	55	Ceased	Removal of breast and glands 1890	Multiple nodules, swelling of arm	March, 1897
41	Berry	54	Ceased	Removal of breast November, 1898	Multiple nodules in scar, etc.	October, 1899
42	A. D. J. W. Smith	40	Regular	None	Inoperable breast	February, 1902
43	Herman (Case 6)	53	Ceased	Breast removed November 1899. Recurrence June, 1900	Recurrence	September, 1900, ovaries atrophied
44	Herman (Case 7)	24	Regular	Complete operation April, 1900	Nodules near scar; glands above clavicle	November, 1900, one contained nodule of cancer

continued.

Thyroid.	Result.	Duration of life after oophorectomy.	Remarks.
None	Diminution of growths, and marked improvement in health	9 months	Local disease again active. Died December, 1897.
None	Shrinkage of tumour, disappearance of glands	11 months	Died of growths in liver, pleura, and spine.
Given steadily	Pain less, shrinkage of growth. 4 days after the operation complained of pain in the breast and axilla. 5 days later the pain was less	4 months	Died June, 1901. Intestinal obstruction, ? due to band or adhesions.
None	Excellent health for 1 year. In March, 1904, two enlarged glands were noticed above the clavicle and one in the anterior triangle	16 months	Died July, 1904. Generalised carcinoma.
—	Greatly improved health; the nodules in the scar disappeared; the glands diminished in size. November 14th, 1902. —Fresh nodules in the scar; enlarged glands in left axilla; œdema of the arm	About 16 months	Died autumn, 1903. Intrathoracic growths, etc.

TABLE C.

None	Nil	14 weeks	Died.
Given steadily	?	40 months	Growth at first stationary, then increased, ulcerated. bled, dying July, 1900.
None	?	6 months	Died from extension of disease.
None	Diminution of growths	1 month	Died. Growths in omentum, stomach, and liver.
Given	?	5 months	Hemiplegic and dying, March, 1900.
None	None	—	—
Given fair trial	No benefit	7 months	Disease progressed, and died April, 1901
—	Temporary diminution of nodules; disease retarded	—	July, 1901.—Mass in pelvis.

TABLE C—

No.	Surgeon.	Age.	Menses.	Previous operations.	Nature of disease for which operation was performed.	Date of oophorectomy.
45	Percy Paton	43	Recent pregnancy	Breast removed January 1900	Ulceration of chest wall	October, 1900
46	E. H. Fenwick	54	?	March, 1898, removal of breast, part of pectoralis major and axillary glands. Sixteen months' duration. July 13th, 1899, several recurrent nodules around scar removed	Several hard nodules in the scar and in the skin around	October 11th, 1899
47	F. Eve	53	?	None	Extensive carcinoma of breast, fixed, small nodules in the skin over the breast. Many enlarged glands. Ulceration in the centre of the breast. Noticed for more than a year	February 8th, 1901
48	H. P. Dean	55	?	June, 1898, scirrhus left breast 2½ years' duration. A little ulceration. Enlarged glands in axilla. Removal of breast, pectoral fascia, and axillary glands	Carcinomatous erythema of skin on left side of chest. Multiple nodules. Enlarged supra-clavicular glands. Very little pain	August 29th, 1899
49	Stanley Boyd	46	Regular	Removal of breast May, 1897	Disease in axilla and in neck	May, 1897
50	Stanley Boyd	32	Irregular	Breast removed March, 1896. Removal of glands above clavicle. Amputation of arm	Glands above clavicle	November, 1897
51	Stanley Boyd	40	Regular	Breast removed November, 1898. Axilla cleared January, 1899. Recurrence removed March, 1899	Recurrence in scar, glands above clavicle	March, 1899
52	Stanley Boyd	39	Regular	Breast and glands removed November, 1894. Removal of nodules July, 1898. Amputation of arm	Nodules in scar, glands above clavicle	June, 1899
53	Stanley Boyd	41	Regular	Removal of breast and glands June, 1899	Nodules in skin, etc. Glands above clavicle	April, 1900
54	Stanley Boyd	39	Regular	Complete operation July, 1900	Involvement of skin and glands	July, 1900

continued.

Thyroid.	Result.	Duration of life after oophorectomy.	Remarks.
Three weeks	Practically none	5 months	Died March, 1901.
Given	Two to three weeks after operation temporary diminution in the size of the nodules, then they increased, and one or two others appeared	?	Left the hospital 5 weeks after operation. Lost sight of.
—	February 12th, 1901.—Breast and arm hurt her more than ever	3 weeks	Did well, died suddenly March 1st.
Given	No sign of improvement on discharge. Died September, 1900	13 months	—
None	Possibly prevented local recurrence	37 months	Pain in right thigh and lameness from February, 1899, otherwise excellent health, July, 1899. Dying of generalised cancer, June, 1900. Paraplegia, etc.
None	?	32 months	Died June, 1900. Extension of disease, (?) metastasis in liver.
None	?	12 months	Dying of generalised carcinoma, March, 1900.
Given for some weeks	Probably none	11 months	Died April, 1900; extension of disease.
None	Probably none	—	Disease progressing when last seen.
None	Probably none	—	Recurrence within 2 months.

TABLE C—

No.	Surgeon.	Age.	Menses.	Previous operations.	Nature of disease for which operation was performed.	Date of oophorectomy.
55	Beatson	40	Regular	None	Extensive disease in breast and glands	October, 1895
56	Watson Cheyne	32	Regular	None	Inoperable breast	May, 1897
57	Eve	45	Regular	Complete operation April, 1899	Extensive recurrence in chest wall. Ulceration	August 1st, 1899
58	Waterhouse	44	Regular	Complete operation November, 1897	Recurrence in scar; swelling of arm	July, 1899
59	Neatby	44	Regular	Removal of breast and recurrences	Recurrent nodule in scar	August, 1899
60	Bloxom	45	Regular	Complete operation December, 1896	Nodules in scar; glands above clavicles	June, 1897
61	Mrs. S. Boyd	42	Regular	Complete operation December, 1899	Extensive disease in glands and skin	December, 1899
62	Bidwell	36	Regular	Complete operation January, 1900	Extensive infiltration of skin	May, 1900
63	Carless	38	Regular	Complete operation February, 1898	Extensive disease of skin	December, 1898
64	Clutton	34	Regular	Complete operation 1898	Disease in axilla	1898
65	Battle	39	Regular	Complete operation 1899	Disease in supra-clavicular glands	December, 1899
66	Pitts	27	Regular	Complete operation 1899	Disease in scar and glands	1899
67	T. K. Dalziel	51	?	Complete operation May, 1897	Recurrent nodules and glands in neck	March, 1899
68	T. K. Dalziel	54	?	Complete operation May, 1899	Ulcerated recurrence in scar	October, 1899
69	R. H. Hodgson	54	?	Complete operation May, 1901	Glands in neck, etc.	October, 1901
70	Frank C. Madden	40	—	None	Ulcerated breast; œdema of arm	March, 1902
71	Frank C. Madden	40	—	None	Inoperable breast	May, 1902
72	Alec Miles	42	?	Complete operation November, 1900	Recurrence in scar and glands	June, 1901
73	Alec Miles	31	?	Complete operation February, 1901	Recurrence in wall of thorax	March, 1901
74	Lynn Thomas	?	?	Complete operation 3 years before	In scar and glands	?
75	Lynn Thomas	38	?	Complete operation 2½ years before	In scar and glands	?
76	Annandale	42	?	Complete operation	Nodules in skin, etc.	September, 1896
77	Annandale	?	?	Complete operation	Leathery skin, etc.	October, 1896

continued.

Thyroid.	Result.	Duration of life after oophorectomy.	Remarks.
Given	Relief of pain	12 months	Died 1896; secondary growths in liver.
Given	Very slight	5 months	Died October, 1897; growths in lung and liver.
Given	None	5 weeks	Died September, 1899.
?	None	9 months, +	Worse in all respects at last note. Under care for religious melancholia, which had threatened for years.
?	None	3 months	Recurrences growing fast.
—	Slight, if any	4 months	Died. Cancer of spine; recurrent nodules remained small.
None	Disappearance of nodules in axilla	11 months	Signs of growth in spine; no local recurrence. Died November, 1900.
—	Improvement	24 days	Sudden death from dyspnœa.
—	—	Few months	Died from progress of disease.
Given	Nodules stationary	—	Lost sight of after a few months.
Given	Little improvement	—	August, 1900. Growths in spine.
—	—	—	—
None	None	8 months	Died.
None	None	4 months	Died.
?	None	?	Disease progressing.
—	—	6 weeks	Died. Cancerous mass beneath liver.
—	No improvement	—	Still under observation.
—	No improvement	9 weeks	Died of extension of disease.
—	No improvement	3 months	Died of disease in spine.
Given	No improvement	3 months	Died. Secondary growths in pleura and liver.
Given	No improvement	?	Died. Secondary growths.
Given	No improvement	7½ months	Died. Exhaustion.
Given	No improvement	4 months	Died. Progress of disease.

TABLE C—

No.	Surgeon.	Age.	Menses.	Previous operations.	Nature of disease for which operation was performed.	Date of oophorectomy.
78	Annandale	?	?	Complete operation August, 1900	Nodules around scar	December, 1900
79	J. M. Cotterill	35	?	Complete operation May, 1899	Nodules; scar; swelling of arm	December, 1899
80	D'Arcy Power	52	?	Removal of breast	Fungating breast; cancer of peritoneum; ascites	?
81	Watson Cheyne	33	?	—	Nodules in scar; glands in neck	May, 1897
82	Abbe	50	?	Removal of breast December, 1900	Glands root of neck	May, 1901
83	Stanley Boyd	46	Regular	Breast removed November, 1897	Disease in axilla, etc.	November, 1897
84	Lawrie McGavin	42	Regular	None	Several nodules in the course of the scar. A mass the size of a walnut in the axilla	May 24th, 1901. Also removal of area of recurrence and partial removal of axillary glands
85	C. Mansell Moullin	45	Still menstruating	None	Scirrhus in the upper half of the breast—one or two nodules in the skin—axillary and supraclavicular glands enlarged. Noticed four months ago while suckling	June 7th, 1900
86	C. Mansell Moullin	48	?	—	Left breast hard and indurated, attached to the skin; movable on the deep structures. Enlarged axillary glands	—
87	A. B. Roxburgh	42	?	Removal of breast and axillary glands March 1st, 1900; recurrent nodules excised April 27th, 1900	Nodules around the scar	June 8th, 1900
88	G. E. Herman	39	—	Left breast removed 6 years ago. ? Axillary glands removed 10 weeks later. Right breast removed three years ago. Recurrences removed from both axillæ and left side of neck 2 years ago	Cachectic and very anæmic. Extensive ulceration on the right side. Collections of recurrent nodules on both sides of chest round the scars of previous operations also nodules on back. Hard fixed glands both axillæ and supra-clavicular regions. Secondary growths in liver	May 7th, 1899

continued.

Thyroid.	Result.	Duration of life after oophorectomy.	Remarks.
Given	Temporary improvement	2 months	Died. Progress of disease.
Given	Temporary improvement	4 months	Died. Progress of disease.
—	No improvement	6 months	Died. Progress of disease.
Given	Temporary improvement	5 months	Died. Growths in liver and pleura.
—	Temporary improvement	5 months	Died.
Given steadily	?	14 months, +	Fungating local recurrences, September, 1898
—	Three weeks later further nodules appeared; secondary deposits in upper dorsal spine and left lung. Mass in axilla had disappeared	? 8 months	Six months after the operation there were secondary deposits in the spine and pleura. She was not expected to live more than six to eight weeks.
Given	Two weeks later measurements indicated some contraction going on in the right upper quadrant. The tissues towards the axilla were softer, as also were the outlying nodules. Died March 27th, 1901	9½ months	—
—	Death on third day	3 days	—
—	Death three and a half weeks later, exhaustion	3½ weeks	—
Given	Death five days after operation, exhaustion	5 days	—

TABLE C—

No.	Surgeon.	Age.	Menses.	Previous operations.	Nature of disease for which operation was performed.	Date of oophorectomy.
89	Eve	44	—	Carcinoma of left breast. Removal of breast and axillary glands 1902	Recurrence in the glands on the left side of the neck	November 21st, 1902, glands excised
90	D'Arcy Power	55	Menopause eight years ago	None	Extensive carcinoma of left breast fixed to the pectoral muscle. Skin brawny and puckered	—
91	Percy Paton	31	Recent pregnancy	Breast removed May, 1901	Inoperable breast	July, 1901
92	Lockwood (Butlin's case)	40	Regular	Breast removed	Recurrence on chest	—
93	Butlin	—	—	—	—	—
94	John Chiene	51	Ceased	Breast removed January, 1897. Recurrence removed November, 1900	Nodules near scar and above clavicle; involvement of pleura	November, 1901
95	John Chiene	35	—	None	Inoperable breast and glands	November, 1897
96	T. H. Openshaw	50	—	Breast removed eight years ago; six operations since for recurrence, the last nine months ago	Hard nodules. Glands felt above right clavicle and beneath pectoralis; also along sub-scapularis muscle. Œdema of upper arm	June 23rd, 1899
97	J. Hutchinson	48	Regular	Removal of breast and axillary glands January 3rd, 1896. Carcinomatous gland removed from axilla December 21st, 1898	Many nodules over the whole trunk. First noticed four months ago round the scar. At the operation some nodules were seen on the parietal peritoneum and in omentum. Carcinoma with myxomatous degeneration	November 27th, 1901

continued.

Thyroid.	Result.	Duration of life after oophorectomy.	Remarks.
Given	June 12th, 1903: Patient having Light treatment for recurrent nodules on left side of breast. June 12th, 1903: Removal of right breast, pectoral fascia, and axillary glands. Large, ill-defined hard lump, movable on pectoralis major. September 30th, 1903: Much œdema of left arm and pain. Diffuse infiltration and recurrent nodules on the left side of the chest	—	—
Not given	Ten days after operation, breast much softer than before, but soon returned to its original hardness. Died November 25th, 1902	—	Pain in the left breast on the second day and for two weeks after removal of ovaries.
—	Felt better; speedy recurrence in scar	5 months	Died December, 1901. Progress of disease.
—	No relief	—	Died.
—	No relief	—	Died.
—	No improvement	—	—
—	No improvement	—	—
—	Mania. Death two weeks after operation	2 weeks	Wound healed first intention. No pyrexia.
—	Some of the nodules diminished in size after the operation. Died January 27th, 1902	2 months	—

TABLE C—

No.	Surgeon.	Age.	Menses.	Previous operations.	Nature of disease for which operation was performed.	Date of oophorectomy.
98	F. Eve	44	?	—	Skin over the right breast is hard, brawny, and indurated. In the outer half of the breast is an ulcer with hard raised edges, the base being covered with a foul slough. The breast is firmly fixed. Hard deposits in the skin up to the axilla. Axillary glands enlarged	November 17th, 1900
99	Abbe	40	?	Removal of breast February, 1900	Glands above clavicle	May, 1901. Ovaries cancerous

DISCUSSION.

Mr. W. BRUCE CLARKE showed a case of a woman five years after oöphorectomy for inoperable cancer. In 1895 the patient had some bloody discharge from the nipple, but no evidence of growth. In 1897 a small nodule was detected and removed, and three months later the whole breast was removed, and the axilla was cleared out. The growth on section proved to be a duct cancer. In 1898 the posterior triangle became involved, and the growth was removed from this situation. In 1899 a further recurrence took place in this situation, and the growth was again removed. In November, 1899, the growth had again recurred, and involved the right side of the chest and abdomen down nearly to the level of Poupart's ligament. Oöphorectomy was now performed. The pain, which had been a marked feature, rapidly diminished; the growth shrivelled in six weeks, and had remained in about the same condition till the present time, some five years after the operation. She had remained well without any important treatment, and she was quite fit for her ordinary work. This was the most successful case upon which he had operated. He never used this method except in absolutely hopeless cases. Of the five or six cases in which he had done the operation, though all improved for a time, this was the only one that survived more than three years after the operation.

Mr. STANLEY BOYD was pleased to note that Mr. Lett was

continued.

Thyroid.	Result.	Duration of life after oophorectomy.	Remarks.
—	Four weeks later the cutaneous nodules distinctly less indurated and had disappeared to a large extent. The main mass smaller and the ulcer cleaner	—	Lost sight of.
—	—	Few months	Died.

able to substantiate the conclusions which he had himself drawn a few years back from many fewer cases. He thought the most important point to note with regard to prognosis was the duration of the cancer before the oöphorectomy. Reference was made to all the most successful cases in Mr. Lett's table (Beatson's, Boyd's, Herman's, Edmunds', Bruce Clarke's, and Waterhouse's) to prove that all had run a slow course up to the time of the oöphorectomy—except Waterhouse's, in which the tumour had been noted only recently. In all these the resistance to the disease was high from the start; they were, in short, very chronic cases. In reference to the most favourable age limits more cases were required on which to base an opinion. There was almost nothing which was an absolute bar to the operation. He had employed a combined oöphorectomy and the complete removal of the breast in three cases, and his impression was that a better result was sometimes obtained than would have resulted from either alone.

Mr. Jonathan Hutchinson, jun., considered that the paper gave encouragement to surgeons to persist with the operation in selected cases. Reference was made to the difficulties in obtaining accurate information as to the date of onset of a malignant tumour. A recent patient, a very intelligent woman with a large cancer of the breast, stated that it had been present only one week. One difficulty in estimating the value of oöphorectomy for advanced cancer lay in the fact that now and then cases of recovery occurred without any treatment. There was no time

limit for recurrence after operation; in one patient of his death occurred from recurrence at the root of the neck after eleven years' immunity, following the complete operation of excision of breast and axillary glands.

Dr. HERMAN said that although the cases were not numerous enough to prove the best age for operation, yet they gave strong indications. The conclusion pointed to was that those cases did best in which the operation was performed at about the menopause. And that the cases operated on did better with the administration of thyroid gland than without it.

Mr. W. MCADAM ECCLES said that there were three points to which he would allude. First, he attached considerable importance to the nature of the carcinoma, for the variation in the rapidity of growth was considerable; secondly, he thought that it was possible that more might be learned from the microscopical examination of the ovaries removed; and, thirdly, the examination of any removed shrunken portions of growth was important, and so far as he knew only one such piece had been microscoped, and the results published. He mentioned a case in which he had performed oöphorectomy for extensive secondary surface cancer, in which all the nodules disappeared except one or two, which, however, atrophied. The patient lived for twenty-six months after the oöphorectomy, and thirty-five months after the primary growth had been removed. This patient had been the subject of a most violent "nerve storm" after the oöphorectomy, but recovered completely in about ten days, and died eventually from secondary deposits in the lungs and pleuræ.

Mr. PEARCE GOULD pointed out the fallacy of the statistical method of collected cases, for only the more successful ones were published. The paper, however, showed that certain striking results followed the operation in some cases. His experience of the results of this operation in several cases had not been so favourable as that of Mr. Bruce Clarke; he had seen but one case with a striking result. In this instance rapid diminution of the growth took place commencing almost immediately after the operation, but the patient unfortunately died from intestinal obstruction. Sections of the atrophied growth showed considerable fibrosis, and atrophy of the cell elements. Until the nature of cancer was known the operation could not be discussed quite satisfactorily. It was a noteworthy circumstance that although cancer of the breast was most common when the ovarian function was ceasing it was just at that time that most benefit had been obtained from oöphorectomy. He had not infrequently seen arrest of the disease without any operation. He never felt able to press the operation on a patient, whilst the knowledge of the nature of cancer was so indefinite. There should be reluctance to raise hopes in patients which might not be realised. In his

opinion the chief value of the paper was in emphasising the fact that there are physiological means by which cancer can be influenced, and he looked forward to the time when these physiological means would be definitely known and could be confidently applied.

Mr. LETT, in reply, agreed that the cases were too few to warrant any definite statement, but thought that they were sufficiently numerous to afford some help in deciding for or against the operation. The cases in his paper included all those operated upon at the London Hospital in addition to the cases already published. The interval between the first operation and oöphorectomy was of even greater importance, he considered, than that between the first onset of the disease and oöphorectomy.

A CASE OF "SPLENOMEGALIC" OR "MYELOPATHIC" POLYCYTHÆMIA, WITH TRUE PLETHORA AND ARTERIAL HYPERTONIA, WITHOUT CYANOSIS

BY

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In the absence of cyanosis the following case differs from the cases of chronic cyanosis with polycythæmia and splenomegaly recorded by Vaquez, Saundby and Russell, Rosengart, Osler, and others. I have had the advantage of being able to study a typical example of such cases,[1] and believe that the present case is really of the same nature but at an earlier stage, and occurring in a fairly robust subject whose circulatory system is acting efficiently. I would temporarily include all these cases, with or without cyanosis, under one heading, namely, "Splenomegalic polycythæmia," although I think it possible that the spleen need not be obviously enlarged to clinical examination in all cases. In many respects cases of this group, like the present one—that is to say, cases with-

[1] F. Parkes Weber and J. H. Watson, "Chronic Polycythæmia with Enlarged Spleen," 'Transactions of the Clinical Society of London,' 1904, vol. xxxvii, p. 115; and 'International Clinics,' 1905, vol. iv, p. 47.

out cyanosis—resemble the case of "hypertonia polycythæmica" (Geisböck) recently recorded by Hess,[1] but apparently differ from it in the absence of albuminuria and the presence of splenomegaly.

The present patient, a Jewish woman, aged 37, of medium height and weight, first came under my care in July, 1903, for acute erythromelalgia of the left foot.[2] This condition under rest in bed and other treatment became less acute, and finally gradually disappeared.

The existence of the polycythæmia was first detected when the blood was examined in March, 1904, but was probably present earlier. From April, 1904, to February, 1905, the patient was constantly under observation in the German Hospital, and on April 22nd, 1904, was shown at a Clinical evening of the Clinical Society. Owing to the disappearance of the erythromelalgia she is no longer confined to her bed. In fact, the condition for which she originally came under treatment is practically cured, though her other symptoms, objective and subjective, persist, and it is with these that the present paper deals.

There is no distinct cyanosis of the face, though the cutaneous blood-vessels are somewhat over-filled, and the tongue is generally of a bright red colour with a bluish tinge resembling the colour of raw butcher's meat. The toes, especially of the left foot, sometimes appear rather livid, but this is possibly connected with the past erythromelalgia, which, it should be remembered, was not entirely confined to the left foot. No evidence of disease has been discovered in the heart or lungs. There is no dyspnœa. By examination of the abdomen nothing abnormal can be detected excepting moderate enlargement of the spleen, which can be felt, one or two finger-breadths below the ribs. For diagnostic purposes, owing to a question of the possibility of splenic tuberculosis, Koch's

[1] Abstract by Pappenheim in the 'Folia Hæmatologica,' 1905, vol. ii, p. 47. *Cf.* F. Geisböck, 'Verhandl. d. XXI Kongresses f. inn. Med.,' 1904, p. 97.

[2] I described this part of the case in the 'British Journal of Dermatology,' February, 1904, p. 70.

old tuberculin was employed in December, 1904, but the injection of five milligrammes failed to produce a reaction. Menstruation regular. Bowels sometimes confined. The urine is usually rather pale, acid, of low specific gravity (about 1010), somewhat increased in quantity, and free from albumen and sugar. The percentage of urea has not been regularly estimated. On one occasion it was 1·9 per cent., making the total daily excretion up to or somewhat above the average. The body weight on June 21st, 1904, was 10 st. 6 lbs.: on August 23rd it was 10 st. 10 lbs.; on November 21st it was 11st.; on February 6th, 1905, it was 11 st. 2 lbs. Slight enlargement of the thyroid gland was temporarily noticed in October, 1904. There is considerable deafness in both ears, possibly connected with chronic dry catarrh. The patient's subjective symptoms consist in a disagreeable noise in her ears and occasionally headaches and slight vertigo, also apparently feelings of prostration. The noise in her head is always present, but varies in character from a whizzing or rushing to a roaring or rumbling sound, and is rhythmical with the heart's action.

I will now give an account of the examination of the blood and circulatory system whilst the patient has been under observation.

Circulatory system.—As already mentioned, nothing abnormal has been found by physical examination of the heart, the apex beat being in the fifth left intercostal space, internal to the nipple line, and the area of cardiac dulness not being increased. The pulse at the wrist is of medium volume and increased tension; it is regular, the rate being about 80—90 in the minute, but affected by mental excitement. Pulse tracings made with a Dudgeon's sphygmograph in June, 1904, showed a pulse of high tension. On December 20th, 1903, Hill and Barnard's pocket sphygmometer on the radial artery gave the blood-pressure as about 140 mm. Hg. (that is, the pressure at which the oscillations were greatest), but their larger instrument on the arm showed a pressure of about 165 mm. Hg. On

December 16th, 1904, Dr. Haldane kindly estimated the maximum brachial blood-pressure by Martin's modification of the Riva Rocci apparatus, and found it to be 157 mm. Hg., and on January 21st, 1905, he found it 152 mm. Hg. In June, 1904, Dr. Gruber kindly made an ophthalmoscopic examination of the blood-vessels in the fundus oculi and reported that the veins were markedly congested and slightly tortuous, but the macular region did not show any decided enlargement of capillaries such as he had noted in the case of chronic cyanosis and polycythæmia[1] already alluded to.

The blood.—The following table is intended to show the results of blood counts[2] and microscopic examination of the blood in relation to diet and treatment from March, 1904, to February, 1905. It must be remembered that, owing to the improvement in the condition of the left lower extremity, the patient was able to be up and to get about much more at the end of this period than at the beginning, and this may have exercised an influence on the general condition.

In addition to the drugs mentioned on the table, bromides, aspirin, and valerian were sometimes used. At the commencement of July, 1904, minute doses of calomel were tried for about a week, but without any obvious effect on the general condition.

The red cells and hæmoglobin value.—The red cells varied abnormally in size, and, according to Dr. Boycott, in shape and staining capacity. These changes, together with the

[1] Weber and Watson, *loc. cit.*

[2] The blood counts were made by a Thoma-Zeiss hæmocytometer, and the hæmoglobin value was obtained either by a Gowers' hæmoglobinometer or by Haldane's modification. In the latter estimations (November 30th and later) either a correct Haldane's instrument was used or an old Gowers' instrument freshly standardised by the kindness of Dr. Haldane and Dr. A. E. Boycott. The instruments used in the earlier estimations were not freshly standardised. I had the pipette of a Gowers' hæmoglobinometer graduated so that only half the usual amount of blood might be sucked up. In this way the diluted blood does not rise above the scale.

presence of a few nucleated red cells, might, as Dr. Boycott points out, be ascribed to unusual activity in the erythroblastic functions of the bone marrow. The nucleated reds seen were all normoblasts, and in the blood-films from November 30th, 1904, as many as three were noticed during a differential count of 500 white cells; in the blood from December 16th eleven normoblasts were found on two

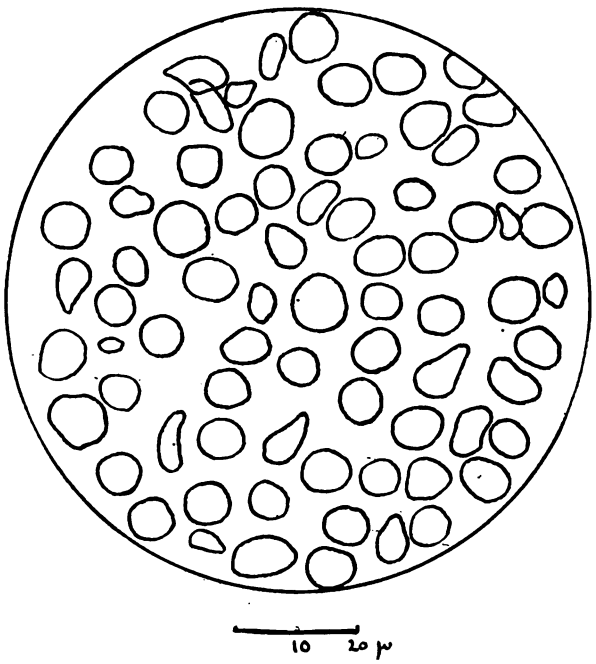

FIG. 1.—Red blood corpuscles, showing variations in size and shape, in a film taken on August 15th, 1904. Drawn to scale by Dr. A. E. Boycott.

slides in the course of a search of about three quarters of an hour. Erythroblasts have likewise been observed by Türk and some others in cases of splenomegalic polycythæmia. In regard to the occasional presence of nucleated red cells in normal human blood, Dr. Boycott tells me that though probably present in all persons they are

Date.	Hæmoglobin per cent. of normal standard.	Red cells per c.mm.	White cells per c.mm.	Polymorpho-nucleears per cent.	Small lymphocytes per cent.	Large lymphocytes, large mononuclears, and "transitionals" per cent.	Coursely granular eosinophiles per cent.	Mast-cells per cent.	Erythroblasts (all normoblasts).
1904 April 13th	120	8,240,000	6000	73·75	17·25	8·5	0·5	Only one seen	—
April 17th	125	9,440,000	8100	—	—	—	—	—	—
April 19th	—	8,660,000	9000	77·6	18·5	3·5	0·3	—	—
May 30th	165	10,600,000	7200	68·8	26·5	4·0	0·7	—	—
June 12th	170	—	—	—	—	—	—	—	—
June 20th	148	10,960,000	8800	—	—	—	—	—	—
July 8th	175	9,440,000	8400	—	—	—	—	—	—
Aug. 3rd	148	8,016,000	8000	—	—	—	—	—	—
Aug. 15th	177	9,840,000	—	77·0	13·0	8·0	0·4	1·6	Present

Average diameter of erythrocytes.	Diet and Treatment.	Remarks.
—	—	The differential count was of 400 white cells by Dr. G. L. Eastes. During the count one mast cell was found. No myelocytes or erythroblasts. Three or four of the white cells counted were "intermediate forms," and Dr. Eastes enumerated these with the type which they most resembled.
—	—	—
—	At the end of April, 1904, a milk diet was ordered, but was only continued for a few days owing to the patient's objection to it. She was then put on ordinary meat diet, and from May 9th was given two to three drops of liquor arsenicalis three times daily. The arsenic was discontinued on May 30th	This blood-count was made by Dr. J. H. Drysdale when he kindly came to see the patient. The large lymphocytes were counted with the small lymphocytes as "lymphocytes." I have, therefore, included them in the group of small lymphocytes; Dr. Drysdale is not responsible for this.
Not greater than the normal.	—	Dr. G. L. Eastes made the differential count and reported that no myelocytes or erythroblasts were seen, and that there was no poikilocytosis. Films were likewise sent to Prof. H. Vaquez, who mentioned in his paper with Dr. Laubry that they confirmed his opinion that in splenomegalic polycythæmia there was no hyperglobuly, that is to say, increase in the average diameter of 100 red cells, though microcytes and macrocytes might be present.
Great variety in size noted	—	—
—	At the end of June, 1904, meat was discontinued, and the diet was made to consist of milk, milk puddings, bread, butter, potatoes, green vegetables, and stewed fruit, with every day two eggs, and about twice weekly some fish. From July 8th, 1904, till Feb., 1905, she took the juice of one lemon daily	Prof. A. E. Wright kindly took away specimens of the blood and urine for examination (*see* later). His count made the red blood corpuscles just over 11,000,000 in the c.mm., a result not very different from that of the count at the hospital.
—	—	—
—	—	This count was taken after unusually copious menstruation.
7·48 μ	—	The differential count was of 500 white cells by Dr. A. E. Boycott. He found the average diameter of 50 red cells to be 7·48 μ; maximum diameter, 8·57 μ; minimum diameter, 5·95 μ.

Date.	Hæmoglobin per cent. of normal standard.	Red cells per c.mm.	White cells per c.mm.	Polymorpho-nucleears per cent.	Small lymphocytes per cent.	Large lymphocytes, large mononu-cleers, and "tran-sitionals" per cent	Coarsely granular eosinophiles per cent.	Mast-cells per cent.	Erythroblasts (all normoblasts).
1904									
Aug. 26th	177	9,680,000	6000	—	—	—	—	—	—
Sept. 9th	175	9,850,000	6000	—	—	—	—	—	—
Sept. 29th	185	9,800,000	—	—	—	—	—	—	—
Oct. 14th	184	9,968,000	7320	—	—	—	—	—	—
Nov. 16th	177	9,976,000	8320	—	—	—	—	—	—
Nov. 30th	156	8,480,000	8200	81·0	12·6	5·6	0·6	0·2	Several
Dec. 9th	—	—	—	73·0	15·0	8·4	3·0	0·6	Several
Dec. 16th	158	—	—	72·6	14·0	7·0	1·0	0·4	Several
Dec. 23rd	161	9,280,000	4800	—	—	—	—	—	—
1905									
Jan. 3rd	156	8,625,000	7500	75·4	18·6	5·4	0·6	Present	Present
Jan. 12th	145	9,568,000	6000	66·0	25·4	6·6	2·0	Present	Present
Jan. 21st	148	8,568,000	4000	75·0	11·0	11·6	2·0	0·4	Present
Feb. 6th	164	8,680,000	4800	80·4	14·0	4·0	1·0	0·6	Present

Average diameter of erythrocytes.	Diet and Treatment.	Remarks.
—	—	In August it was noted that patient could walk about without the left foot becoming hot or different in colour from the right foot.
—	—	The specimen of blood from which this count was made was obtained direct from a superficial vein in the right fore-arm by a Pravaz syringe.
—	In Sept. the diet was changed so as to include fish or meat every day at the mid-day meal. From Sept. 15th to Oct. 8th Röntgen ray treatment was carried out; 21 sittings of 5 to 10 minutes' exposure of the splenic region; at several sittings the lower parts of the legs were likewise exposed for 5 minutes	—
—	—	—
—	From Oct. 31st to Dec. 5th; 1904, three grains of potassium iodide were given three times daily	—
7·74 μ	—	The blood examination is by Dr. A. E. Boycott. During the differential count of 500 white cells he found 3 normoblasts, one with polychromatic cytoplasm and about 12 polychromatic red cells. The red cells varied from 4 μ to 10 μ in diameter, and there were too many oval and irregular shapes. The hæmoglobin was estimated by Dr. Haldane (*see* also later).
—	—	The red cells Dr. Boycott said showed the same abnormal variation in size, shape, and staining capacity as at the last examination. He soon found several normoblasts and some polychromatic red cells.
—	—	The hæmoglobin estimation was by Dr. Haldane.
—	Venesection 100 c.c.	The blood examination was of blood obtained by venesection containing 1 per cent. of a 50 per cent. aqueous solution of potassium citrate.
—	During last days of Dec. till Jan. 12th patient was taking 15 grs. of potassium bromide and 7½ grs. of aspirin thrice daily. From Jan. 12th till Feb. 6th, 1905, she took 10 grs. of sodium salicylate thrice daily	The differential counts in 1905 were all of 500 white cells, by Dr. A. E. Boycott, as were all the others from Nov. 30th. Dr. Haldane estimated the hæmoglobin on Jan. 21st. In the blood-films of Feb. 6th Dr. Boycott thought the changes in the red cells were much less marked than before, and could only discover one normoblast with great difficulty.
—		
7·6 μ		
—		

extremely rare. During the winter 1903–1904 he searched about 500 blood-films with this point in view, and only found three or four altogether, though 500 leucocytes were counted in each specimen.

The hæmoglobin values of the blood noted in April, 1904, were relatively low; probably the colour index of the corpuscles, and perhaps their size also, increased soon after this. The highest hæmoglobin values recorded were on October 14th, 1904 (184 per cent.), and on September 29th (185 per cent.), but the hæmoglobinometers used on these occasions had not been specially standardised. The recent figures have varied from 145 to 164 per cent. The hæmocytometer readings have on the whole varied less than the hæmoglobinometer readings. The largest number of red cells recorded was 10,960,000 on June 20th, 1904, and the lowest was 8,016,000 on August 3rd; at present (February 6th, 1905) there are about eight and a half millions in the cubic millimeter, and the average colour index of the cells is nearly up to the normal.

Vaquez[1] is probably right in saying that in splenomegalic polycythæmia, even when combined with marked cyanosis, there is no "hyperglobuly"—that is, that the average size of the red cells, judged by their average diameter, does not exceed normal limits. In my last case[2] it did not, and in the present case it does not; for Dr. Boycott estimated the average diameter at 7·7 μ on November 30th, 1904, and at 7·6 μ on January 21st, 1905.[3] In an old preparation from August 15th, 1904, he made it only about 7·5 μ. In this connection it is interesting that Professor A. E. Wright,[4] who kindly examined the patient's blood on June 20th, 1904, counted the red cells as just over 11,000,000 in the cubic millimetre, and

[1] Vaquez, "Du volume des globules rouges dans les polyglobulies avec cyanose." Société de Biologie, Paris, July 16th, 1904.

[2] *See* Weber and Watson, *loc. cit.* The average diameter was 7·1 μ.

[3] Dr. Boycott used ordinary stained blood-films for this purpose, measuring fifty to one hundred cells on each occasion.

[4] On the volumetric estimation of the corpuscular elements, *see* A. E. Wright, 'Lancet,' January 23rd, 1904, p. 21

in the sedimentation tube obtained 9·25 volumes instead of the ordinary five volumes of corpuscles in ten volumes of blood. It may here be recalled that J. A. Capps in his 'Study of Volume Index'[1] concluded that the volume of the individual erythrocyte is best obtained by using the centrifuge in conjunction with the hæmocytometer.

The white cells.—In regard to the white cells the first thing to be noticed is the relative leucopenia, which has lately been very pronounced, the count having been on three occasions as low as four to five thousand in the cubic millimetre of blood. Relative leucopenia has, however, not been a feature in all cases of splenomegalic polycythæmia. The second point is the high percentage of polymorphonuclears, 66 to 81 per cent. This, Dr. Boycott thinks, forms additional evidence of unusual activity in the bone marrow. In my previous case of splenomegalic polycythæmia[2] the polymorphonuclears constituted 82·4 per cent. of the total white cells, and Vaquez[3] found the proportion 79 to 82 per cent. No myelocytes were found in the blood from either of my cases.

Total volume of the blood.—Dr. Haldane kindly came three times to estimate the total quantity of the patient's blood by his carbon monoxide method.[4] After his last visit he wrote that not only was there no doubt at all as to the enormous increase in the red corpuscles and hæmoglobin, but that he also felt convinced that the last blood-volume-determination (January 21st, 1905) left no loophole of error as to there being also a large increase in the blood-volume, though not so large as in many cases of chlorosis according to Dr. Lorrain Smith's estimations. Following are Dr. Haldane's figures of his first determination, November 30th, 1904, and of his third determination, January 21st, 1905. He

[1] 'Journal of Medical Research,' Boston, December, 1903, vol. x, p. 367.

[2] Weber and Watson, *loc. cit.*

[3] Vaquez and Laubry, "Cyanose avec Splénomégalie et Polyglobulie," 'Tribune Médicale,' Paris, August 13th, 1904, p. 517.

[4] *Vide* J. Haldane and J. Lorrain Smith, "The Mass and Oxygen Capacity of the Blood in Man," 'Journal of Physiology,' August 29th, 1900, vol. xxv, p. 331.

thinks there was certainly an error in his second determination December 9th, 1904, which has, therefore, been omitted.

Date.	Value of CO in c.c. at 0° C. and 760 mm. barometric pressure.	Saturation of haemoglobin per cent.	Total oxygen capacity of blood in c.c.	Percentage of haemoglobin.[1]	Total volume of blood in c.c.[2]	Red corpuscles in c.mm. of blood.	White corpuscles in c.mm. of blood.	Body weight in kilos (clothes allowed for).	C.c. of blood per 100 grammes body weight.	Oxygen capacity per 100 grammes body weight in c.c.
1904 Nov. 30	74·0	4·6	1610	156	5600	8,480,000	8200	68	8·2	2·4
1905 Jan. 21	127·0	7·0	1810	148	6000	8,568,000	4000	68	9·7	2·7

The residue of carbon monoxide was analysed after each experiment to ascertain its degree of purity, and the air left in the bladder was also analysed to make certain that the carbon monoxide had been actually absorbed.

Amount of iron in the blood.—Some blood (45¾ grammes), after serving for the viscosity and cryoscopy examinations, was used by Mr. J. H. Ryffel, B.Sc., to obtain a quantitative estimation of the iron. He dried the blood in a platinum dish in the water oven, then ignited in the dish, till all carbon was burnt away, dissolved the ash in strong hydrochloric acid, diluted, filtered, evaporated in a porcelain dish with a few drops of strong nitric acid, redissolved in dilute hydrochloric acid, precipitated with ammonia, filtered, washed, dissolved in dilute sulphuric acid, reduced with pure zinc, filtered through asbestos, and titrated with $\frac{N}{10}$ permanganate solution. Mr. Ryffel found that the percentage of iron, calculated for the undiluted blood, was

[1] The normal percentage of hæmoglobin is reckoned as 100. It may be added that 100 c.c. of blood with this normal percentage of hæmoglobin can take up 18·5 c.c. oxygen.

[2] The normal individual is estimated to possess 4·6 c.c. of blood per 100 grammes body weight.

0·0673 grammes per cent. Dr. Boycott points out that supposing hæmoglobin to contain 0·33 grammes per cent. iron, Mr. Ryffel's result would correspond to 23·9 grammes hæmoglobin per cent. in the blood; the 100 per cent. of Haldane's hæmoglobinometer standard corresponds to an oxygen capacity of 18·5 per cent., which is believed to be equivalent to 13·7 grammes hæmoglobin per cent. in the blood; Mr. Ryffel's result would, therefore, correspond to 174 per cent. hæmoglobin on Haldane's scale. The hæmoglobin actually found in the venesection blood was 161 per cent. Allowing, therefore, for a little iron in the white blood corpuscles (normal blood-plasma is said to be free from iron) and for some evaporation (concentration of the blood) during the viscosity estimation, Mr. Ryffel's estimation of the total iron in the blood corresponds with the percentage of hæmoglobin found.

Specific gravity of the blood.—On June 27th, 1904, the specific gravity of a sample of the patient's blood obtained by pricking the finger was found to be 1·078, by the help of Hammerschlag's method. By the more accurate (pyknometer) weighing method the specific gravity of the blood obtained at the venesection on December 23rd, 1904 (after being mixed with 1 per cent. of a 50 per cent. aqueous solution of potassium citrate), was found to be 1·072.

Viscosity of the blood.—I made use of the citrated blood obtained at the venesection on December 23rd, 1904, to examine its viscosity by means of a viscosity tube exactly similar to the one suggested to me by Professor Arthur Schuster and made for me by Messrs. Baird and Tatlock, which I had employed with Dr. J. H. Watson in 1904[1] to ascertain the influence of the proportion of corpuscles on the viscosity of blood (in blood from a horse citrated to hinder coagulation). I found that the citrated blood from the patient took 203 seconds to run through the bulb of the tube, from one mark to the other, water taking only

[1] *Vide* Weber and Watson, 'Clin. Soc. Trans.,' 1904, vol. xxxvii. The apparatus used is described and figured on page 131.

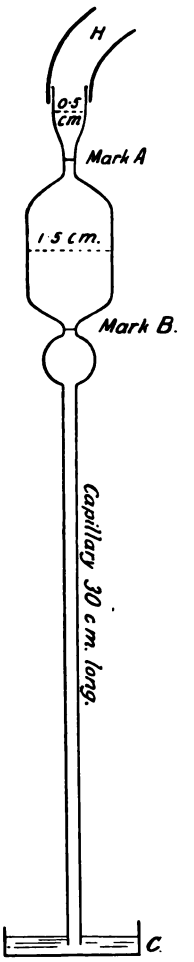

FIG. 2.—The blood is sucked up from an open vessel, C, by means of an india-rubber tube, H. The liquid is then allowed to fall whilst the tube is kept in a vertical position. The times at which the upper surface of the liquid passes the marks A and B are noted. If the time taken in two different liquids be t_1 and t_2 respectively, and p_1 and p_2 be the respective densities of the two liquids, and n_1 and n_2 their coefficients of viscosity, then $\frac{n_1}{n_2} = \frac{t_1 p_1}{t_2 p_2}$; so that, if for one liquid (as in our case for water), n_2 is known, n_1 may be calculated out.

19 seconds.[1] The specific gravity of the citrated blood was 1·072. Therefore, according to the formula given me by Professor Schuster, if n_1 = the coefficient of viscosity of the citrated blood and n = coefficient of viscosity of water at the temperature at which the experiment was made, $n_1 = \frac{n \times 203 \times 1{\cdot}072}{19 \times 1{\cdot}000}$. Therefore the coefficient of viscosity of the citrated blood was 11·45 times the coefficient of viscosity of the water used. Unfortunately, the temperature of the water used was not taken, and it may have been below the temperature of the room, which was about 18·4° C. Evidently, however, the viscosity of the blood was excessive, much higher than that of blood in ordinary diseases and conditions.

Cryoscopy of the blood.—Some blood obtained at the venesection on December 23rd, 1904 (mixed with 1 per cent. of a 50 per cent. aqueous solution of potassium citrate) was employed by Dr. Emery, Clinical Pathologist at King's College Hospital, to determine the freezing point, which he very kindly did on the day on which the blood was obtained. After the slight correction necessary for the presence of potassium citrate the freezing point was found to be minus 0·53° C., and therefore not very different to that of normal blood, which is about minus 0·56° C. The urine passed by the patient immediately after the venesection was of specific gravity 1008, faintly acid, and free from albumen and sugar. Dr. Emery found its freezing point was minus 0·79° C. Dr. Emery added that the blood plasma could not be collected in amount sufficient for the estimation of the freezing point, as after thorough centrifugalisation of a specimen of the citrated blood the plasma layer was only about 2 mm. thick, or, roughly speaking, only about 3 per cent. of the blood-column.

The salts in the blood and urine.—On June 20th, 1904,

[1] The tube previously used for the experiments with horse's blood was not used on this occasion, because water took about 44 seconds to run through the bulb, and the estimation of the viscosity of the patient's blood would have taken an unnecessarily long time.

Professor A. E. Wright[1] estimated the salts in the blood as equivalent to 0·58 per cent. sodium chloride (in lieu of the normal, about 0·78 per cent.), the salts of the urine[2] coming out as equivalent to 0·46 per cent. sodium chloride. This, Professor Wright says, gives an excretory quotient of about 0·8 instead of the normal of over 2·0. However, on June 26th, 1904, he estimated the salts in the blood-serum as equivalent to 1·14 per cent. sodium chloride, whilst the salts in the fluid from a blister were equivalent to 0·95 per cent. sodium chloride.

The alkalinity of the blood.—Professor Wright estimated this on June 26th, 1904, as equivalent to that of a normal alkaline solution diluted thirty-five times. He expresses it as $\frac{N}{35}$.

The amount of albuminous substance in the blood-plasma.—Professor Wright found the amount in the citrated blood obtained on December 23rd, 1904 (at the venesection) to be normal. His method of measuring the albuminous substances is by the resistance of the clot obtained by heating a graduated series of dilutions of the plasma.[3]

The coagulation of the blood.—Professor Wright thought its occurrence was very much delayed on June 20th, 1904. Afterwards it appeared to occur fairly readily, but the exact coagulation-time was not estimated.

The resistance of the red cells to hæmolytic agents.—Professor Wright examined the citrated blood from December 23rd, 1904, by a method of his own, and found the resistant power of the red cells to be about normal. One volume of centrifugalised sediment of red cells (the red cells were by repeated centrifugalisation washed fairly clean of blood-plasma) was suspended in sufficient $\frac{N}{10}$ salt

[1] *Vide* Wright and Kilner, "On a New Method of Testing the Blood and the Urine," 'Lancet,' April 2nd, 1904, p. 921.

[2] This was the urine passed directly after the examination of the blood, the bladder having been emptied before the examination. It was of specific gravity 1010, clear, rather pale, acid, and free from albumen.

[3] A. E. Wright, 'Lancet,' January 23rd, 1904, p. 218.

solution to bring the total volume of the suspension up to three volumes. Complete hæmolysis was then obtained by adding one volume of the suspension in a capillary tube to one volume of a $\frac{N}{30}$ salt solution.

Effect of Treatment and Progress of the Case.

As already stated, it is exceedingly difficult to estimate how much the patient's condition has really altered apart from the improvement in the erythromelalgic extremity. She can now walk about quite well, and has gained in weight, the erythromelalgia has disappeared, but the polycythæmia, high arterial tension, and the other phenomena in the blood and circulatory system persist, as do likewise the subjective symptoms (headache, etc.), although they vary in degree from time to time.

Arsenic, which was employed by W. Türk[1] in his cases, was soon discontinued in the present case on account of a considerable apparent increase in the number of red corpuscles which followed its use. On the whole, the patient has seemed to be better when on a diet containing relatively little meat, and when taking lemon-juice daily. It is possible that small doses of iodide of potassium or salicylates have a favourable influence. I have little doubt that absolute rest in bed, which was at one time required owing to the erythromelalgia, has an injurious influence on the general condition, and some of the slight apparent improvement in the general condition may be due to the patient having been able to take a little exercise (favouring metabolic processes). Opium and its derivatives have not been given a trial. In regard to drugs such as phenacetin and antifebrin (acetanilide) it must not be forgotten that the chronic use of antifebrin seems to give rise to cyanosis and blood changes.[2]

[1] 'Wiener klin. Wochenschrift,' 1904, Nos. 6 and 7.

[2] Stengel and White, "A Report of a Case of Chronic Acetanilide Poisoning, with marked Alterations in the Blood," 'University of Pennsylvania Medical Bulletin,' Philadelphia, February, 1903, p. 462.

The venesection in the present case seemed to make no difference in the subjective or objective condition, and it was not repeated, but perhaps the amount of blood (100 c.c.) withdrawn was too little to produce any decided change. It may be noted also that the diminution in the number of red blood corpuscles observed on August 3rd, 1904, followed unusually copious menstruation.

The employment of Röntgen rays, which has lately been found to have such a decided effect in many cases of leukæmia, seems to have made no change in the number of red blood corpuscles in the present case. This is not to be wondered at, since the action of these rays on the spleen and hæmopoietic tissues in leukæmia seems to be chiefly on the lymphocytes ("lymphocytolysis") and lymphadenoid tissues, and on the leucocytes generally ("leucolysis").[1] It is, however, to be noted that after a good many Röntgen ray sittings the patient complained more of headache or feeling of congestion in the head, and this decided us to discontinue the treatment. The number of white cells in the cubic millimetre has been particularly low at recent counts (see the table), and the spleen has not been so easy to feel. It is possible, therefore, that the Röntgen rays, though they had no influence on the number of red blood cells, may have had a slight tardy effect on the spleen and on the formation of white cells, analogous to that recorded in cases of leukæmia. The Röntgen ray treatment was carried out by Dr. Mülberger, senior house surgeon at the German Hospital, who employed apparatus of Siemens and Halske, of London, with direct street main supply; spark gap, 25 cm.; mercury dip break; 220 volts; 10 ampères; frequent breaks; hard tube (C. H. Müller, of Hamburg, No. 13 with vacuum-regulating apparatus); distance of the patient's skin from anticathode, 50 cm.; diaphragm compressor according to Dr. Faulhaber, of Würzburg. The splenic region was subjected to the treat-

[1] *Cf.* especially A. Wolff, "Theoretisches über die Behandlung der Leukämien und Anämien mit lytischen Methoden durch Röntgenstrahlen und leukolytische Sera," 'Wiener klin.-ther. Wochenschrift,' 1904, No. 49.

ment for five minutes at every sitting, and for one minute in addition every subsequent sitting till ten minutes were reached, after which the time of exposure was not changed. The treatment was carried out regularly, one sitting every day (Sundays excepted) from September 15th to October 8th, 1904, twenty-one sittings altogether. At nearly all of these sittings the lower parts of the legs were likewise exposed to the rays for five minutes.

In regard to future treatment it will be important to observe how the patient progresses now that she has left the hospital; in fact, the question is whether any special treatment is at present advisable or not. In the absence of all evidence that the disease is primary in the spleen, I do not think that the operation of splenectomy can be recommended.

Remarks and Conclusions.

As stated at the commencement of this paper, I regard the present case, in spite of the absence of cyanosis, as similar in nature to my previous case[1] of splenomegalic polycythæmia. The excess of red corpuscles, true plethora ("polyhæmia") and arterial hypertonia were present in both cases. In the previous case the polycythæmia, with the resulting increased viscosity of blood and increased strain on the circulatory mechanism, was, doubtless, of older standing, the patient's vital powers were probably on the decline, and the cyanosis and relatively scanty urine may have been a result of a gradually developing inadequacy of the circulatory mechanism to compensate for the great viscosity of the blood, in spite of the presence of high arterial blood-pressure.

The conclusions arrived at in my paper with Dr. J. H. Watson as to a pathological activity in the production of erythrocytes in the bone marrow being the cause of the blood and circulatory phenomena have, I think, been amply confirmed by the present case. Our theoretical observations on a possible alteration in the osmotic tension of the

[1] Weber and Watson, *loc. cit.*

blood may not have been required to explain the phenomena observed, and, indeed, I shall not discuss that question here, as I have no fresh evidence to offer that there is any special change in regard to osmotic tension to be found in the symptom-complex under consideration.

In my previous case the existence of most extensive bone-marrow changes was proved by examination after death. A great portion of the normal bone-marrow of the shafts of the long bones was found to have been replaced by red bone-marrow, relatively free from fat, in which very active formation of red corpuscles was in progress, as evidenced by the large quantity of erythroblasts. The changes were, however, not exclusively of an erythroblastic kind, and it may be doubted whether the erythrocyte-producing functions of the bone-marrow can ever be greatly increased without the myelocytes being to some extent involved in the unusual activity. Dr. Boycott has pointed out that in the present case an abnormal activity of the bone-marrow may not only account for the excess of red cells in the blood, for the great variations in their size (and for the variations noted in lesser degree, in their shape and staining capacity) and for the presence of nucleated red cells, but likewise for the high percentage of polymorphonuclear leucocytes. These changes have been found in other cases of splenomegalic polycythæmia.

There is no evidence pointing to diminished destruction or lessened wearing out of red blood cells as a factor in the production of the anomalous blood condition.

If the polycythæmia were due merely to concentration of the blood, it would doubtless be only temporary, and the blood would rapidly be diluted by fluid absorbed from the alimentary canal either directly into the blood-capillaries or else into the lacteals to be thrown into the blood-stream by way of the thoracic duct. Moreover, the fact that the total volume of blood in the body is abnormally great precludes the possibility that mere concentration of blood can be the cause of the blood changes. The clinical investigations of these blood changes, as already pointed

out, as well as post-mortem examination, show that in cases of splenomegalic polycythæmia there is increased production of red corpuscles, and I think the evidence is now really conclusive that the symptom-complex is always accompanied by, and at all events mainly due to, a pathological activity in the bone-marrow. Whether the latter condition can or cannot be regarded as the primary factor is a question to which I shall refer later on.

The objection to calling the symptom-complex "primary myelopathic polycythæmia" is that we are not sure that the disturbance of the bone-marrow is necessarily the primary pathogenic factor; whilst the term "myelogenic polycythæmia" is insufficient, for every polycythæmia, excepting temporary states due to mere concentration of the blood, and states (if there really are any) due to diminished destruction of erythrocytes, is "myelogenic" in the sense that the excess of red blood corpuscles is due to unusual activity of the bone-marrow. On the other hand, the term "splenomegalic polycythæmia" only signifies that the spleen is usually enlarged, not that it must necessarily be found enlarged in every case.

I now believe the *order of development of the main symptoms* and their *causal connection* to be as follows:

(1) Increased erythroblastic activity involving a great part, but not necessarily the whole, of the bone-marrow.[1]

(2) Increased viscosity of the blood resulting from the polycythæmia.

(3) Dilatation of small blood-vessels, partly to lessen resistance to the abnormally viscous blood, partly to make more room for dilution of the blood.

(4) The "plethora vera" or "polyhæmia" is probably to be regarded as an attempt to compensate for the

[1] In post-mortem investigations on these cases it would obviously, therefore, be a great mistake to be content with the examination of one portion of the bone-marrow. Part of the shaft of one long bone might be filled with bone-marrow of the ordinary yellow fatty variety, and yet the total active red-cell-forming bone-marrow in the body might be more than three times the normal amount.

increased viscosity of the blood and for the excessive percentage of the total blood-volume occupied by the cells. In fact, it is necessary, firstly, that there should be sufficient blood-plasma to nourish the tissues and make metabolism possible, and, secondly, that the viscosity may not become so great as to render sufficient circulation impossible.

(5) The arterial hypertonia is to be regarded as a result of the greater strain thrown on the circulatory mechanism.

(6) Cyanosis, when this occurs, is probably due to inadequacy of the series of compensatory changes, which, according to my view, precedes it.

In my opinion the evidence afforded by this and other cases strongly supports the foregoing conclusions, but the question now arises, *What is the nature of the pathological activity in the erythroblastic function of the bone-marrow?* At least two theoretical explanations suggest themselves. In the first place that the bone-marrow activity is a primary one, allied to a tumour formation, or the result of an "idiosyncrasy" of the patient. One can suppose, for instance, that some persons have a bone-marrow which reacts to ordinary erythroblastic stimuli to an excessive degree, viz. by throwing almost double the normal quantity of red cells into the blood-stream. If, however, as H. Ribbert[1] believes, there is a form of "myeloma" (that is to say, of growth originating in the elements of the bone-marrow) which should be termed "erythroblastoma," because the tumour-cells are related to erythroblasts, it seems possible that cases of splenomegalic polycythæmia, such as our two cases, may bear a relation to cases of erythroblastoma similar to that which lymphocytic leukæmia bears to lymphocytic myeloma.[2]

The other explanation is that some toxin of a hæmolytic

[1] H. Ribbert, 'Centralblatt für allg. Pathologie,' Jena, 1904, vol. xv, No. 9.

[2] *Cf.* F. Parkes Weber, "A Case of Acute Leukæmia, with a Scheme of Classification of Leukæmias and Pseudoleukæmias," 'Path. Soc. Trans.,' 1903, vol. liv, p. 286.

nature manufactured in the enlarged spleen or alimentary canal is absorbed into the circulating blood in minute quantities, not sufficient to cause much hæmolysis, but in amounts just sufficient to excite reaction in the hæmopoietic (erythroblastic) tissues. Metchnikoff quotes Belonovsky,[1] of St. Petersburg, as having increased both the number of corpuscles and the amount of hæmoglobin in the blood of anæmic persons by the injection of minute doses of hæmolytic serum.

I have just mentioned these possible alternatives in regard to the nature of the bone-marrow activity in splenomegalic polycythæmia, but they are merely theoretical suggestions, and I do not think it will be profitable to discuss them further without additional evidence.

The cause of the splenic enlargement.—If one inclines to the view that the bone-marrow condition in splenomegalic polycythæmia is the result of reaction to toxins circulating in the blood, it is natural to suggest that the enlargement of the spleen is due to the same toxins, whether they enter the circulation from the intestines or elsewhere, or else that there is a primary disease of the spleen, such as tuberculosis (as there actually was in the case of Rendu and Widal[2] and some other cases), which gives rise to a condition of toxæmia to which the bone-marrow reacts (excessive reaction being explained by idiosyncrasy) by an erythroblastic reaction resulting in polycythæmia. In favour of such a view there is the fact that in Saundby and Russell's case[3] of splenomegalic polycythæmia with cyanosis, Dr. Russell saw the patient with an enlarged spleen several years before cyanosis developed. To this I would answer that the polycythæmia was probably likewise present for years before the cyanosis developed, and that cyanosis, if the views I have brought forward are correct, is not an essential part of the symptom-complex.

[1] 'Sur l'Influence de l'Injèction de Diverses Doses de Sérum Hémolytique sur le Nombre des Elements du Sang,' St. Petersburg, 1902.

[2] 'Bulletins de la Soc. Médicale des Hôpitaux,' Paris, 1899, p. 528.

[3] "An Unexplained Condition of Chronic Cyanosis," 'Lancet,' 1902, vol. i, p. 515.

In my present case the enlargement of the spleen is not excessive, and does not appear to be progressive, and there is no fever or reaction to tuberculin to suggest the presence of tuberculosis. Splenic tuberculosis is certainly not necessarily present in cases of splenomegalic polycythæmia and primary tuberculosis of the spleen is not necessarily accompanied by polycythæmia.[1]

Possibly in the cases in which the splenic enlargement is due merely to increase of the pulp and engorgement with blood, as it seems to have been in my previous case, it may be explained as being due to the plethora vera (polyhæmia) and high blood-pressure, or as resulting from excessive functional activity in attempting to compensate for the excessive production of red corpuscles by increased destruction. Of these two last alternatives the former seems to me the most probable, as there is as yet no evidence of greatly increased destruction of red cells in these cases either occurring in the spleen or elsewhere.

Relation of erythromelalgia to polycythæmia.—There is no certain causal relationship, and in the present case the association of the two conditions may have been a chance one. Yet it must be remembered that the association of splenomegalic polycythæmia with erythromelalgia has already been noted by W. Türk,[2] of Vienna. I have elsewhere[3] given my reasons for believing that in the so-called "idiopathic or neuropathic erythromelalgia," as well as what might be termed "symptomatic erythromelalgia" (that is, severe pain and redness in an extremity affected with decided arterial obstruction), there is obstruction to the supply of arterial blood, at all events during the chronic stages. In the present case the muscular atrophy and absorption of bone-salts in the affected extremity[4] at

[1] *Cf.* J. Bayer, "Ueber die primäre Tuberkulose der Milz," 'Mitteil. aus den Grenzgebieten d. Med. u. Chir.,' 1904, vol. xiii, p. 523.

[2] 'Wiener Klinische Wochenschrift,' 1904, Nos. 6 and 7.

[3] 'British Journal of Dermatology,' February, 1904, p. 70.

[4] This was shown by radiograms of the two feet. *Vide* 'Clin. Soc. Trans.,' 1904, vol. xxxvii, p. 250.

one time pointed to local deficiency of arterial blood supply. It is possible that when for any reason the supply of blood to the bone-marrow of a long bone is greatly diminished the bone-marrow may make an attempt to manufacture more blood—in fact, may undergo a hæmopoietic (erythroblastic and leucoblastic) reaction. Through the kindness of my colleague, Dr. Michels, in 1904 I had the opportunity of examining the tibia removed by amputation from an extremity affected by chronic arterial obstruction. There was some red metaplasia of the bone-marrow at the ends of the shaft. In this case, however, the bone-marrow reaction may have been connected with the septic pyrexia preceding the amputation. Dr. J. Galloway also kindly showed me a patient with pain and redness in one foot, undoubtedly due to arterial obstruction, and informs me that in that patient the blood has been repeatedly examined, and the number of red cells has always been found considerably above the normal. Such an increase in the number of red blood corpuscles was, however, not present in another somewhat analogous case, and further information is needed to find out whether chronic ischæmia of an extremity, which can notoriously give rise to local muscular and osseous atrophy, can likewise produce changes in the bone-marrow of the affected part besides those due to absorption of fat-cells.

I have to thank all those who have so kindly assisted me in the examination of this case, without whose assistance I could not have arrived at my few conclusions—in the first place, Dr. J. Haldane, Dr. A. E. Boycott, and Prof. A. E. Wright, and then Dr. Drysdale, Dr. Emery, Mr. Ryffel and Dr. G. L. Eastes, and also (not least) the house physicians at the German Hospital, Dr. Blendinger and Schuh.

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[1] This does not include all the articles referred to in the footnotes.

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polycythæmia with enlarged spleen might, therefore, almost be called "Maladie de Vaquez."

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ADDENDUM.

In cases of splenomegalic polycythæmia the onset of chronic cyanosis probably still further increases both the viscosity and the total volume of the blood. The effect of carbonic acid gas in increasing the viscosity of the blood has been clearly demonstrated by J. Bence ('Deut. Med. Wochenschrift,' April 13th, 1905, p. 590), who states also that, according to Limbeck, an increase in the total volume of the blood accompanies the increase in the viscosity, and adds that, according to Hamburger, this volumetric increase depends on an alteration in the osmotic relation between the red corpuscles and the blood-plasma.

Since writing the present paper, I have come across an interesting account, by W. Weintraud, of three cases of splenomegalic polycythæmia ('Zeitschrift f. klin. Medicin,' 1904, vol. lv, p. 91). The second of his patients suffered also from a condition said to resemble erythromelalgia. In the same case, on one occasion, a remarkable improvement in the subjective symptoms followed a sharp attack of hæmatemesis.

DISCUSSION.

Dr. J. S. HALDANE said that he wished first to congratulate Dr. Weber on the completeness of his investigation, which was evidently a piece of original work of great importance, and threw much new light on the subject. He thought that the facts recorded entirely bore out Dr. Weber's general conclusions as to the pathology of the disease. He did not think, however, that there was evidence of a plethora of blood-plasma. The proportion of plasma in the blood was clearly much diminished, so that in spite of the great increase in the total volume of the blood it might well be that the total volume of plasma was diminished rather than increased. When blood was injected from one animal into the vessels of another animal of the same species plasma rapidly disappeared, so that the percentage of red corpuscles for some time was greatly increased. The physiological tendency to keep the volume of the blood nearly constant seemed to overcome the tendency to keep the proportion of plasma to corpuscles normal, and this had probably occurred in the patient. With regard to the carbonic oxide method of measuring the total blood volume and total hæmoglobin in the body, he might perhaps mention that an experimental comparison of this method with the older method of Welcker (which, of course, implied killing the animal) was being carried out on animals by Mr. Gordon Douglas at the Oxford Physiological Laboratory. Although the investigation was not yet quite complete, the results attained showed that the two methods gave practically identical results. He would like to lay particular stress on the enormous excess in the total hæmoglobin contained in the blood of the patient. The hæmoglobin was most accurately measured in terms of its capacity for absorbing oxygen, and Professor Lorrain Smith and he had found that in normal men the oxygen capacity in c.c. per 100 grammes of body-weight was extraordinarily constant, and only varied in different individuals by about 10 per cent. from the average figure of 0·83 c.c. ('Journal of Physiology,' xxv, p. 340). In this patient the oxygen capacity was 2·7 c.c. There was thus an increase of more than 200 per cent. in the total hæmoglobin. Part of this hæmoglobin might be contained in the bone-marrow, outside the circulation, but in any case the result was very remarkable. Lorrain Smith and McKisack ('Path. Soc. Trans.,' 1902, liii, p. 136) had described a case of old pericarditis with cyanosis, in which the oxygen capacity was 2·0 c.c. In this case, however, the percentage of hæmoglobin was about normal, so that the increase in total hæmoglobin may have been primarily due to an increased blood-volume secondary to backward pressure and consequent increase in the capacity of the blood-vessels. He regretted that it had not been possible to give a fair trial to the therapeutic effects of bleeding the patient copiously, and possibly

repeatedly. By copious bleeding, repeated as often as necessary, it would undoubtedly be possible to keep the percentage of hæmoglobin normal, and he thought that probably the patient would benefit correspondingly. It must not, however, be forgotten that in the case of perfectly healthy individuals living at great altitudes, the percentage of hæmoglobin was commonly increased by about 40 or 50 per cent. without any apparent inconvenience occurring. Curiously enough, he had just received a letter on this subject from Mr. Richards, a Cornish mining engineer, who had recently gone out to manage a mine in Bolivia at an altitude of 15,000 feet, and had undertaken to make observations with a hæmoglobinometer, which had been carefully standardised at the Oxford Physiological Laboratory. His hæmoglobin had already, after a few weeks, risen gradually to 145 per cent., and his red corpuscles to 7,200,000, and he reported himself as feeling particularly well.

Dr. A. J. WHITING said there were many points in Dr. Parkes Weber's interesting paper which called for full discussion. He wished to refer to one. It was a little extraordinary that, although the adjectival term attached to the condition described was "splenomegalic," but little attention was given to the nature of the splenic enlargement present. There was no doubt that under certain circumstances the spleen had marked hæmopoietic functions. It was well known that normally during late intra-uterine and early extra-uterine life the spleen was a seat of the formation of red blood corpuscles. After bleeding animals copiously, the abstraction of blood being held to be an excitant of the hæmopoietic power of the body, the bone-marrow showed marked evidence of increased activity, but the same was also to be found in the spleen. His own experiments had been made mainly on dogs. The spleen in these animals became swollen, tense, of a rosy red colour, and more friable than natural after hardening. Microscopically, it showed large numbers of nucleated red cells, erythroblasts, and giant cells. Dr. Parkes Weber had referred to the presence of giant cells in the bone-marrow of his earlier cases; his own experience was that wherever blood-formation was taking place giant cells were to be found. In the spleens in question, while normally one or two might be seen in a section, after artificial hæmorrhage a thousand might be counted, and this seemed to offer a partial explanation of the mechanical increase in size of the organ. He had been interested to see that in regard to a recent case of leukæmia recorded abroad, in which large numbers of nucleated red cells were present in the blood, the author had said the splenic pulp was almost indistinguishable from the bone-marrow, and this was really so. He would like to ask if Dr. Parkes Weber had examined the spleen of his former case microscopically, and if so what were its characters?

Dr. A. E. Boycott, speaking from the point of view of the histology of the blood alone, considered that the condition described was a clinical entity. Apart from the increase in the hæmoglobin and in the number of cells he had found marked variation in size between individual red-blood corpuscles, their staining reactions were altered, and a small number of nucleated red-cells were constantly present. Polymorphonuclear neutrophile leucocytes were increased; this also indicated an increase in the activity of the bone-marrow.

[By permission of the President the two following letters, in the unavoidable absence of the writers, were admitted as contributions to the discussion.]

May 8th, 1905.

Dear Dr. Parkes Weber,—Let me thank you very warmly for your kindness in sending me the proof of the paper you are about to read to-morrow on "'Splenomegalic' or 'Myelopathic' Polycythæmia." When the subject first emerged, a few years ago, it seemed to me that it might be simply a form of cyanosis due to disturbance of the circulation by one of the many mechanical factors which may disturb the equilibrium. The consideration, however, of certain of the more recently published cases, the careful examination of an instance of the disease under the care of one of my friends here, which he recently published in the 'Edinburgh Medical Journal,'[1] and, above all, the study of your own work upon the subject, have led me to the conclusion that a purely mechanical hypothesis will not account for the phenomena. Your last paper has convinced me that increased blood-formation is the real cause of the disease, and I have no hesitation in accepting your suggestion of increased erythroblastic activity of the bone-marrow. Your conception of the order of development of the main symptoms appears to me to be perfectly correct, as increased viscosity of the blood must lead to increased strain on the circulation and to real plethora as an attempt to compensate for the viscosity.

G. A. Gibson.

Dear Dr. Parkes Weber,—A year and a half ago I had under my care at Westminster Hospital a case of splenomegalic polycythæmia, which presented many points of similarity with the case described in your paper.

The patient was a woman, aged 53, who had apparently been in good health until the time of the menopause, five years previously. At that time she had an attack similar to the one for which she was admitted to hospital, consisting of headache, vomiting, and giddiness, with marked weakness of all four limbs, lasting about a week; there was no loss of consciousness. In

[1] Ronaldson, 'Edin. Med. Journ.,' vol. xvi, 1904, p. 244.

about two months the patient appeared to recover completely. Three years before admission she had a second attack, similar to the first, but this time she was confined to the house for six months, and became permanently weak and unable to work. A year before admission she noticed a dragging pain in the left side of the abdomen, doubtless associated with the splenic enlargement. She noticed the abdomen to be red and swollen at this time. The pain passed off and did not recur.

Five weeks before admission the attack commenced for which she sought admission to hospital. It commenced with intense headache, dimness of vision, more marked in the right eye, and vomiting unassociated with food. Occasionally she noticed a little blood in the vomited material. She lost weight considerably during the five years of her illness.

On admission she was found to be somewhat emaciated; her lips were cyanosed, and the face had a dull, leaden flush, with dilated venules. The hands and feet were slightly red. The tongue and buccal mucosa were bright red. There was no dropsy or jaundice. She had numerous minute bright-red nævoid elevations, scattered over the skin of the trunk. Her skin was dry. No enlarged lymphatic glands could be detected anywhere. The heart was normal, save for some accentuation of the aortic second sound; the pulse was regular, of high tension and with thickened arterial walls. The superficial veins of the abdomen were markedly dilated and tortuous. The spleen was markedly enlarged, its notched border extending to one inch on the left of the umbilicus, the lower border reaching a level two inches below the umbilicus. The right lobe of the liver was also markedly enlarged and palpable, the vertical dulness extending from the fifth space in the nipple line to four inches below the costal margin. There was no ascites. The urine had a sp. gr. of 1024, was high-coloured, neutral in reaction, loaded with albumen, and microscopically showed granular, hyaline, and cell casts. There was double optic neuritis with retinal hæmorrhages. The nervous system at that time was otherwise normal. There was no tenderness of any of the long or flat bones, nor any joint affection. The blood was repeatedly examined by Dr. Hebb. It was very dark in colour, and of a viscous consistence. On October 23rd the erythrocytes were found to number 8,750,000 and the leucocytes 24,000 per cubic mm. (polymorphs 92 per cent., large monomorphs 2 per cent., small monomorphs 6 per cent.) On October 28th another count showed the red cells to number 10,333,000, the whites 23,400. The hæmoglobin was 130 per cent. The polymorphs were 94 per cent. granular (mostly finely granular), the monomorphs 6 per cent.

The patient left hospital against advice, and subsequently was admitted to Wandsworth Infirmary, where she died in April, 1904, five months after her admission to Westminster.

Shortly after she left Westminster she had an attack of right hemiplegia, with aphasia, apparently due to cerebral hæmorrhage. She gradually became totally blind in both eyes. The aphasia passed off, but the hemiplegia persisted. No autopsy could be obtained.

At the time when this patient was under my observation it was obvious that her case corresponded to the class described by Osler in the 'American Journal of the Medical Sciences' (August, 1903, p. 187). In connection with the erythromelalgia which has been noted by you in your case, my patient frequently observed that her hands and feet never felt cold, and they were certainly persistently flushed.

PURVES STEWART.

Dr. PARKES WEBER, in reply, said, in regard to Dr. Haldane's recommendation of very copious bleeding, that perhaps several smaller bleedings might be as effective as one large one, and would not be so terrifying. A single small bleeding may doubtless, however, be ineffectual. A few hours after one small bleeding in a patient (then under Dr. Weber's care) with chronic cyanosis, polycythæmia, and increased viscosity of blood, probably partly of pulmonary origin, an apparent increase in the number of red blood corpuscles was observed—that is to say, in the blood from pricking the finger as compared with the number in the venesection blood of a few hours previously. It seemed to be almost an impossibility to measure the amount of the plasma in the thick, sticky blood of extreme cases of polycythæmia, a great part of the plasma remaining, even after thorough centrifugalisation, in the interstices between the red corpuscles. In regard to Dr. Whiting's question, the enlargement of the spleen in his (Dr. Weber's) previous case of splenomegalic polycythæmia, which was examined microscopically, was due to increase in the ordinary splenic pulp, and largely, he thought, to engorgement of the organ with blood. He compared the myeloid transformation, mentioned by Dr. Whiting, in the spleen of animals rendered anæmic by repeated bleedings, to the myeloid change found in the spleen from a case of leukanæmia, which he had described in the 'Transactions' of the Pathological Society of London (1904, vol. lv, p. 287). In that case both the spleen and the liver were found to be crammed with erythroblasts and other cells of the bone-marrow kind (the liver containing numbers of giant cells), whilst the relatively small number of these bone-marrow elements present in the circulating blood during life made it probable that their presence in such great abundance in the spleen and liver after death was not merely the result of a metastasis or infiltration of these organs from the bone-marrow, but was to be regarded as representing a true myeloid transformation of the organs in question.

ACTINOMYCOSIS OF THE GALL-BLADDER

BY

A. W. MAYO ROBSON, D.Sc., F.R.C.S.,

SENIOR VICE-PRESIDENT ROYAL COLLEGE OF SURGEONS OF ENGLAND

Received November 10th, 1904—Read April 11th, 1905.

So far as I can ascertain by a search through medical literature bearing on the subject, no case of actinomycosis of the gall-bladder has hitherto been recorded.

The disease in this situation therefore is rare, though when it is borne in mind that no part of the body is exempt from the ravages of the fungus and that, next to the mouth and its neighbourhood, the abdomen is the most frequent site of disease, one might have reasonably expected that, like the appendix, the gall-bladder would occasionally have become invaded from the intestine.

In the case now under consideration it is, of course, a mere conjecture as to the fungus having been swallowed and having found a nidus in the duodenum, whence it had invaded the biliary passages through the common duct, for it may have invaded the gall-bladder by direct continuity from the intestine. This, however, I think is improbable, as otherwise, when the gall-bladder had been opened, curetted, and drained, I think a fæcal fistula would have formed.

The invasion did not occur through the skin, and as

there was no evidence of any other lesion, I do not think it likely that it occurred either through the blood or lymphatics.

As in other successfully treated actinomycoses, the treatment was at first surgical and subsequently medical. The history of the case is as follows:

Frederick N—, aged 47, was sent to me by Dr. Dickey, of Colne, in July, 1901, on account of a tumour in the region of the gall-bladder associated with severe pain and loss of flesh.

He gave the history that for eighteen months he had been losing weight and feeling ill; at first he had neither pain nor any signs by which he could localise his disease. After a time he began to have occasional attacks of pain in the region of the liver, but until three months before seeing me the general symptoms of malaise only engaged his attention.

For three months he said that he had never been a day free from bad attacks of colic and between these seizures there was a constant aching pain which he described as inflammatory, and in fact the tenderness on the right of the abdomen at first led to a suspicion of appendicitis. There had been neither jaundice nor vomiting and his bowels were regular.

When I saw him he looked pinched and ill, and on palpating the abdomen a decided swelling was at once found beneath the right costal margin, extending two inches below the level of the umbilicus. The tumour was continuous with the right lobe of the liver, was tender on pressure, and felt hard and irregular. The stomach was somewhat dilated, but no succussion splash was obtained. There was no jaundice and no evidence of ascites could be obtained.

A diagnosis of cholecystitis was made, and on July 19th I made an incision through the right rectus on to the swelling, which I found to be an enlarged gall-bladder adherent to the abdominal wall and containing pus and granulation-like material, with here and there suspicious

yellow particles in it. The abdominal wall was not invaded, but the omentum, gall-bladder and right border of the liver were intensely bound together and invaded by the same granulation-like tissue. By means of a blunt-edged scoop the cavity was scraped out and afterwards irrigated, a moderate-sized drainage-tube being inserted and the rest of the wound closed. His progress was uneventful, the temperature and pulse being normal throughout convalescence. The tube was shortened and removed in the second week, when the wound was packed with gauze dipped in hazeline, and he returned home on August 10th, 1901.

The following is a Report by Dr. Maule Smith of the material removed at the operation: "Films show numerous filaments, some radiating from a common centre, others broken off. At their ends they show thickenings (clubs). There are some rounded bodies which stain more darkly and are probably coccus bodies. Diagnosis—Actinomycosis."

Iodide of potassium was prescribed on July 24th and continued for some length of time. On November 11th, 1901, I had a letter from Dr. Dickey to say that N— was well and going regularly to his work.

In order to ascertain the present condition of the patient I wrote to Dr. Dickey in November, 1904, and on the 6th I received a letter to say "N—, whom you operated on in July, 1901, has got on splendidly since. He never had a return of the severe colic, which generally used to last two or three days."

DISCUSSION.

The PRESIDENT.—Cases of actinomycosis of the gall-bladder must be very rare. Can Mr. Mayo Robson give us any information as to their prevalence?

Mr. MAYO ROBSON.—As far as I have been able to ascertain no other case has been recorded.

ADENOMA OF THE GALL-BLADDER

BY

A. W. MAYO ROBSON, D.Sc., F.R.C.S.

SENIOR VICE-PRESIDENT, ROYAL COLLEGE OF SURGEONS OF ENGLAND

Received February 28th—Read April 11th, 1905

ADENOMA of the gall-bladder has been described as a pathological curiosity; hence I believe that the case I am about to relate is sufficiently uncommon to merit being reported.

The only other cases of which I have been able to find a description are one of Dr. L. R. Sutherland's, in the museum of the Western Infirmary, Glasgow; one of Wiedmann's, referred to by Terrier and Auvray; and one of Stanmore Bishop's, described in the 'Lancet' for 1901.

The interesting point in my case is that the tumour produced symptoms exactly resembling gall-stones, characterised by spasmodic pains in the gall-bladder region, transient icterus, and deterioration of health. These might either have been due to the tongue-like process of the growth acting as a foreign body, or to the chronic catarrh set up by the neoplasm. The case is as follows:

Miss C—, aged 53, seen with Dr. Waterhouse on the 3rd of October, 1904. The patient, otherwise healthy, had been subject to attacks of pain beneath the right costal margin for some months, the seizures recurring

frequently, and being associated with slight jaundice. Before my seeing her, the attacks had occurred more frequently, and the pain had radiated round the right side to a spot beneath the right shoulder blade. A physical examination showed a slight enlargement of the right lobe of the liver, but the gall-bladder itself could not be definitely made out. There was slight tenderness in the usual position an inch above and to the right of the umbilicus in a line between the umbilicus and the ninth costal cartilage. As the patient had been under medical treatment for gall-stones for some time without obtaining any relief, an operation was advised. Operation 19th of October. A sand bag was placed under the loin, and my usual vertical incision for cholecystotomy was made, which enabled the gall-bladder to be thoroughly exposed. On opening the abdomen I found a thickened gall-bladder under cover of an elongated right lobe of the liver, but no gall-stones could be felt either in it or in the ducts. On incising the thickened fundus the incision was made through a multilocular cystic tumour occupying the wall of the gall-bladder. It then became evident that the fundus of the gall-bladder was occupied by an adenomatous tumour, which is shown in the photograph. The loculi contained some particles of cholesterin. The cystic duct seemed to be narrowed, and the remainder of the gall-bladder was small and thickened. Cholecystectomy was therefore performed in the following manner:—The cystic duct was grasped within half an inch of the common duct by means of a pair of pressure forceps. The peritoneum of the duct was incised one third of an inch above this, and, after stripping the peritoneum off the duct, the duct was divided and ligatured close to the forceps. The peritoneum connecting the gall-bladder to the liver was divided one third of an inch from its attachment to the liver. The gall-bladder was then separated from the liver by the finger, working from below upwards, leaving about one third of an inch of its peritoneal covering connected to the margins of the gall-bladder fissure

of the liver. The peritoneum covering the cut end of the cystic duct was then brought together by a ligature, so as to bury the ligatured duct. After this the peritoneal margin of the gall-bladder fissure were stitched together from side to side, so as to completely obliterate the groove in the liver where the gall-bladder had been attached. As no soiling of the peritoneum had occurred and all oozing had ceased, it was not thought necessary to adopt any drainage, and the wound was closed in the usual way. The after-history gave nothing special to record. The wound healed by first intention, the patient lost all her pain, and left the nursing home at the end of the third week.

The specimen, of which a photograph is appended, is now in the Hunterian Museum at the Royal College of Surgeons, and for the micro-photographs of sections of the tumour I am indebted to Dr. P. J. Cammidge.

Mr. Mayo Robson's Adenoma of Gall-bladder.

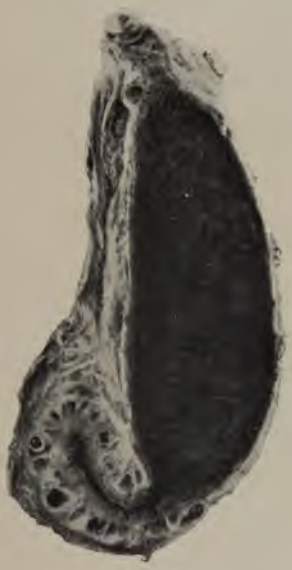

FIG. 1.

FIG. 2.

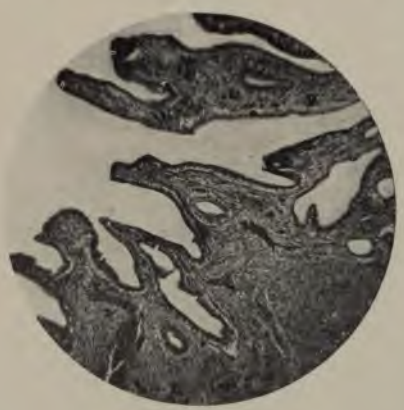

FIG. 3.

Adlard & Son, Imp.

THE OPERATION OF JEJUNOSTOMY,

WITH A DESCRIPTION OF A NEW METHOD

BY

A. W. MAYO ROBSON, D.Sc., F.R.C.S.

SENIOR VICE-PRESIDENT OF THE ROYAL COLLEGE OF SURGEONS OF ENGLAND

Received November 4th, 1904—Read April 11th, 1905.

JEJUNOSTOMY is an operation occasionally called for as a means of giving relief and prolonging life in patients suffering from advanced disease of the stomach, where on exploration it is discovered to be impracticable to perform gastrectomy, gastrostomy, or gastro-enterostomy. The indications for the operation are:

1. Extensive cancer of the stomach too advanced for gastrectomy, and in which no healthy spot of sufficient size on the stomach wall can be found for the purpose of gastrostomy or gastro-enterostomy.

2. General cicatricial contraction of the stomach, simple in character and due to the swallowing of caustic fluid, in which the stomach has been so far damaged that it no longer performs its functions or even allows of the proper passage onwards of food.

It has also been suggested in pronounced hyperchlorhydria, in preference to gastro-enterostomy in order to avoid peptic ulcer of the jejunum; but as the latter is extremely rare and practically only associated with anterior gastro-enterostomy, a method that is being replaced by

the posterior operation, I do not think surgeons generally will be likely to endorse Neumann's suggestion. I do think it probable that jejunostomy will be likely to replace

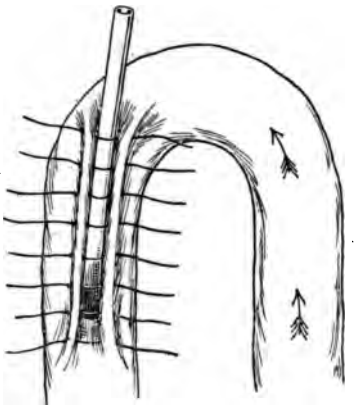

FIG. 1.—A method of performing jejunostomy by a modification of Witzel's operation for gastrostomy. First stage.

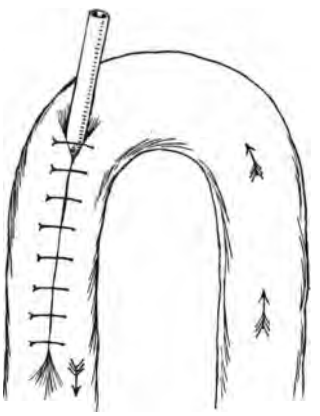

FIG. 2.—Second stage of Fig. 1.

other methods of securing rest in the treatment of gastric ulcer, or of hæmatemesis, as Cackovic has suggested, and as has been adopted on one occasion with success by Mr. Willoughby Turner.

Modes of Operation.

For any operation to be a success the bowel must be so placed that it will serve the two purposes (1) to permit the passage onward of the bile and pancreatic fluid poured into the intestine above the artificial fistula, (2) to allow of food being introduced through the fistula without fear of regurgitation, either of the food or of the intestinal contents.

In my first operation, performed in 1891, I used a

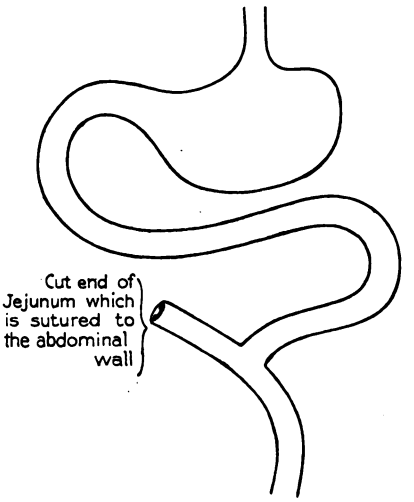

FIG. 3.—Jejunostomy by Maydl's method.

method which though successful in prolonging life was inconvenient and cumbersome, and I need not mention it further. The operations now used are :

1. A modification of Witzel's method of gastrostomy in which a No. 12 rubber catheter is stitched into an opening in the jejunum, and afterwards the catheter is buried in a groove in the bowel for a distance of about two inches, the line of suture being fixed to the abdominal wall (see Figs. 1 and 2).

2. Maydl's method of dividing the jejunum, implanting

the proximal cut end into the distal portion a few inches from the original line of section, the open end of this section being fixed to the skin (see Fig. 3).

3. The operation I have performed, and which I believe is new, consists in taking a loop of the beginning of the jejunum just sufficiently long to reach the surface without tension; the two arms of the loop are short-

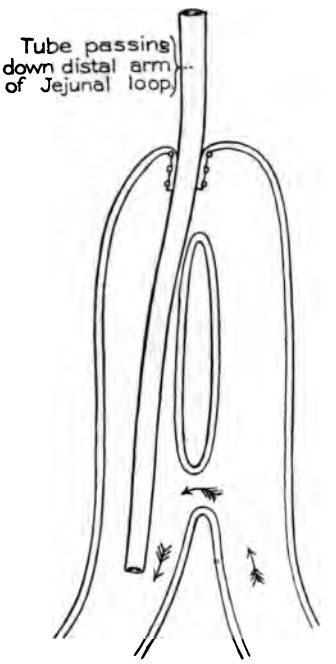

FIG. 4.—Jejunostomy by the author's method now described.

circuited about three or four inches from the surface, the short circuiting being done either by means of sutures around a decalcified bone bobbin or by sutures alone; personally I prefer the former.

A small incision is then made into the top of the loop just large enough to admit a No. 12 Jaques catheter, which is inserted and passed for three inches down the distal arm of the loop; this is fixed to the margin of the

incision in the gut by a silk or Pagenstecher's suture, and the entrance of the tube into the bowel is further guarded by two purse-string sutures, one over the other. The top of the loop is fixed to the skin by one or two stitches and the wound closed. The patient can then be fed at once with some peptonised milk and brandy. The whole operation can be done in from fifteen to twenty minutes and with very little visceral exposure (see Fig. 4).

Should the patient be too ill to bear the little extra time occupied by the short circuiting, the tube may be inserted as directed and surrounded by two or three purse-string sutures, a proceeding which can be accomplished in a few minutes. In this case the loop of bowel must not be brought to the skin, but had better be fixed by sutures to the peritoneal margin and the aponeurosis, in order to leave part of the lumen of the attached loop within the abdomen for the direct passage onwards of the intestinal fluid with the bile and pancreatic secretion.

The following case affords an example of the relief that may follow this operation :

Mrs. C—, aged 46, the wife of a sea captain, was sent to see me on April 8th, 1904, when she was suffering great pain, which came on at frequent intervals, and she was vomiting five or six times a day ; in fact, whenever she took food it was shortly vomited, the vomit being at times coffee-ground in character. A tumour in the epigastrium about the size of a large flat orange could be readily felt, and at short intervals the whole stomach became hard and rigid. No enlarged glands could be felt in the groin or above the clavicle. There was no tenderness on pressure, and the hard, nodular tumour was suggestive of cancer. Though she gave a history of indigestion and loss of health for eight years or even longer, the acute symptoms had only existed for six months.

On April 13th the abdomen was opened by a vertical incision through the inner margin of the right rectus, when the stomach was found to be involved in cancer from

end to end, and as the glands along the lesser curvature were involved and others could be felt passing up through the opening in the diaphragm and a number also in the great omentum, it was clearly impossible to perform gastrectomy and impracticable to do a gastro-enterostomy. A loop of jejunum was therefore brought up and short-circuited by suture over a decalcified bone bobbin, and a No. 12 Jaques catheter was inserted as just described. The wound was rapidly closed and the patient was put to bed in good condition, the whole operation only having occupied half an hour or less. A meal of peptonised milk was given at once and repeated every two hours. From the time of operation up to now the vomiting has never been repeated, and she has completely lost her pain. On April 28th, fifteen days after operation, I wrote to Dr. C—, "Mrs. C— has made a good recovery and is now able to take a little food by the mouth, though the feeding is chiefly by the tube. She is free from pain and the tumour is less." She returned home on the nineteenth day, having gained flesh and strength.

After the patient's return home, on May 17th I received a reply from Dr. C—: "Mrs. C— is doing wonderfully well. No pain or sickness, and taking as much food as she requires. She is very content, and is, I think, gaining flesh and strength, so that the result is even better than you had hoped for and much better than I ever expected. She is very grateful."

On October 10th I had a letter from Dr. S— to say that Mrs. C— had come under his care in North Devon, and expressed herself as much improved, though there was a tumour manifest.

The case is of interest, not only from the relief afforded by jejunostomy, but also as showing the sequence of cancer on ulcer, a not uncommon incidence, and one which might be prevented by timely intervention in the pre-cancerous stage. It is now clearly proved that ulcer of the stomach is a much more serious disease than was once thought and that many cases fail to yield to medical

treatment, but I do not think it is fully recognised how frequently cancer follows on ulcer.

In no less than 59·3 per cent. of the cases of cancer of the stomach on which I have operated, the history, as in this case, clearly points to antecedent ulcer, the cancer being a later manifestation.

The operation of jejunostomy, which has a mortality of from 10 to 20 per cent., should be very seldom called for, and would not be if the early diagnosis and treatment of gastric cancer were insisted on; but it must be distinctly recognised that if we intend to do the best for these cases we must make the diagnosis before tumour or coffee-ground vomiting leave no doubt as to the nature of the ailment.

Whenever a patient at or after middle age complains somewhat suddenly of indefinite gastric uneasiness, pain and vomiting followed by progressive loss of weight and energy, associated with anæmia, the possibility of cancer of the stomach should be recognised, and if no improvement takes place in two or three weeks an exploratory operation is more than justified. Our diagnosis can only be rendered certain by a digital examination, which may be effected through a small incision that can if needful be made under cocaine anæsthesia with little risk. Let us not forget that the early recognition of cancer of the stomach means not only the probability of relief but the possibility of cure in a very considerable proportion of cases.

A SERIES OF 123 CASES OF CHOLEDOCHOTOMY FOR CALCULI IN THE COMMON DUCT

BY

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On March 25th, 1902, I read a paper before this Society on "The Surgical Treatment of Obstruction of the Common Bile-duct by Concretions, with especial reference to the advantages of an operation for the more complete exposure and treatment of the deeper parts of the biliary passages," which I then described. The advantages of the modifications suggested have since been accepted and freely acknowledged by many operators both at home and abroad. I hope that the report of an additional series of operations on the common duct for the removal of gall-stones by this method will afford me a sufficient excuse for inflicting on the Society another paper on the same subject.

The operation is fully described in the 'Transactions' of the Society for 1902, and in the third edition of my work on "Diseases of the Gall-bladder and Bile-ducts," so that I need not further describe it now.

When I began to employ this operation the mortality

of choledochotomy including every case, whether complicated or simple, was 16·2 per cent. (21·4 per cent. in the first series, and 9·3 per cent. in the second series, or together 16·2 per cent.), but since adopting the operation for complete exposure of the bile passages, I have performed a continuous series of 76 choledochotomies for gall-stones with only three deaths, a mortality of 3·9 per cent., thus amply verifying the forecast that I then made, when I stated that I expected the mortality would be reduced to 5 per cent. or less.

After I had had 53 consecutive recoveries from choledochotomy, nearly all the patients being deeply jaundiced and extremely ill at at the time of operation, and a number of them being aged and very feeble, I began to hope that I might be able to reduce the mortality of the common duct operation to the same figure, 1 per cent., which has been my experience in the more simple operation of cholecystotomy for the removal of gall-stones from the gall-bladder and cystic duct, in the absence of cancer and suppurative or infective cholangitis ; but this has not yet been realised, for the 54th, 59th, and 61st cases proved to be desperate ones, in which immediate operation gave the only chance of life. The first case, a lady aged 65, was suffering from pyæmia at the time of operation, having had two rigors a day for several days ; and, besides a suppurating parotitis, she had serious heart disease and albuminuria ; on opening the common duct extremely offensive pus and bile flowed from the ducts in the liver. The next patient, a gentleman of 65, had suppurative pancreatitis with high temperature and gangrene of the gall-bladder, accompanied by rigors and septicæmia at the time of operation. He also had albuminuria and cirrhosis of the liver, the last being probably the cause of death, from symptoms resembling those of acute atrophy of the liver a fortnight after operation.

In both of these cases not only the *Bacillus coli communis* but also *Streptococcus pyogenes* were found in the pus from the common duct.

The third case occurred abroad, and although I had gone a long distance to operate I said I thought the patient too ill to bear surgical treatment, but when I was about to leave the next day, intending to see her again after a trial of general means, I got a message from her medical attendant to say that unless I could see my way to operate she would certainly die. She was aged 53, and had been operated on and gall-stones had been removed from the gall-bladder by another surgeon sixteen months previously, but she had never completely recovered. Recently she had had rigors and deep jaundice associated with profound weakness. After removing a large gall-stone from the common duct I found the passage into the bowel so free that I thought I could do without drainage of the duct. The immediate progress after operation was all that could be desired and I left feeling very hopeful, but the jaundice became more intense at the end of a week, and the patient died a fortnight later, choleæmic. I suspect that a secondary interstitial pancreatitis was responsible for the jaundice and for the untoward ending. It would have been better to drain the common duct in this case as I generally do when I cannot drain the gall-bladder, and had I done so I think the result would have been different.

In several cases I have found gall-stones in the common duct where there was no jaundice, and where, except for the rule that I always follow, to carefully examine the common duct in every gall-stone operation, I should inevitably have left the patients unrelieved and probably with a biliary fistula, which never follows operation if all obstruction is removed. On five occasions I have removed gall-stones from the common duct, although the patient would not confess to having had any pain, the operation being undertaken for deep jaundice associated with frequent rigors and high temperature. As these cases were all in men who had resided in India or in tropical countries and who had had fever, they naturally believed themselves to be suffering from ague, a diagnosis which

was supported in three of the cases by the presence of enlarged spleen, and in one by the discovery of ague organisms in the blood. The removal of gall-stones from the common duct and drainage of the bile-ducts led to recovery without any fever in all the cases.

In more than half the cases of common-duct cholelithiasis, pancreatic catarrh or interstitial pancreatitis complicated the gall-stone trouble, and was cured by the drainage of the bile-ducts.

In a number of cases where the jaundice had existed for a long period, cirrhosis of the liver was found to be present. In one case, well-developed ascites existed at the time of operation, and the omentum was at the same time stitched to the abdominal wall to establish a collateral circulation, which it did so effectually that the patient is well and has had no return of fluid in the peritoneum.

Quite a number of the patients have been advanced in years, so that age, even up to seventy or beyond, seems to be no barrier to a successful operation.

Drainage of the biliary tracts in common-duct cholelithiasis is always advisable, as the bile is infective and the head of the pancreas is frequently swollen; but when I am able to use the gall-bladder for the purpose I prefer to drain it rather than to drain the common or hepatic duct directly. If, however, the gall-bladder is seriously diseased or shrunken, or if it be acutely inflamed or gangrenous, I prefer to remove it and then to insert a No. 12 Jacques' catheter into the common duct for drainage.

Although the disease is essentially a septic one from infection of the bile in the ducts, I am quite sure that the nearer complete asepsis can be reached by the operator so much the better, for under such circumstances these cases nearly always pursue a completely aseptic course subsequent to operation. In order to effect this I now always operate in boiled rubber gloves and observe rigidly the usual precautions, with the result that primary union is the rule, and that all signs of the ailment being of a septic nature disappear immediately after operation.

It has to be borne in mind that common-duct cholelithiasis is, without operation, usually a fatal disease, and that nearly every recovery is a life saved; it affords, therefore, great satisfaction to be able to tell patients suffering from so serious an ailment that by means of operation their chances of recovery are at least 95, and may be 97 or 98 per cent.

DISCUSSION

The PRESIDENT alluded to the presence of gall-stones in the common duct without jaundice, and asked for an explanation. He also asked what extent of surface of adhesion between the surface of the liver and the abdominal wall he considered adequate to effect relief of the ascites? Was the capsule of the liver removed in the cases in which the liver was fixed to the abdominal wall on the analogy of decapsulation of the kidney?

Mr. J. WARRINGTON HAWARD emphasised the importance of examining for gall-stones in the common duct even though jaundice were not present; he referred to two cases occurring in his own experience. He testified to the value of the operation of choledochotomy as advised by Mr. Robson. In one case of ascites, due to cirrhosis of the liver, he had obtained the advantages of a collateral circulation by securing adhesion of the surface of the liver to the abdominal wall.

Mr. C. B. KEETLEY referred to a case of a man, who had suffered from hydatids of the liver for years, who was taken acutely ill with signs of suppuration in the upper abdomen. Laparotomy was performed, an abscess was found under the liver, behind which were the hydatids. For some time apparently all the bile secreted came away from the wound, suggesting that the abscess might be due to perforation of the common bile-duct, possibly by a stone.

Mr. MAYO ROBSON, in reply, said he had frequently found gall-stones in the common bile-duct without jaundice when the duct was dilated, and the explanation was probably that the stone had not become impacted. The absence of pain in some of the cases was even more remarkable. For the relief of ascites he usually stitched a loop of omentum of two or three inches in extent to the incision. He had never removed the capsule of the liver. By thoroughly exposing the common duct and freeing adhesions stones could as easily be removed from it as from the gall-bladder. Might not the abscess in Mr. Keetley's case have been of the dilated common duct, the dilatation being due to the hydatids?

A CASE OF CHRONIC JAUNDICE AND GREAT ENLARGEMENT OF THE LIVER DUE TO PRIMARY CARCINOMA OF THE EXTRA-HEPATIC BILE-DUCTS COMMENCING AT THE JUNCTION OF THE HEPATIC DUCTS

BY

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THE patient, George W—, aged 35, was admitted into the German Hospital on July 24th, 1904, for deep jaundice and enlarged liver.

The jaundice commenced early in July, without pain or any gastric disturbance. Dr. Leonard Williams, who saw the man in the Out-Patient Department two weeks or so before his admission, says that at first the liver appeared not to be enlarged.

There was no history of gall-stones or previous jaundice, and the general health was said to have been good. There was no evidence of syphilis, alcoholism, or hereditary tendency to disease of the liver.

Condition in the hospital.—The jaundice was extremely deep. The liver was greatly and uniformly enlarged, extending downwards on the right side of the abdomen to

the anterior superior spine of the ilium. It seemed hard, and its surface was smooth. The spleen was also somewhat, but not greatly, enlarged. The fæces were invariably colourless, and the urine deep brown from the presence of bile. It was free from sugar, and practically free from albumen, but, as in most cases of deep chronic jaundice, it showed a marked "cloud" of nucleo-albumen on adding acetic acid, either in the cold or after boiling. It likewise contained the usual golden-orange pigmented cells and a few hyaline tube casts, as urines of severe jaundice always do.[1]

Apart from the jaundice, the patient seemed to be in fairly good health. There was never any xanthoma, practically no prurigo, and no bleeding from the gums or tendency to cutaneous hæmorrhages. The only peculiarity, apart from the disease for which he was admitted, was the very tall or "turriform" shape of his head (German, "Turmschädel"), which, however, has no bearing upon

[1] It is perhaps only in the most extreme degrees of jaundice, with chronic obstruction in the common bile-duct, that the urine gives the very marked reaction for a "mucinoid" substance, or a nucleo-albumen, to which one of us alluded in 'Path. Soc. Trans.,' 1900, vol. li, p. 176. On the other hand, in all, or nearly all, cases of jaundice, with complete obstruction to the escape of bile (even when of relatively short duration), casts can be found in the urine, if carefully searched for, though albumen be absent. These casts may contain pigment granules and pigmented cells. Nothnagel ('Deut. Arch. für Klin. Medicin,' 1874, vol. xii, p. 326) thought that in every marked case of jaundice, whatever the cause of the jaundice may be, casts appear in the urine. (*Cf.* Dr. P. S. Wallerstein, "Ueber reine Cylindrurie bei künstlich erzeugter Gallenstaung," 'Berliner Klin. Wochenschrift,' 1902, No. 14, p. 310.) Zeri ('Il Morgagni,' October 1904), from observations of thirty cases of jaundice, concluded that renal epithelial cells and casts formed from degenerated renal epithelium could frequently be found in bilious urine, even in the absence of any albumen. He thought that the epithelial cells and casts in the urine of icteric cases signified damage to the renal tubules, whilst albuminuria, when it occurred in such cases, pointed to damage of the glomeruli. In the present case we were able to note the disappearance (except for the ordinary faint trace) of the nucleo-albumen from the urine when the biliary retention was relieved. It is possible that the nucleo-albumen of bilious urines may like the bile-pigments, be derived from the bile-passages.

the subject of our paper. Nothing abnormal was noted in the thoracic organs. There was no enlargement of any of the accessible lymphatic glands. Examination of the blood (September, 1904) gave the hæmoglobin value as 80 per cent. of the normal. The red cells were 3,880,000 in the cubic millimetre and the white cells 7000.

Owing to the possibility of the obstruction to the outflow of bile being due to a syphilitic lesion at the hilum of the liver, prolonged treatment by iodide of potassium was tried, but with no obvious result. Olive oil given by the mouth likewise had no effect.

On October 7th, 1904, Dr. Michels performed an exploratory laparotomy, with the object of discovering the cause of the biliary obstruction, and remedying it if possible. At the operation the liver was found very large and rather hard; it had a dark congested appearance; its surface was not granular. No biliary calculus was felt after full exposure of the bile-ducts, nor could Dr. Michels make out any swelling or tumour in the pancreas, or at the hilum of the liver, which might be compressing the extra-hepatic bile-ducts. There were no adhesions about the gall-bladder, which was small, not much distended, and so far behind the edge of the liver that cholecystostomy could not have been safely undertaken, even had it been otherwise desired. Neither the common bile-duct nor any of the bile-ducts which could be felt and seen outside the liver were distended.

The patient did not appear to suffer from the operation, though for a time the jaundice seemed still more intense. The wound healed fairly rapidly, and he was soon able to get about again. The jaundice and his general condition were apparently about the same on November 5th, 1904, when he left the hospital, as they were on admission.

He was re-admitted on November 24th, 1904, with the idea that an operation might be performed to drain off the bile externally. The complete and constant absence of bile from the bowel and the history of the case made it practically certain that the condition of the liver was

due, not to a form of Hanot's cirrhosis nor to any form of cholelithiasis, but that it was caused by complete obstruction in the large bile-ducts from a cause which could not be ascertained at the exploratory operation in spite of careful examination of the region of the transverse fissure. (The result showed that the tumour causing the jaundice must at the time of the first operation have been very small, and situated deep in the transverse fissure at the junction of the two hepatic ducts.) It was finally decided, after a consultation with Dr. zum Busch, to establish a biliary fistula by the method suggested by M. Hirschberg[1] and J. Rotter.[2]

This operation was performed by Dr. Michels on December 13th, 1904.[3] He made a small incision above the umbilical level to the left of the scar of the first operation. The liver was found to be partly, but not altogether, adherent, and as attempts to stitch the liver to the abdominal wall led to bleeding from the liver, he shut off the peritoneal cavity with tampons. He then bored a hole into the liver with a Paquelin's cautery about 3 centimetres deep, and on probing this a great deal of serous-looking fluid suddenly welled up from the opening, and the probe then passed down for 6½ centimetres from the surface, as if one of the large bile-ducts or a cyst filled with serous-like fluid had been entered. A drainage-tube was left in the opening.

Some of the fluid which escaped at the operation was examined and found to contain very little albumen—in fact, not more than could be accounted for by admixture with blood. No hydatid hooklets were seen.

The patient rapidly recovered from the effects of the anæsthetic, and seemed quite comfortable. In the evening

[1] "Die Behandlung schweren Lebererkrankungen durch die Anlegung einer Leber-Gallengangsfistul," 'Berliner Klinik,' October, 1902, Heft. 172.

[2] See H. Scheuer, "Casuistisches zur Chirurgie der Gallenwege," 'Berliner klin. Woch.,' 1902, No. 7, p. 138.

[3] Calcium chloride was, as usual, employed before this operation, and likewise before the previous operation, in order to lessen the tendency to hæmorrhage.

following the operation his temperature was 100° F. The next morning (December 15th) it was 99° F., rising in the evening to 100·4° F., after which it remained below 100° F. The amount of fluid discharged from the liver was enormous, and on December 16th it was all allowed to run through a long drainage-tube from the wound into a bottle beneath the bed, where it was collected and measured every day. On December 16th it was clear orange-brown in colour and not tinged with blood; specific gravity 1006, of neutral or slightly alkaline reaction, yielding a very considerable "cloud" of nucleo-albumen on the addition of acetic acid in the cold. As the precipitate, after boiling and acidifying, was scarcely greater than after adding acetic acid in the cold, it was concluded that the fluid contained hardly any serum-albumen. Though it yielded an intense Gmelin's reaction, no Pettenkofer's reaction for bile-salts could at first be obtained. It still contained some red blood cells.

The discharge of the bilious fluid continued, but the daily amount greatly diminished, and it became darker in colour. On December 26th, 1904, we noted that the fluid was clear, limpid, of dark colour from the presence of bile pigments, of specific gravity 1010, and of neutral reaction. A drop or two of glacial acetic acid, added in the cold, produced a considerable precipitate of nucleo-albumen, which was redissolved in an excess of the acid. On boiling without the addition of acid only a very faint "cloud" appeared, but on adding acetic acid the same precipitate was produced as by the acetic acid in the cold; a very faint "cloud," however, remained after adding excess of acid to the boiling fluid, and this very faint "cloud" may have been due to a trace of serum-albumen, in addition to the nucleo-albumen. Sediment obtained by the centrifuge showed a few red blood corpuscles, but no cholesterin crystals. Some of the fluid, which had been allowed to become concentrated by slow evaporation, showed the presence of leaf-shaped crystals and of "rosettes" and "horse-comb" clusters similar to clusters of uric acid

crystals sometimes observed in artificially concentrated urines. On some occasions octahedral crystals, like those of oxalate of lime, were noted, as well as cholesterin plates.

Some of the fluid from the liver, on December 22nd, 1904, was sent to the Clinical Research Association to be examined for bile acids. The report then was that a positive Pettenkofer's reaction was obtained, and that, after treatment by Dr. Tyson's method, the fluid also answered to Oliver's test (peptone solution), and to Pettenkofer's test, so that there could be no doubt that bile-salts were present.

The specific gravity of the fluid varied slightly. On one occasion, as already mentioned, it was 1010, but on December 31st it was again only 1006. On January 5th the daily quantity of fluid from the biliary fistula was only 200 cubic centimetres, and as it escaped at the sides of the long drainage-tube the latter was discontinued after that date, the fluid afterwards being allowed to collect in the dressings, which had to be changed twice daily.

The following table shows the daily quantity of the

Date.	Daily amount of fluid from biliary fistula.	Daily amount of urine.
December 17th, 1904	2300 c.c.	—
December 18th, 1904	2000 c.c.	—
December 19th, 1904	2400 c.c.	—
December 20th, 1904	2000 c.c.	1000 c.c.
December 21st, 1904	1650 c.c.	1100 c.c.
December 22nd, 1904	700 c.c.	750 c.c.
December 23rd, 1904	—	2000 c.c.
December 24th, 1904	—	—
December 25th, 1904	400 c.c.	1700 c.c.
December 26th, 1904	750 c.c.	700 c.c.
December 27th, 1904	250 c.c.	750 c.c.
December 28th, 1904	400 c.c.	1500 c.c.
December 29th, 1904	—	1000 c.c.
December 30th, 1904	300 c.c.	1100 c.c.
December 31st, 1904	200 c.c.	1300 c.c.
January 1st, 1905	250 c.c.	1250 c.c.
January 2nd, 1905	—	1750 c.c.
January 3rd, 1905	400 c.c.	1750 c.c.
January 4th, 1905	—	1700 c.c.
January 5th, 1905	200 c.c.	1400 c.c.

fluid from the biliary fistula, as long as it was collected in a vessel—that is, until January 5th. The daily amount of urine is likewise given.

We must now describe the patient's condition after the operation, an operation which might be termed an "intra-hepatic cholangiostomy." This was not satisfactory. Though he was free from fever, and quite cheerful, and took his food fairly well, and though the jaundice diminished, and the liver became smaller, he steadily became more emaciated. The fæces, of course, remained perfectly colourless, but the urine became less bilious. Occasionally there were attacks of vomiting, and on one occasion the vomit apparently contained altered blood. At the commencement of January there was much purulent expectoration and impairment of resonance, with crepitation, in the right infrascapular region.

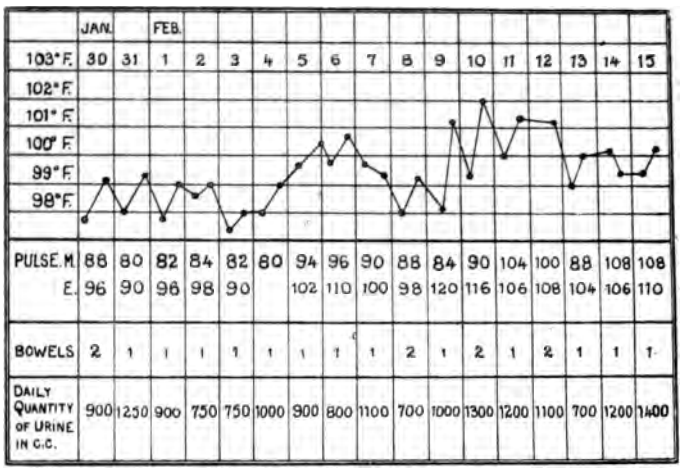
In the middle of January, 1905, the diminution of the jaundice was very striking, and on February 2nd the icteric tinge had practically disappeared from his skin and sclerotics. About this time the liver had decreased so much in size that the lower border, in the right nipple line, was about the level of the umbilicus; in fact, the lower border had risen as much as the attachment of the front of the liver to the abdominal wall in the neighbourhood of the biliary fistula would allow. The urine (February 2nd, 1905) gave no distinct Gmelin's reaction, though it turned red on the addition of nitric acid. It was clear, of medium colour, acid reaction, and free from albumen, and—what we believe to be a very worthy point—free from nucleo-albumen, the addition of acetic acid in the cold giving no precipitate or turbidity.

By the middle of January the patient had lost about 14 kilogrammes in weight since the operation, but after that he began gradually to increase in weight, gaining 5 kilogrammes by the end of the month.

Early in February he commenced to have irregular fever. On February 7th slight œdema and purpura of the legs were noted after he had sat up in the ward. On

February 13th there was much œdema of the loins, and the patient was manifestly losing strength. The slight purpuric eruption on the legs was still present. There was impairment of resonance in the left infrascapular region. The urine was of specific gravity, 1020, acid, clear, of medium colour, giving no Gmelin's reaction, and free from albumen and excess of nucleo-albumen. Increased feebleness was followed by death on the morning of February 16th.

The accompanying chart shows the temperature, pulse, and daily amount of urine during the last weeks of the patient's life:



	JAN. 30	31	FEB. 1	2	3	4	5	6	7	8	9	10	11	12	13	14	15
PULSE. M.	88	80	82	84	82	80	94	96	90	88	84	90	104	100	88	108	108
E.	96	90	96	98	90		102	110	100	98	120	116	106	108	104	106	110
BOWELS	2	1	1	1	1	1	1	1	1	2	1	2	1	2	1	1	1
DAILY QUANTITY OF URINE IN C.C.	900	1250	900	750	750	1000	900	800	1100	700	1000	1300	1200	1100	700	1200	1400

Necropsy (*by Dr. Weber, February 16th,* 1905).

Body much emaciated; front of the chest somewhat sunken on the right side.

Examination of the head and brain showed nothing special, excepting the "turriform" shape of the skull, already alluded to, which was found to be associated with complete synostosis of the frontal and parietal bones, with obliteration of the sagittal and coronal sutures. The brain weighed 48 oz.

The heart was rather small, weighing only 8 oz. Otherwise it showed nothing peculiar.

The right lung was completely and very firmly adherent to the chest wall, and was not removed for careful examination.

The left pleura contained some serous effusion. There were some nodules, which to the naked eye appeared like metastatic tumours, in the left pleura, and in the left lung itself. At the apex of the left lung was a little old scarring, and some of the bronchial glands were enlarged, partly pigmented, and partly caseated.

The liver was firmly adherent to the abdominal wall in front. The fistulous opening on the front of the abdomen, for the production of which the second operation had been performed, was found to communicate by a very narrow channel with one of the intra-hepatic bile-ducts. Great contraction had evidently taken place since the time of the operation, but the track was still open. Evidently owing to this biliary fistula none of the bile-ducts in the liver were dilated. The liver was apparently not enlarged, as when separated from the surrounding parts it weighed only about 50 oz. Its substance was rather firm and everywhere of a dark green colour. It contained no new growth of any kind or calculus.

The gall-bladder was of moderate size, and filled with very sticky inspissated blackish-green bile. The transverse fissure of the liver, including the whole region of the junction of the cystic, common, and hepatic bile-ducts, was occupied by a non-encapsuled rather tough growth resembling connective tissue. With probe and scissors it was possible to follow the channels of the various ducts most of the way through this growth. No calculus was found anywhere. The duodenal end of the common bile-duct was pervious, and apparently not in any way diseased. In the neighbourhood of the hilum of the liver were some enlarged lymphatic glands.

There was a good deal of serous peritoneal effusion. Scattered over the peritoneum were nodules, supposed at

the time of the necropsy to be metastatic tumours. The omentum was collected into a tough mass and seemed to the naked eye to be infiltrated with growth, and there was evidence of chronic peritonitis, with considerable thickening of the whole serous coat of the stomach[1] such as is not rarely met with in cases of diffuse peritoneal cancer (the so-called "cancerous peritonitis"), sometimes after operative attempts to remove a primary growth.

The spleen only weighed 4 oz., and contained one nodule of apparent growth.

The kidneys weighed together 11 oz., and one of them, it may be remarked, showed a peculiar congenital abnormality, the pelvis and attachment of the ureter being situated on the ventral surface instead of in the usual position.

The pancreas appeared to be quite normal.

Of the suprarenals one only was examined, and appeared normal.

The peritoneal coat of the stomach was, as has already been mentioned, considerably thickened. The intestines showed nothing peculiar (excepting the growths on their peritoneal surfaces already mentioned), neither did the testicles.

The left humerus was sawn through longitudinally, so that the bone-marrow of the shaft might be examined. This was of red colour throughout; and of gelatinous consistence.

Microscopical Examination.

Sections of twenty-four different pieces from various parts were prepared. The tough connective-tissue-like mass from the hilum of the liver filling up the transverse fissure was found on microscopic examination to consist of adenocarcinomatous tissue (malignant adenoma).

[1] Superficially the stomach resembled an early stage of the so-called "leather-bottle stomach"—*vide* A. W. Nuthall and J. G. Emanuel, "Diffuse Carcinomatosis of the Stomach and Intestines," 'Lancet,' January 17th, 1903, p. 159.

The microscopic sections (two pieces of the growth were examined) showed tubules (Fig. 1) lined by columnar-celled epithelium, cut across in various directions. The tubules were of various sizes and shapes, some of them being large enough to appear in transverse section as cysts (lined by columnar epithelium). In some parts tubules were to be seen inside larger tubules (intra-cystic growth), and a few of the tubules were lined by less distinctly columnar (more spheroidal-like) epithelium, and were filled up with cells so as to show no lumen. The tubules were separated from each other by a variable amount of connective tissue; in some parts they were close together, as in adenomata, but in other parts they were widely separated by connective tissue. Though the tubules were mostly lined by only a single layer of cylindrical epithelium, and in their irregularity reminded one of adenomatous growths,[1] they were found to have invaded the structures at the hilum of the liver, penetrating between, and in one or two places into nerves (Fig. 2). The growth was not encapsuled and was evidently malignant, although, as we shall presently have to point out, no metastasis had as yet occurred—at least, none that could be recognised. It was therefore a primary adenocarcinoma or malignant adenoma, which, owing to its position, doubtless originated from the extra-hepatic bile-ducts at the hilum of the liver. We shall return to this subject later on.

Examination of the liver (Fig. 3) itself showed a certain amount of fibrosis between the acini, with increase (at least, apparent increase) in the number of the biliary canaliculi—in fact, the appearance found in cases of biliary cirrhosis, due, directly or indirectly, to obstruction in the large bile-ducts. One or two circular spots of cellular infiltration suggested commencing tubercle-formation, but a section was specially stained for tubercle bacilli with a negative result.

[1] The tubules of adenocarcinomata of the rectum may, of course, also be well formed and lined by a single layer of epithelium.

[2] F. Parkes Weber, "Biliary Cirrhosis of the Liver, with or without Cholelithiasis," 'Path. Soc. Trans.,' 1903, vol. liv, p. 103.

The supposed metastatic growths from the lungs and spleen, and from the peritoneal surfaces of the jejunum and ileum, as well as the thickened omentum, the thickened outer wall of the stomach, and the lymphatic glands from near the hilum of the liver, were found by microscopical examination to show the typical changes of tuberculosis, with giant-cell formation, more or less caseation, etc. Sections stained for tubercle bacilli showed their presence in the thickened wall of the stomach, in the omentum, and in nodules from the lung, spleen, and jejunum.

Sections of the bone-marrow from the shaft of the left humerus showed typical "gelatinous degeneration." The degenerated tissue consisted of an almost homogeneous substance, containing only a few erythroblasts and other cells and the remains of fat vesicles. At one part of the field, shown in Fig. 4, there is, however, a decided erythroblastic reaction. The tissue at this part is rich in cells, almost all of them being erythroblasts, with typical deeply-stained homogeneous-looking nuclei, some of them with two nuclei or a lobed nucleus, probably in process of division. Here and there large numbers of erythroblasts are grouped together in dense clumps.

Remarks.—The question of diagnosis claims our attention. At one time, especially after the negative result of the first operation (exploratory laparotomy), we almost regarded the case as one of Hanot's disease—that is to say, hypertrophic cirrhosis of the liver, with chronic jaundice. The points against this diagnosis were: the absence of great enlargement of the spleen, the comparatively recent and sudden development of the jaundice and of the hepatic affection altogether, and thirdly, the complete absence of biliary pigments from the fæces. It was this latter point which finally made us come to the conclusion that the cause of the jaundice and hepatic enlargement was some mechanical obstruction (probably not a calculus[1]) at the hilum of the liver, and owing to

[1] There was no history of biliary colic or of previous jaundice, or of attacks of fever such as are frequently met with in cholelithiasis cases.

the absence of distension of the common bile-duct and gall-bladder, Dr. Michels thought that this obstruction was situated proximal to the junction of the cystic and common ducts. We therefore concluded that the bile ought to be drained off externally, and Dr. Michels succeeded in effecting this by the establishment of a biliary fistula in the way described. In fact, the jaundice practically completely disappeared, the urine became free from biliary pigments, and the liver shrank, so as to weigh about the normal amount at the time of death. It is probable that the spleen also became smaller with the relief of the jaundice, as, though it had been apparently enlarged by physical examination in the earlier part of the illness, it was found to weigh rather less than the normal at the necropsy.

There is not much to be said in regard to the tuberculosis which developed. Minute carcinomatous metastases on the peritoneum and pleura often have the appearance of tubercles when examined by the naked eye at the time of a necropsy, or during an operation, but in the present case it was the tuberculous growths which at the necropsy appeared to us to be carcinomatous metastases. A striking example of the macroscopic resemblance of tuberculous growths to carcinomata has recently been recorded in Germany by Dr. E. Ruge.[1] His patient presented all the signs of carcinoma of the pylorus; the typical tumour was felt, and there was absence of hydrochloric acid and lactic acid from the gastric contents. The diagnosis of cancer of the pylorus was confirmed by laparotomy, and even at the post-mortem examination the disease was supposed to be carcinoma of the pylorus, with numerous metastases. It was only the microscopical examination which showed that not only the supposed primary tumour, but all the metastases, were in reality of tuberculous inflammatory origin. Ruge compared the carcinoma-like tuberculous nodules to perlsucht in cattle.

Moreover, it must be remembered that gall-stones in the hepatic and common bile-ducts nearly always allow some bile to pass now and then.

[1] "Beiträge zur Klinik der Tuberkulose," 1905, vol. iii.

The explanation of the development of the tubercle in our case is not quite obvious. The presence of some partially caseous bronchial glands and remains of pulmonary (apical) tuberculosis show that the patient had previously had old quiescent tubercle in his body. The outbreak may, perhaps, be regarded as a rewakening of quiescent and latent tuberculosis facilitated by the patient's cachexia and diminished power of resistance. Secondary infection from without appears extremely improbable, but it is just possible that at the first operation tuberculous glands in some part of the abdomen or thorax may have been damaged. On the other hand, no bad signs were noted until after the second operation, and at this operation it is exceedingly unlikely that anything of the kind can have taken place, as the organs were not moved about or pressed upon. The type of the tuberculosis was remarkable, as we have already noted, especially in respect to the tumour-like growths on the peritoneal surface of the intestines and the diffuse tuberculous thickening of the outer coat of the stomach.

Primary carcinoma of the bile-ducts at the hilum of the liver without metastases and without the liver itself being invaded by the new growth is not a common cause of chronic jaundice. H. D. Rolleston, in his recent work on 'Diseases of the Liver,'[1] has pointed out that some cases described as primary cancer of the liver might possibly in reality have been cases of single large adenomata of bile-ducts. In our case, however, the growth was a typical cylindrical-celled adenocarcinoma or malignant adenoma,[2] infiltrating the tissues at the hilum of the liver and even penetrating into nerves.

J. Orth[3] says that primary cancer of the bile-ducts, which is generally a cylindrical-celled adenocarcinoma,

[1] Saunders & Co., Philadelphia and London, 1905, p. 455.

[2] Rolleston (*loc. cit.*, p. 688) thinks that spheroidal-celled carcinoma of an extra-hepatic bile-duct may possibly be derived from mucous glands in its wall.

[3] 'Lehrbuch der spec. path. Anatomie,' Berlin, 1887, vol. i, p. 988.

is a rare disease. "Its most usual situation is at the junction of the hepatic ducts or at the orifice of the choledochus." In our present case there can be practically no doubt that the growth originated at the junction of the two hepatic ducts to form the common hepatic-duct, and that at the time of the first operation (exploratory laparotomy, October 7th, 1904) the cystic and common bile-ducts were not yet involved. At that time, although the obstruction to the exit of bile from the liver was complete, Dr. Michels found no dilatation of the gall-bladder or of the common bile-duct, and was able to express some of the bile from the gall-bladder into the common duct. Doubtless the cancer could not be felt at the operation, because at that time it was extremely small and situated deep in the transverse fissure of the liver, though at the time of death (February 16th, 1905) it had already involved the junction of the cystic and common bile-ducts.

We have already alluded to the fact that the present case confirms the view that chronic obstruction of the larger bile-ducts leads gradually to more or less interstitial hepatic fibrosis, in the same way that chronic obstruction of a ureter with hydronephrosis leads to chronic fibrosis of the affected kidney.[1] We lay stress on this point, as it

[1] *Vide* F. P. Weber, 'Path. Soc. Trans.,' *loc. cit.* In regard to this subject our colleague Dr. Fürth has kindly allowed us to allude to the following striking case, which was under his care at the German Hospital. The patient, a married woman, aged 66, commenced to suffer from jaundice for the first time in March, 1904. Apparently no bile passed into the bowel, and jaundice, emaciation, and weakness, became extreme. She was tapped four times for ascites. The liver, which was at first greatly enlarged, with a somewhat uneven edge, diminished so much in size that just before her death (October, 1904) the edge could only just be felt below the costal margin. At the necropsy the gall-bladder, full of calculi, was surrounded by malignant growth, but the point which specially concerns us is that all the bile-ducts, intra-hepatic as well as extra-hepatic, were so dilated as to remind one of the appearance seen in a hydronephrotic kidney. Microscopically the minute biliary ducts were seen to be full of inspissated bile (by naked-eye examination the substance of the organ was of a deep green colour as usual) and there was a

has been disputed by many. Amongst other writers on the subject Scagliori[1] has quite recently drawn attention to biliary cirrhosis as a result of obstruction by columnar-celled carcinoma of the large bile-ducts.

As far as it went the unusual operation (biliary drainage by intra-hepatic cholangiostomy) was successful. It was only intended to relieve the jaundice and the retention of bile in the liver. The jaundice practically disappeared, the liver, though it could not, owing to adhesions, return to its normal position, returned to its normal dimensions (as judged by its weight) and the urine became practically free from bile and nucleo-albumen.[2] But the tuberculous complication prevented the result from being really satisfactory, and we must also admit the possibility of the biliary fistula closing up spontaneously had the patient lived longer, for the fistulous channel at the time of death had become extremely narrow.

The surgical aspect of the case requires but little comment. Where the situation of the obstruction is proximal to the junction of the hepatic and cystic ducts it would, of course, be futile to attempt to drain the gall-bladder or to make a cholecystenterostomy. Drainage of the intra-hepatic ducts means a permanent biliary fistula, at least, as long as the fistulous channel can be kept open, except in those cases where from the nature of the obstruction a re-opening of the natural biliary channels may be expected, as in the case published by Rotter.[3] With a view to avoid a biliary fistula, Kehr excised a piece of the edge of the liver, 6 cm. long and 3 cm. broad, made a corresponding opening into a loop of small intestine, and stitched

certain amount of biliary cirrhosis. Yet so much had the liver diminished in size before death, that it weighed only about 40 oz. (The heart was also very small, weighing only 5 oz.)

[1] 'Il Policlinico,' November, 1904.

[2] As if the excess of nucleo-albumen, which constitutes a striking feature of some bilious urines, were, like the bile-pigment, absorbed from the biliary passages into the blood, and then (again like the bile-pigment) excreted with the urine.

[3] *Loc. cit.*

the edges of the intestinal opening to the margin of the wound in the liver. Kehr terms the operation "hepato-cholangio-enterostomy."[1] In his case the jaundice was soon relieved, the bile passing from the exposed intra-hepatic ducts straight into the intestine, but the patient survived only four weeks. It seems a somewhat hazardous proceeding to bring the wound of the liver into contact with the contents of the bowel. The establishment of a biliary fistula is certainly the safer proceeding, and the operation should be performed on the left side of the liver, as the chance of reaching a large bile-duct is supposed to be better on the left side than on the right. The operation can be done with a trocar (Hirschberg) or preferably (as in the present case) with the thermocautery.

We will only add a few words on the change in the bone-marrow. This consisted in a mixed erythroblastic reaction and "gelatinous degeneration," the latter predominating. It is well to ascertain, as far as possible, the bone-marrow changes associated with various clinical conditions. According to Roger[2] and Muir[3] extreme inanition and emaciation seem to be the chief causes of gelatinous change in the bone-marrow, and great emaciation was certainly present in our case.

In conclusion, we have to thank the residents at the German hospital, Dr. Schuh, Dr. Mülberger, Dr. Schenck, and Dr. Daser, for their many and excellent microscopic preparations from the case, and we are also greatly indebted to Dr. H. D. Rolleston and Mr. S. G. Shattock for their kindness in looking through the sections with us.

[1] 'Centralblatt für Chirurgie,' 1904, No. 7, p. 185. See also Enderlen, and Zumstein, "Ein Beitrag zur Hepato-Cholangio-Enterostomie," 'Mitteilungen aus den Grenzgebieten der Med. und Chir.,' Jena, 1904, vol. xiv, p. 104.

[2] Roger and Josué, 'Comptes rendus de la Soc. de Biologie,' Paris, 1900, p. 417.

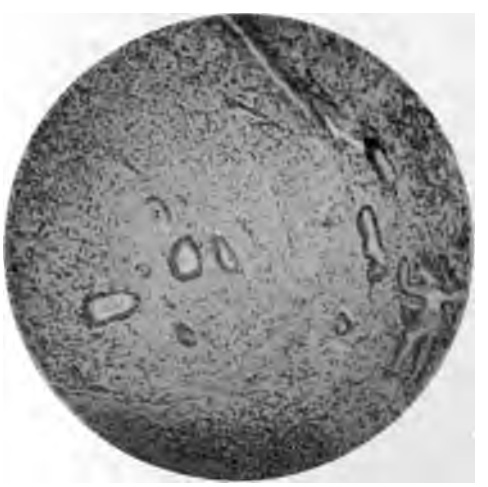
[3] R. Muir, 'Path. Soc. Trans.,' 1902, vol. liii, p. 392.

Dr. Parkes Weber and Mr. Michels: Chronic Jaundice. Plate I.

FIG. 1.

The primary adeno-carcinoma at the hilum of the liver, showing tubules of various sizes and shapes; one with its lumen filled up with cells or intra-cystic growth. (× 105 diam.)

FIG. 2.



Portion of the growth, showing carcinomatous tubules having penetrated into a nerve. (× 105 diam.)

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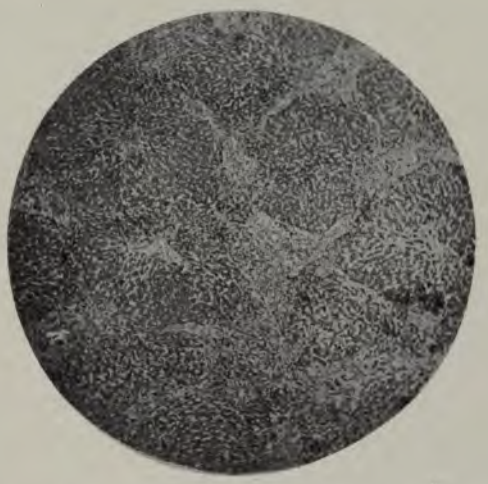
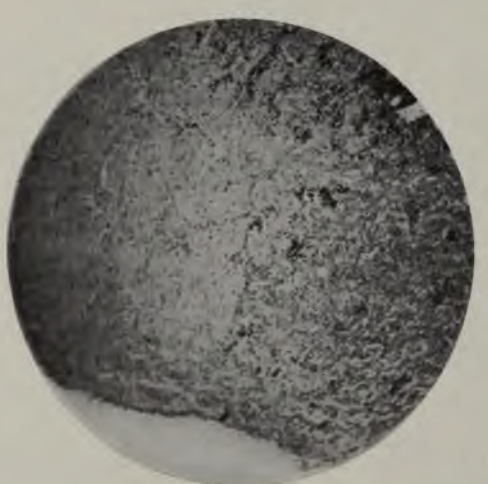
Dr. Parkes Weber and Mr. Michels: Chronic Jaundice. Plate II.

FIG. 3.



Section of the liver, showing a certain amount of interacinous biliary cirrhosis. (× 105 diam.)

FIG. 4.



Section of bone-marrow from the shaft of the left humerus, showing an erythroblastic reaction on one side and "gelatinous degeneration" on the other. (× 114 diam.)

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THE REACTION OF PHENYLHYDRAZIN WITH OTHER SUBSTANCES THAN DEXTROSE OCCURRING IN THE URINE

BY

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THE application of phenylhydrazin as a reagent for the detection of sugar in the urine marked an epoch in the investigation of glycosuria and allied conditions; for not only is it much more delicate than any test previously employed, but it is unaffected by other substances such as creatin, creatinin, hippuric acid, homogentisic acid, and excess of uric acid or urates, as met with in human urine, that are liable to give rise to difficulties when most other methods of detecting sugar are relied upon. This reagent has also the advantage of allowing the various forms of carbohydrate to be distinguished. This is a matter of some importance, for although dextrose is undoubtedly the commonest sugar met with in the urine, there is considerable evidence to show that other forms of carbohydrate and allied substances occur more frequently than has been supposed, and that their presence is associated with various abnormalities of metabolism. As more detailed attention is paid to the investigation of the urine in cases of supposed diabetes, and some diseases in which glycosuria is likely to occur, our knowledge of the true significance of these rarer

forms will be increased, and possibly at the same time our information as to the mechanism of normal carbohydrate metabolism may also be added to. A brief survey of the more important characters of the phenylhydrazin compounds which may be derived from the urine may be of some service; for although the test is now frequently employed clinically, it is not often that the investigation is carried further than the preparation of the osazone crystals, and it is probable that much valuable information is missed in consequence.

The method of performing the phenylhydrazin test described by von Jaksch has been much modified by subsequent writers, and since the physical conditions under which it is carried out materially affect the result it will be necessary to refer to the chief variations proposed. Von Jaksch originally recommended (1) that 50 c.c. of the urine to be tested should be mixed with 2 grammes of sodium acetate, and from 1 to 2 grammes of phenylhydrazin hydrochloride, and that the mixture be heated in a water bath for twenty minutes to half an hour. If glucose is present, the osazone then separates out on cooling as an amorphous or crystalline deposit of a yellow or reddish colour. If amorphous, the precipitate can be recovered in a crystalline form by dissolving it in hot alcohol, diluting the solution with water, boiling to expel the excess of alcohol, and then cooling.

This method of performing the test takes time and requires the use of special apparatus which is not always available. To obviate these difficulties and render the reaction convenient for clinical use it has been suggested (2) that 0·5 grammes of phenylhydrazin hydrochloride and 1·5 grammes of sodium acetate should be dissolved by gentle heat in a few c.c. of water in a test-tube, and then 5 to 10 c.c. of the urine added. The mixture is brought to the boiling point and maintained there for three minutes with strong and five minutes with weak solutions of sugar. The test-tube is then set aside to cool, and the deposit examined for osazone crystals in five or ten minutes. Some

authorities, following E. Fischer (3), have preferred to use phenylhydrazin and not the hydrochloride, but it has the disadvantage of not keeping well. A knife-point of sodium acetate is added to 10 c.c. of the urine, then 1–2 c.c. of 10 per cent. acetic acid, and 5 drops of pure phenylhydrazin introduced. The mixture is heated in the water bath or over the free flame in the same way as when the hydrochloride is employed. A recent modification of this method suggested by Kowarski (4), which gives very satisfactory results and is more delicate, consists in mixing 5 drops of pure phenylhydrazin in a test-tube with 10 drops of acetic acid, gently shaking and then adding about 1 c.c. of a saturated solution of sodium chloride. To the solid mass that forms is added 3–5 c.c. of the urine, and the test-tube heated in the free flame for two minutes after its contents begin to boil. On cooling, the osazone crystals separate from urines containing over 0·2 per cent. of sugar in one minute and from weaker solutions in about five minutes.

In whichever way the test is carried out, a difficulty occasionally arises from the formation of a crystalline phenylhydrazin compound of glycuronic acid which is liable to be mistaken for the osazone of a sugar. It has been stated (5) that prolonged heating tends to obviate this source of error, the glycuronic acid compound being precipitated on cooling as a brown amorphous mass and not in the crystalline form. In the examination of a hundred normal urines I sought to test the truth of this statement, and found that when heated in the water bath for an hour four of them showed a crystalline deposit, while by boiling in the free flame for five minutes six specimens gave a positive result. Using the same methods but shortening the period of heating to twenty minutes and two minutes respectively, exactly the same results were obtained. So that the mere time or method of applying the heat cannot be relied upon to differentiate glycuronic acid from the sugars.

In addition to dextrose, the sugars levulose, lactose, maltose, isomaltose, and the pentoses, arabinose and

xylose, have been described as occurring in the urine and forming compounds with phenylhydrazin. Glycuronic acid, as I have already mentioned, also forms a compound with this substance, and exceptionally combinations with acetone, aceto-acetic acid, oxalic acid, and in very concentrated urines with uric acid may also be encountered. As human urine is relatively poor in uric acid, the last named does not call for further remark. The acetone compound occurs as needles which melt to oily globules at 16° C., while the oxalic acid salt separates as insoluble, glancing, colourless plates which melt at 172–173° C. The phenylhydrazin compounds of the remaining substances possess certain physical and chemical characters by which they can be more or less readily differentiated. The chief of these are: (1) the rate of osazone formation; (2) the microscopical characters of the crystals; (3) the solubilities of the osazones in various reagents; (4) the melting points of the purified products; (5) the percentage content of nitrogen.

(1) The rate of osazone formation is a point of considerable importance in determining the variety of sugar present in a particular solution, for, as Marquenne has shown (6), this is a property which has very characteristic variations for the different carbohydrates. Under experimental conditions, with solutions of definite strength, exact time-limits for the appearance of the osazone can be laid down. Although this is not feasible with such a liquid as urine, where the proportion of sugar present is unknown, valuable information can be obtained by observing the conditions and rate of osazone-formation. The compound formed by dextrose and levulose separates from the hot solution after a comparatively brief interval, the former in most cases with characteristic suddenness. The osazones of maltose, lactose, and the pentoses only form after much more prolonged heating, and although crystals of pentosazone may eventually separate from the hot solution, maltosazone and lactosazone never appear until the fluid cools, no matter how long it may be boiled. In my experience the rapid

method of performing the phenylhydrazin test gives a reaction with all sugar when 10 c.c. of the urine is used and the heating continued for at least five minutes. In the water bath, however, even an hour is not sufficient to obtain a satisfactory yield with maltose, lactose, and pentose, an hour and a half or even two hours being required to demonstrate their presence satisfactorily, especially when the solution is weak. Those who prefer to use the water-bath method, therefore, and limit its time of application to fifteen or thirty minutes, can only expect to demonstrate dextrose or the more readily precipitated sugars and possibly also any glycuronic acid compounds that may be present in the urine.

(2) Microscopical examination of the crystalline deposit obtained after treatment with phenylhydrazin shows certain differences in the characters, size, and arrangement of the crystals which are suggestive. The large yellow needles arranged in sheaves and rosettes yielded by dextrose are well known. Levulosazone resembles glucosazone, but there is less tendency to rosette formation and the crystals are somewhat longer and more slender. Lactosazone occurs as spherical masses of crystals resembling a shaggy yellow chrysanthemum. At the periphery the separate crystals can be distinguished and are seen to be slender, flexible, and hair-like, but in the centre they are felted together into a brown semi-opaque mass. In preparations made by the rapid method the individual crystals are usually not as distinct as those formed after prolonged heating in the water-bath, the appearance presented being that of a brown central boss surrounded by a light yellow halo showing "fine" radial striations. The crystals of maltosazone are short, stiff, and sword-like and are arranged in small rosettes when prepared by the water-bath method. Preparations made by heating in the free flame are less characteristic, consisting of narrow crystals grouped in small bushy sheaves. Isomaltose yields masses of aggregated flexible needles of a golden-yellow colour, mostly arranged in spheres. The pentose crystals are silky, tangled, curved needles,

generally arranged in rosettes. The shape, size, and arrangement of the crystals is influenced to a certain extent by the manner in which the test is carried out and by the rate of cooling, so that not only is some experience of typical preparations required in forming an opinion, but the physical condition under which the sample under examination was prepared must also be taken into account. In some cases the crystalline form can only be determined satisfactorily after the specimen has been purified by recrystallisation.

3. The osazones can be differentiated to a certain extent by their solubilities. Thus pentosazone is readily soluble in water at 60° C., but dextrosazone is almost insoluble. One part of lactosazone dissolves in eighty to ninety of boiling water, while maltosazone is still more soluble (1 in 70–75). Isomaltose dissolves one part in four of water at 100° C. I have found that the rate of solution of the osazones in dilute sulphuric acid is of some value in distinguishing the phenylhydrazin compounds of dextrose and levulose from those of other carbohydrates. On irrigating a preparation of the latter with a 33 per cent. solution of the acid the crystals turn brown and dissolve rapidly, while dextrosazone and levulosazone only slowly assume a brown coloration and take several minutes before they disappear. All the osazones are soluble in hot 50 per cent. alcohol and advantage is taken of this fact to prepare them in a pure form.

4. The melting point of the crystals obtained from urine by the phenlhydrazin reaction is one of their most useful and characteristic properties. The product employed for the purpose must, however, be pure, or doubtful and misleading results will follow. Thus the melting point of pure dextrosazone is 204–205° C., but the impure crystals obtained direct from the urine generally melt somewhere between 173° and 194° C. Levulosazone melts at the same temperature as dextrosazone. The melting point of the phenylhydrazin compound of lactose is about 200° C., and of maltose 206°–207° C. Isomaltose begins

to form drops at 140°–145° C., melts at 150°–153° C., and blackens at 200° C. The osazone of the pentoses in a pure form melts at 160° C., but as obtained from the urine the melting point lies between 156° and 158° C. As the osazones undergo decomposition on prolonged heating it is necessary that the temperature should be rapidly raised at first and then gradually increased as the point of fusion is approached, or charing of the specimen may obscure the change of state.

5. The formula of the osazones formed by the monosaccharides dextrose and levulose is $C_{18}\ H_{22}\ N_4\ O_4$, while that of the disaccharides lactose, maltose, and isomaltose is $C_{24}\ H_{32}\ N_4\ O_9$. Hence the former may be expected to yield 15·64 per cent. and the latter 10·76 per cent. of nitrogen. In practice slightly lower readings are found to be the rule, about 15·58 per cent. being obtained for dextrose and levulose and 10·67 per cent. or thereabout for the disaccharides. The percentage of nitrogen contained in the pentosazones is 17·07, as the formula is $C_{17}\ H_{20}\ N_4\ O_3$. By determining the percentage of nitrogen in an osazone it is therefore possible to decide to which of these three classes of carbohydrate the sugar belongs. A satisfactory determination is, however, only possible when a sufficient amount of the pure product and the necessary apparatus are available for a combustion experiment, as Kjeldahl's process is useless for the purpose, the separation of the nitrogen not being complete.

Consideration of the properties of the compound that a sugar forms with phenylhydrazin therefore enables one to distinguish between all the recognised varieties occurring in the urine save only dextrose and levulose. Here reliance must be placed on other tests, such as the polariscope, Seliwanoff's test, and the reaction with methyl-phenylhydrazin. It is evident, however, that unless special care is taken, the rarer forms of carbohydrate may readily be overlooked, especially if only small quantities are present, and that in some instances the method usually employed for demonstrating glucose will not suffice to reveal all that

a more extensive use of the test would teach. It is consequently not surprising that comparatively little is known as to the occurrence in the urine and real significance of other varieties of sugar than dextrose.

Recent research has shown that levulose is often present in diabetes along with dextrose, and it has also been described as occurring alone in mental depression, melancholia, and sleeplessness. Lactose is present in the urine of a considerable percentage of women during the puerperal period, and is most marked when the breasts are gorged. Sucking infants with gastro-intestinal catarrh are also said to pass lactose in their urine occasionally. Hitherto maltose has only been met with in a few cases of disease of the pancreas, and, so far as is known at present, its occurrence is confined to this class of case. Unfortunately, maltosuria is by no means a constant symptom of pancreatic disease, and cannot therefore be relied upon for diagnostic purposes. In an examination of 245 specimens of urine from cases of disease of the pancreas by the phenylhydrazin method I have only met with two in which an osazone having the characters of maltosazone was obtained in sufficient quantity for an accurate investigation, and five in which a small deposit of crystals, probably also maltosazone, was given. One of the former was a patient in whom an operation for stone in the common duct with chronic pancreatitis had been performed four years previously. Isomaltose is prepared artificially along with maltose, in the reversion of glucose by the action of malt diastase, pancreatic juice, saliva, or blood serum on starch. It has been found in small quantities in apparently normal urine, and up to the present it has not been determined whether its presence is associated with any particular metabolic disturbance, or if an excess occurs under pathological conditions. Two varieties of pentose are encountered in the urine, a dextrorotary and an optically inactive form. The former is found in alimentary pentosuria following in healthy persons the ingestion of pears, plums, cherries, bilberries, and other food materials containing pentose, while the latter is the

form met with in chronic pentosuria and under pathological conditions. Only a few cases of chronic pentosuria have been described, but more careful routine examination of the urine would probably show that the condition is more commonly mistaken for ordinary diabetes than is generally supposed. In a few cases of pancreatic disease and frequently along with dextrose in severe diabetes pentoses have been reported as present in the urine. This is interesting in view of the fact that extirpation of the pancreas in dogs is followed by excretion of a pentose in the urine. In none of the cases of inflammation or malignant disease of the pancreas that I have examined has any evidence of the presence of this substance been found.

Glycuronic acid, the only other important substance forming a compound with phenylhydrazin that is met with in fresh urine, is closely related to the sugars. It represents an early stage in the oxidation of glucose where one atom of oxygen has replaced two hydrogen atoms in the group CH_2—OH. It is not found free in the urine, but always in combination with various bases, and with fatty and aromatic alcohols, especially the latter. In perfect health the amount excreted is small, but when the conditions are such that there is an excessive formation of phenol, indol, and similar products of putrefaction, the excretion is correspondingly increased. It is well known that the increased reducing power of the urine after chloroform anæsthesia and the administration of certain drugs such as camphor, chloral, salicylic acid, turpentine, thymol, etc., is due, not to sugar, but to conjugated glycuronic acid which these substances cause to pass into the urine. In cases of respiratory difficulty and in febrile diseases an increased output of glycuronic acid is said to take place. The urine of diabetics often contains glycuronic acid, and even in severe cases the tissues appear to be able to carry the oxidation to this point, for the administration of appropriate drugs will cause a large amount to be passed. Large doses of sugar in the apparently healthy may be reflected in the urine by an

increased excretion of glycuronic acid. Alkaline salts of the acid form a compound with phenylhydrazin, which slowly separates on cooling as yellow needles usually of a somewhat darker colour, of smaller size, and more irregular shape than the osazones of the sugars. Some compounds of glycuronic acid undergo the decomposition that must occur before it combines with phenylhydrazin more easily than others, so that the readiness with which the reaction takes place varies. A urine containing a compound such as urocholic acid, which readily splits up, quickly responds to the test; but the more resistant phenol and indol compounds require prolonged treatment before they react. When the reaction takes place in a solution containing free acid the phenylhydrazin compound is precipitated in the form of dark brown granules and does not assume the crystalline form. Prolonged heating is said to bring about a similar result, but in practice this cannot be depended on to distinguish the sugars from glycuronic acid.

The crystals are readily soluble in hot alcohol, but generally separate from this solution after dilution with water and boiling, in an amorphous form. In 33 per cent. sulphuric acid they dissolve in about one to two minutes. The melting point of the crystalline variety is 114-115° C., but the amorphous form requires heating to 150° C. before any change is observed. Since the formula of the phenylhydrazin compound of glycuronic acid is C_{42} H_{48}, N_{10} O_{10}, it is calculated to yield 16·4 per cent. of nitrogen on combustion. It is not at all an easy matter, however, to obtain a sufficiently pure specimen from the urine to allow of an accurate determination of either the melting point or the nitrogen content.

The freeing of glycuronic acid from its combinations is most readily effected by heating with dilute acids. Hence some urines that do not react with phenylhydrazin alone may do so after such treatment, and in others the yield may be materially increased. In an investigation with the phenylhydrazin test of a series of one hundred urines

from apparently healthy people, I found that, in addition to the six already mentioned which gave a reaction with phenylhydrazin alone, thirteen yielded a positive result due to glycuronic acid after treatment with hydrochloric acid. The series, on being divided into an earlier set of 43, derived from persons living in the country with an abundance of fresh air and exercise, and a later set of 57 obtained from city dwellers who lived under less favourable hygienic conditions, showed that none of the former gave a reaction until the urine had been boiled with acid, and then only three yielded a crystalline deposit, but that six of the latter gave a positive result before treatment and nine after.

Glycuronic acid is not the only substance capable of combining with phenylhydrazin that is set free in the urine by heating with dilute acids. Under certain pathological conditions an appreciable quantity of osazone with a definite melting point differing from that of glycuronic acid may be obtained. It has been stated that when normal urines are boiled with one tenth volume of strong hydrochloric acid for five to ten minutes and the excess of acid neutralised very characteristic crystals are "invariably" obtained with phenylhydrazin (7). This is not by any means my experience, for in 94 normal urines that gave no reaction with phenylhydrazin I found that 19 gave a slight crystalline deposit and 7 a more marked result after boiling with hydrochloric acid. Of these, 12, as I have already stated, were glycuronic acid compounds, so that in 14 out of 94 specimens from apparently healthy people there was a reaction due to some other substance. Only three of these were at all comparable in degree with what is met with pathologically and, as all three were derived from persons living a sedentary life and occurred in my later series, I think it is not unlikely that the reaction was an expression of a disturbance of function. In disease, especially where there is inflammatory reaction and marked tissue change or an affection of the kidneys, treatment of the urine with an acid previous to the appli-

cation of the phenylhydrazin test produces a marked and in many instances a most striking effect if the operation is carefully performed. The details of the experiment, however, exert considerable influence on the result, and some experience is required to determine what conditions are best suited to obtain the most characteristic effect with the particular specimen of urine under examination. Insufficient or too prolonged heating after the addition of the acid may prevent the reaction or interfere with it, while unless care is taken in carrying out the neutralisation of the excess of acid the derivatives obtained may be destroyed or their presence obscured. Boiling in the water-bath for 15 to 30 minutes is, in my experience, quite insufficient to secure a characteristic reaction with phenylhydrazin when this test is subsequently applied. To avoid the difficulties attending neutralisation with a caustic alkali I have generally employed a neutral salt that formed an insoluble compound with the acid—lead carbonate for hydrochloric acid and barium carbonate for sulphuric acid. Although lead chloride is soluble in hot water, it is almost insoluble in cold, especially when there is a little free hydrochloric acid present. It is therefore essential in using this method, if difficulties from lead salts are to be avoided, that the solution should be well cooled before the carbonate is added, and it is an advantage if a trace of hydrochloric acid is left un-neutralised. Of course, the usual precautions in filtering off the insoluble lead compound are necessary. Sulphuric acid gives comparable results with hydrochloric and the complete insolubility of barium sulphate is an advantage, but much more care is required to secure constant results. The fact that similar crystals are obtained from the same urine with both acids and with different methods of neutralisation indicates that those produced by the use of hydrochloric acid and lead carbonate are not lead compounds, as does also their not responding to any of the reactions for lead when isolated and purified.

My experience of this reaction has been chiefly gained

in connection with work on diseases of the pancreas which I have carried out in conjunction with Mr. Mayo Robson. Up to December 31st, 1904, I have had the opportunity of examining 479 specimens of urine from 295 cases, most of whom were under Mr. Robson's care, and on many of whom he performed an abdominal section.

In 130 cases the urine when treated with hydrochloric acid and neutralised with lead carbonate yielded with phenylhydrazin hydrochloride and sodium acetate a considerable flocculent brown deposit which, under the microscope, was found to consist of yellow crystals much smaller than glucosazone and generally arranged in rosettes. In some the crystals were slender and somewhat resembled small lactosazone or pentosazone crystals, while in others they were less delicate, and approached the glucosazone or even maltosazone type in shape. Their time of solution in 33 per cent. sulphuric acid varied from half to one and a half or two minutes when the mean of several observations was taken. On being filtered off, washed, extracted with boiling 50 per cent. alcohol and re-crystallised they gave a melting point of 168° to 170° C.

The clinical symptoms in 116 of these cases pointed to disease of the pancreas, and in 79 it was possible to ascertain the condition of the parts at operation. In 5 the diagnosis was confirmed post mortem, shortly after the last examination of the urine had been made. Three cases from whose urine fine rapidly dissolving crystals were isolated were found to be suffering from acute pancreatitis with fat necrosis. One succumbed shortly after operation and was examined after death. In 37 gall-stones were found impacted in the lower part of the common bile-duct and the pancreas was stated to be unmistakably enlarged and hardened. In 2 cases a similar condition of the gland was found, associated in one with obstruction and considerable dilatation of the common bile-duct, and in the other with a malignant growth of the duodenal papilla which subsequently proved fatal. In two cases pancreatic calculi were found, and in two others

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a cyst of the pancreas was dealt with. The pancreas was described as being enlarged and harder than usual in 29 cases, but no obstruction of the ducts could be found at the time of operation. In 1 case a malignant growth of the stomach was found adherent to and growing into the pancreas. In 3 cases, although there were gall-stones in the gall-bladder or cystic duct, no abnormality of the pancreas to sight or touch could be detected. Three cases in which the urine during life gave a well-marked deposit of crystals were not operated on, but chronic pancreatitis was found after death. In another case in which the urine gave a similar reaction malignant disease of the pancreas was found post mortem. A clinical diagnosis of cancer of the pancreas was made in 2 patients whose urine gave many fine rapidly dissolving crystals. In one of these a large mass was found at operation occupying the head of the gland and probably compressing the duct. The clinical symptoms in 14 control cases did not suggest pancreatic disease, but the urine gave a well-marked reaction. Of these patients, 3 were suffering from mumps, 2 from cancer with secondary deposits, 2 from disease of the kidney with albuminuria, 2 from fractured bones, 1 from peritonitis, 1 from chronic colitis, and 1 from pneumonia.

A small deposit of crystals, considerably less than in the previous group, was obtained in 35 cases. In 3 of these the pancreas was probably diseased. One showed a small stone in the common duct and some enlargement of the gland. A history of gall-stone colic was present in the other 2, and when examined at operation the pancreas was found to be enlarged and harder than normal. In the remaining 32 a lesion unconnected with the pancreas was found.

A deposit consisting of broad flat sword-like crystals, soluble in 33 per cent. sulphuric acid in 4-5 minutes, was obtained in 21 cases. Clinically these were all diagnosed as malignant disease of the pancreas, but only 2 were operated on. One died a few days after the

operation, and the other lived for several weeks, but finally died of the disease. Thirteen are known to have steadily got worse and died at later periods with secondary symptoms of malignant growth. In 2 a post-mortem examination was obtained, and in both the diagnosis was confirmed. The crystals were difficult to secure in several instances, and without the exercise of considerable care and patience it is quite possible that no result would have been obtained. In these cases of pancreatic cancer and in many others of suspected pancreatic disease much assistance in diagnosis was derived from a microscopical and chemical examination of the fæces.

A few crystals were seen microscopically in the urine from eleven cases, but none of them showed any evidence, either clinically or at operation, of disease of the pancreas.

No reaction whatever was obtained in 93 cases. Two of these died and were examined a short time subsequent to the examination of the urine. The pancreas in both was found to be normal macroscopically and microscopically. Thirty-two were operated on. In 9 gall-stones were found in the gall-bladder or cystic duct, but the pancreas was said to be normal. In three a calculus was found above the pancreatic portion of the common bile-duct and 6 showed a biliary concretion floating in the common duct, but no affection of the pancreas. No obstruction of the common duct or disease of the pancreas could be found to explain the jaundice present in three cases. In two patients there was said to be pancreatitis when the gland was examined at operation. The remaining 59 cases were not operated on. In 8 there was at the time the urine was examined a suspicion of pancreatic disease, which the subsequent course of the case dispelled. Fifty-one were control cases examined for the purpose of comparison, 2 had cancer of the liver, 5 gastric ulcer, and 2 ulcer of the duodenum, while the remainder suffered from diseases unconnected with the pancreas.

From the urine of 3 cases of pancreatitis and 2 cases

of diabetes I succeeded in isolating a substance which gave the reactions of a proteid, but none with phenylhydrazin. Solutions of this on hydrolysis with hydrochloric acid yielded a crystalline phenylhydrazin compound resembling that obtained from the urine direct. In consequence of this observation I was led to try the effect of treatment of the urine with mercuric chloride before hydrolysis. In 24 out of 126 cases in which the urine gave a reaction before treatment it was found that the addition of half its volume of a saturated solution of corrosive sublimate entirely prevented the reaction. Three of the patients were suffering from diabetes, and in one, whose pancreas was examined after death, it was found that almost the whole of the glandular substance had been replaced by fibrous tissue. In another the glycosuria had supervened eight and a half years after an operation for pancreatitis. Active pancreatitis associated with obstruction of the common duct was found in 17 cases. In 4 the pancreas was enlarged and hard, but there was no obstruction. The reaction, although not completely prevented, was appreciably diminished in 30 cases. Twenty-four of these were found at operation to have an enlarged and hardened pancreas, and in eleven there was a gall-stone in the common duct. Two were diagnosed as cancer of the pancreas and ended fatally. One case with an affection of the kidney, 1 with mumps, and 1 with advanced carcinoma of the breast, showed some diminution in the amount of the deposit. No appreciable difference was observed in the urine of 69 cases. Of these 10 were found to be suffering from pancreatitis, which in 4 was known to be of some standing. In 14 a diagnosis of malignant disease of the pancreas was made. The remainder were cases in which the results of operation or the clinical condition and after-history of the case negatived pancreatic mischief.

With nitric acid (0·25 c.c. to 10 c.c. of urine) similar results to those described with hydrochloric acid and sulphuric acid have been obtained, but the reaction has been found to be more difficult to control. Crystals of a kind

can be prepared under most circumstances from suitable specimens of urine, but, with care, those cases in which there was obstruction of the common duct and marked swelling of the pancreas have been found to yield small masses of fine lemon-yellow needles quite distinct from the brownish sheaves with which they were generally mixed. As the excess of nitric acid left after heating cannot be removed as an insoluble salt, and as even a slight excess of alkali has been found to interfere with the subsequent course of the reaction, the fluid is diluted with 10 c.c. of urine, made up to 15 c.c. with water and a slightly larger amount of sodium acetate added with the phenylhydrazin hydrochloride than usual. [I may here mention that by heating a solution of glycerine with nitric, *not* hydrochloric, acid and neutralising with urea I have repeatedly obtained on treatment with phenylhydrazin an osazone with a melting point of 131°C., and that it was on this fact that the original investigations on the urine in diseases of the pancreas were founded.]

It is evident that in phenylhydrazin we possess a most useful reagent for inquiring into variations in the urine accompanying certain changes in metabolism. In the hands of those who have had some experience of its application and the interpretation of the results that may be obtained by its use, the test is capable of more than the detection of traces of dextrose in slight or doubtful cases of diabetes, important as this may be. Although advocating a more extended use of this test in the examination of urine, I do not wish it to be thought that I am under-estimating the value of other reactions or methods of investigations. Like most laboratory methods in the present state of our knowledge, it has its limitations and needs to be interpreted in the light of all the available evidence, not only from a complete chemical and microscopical examination of the urine but also from the clinical aspects of the case and under certain circumstances from the investigation of the fæces, or blood, or other "products."

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THE CONSUMPTIVE WORKING MAN

A STUDY OF THE SOCIAL AND ECONOMIC CONDITIONS WHICH GOVERN HIS LIFE

BY

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DETAILED RECORD OF THE TEN PATIENTS.

CASE 1.—*Age.*—30.
Occupation.—Officer in the Salvation Army.
Type of disease.—Early (infiltration) disease of one lobe.
Duration of disease.—3 months.
Number of weeks in Cottage San.—12.

	Condition on admission.	*Condition on discharge.*
Fever	*Nil.*	*Nil.*
Digestion	Normal.	Normal.
General health	Very fair.	Excellent.
Weight	11 st. 5 lbs.	11 st. 12 lbs.
Relation of weight to highest known weight	— 7 lbs.	= to.

SOCIAL AND ECONOMIC CONDITIONS AT DATE OF CONTRACTION OF TUBERCULOSIS.

Family.—Patient was a widower with one child.
Work.—This man was an officer in charge of a corps, which means that he was responsible for a branch of Salvation Army activity, work in which he was assisted by the junior officers of the same denomination. This patient was stationed in a small country town, and the following was his routine:

Rises at 6.30 to 7. Breakfast about 8. From 8.30 to 10, correspondence, reading, study, etc. 10 to 12, visitation of the sick, etc. 12 to 1, dinner. 2 to 4.30, visitations. 4.30 to 5, tea. After 5 he prepares for his meeting, which takes place at 6. 7.15, open-air services on four nights a weeks, followed by an indoor service which lasts from 8 to 9.30. On the whole he worked about twelve hours daily, and such work, as far as one can judge, is distinctly hard, and involves a good deal of responsibility. The visiting of the poor in their own homes, of which a good deal is done by Salvation Army officers, necessitates a good many hours being spent in very bad hygienic conditions, and, no doubt, accounted for this man becoming infected with tuberculosis.

Home conditions.—The patient lived rent free at the Salvation Army Headquarters, and his conditions in this respect were quite satisfactory.

Wages.—Officially, this man had 28*s.* per week, with free lodgings and allowance of 1*s.* per week for his child. It is, in practice, however, a difficult matter to accurately estimate the income of a Salvation Army officer in charge of a corps. To give an idea of what this man's financial possibilities were it is perhaps best to briefly describe the lines upon which he had to run his command, which is typical of the work of all officers in the Army. In the first place, no salary is guaranteed to anyone; men join on this understanding. An officer in charge of a corps has to make himself responsible for all current expenses connected with his work—*e.g.* rent, taxes, and up-keep, etc., of the headquarters. The income to meet these charges is obtained by the officers from collections at meetings and money given by local sympathisers with the cause. From the weekly income which remains after defrayment of this first charge, viz., current running expenses, the officer is entitled to keep 28*s.* per week for his own salary, and whatever remains after this has been taken out is handed over to the general funds of the Army. In addition to this salary the officer is allowed to take gifts in kind from the members of the congregation—*e.g.* coals, food, etc. —and in many instances this is a material asset in their finances. A further source of income, possibly 1*s.* to 2*s.* per week, is that derived from the sale of literature and uniforms to local members of the Army. From this it will be seen that the officer depends for his wage upon his own capacity to do profitable work, and, as one would expect, the income made by different men in different places varies very greatly. In the majority of cases it would seem that officers have no difficulty in collecting sufficient money to provide themselves with their official allowance, after payment of

the current expenses. In districts, however, which are unfriendly to the Salvation Army the income may be insufficient to provide the officer in charge with his salary, and in such case he can have this deficit rectified by applying to headquarters. In practice, however, it seems that officers who fail to make both ends meet frequently do not apply for financial assistance, preferring to get along as best they can. This is really unnecessary self-denial; at the same time, one understands from Salvationists that for an officer to apply for financial help is to some extent a confession of failure, and not calculated to help him up the ladder of promotion. From this cause some officers in the Salvation Army have a weekly income which is, in my opinion, inadequate to keep them physically efficient. The officer whose case we are now considering was a popular man, who did well, and had no difficulty in obtaining his salary, which in all amounted to about 30*s.* per week.

The following is an approximate statement of his income and expenditure at this date:

Income.	£	*s.*	*d.*	Expenditure.	£	*s.*	*d.*
Salary . . .	1	10	0	Rent (*nil*) . .			
				Household expenses	0	5	0
				Clothes . . .	0	5	0
				Balance for food .	1	0	0
	£1	10	0		£1	10	0

No. in family in terms of men . . . . . 2·1
Approximate weekly sum available for food per man . 10*s.* 6*d.*

Financial Condition of Patient and his Family during his stay in the Sanatorium.

During this patient's stay in the sanatorium the expenses there were paid entirely by the Salvation Army, who, in addition, kept his child during the same period.

Condition on Discharge from the Cottage Sanatorium in May, 1903.

Lung Disease.—Arrest apparently completed, physical signs limited to impaired resonance, no adventitious sounds being audible.

General health.—Completely restored, walking 10 miles daily.

Normal working capacity.—Completely restored.

After History.

May, 1903.—After discharge, the Salvation Army thought it best not to send him back to his former duties as officer in

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My experience of this reaction has been chiefly gained

in connection with work on diseases of the pancreas which I have carried out in conjunction with Mr. Mayo Robson. Up to December 31st, 1904, I have had the opportunity of examining 479 specimens of urine from 295 cases, most of whom were under Mr. Robson's care, and on many of whom he performed an abdominal section.

In 130 cases the urine when treated with hydrochloric acid and neutralised with lead carbonate yielded with phenylhydrazin hydrochloride and sodium acetate a considerable flocculent brown deposit which, under the microscope, was found to consist of yellow crystals much smaller than glucosazone and generally arranged in rosettes. In some the crystals were slender and somewhat resembled small lactosazone or pentosazone crystals, while in others they were less delicate, and approached the glucosazone or even maltosazone type in shape. Their time of solution in 33 per cent. sulphuric acid varied from half to one and a half or two minutes when the mean of several observations was taken. On being filtered off, washed, extracted with boiling 50 per cent. alcohol and re-crystallised they gave a melting point of 168° to 170° C.

The clinical symptoms in 116 of these cases pointed to disease of the pancreas, and in 79 it was possible to ascertain the condition of the parts at operation. In 5 the diagnosis was confirmed post mortem, shortly after the last examination of the urine had been made. Three cases from whose urine fine rapidly dissolving crystals were isolated were found to be suffering from acute pancreatitis with fat necrosis. One succumbed shortly after operation and was examined after death. In 37 gall-stones were found impacted in the lower part of the common bile-duct and the pancreas was stated to be unmistakably enlarged and hardened. In 2 cases a similar condition of the gland was found, associated in one with obstruction and considerable dilatation of the common bile-duct, and in the other with a malignant growth of the duodenal papilla which subsequently proved fatal. In two cases pancreatic calculi were found, and in two others

Home conditions.—Are equally satisfactory. He lives over the stable in comfortable quarters, and he always keeps his windows open. He takes his meals with the gardener and his wife.

Wages.—He earns 18*s.* per week, but shortly expects to rise to 20*s.*; in addition he lives rent free.

The following is an approximate statement of his income and expenditure at the present time:

Income.	£	*s.*	*d.*	Expenditure.	£	*s.*	*d.*
Wages . . .	0	18	0	Food . . .	0	11	0
				Clothes . . .	0	1	6
				Tobacco, etc. . .	0	2	0
				Beer . . .	0	2	0
				Sends his mother .	0	1	6
	£0	18	0		£0	18	0

The following is approximately the patient's weekly dietary. In this respect the patient stated that his appetite was excellent, and that he is now much more careful about his meals than he was before his illness.

	Breakfast.	Dinner.	Tea.	Supper.
Sunday	Bacon, egg, bread, butter, tea	Roast pork, potatoes, sprouts, blackberry tart	Bread, butter, tea, cucumber, cake	Bread, cheese, mushrooms, cocoa.
Monday	2 eggs, bread, butter, tea	Cold pork, bread, blackberry tart	Bread, butter, cake, jam	Bread, cheese, cocoa.
Tuesday	Bacon, bread, butter, tea	Mutton chop, marrow and potatoes, tapioca	Bread, butter, jam, cake, tea	Bread, cheese, cocoa.
Wednesday	Bloater, bread, butter, tea	Liver and bacon, marrow, potatoes	Bread, butter, jam, cake, tea	Bread, cheese, cocoa.
Thursday	Bacon, bread, butter, tea	Steak, sprouts, potatoes, apple pudding	Bread, butter, jam, cake	Bread, cheese, cocoa.
Friday	Haddock, bread, butter, tea	Sheep's heart, potatoes, marrow, apple pudding	Bread, butter, jam, cake, and tea	Bread, cheese, cocoa.
Saturday	Eggs, bacon, bread, butter, tea	Steak pudding, marrow, potatoes, tapioca	Bread, butter, jam, tea, cake	Tinned salmon, bread, butter, cocoa.

He is drinking no milk as a practice, as he doesn't want the bother of it.

At the present time he drinks about two pints of beer daily.

This patient pointed out in reference to the above dietary that he was fortunate in getting many extras, such as mushrooms, blackberries, etc., on account of living in the country. Had he to buy these things he would not have them.

CASE 3.[1]—*Age.*—38.
Occupation.—Wood sawyer.
Type of disease.—Early disease (infiltration) of upper and lower lobe of right lung.
Duration of disease.—4 months.
Number of weeks in Sanatorium.—14.

	Condition on admission.	*Condition on discharge.*
Fever	*Nil.*	*Nil.*
Digestion	Poor.	Excellent.
General health	Fair.	Excellent; walking 10 miles daily.
Weight	10 st. 6 lbs.	11 st. 9 lbs.
Relation of weight to highest known weight before illness.	−14 lbs.	+ 3 lbs.

SOCIAL AND ECONOMIC CONDITIONS AT DATE OF CONTRACTION OF TUBERCULOSIS.

Family.—His family consisted of himself, his wife, and two young children.

Work.—As a wood sawyer he worked in a close shop along with some 16 other men. The shop is badly ventilated and very stuffy; moreover, the air is constantly full of dust.

Home conditions.—He lived in a small house in Walworth, the patient and his family occupying three rooms. The patient himself is a steady, intelligent man, and his wife appeared to be a sensible and capable person.

Wages.—He earned 32*s.* per week.

The following is an approximate statement of his income and expenditure at this date:

INCOME.	£	*s.*	*d.*	EXPENDITURE.	£	*s.*	*d.*
Wages	1	12	0	Rent, per week	0	6	6
				Clothes, fuel, etc.	0	5	0
				Sick club	0	0	6
				Food, etc.	1	0	0
	£1	12	0		£1	12	0

No. in family in terms of men . . . . 3·3
Approximate weekly sum available for food per man . 5*s.* to 6*s.*

[1] This patient was one of those treated at Mundesley Cottage Sanatorium at an inclusive fee of 17*s.* per week.

Financial Condition of Patient and his Family during his stay in the Sanatorium.

The sole personal resources of this patient in case of illness consisted of 10*s.* a week from his sick and benefit club, a hopelessly inadequate sum for the requirements of his family. The C.O.S., however, allowed him 23*s.* a week throughout his three months' stay in the Cottage Sanatorium, which enabled him to pay for his treatment (17*s.*) and at the same time maintain his family.

The weekly budget of income and expenditure during this period worked out as follows:

Income.	£	s.	d.	Expenditure.	£	s.	d.
Club money . .	0	10	0	Fee at Cottage San.	0	17	0
From C.O.S. . .	1	3	0	Rent . , .	0	6	6
				Household expenses	0	1	6
				Food . . .	0	8	0
	£1	13	0		£1	13	0

No. of family (at home), in terms of men . . 2·3
Weekly sum available for food per man . . 3*s.* 2*d.*

In addition to this assistance, the C.O.S. allowed the man a guinea a week after his discharge from the Sanatorium while he was looking for work, and also allowed him a further 10*s.* a week when his benefit money from the sick club ceased—viz. after 12 weeks' payments.

Condition on Discharge from the Cottage Sanatorium on May 9, 1903.

Lung disease.—Completely arrested, physical signs being limited to impaired resonance over the affected area, and no adventitious sounds being audible.

General health.—Perfectly restored; walking 10 miles a day.

Normal working capacity.—Completely restored.

After History.

May, 1903.—On his discharge he returned to his home in Walworth. He did no work, but kept out of doors as much as possible. His income at this time consisted of 31*s.* per week.

August, 1903.—During August he was at the Gladstone Convalescent Home at Mitcham, where he continued in excellent health.

September, 1903.—In September, thanks to the efforts made on his behalf by the C.O.S., he had an opportunity to emigrate to Canada, but the emigration authorities, after everything else had been arranged, refused to pass him. Having failed in their attempt to get him abroad, the C.O.S. tried to find him some sort of open-air employment by advertising, etc., but without success. In the middle of September the

patient, tired of waiting for work, and being anxious to once more earn money for himself, returned to his original occupation in the saw-mill.

April, 1904.—In April, 1904, he gave us the following replies to a series of questions we sent him: "I started work in the middle of September, and I work eight hours a day. I am back at my old job again at the saw-mills. I kept my windows all open until lately, but find that the wife and baby cannot stand it, so cannot keep so strict as I should like. I kept in fine health until I had been at work a fortnight, and then got very bad with my chest. My firm got me a letter for St. Thomas's Hospital. I saw Dr. Perkins and told him the history of my case. He examined me, and said that wherever I had been they had made an excellent cure of me, and he was pleased to tell me that nothing of my old disease had returned. He signed my paper 'acute bronchitis.' I earn on an average 29*s.* per week. My appetite is not so good as it was at Mundesley, and I wish you could send a little Norfolk air up here."

November, 1904.—In November, 1904, this patient was visited at his own home. The patient was found to be looking in good health, and was working eight hours per day at the saw-mills, and had been doing so almost constantly for the past twelve months. Except for recurrent attacks of bronchitis, apparently brought on by his work, since he never suffered from it while at Mundesley, he said that he enjoyed good health, and was able to do his full day's work without being knocked up. Since his return home from Mundesley he had always kept the front door of his house open, but he was not able to keep his windows open very much on account of the tendency of his wife and baby to catch cold. The baby was born while the patient was at Mundesley, thus making his family three in addition to his wife. The patient said he had learned a good deal at Mundesley as to how to live, and was much more careful in his habits, especially with regard to his food, than he had been formerly. His wages since his return to work have been 30*s.* per week, or two shillings a week less than before his illness. This was due to his having returned to a somewhat easier but less remunerative branch of the saw-mill work.

The following is an approximate statement of his weekly income and expenditure at the present time:

Income.	£	*s.*	*d.*	Expenditure.	£	*s.*	*d.*
Wages . . .	1	10	0	Rent . . .	0	6	6
				Clothes, fuel, &c. .	0	4	0
				Slate club . .	0	0	6
				For food, etc. .	0	19	0
	£1	10	0		£1	10	0

No. in family, in terms of men . . . . 3·6
Approximate weekly sum available for food per man . 5*s.* 4*d.*

At the present time the weekly expenditure upon food is approximately as follows:—

	s.	*d.*
1 lb. of meat daily	5	10
½-qrtn. loaf daily	1	5½
1 quart milk daily	2	4
2 lbs. potatoes per day	1	2
2 lbs. other vegetables daily	1	2
¼ lb. butter daily	2	4
3 lbs. jam per week	0	9
Cocoa for a week	2	2½
Fish, for a week	0	6
Tea for a week	0	2½
Bacon for a week, 1 lb.	0	9
	18	8½

The large amount spent upon cocoa is due to the patient taking this instead of milk, as he found it agreed with him better.

Sample week's dietary taken by patient since his return from the Cottage Sanatorium:—

	Breakfast.	Dinner.	Tea.	Supper.
Sunday	Bloater, bread and butter, cocoa	Steak and kidney pudding, custard and baked apple	Cocoa.	Cocoa.
Monday	Bacon and tomatoes, bread and butter, cocoa	Steak, beans and potatoes	Bloater, bread and butter, cocoa	Cocoa.
Tuesday	Bacon and tomatoes, bread and butter, cocoa	Chop, beans and potatoes	Bread and butter, jam, and cocoa	Cocoa.
Wednesday	Bacon and tomatoes, bread and butter, cocoa	Stewed steak, with vegetables, potatoes, rice pudding	Bread and butter, jam, and cocoa	Cocoa.
Thursday	Bacon and tomatoes, bread and butter, cocoa	Steak pudding, potatoes, and cauliflower	Herring, bread and butter, cocoa	Cocoa.
Friday	Bacon and tomatoes, bread and butter, cocoa	Steak, cauliflower, potatoes	Cocoa, bread and butter	Cocoa.
Saturday	Sausages and tomatoes, bread and butter, cocoa	Cauliflower, potatoes, and steak.	Cocoa, bread and butter, jam	Cocoa.

This diet is taken by the patient himself; the other members of his family are not always able to afford such good food-stuffs.

CASE 4.[1]—*Age.*—28.
Occupation.—Cook in coffee-shop off the Strand.
Type of disease.—Early disease (infiltration) of one lobe.
Duration of disease.—3 months.
No. of weeks in Cottage San.—13.

	Condition on admission.	*Condition on discharge.*
Fever	*Nil.*	*Nil.*
Digestion	Good.	Good.
General health	Good.	Excellent; walking 10 to 12 miles per day.
Weight	11 st. 10 lbs.	12 st. 1 lb.
Relation of weight to highest known weight	= to	+ 5 lbs.

SOCIAL AND ECONOMIC CONDITIONS AT DATE OF CONTRACTION OF TUBERCULOSIS.

Family.—Unmarried. No one dependent upon him.
Work.—His work was that of cook and carver in a coffee-shop in a side street off the Strand, where he had been for the past eight years. His working hours were from 6 a.m. to 8 p.m., but he was off duty from three to four in the afternoon. The kitchen in which he worked is about 12 by 13 ft. in size, situated behind the shop. The windows of the kitchen are very small, and the room itself is always very hot, containing a gas stove in addition to the cooking range.
Home conditions.—He slept over the shop in a room measuring some 12 ft. by 16 ft. The room had a good-sized window, which he always kept closed. He shared this room with a boy aged 7, and on three nights of the week another man also slept in it. He got his meals regularly and had plenty of food.
Wages.—He earned 20*s.* per week, with board and lodging all found.

The following is an approximate statement of his income and expenditure at this time:

INCOME.	£	*s.*	*d.*	EXPENDITURE.	£	*s.*	*d.*
Wages	1	0	0	Subscription to Oddfellows' Club	0	1	6
				Food and rent, *nil.*			
	£1	0	0		£0	1	6

[1] This patient was one of those treated at the Mundesley Cottage Sanatorium for an inclusive fee of 17*s.* a week.

The balance is 18*s.* 6*d.* This man was unable to state his expenses, but his chief expenditure was upon linen aprons, etc. His total expenditure, however, was considerably less than his income. His subscription of 1*s.* 6*d.* per week to the Oddfellows entitled him to a sum of 10*s.* a week for six months when ill.

FINANCIAL CONDITION OF PATIENT DURING HIS STAY IN THE SANATORIUM.

From the Charity Organisation Society . .	7*s.* a week.
From the Oddfellows' Benefit Club . .	10*s.* ,,

CONDITION ON DISCHARGE FROM THE COTTAGE SANATORIUM IN APRIL, 1903.

Lung disease.—Completely arrested; impaired resonance only; no adventitious sounds audible.

General health.—Completely restored; walking ten to twelve miles daily.

Normal working capacity.—Completely restored.

AFTER HISTORY.

April, 1903.—On leaving Mundesley this patient returned to London, and on our advice gave up his work at the coffee-shop and tried to find a healthier kind of employment. This he found a very difficult matter, and for three months he did nothing, and not being at all well off, he had rather a hard time, and was not able to get proper meals. He maintained his health, however, and in July obtained a situation as 'bus conductor.

July, 1903.—As 'bus conductor he worked from twelve to fifteen hours a day, and earned 30*s.* per week. Out of this sum he had to pay for his board and lodging, clothes, etc., and was comfortably enough off. During the autumn of 1903 he married and went to live at Putney. He now found his income of 30*s.* per week to be barely sufficient for his requirements, and in

July, 1904—he gave up his post as 'bus conductor, which he had held twelve months, and returned to his original employment in the coffee-shop.

November, 1904.—When visited in November, 1904, this patient appeared to be perfectly well, and was apparently comfortably off.

SOCIAL AND ECONOMIC CONDITIONS IN NOVEMBER, 1904.

Work.—His work as a cook and carver in the coffee-shop has already been described in the account of his conditions of

work before contracting tuberculosis. He finds the work itself easy enough, but feels the heat much more than he used to do. He has lost 14 lbs. in weight during the three months he has worked in the cook-shop.

Home conditions.—He is living in Putney with his wife and one child. When at home he keeps out of doors as much as possible, and his bedroom has three windows, of which the one nearest to himself is always kept open.

Wages.—Unknown, patient declining to state what his wages are, but stated that he found his present work to be more profitable than 'bus conducting, and, moreover, more to his liking. We are safe to assume that his income is something over 30*s.* per week.

The patient also declined to give any information as to his income and expenditure.

His diet is usually about as follows:

Breakfast.—Eggs and bacon.

Dinner.—Meat and vegetables and pudding.

Tea.—Bread and butter and tea; sometimes jam or celery.

Supper.—Fish, or bread and cheese, or bread and butter.

Drinks about a pint of milk every day and a glass of stout for supper.

CASE 5.—*Age.*—22.

Occupation.—Coal-miner.

Type of disease.—Early disease (infiltration) of apex and left lung.

Duration of disease.—4 to 6 months.

Number of weeks in hospital.—20.

	Condition on admission.	*Condition on discharge.*
Fever	*Nil.*	*Nil.*
Digestion	Good.	Good.
General health	Poor.	Excellent.
Weight	8 st. 12 lb.	9 st. 10 lbs.
Relation of weight to highest known weight before illness	— 12 lbs.	= to.

SOCIAL AND ECONOMIC CONDITIONS AT DATE OF CONTRACTION OF TUBERCULOSIS.

Family.—His family consisted of his mother, himself, and younger brother, and a sister. (The patient was the only one at work, and therefore bread-winner of the family.)

Work.—In the coal-mine his work consisted of picking coal from the seam and taking it to the foot of the shaft, a distance of some 400 yards—a particularly bad occupation for a consumptive. He worked 9 hours a day.

Home conditions.—He lived in a small cottage near the pit, which was in the country. The house had two bed-rooms and one sitting-room. The patient himself is a steady fellow.

Wages.—He earned 16*s.* to 18*s.* per week.

The patient is unable to give even an approximate statement of the expenditure at this time; all he knows is that the total income of the family was his 18*s.* per week, and that the whole family lived upon this sum. This patient and his family must be classed among the very poor, 16*s.* to 18*s.* per week being a totally inadequate sum for their proper maintenance.

No. in family in terms of men . . . 2·8.
Approximate weekly sum available for food per man 3*s.*

Financial Condition of Patient and his Family during his stay in Hospital.

The patient says that his family were not any worse off when he had to go to the infirmary, "for his mother found a home and had sufficient income for herself and the two younger children." She had not enough, however, to afford to keep him on his return from the infirmary.

Condition on Discharge from the Infirmary in October, 1899.

Lung disease.—Arrest apparently complete, there being no physical signs except impaired resonance at the apex.

General health.—Completely restored.

Normal working capacity.—Completely restored.

After History.

December, 1899.—After discharge from the infirmary, he gave up his work in the coal-mine and found employment as an out-door labourer to a local telephone company. His work now consisted of helping to erect telephones, and though out of doors was of a fairly arduous nature, since he had to do a good deal of telephone-pole climbing with climbing irons. He spent most of his evenings out of doors. His working hours were 7 a.m. to 5 p.m. The following were his economic conditions at this important period: He lived with a married sister who was in fairly comfortable circumstances, his home conditions being thus satisfactory. His work, as already stated, was of an out-door nature, and proved to be very satisfactory. His income, too, of a guinea per week was more than sufficient for his wants, and was spent in the following manner:

Income.	£	s.	d.	Expenditure.	£	s.	d.
Income . . .	1	1	0	Board and lodging .	0	12	0
				Tram fares . .	0	1	0
				For support of mother	0	4	6
				Kept for himself .	0	3	6 [1]
	£1	1	0		£1	1	0

The approximate weekly sum available for food was something between 6*s.* to 8*s.*, an ample sum for his requirements. The actual diet he took was approximately as follows:—

Breakfast.—Tea only.

Lunch, 10 a.m.—Bread, butter, and jam.

Dinner at 12.—Pork chop with potatoes, and plenty of milk pudding. No bread or butter.

Tea at 6.—Bread and butter, and egg and tea.

Supper at 10.—Either pork-pie, fish and potato-chips, or bread and poloney sausage, and a cup of cocoa made with water. In all he took 12 ozs. of bread per day.

September, 1900.—He reported himself as being perfectly well, and still at work for the telephone company.

February, 1901.—He again reported himself, this time from Barnsley, as being perfectly well and still at the same work.

January, 1902.—He was visited by Mr. Chapman in his own home in Sheffield. He was then still at work for the telephone company, and had not missed a day's work since commencing it in December, 1899. Examination of his chest showed no evidence of lung disease except slight loss of resonance over the area originally affected. In appearance and in every other respect he was perfectly normal. He weighed 10 st. 2 lbs., or 5 lbs. more than when discharged from the infirmary. He was doing a hard day's work without the least fatigue, and in addition frequently walked home from his work in Barnsley, a distance of fifteen miles. Examination of his blood gave the following result: hæmoglobin 95 per cent., red corpuscles 4,300,000, giving a decimal of 1·1, a very good result and an indication that his diet and life generally were satisfactory.

1903.—Throughout 1903 he fully maintained his health, and remained at the same work. In June of this year he married.

January, 1904.—In answer to a letter, his sister sent us the following report about him. "My brother is now very busy. He has now been promoted from labourer to be motor-man, and he now looks after a large engine which makes electric light for the works. I am sure he has never enjoyed better health than he does now. He never complains of anything,

[1] Of this 3*s.* 6*d.*, the patient now tells me, he was careful to save what he could for fear he should be ill again.

no cough or spit. We never expected him to live before he went to the infirmary, but people won't now ever believe he had consumption."

January, 1905.—In answer to a letter the patient sent the following report of himself and his economic and social conditions.

SOCIAL AND ECONOMIC CONDITIONS IN JANUARY, 1905.

Family.—His family consists of himself and his wife, the only child they had having died.

Work.—He is in charge of a 150 h.-p. motor attached to a large grinding-shop. This work, as he says, is indoors, but he is none the worse for it. He gave up work for the telephone company because he found the going about to different towns entailed a good deal of expense in the shape of lodgings, etc. Though less remunerative than the present work, the telephone company work was certainly healthier. He works eight hours a day, but in addition there is often overtime work, for which he is paid extra. On the average he works some ten hours daily.

Home conditions.—He lives in a four-roomed cottage in Attercliffe, a thickly populated working class suburb in Sheffield, as smoky as Sheffield itself.

Wages.—He earns 22*s.* per week for forty-eight hours, but his income is rather more than this owing to the extra payment for the overtime.

The following is an approximate statement of his income and expenditure :

INCOME.	£	*s.*	*d.*	EXPENDITURE.	£	*s.*	*d.*
Average income .	1	6	0	Rent . . .	0	5	6
				Food . . .	0	13	0
				Coals, light, etc. .	0	1	6
				Balance for clothes and household expenses . . .	0	6	0
	£1	6	0		£1	6	0

No. in family in terms of men . . . 1·8.
Weekly sum available for food per man . . 7*s.*

CASE 6.[1]—*Age.*—32.

Occupation.—Coachman.

Type of disease.—Incompletely arrested disease of three lobes of the right lung.

Duration of disease.—Two years, with periods of more or less complete arrest.

No. of weeks in Cottage San.—15.

[1] This patient was one of those treated at the Mundesley Cottage Sanatorium at an inclusive fee of 17*s.* per week.

	Condition on admission.	*Condition on discharge.*
Fever	*Nil.*	*Nil.*
Digestion	Good.	Good.
General health	Fair, but unfit for work.	Practically quite restored.
Weight	8 st. 6 lb.	8 st. 9½ lb.
Relation of weight to highest known weight.	— 3 lbs.	= to

SOCIAL AND ECONOMIC CONDITIONS AT DATE OF CONTRACTION OF TUBERCULOSIS.

Family.—His family consisted of himself, his wife (suffering from pulmonary tuberculosis), and three young children.
Work.—Worked as a coachman in a livery stables in London.
Home conditions.—Unknown.
Wages.—25*s.* per week, and lodgings.

The following is an approximate statement of his income and expenditure at this date:

INCOME.	£	*s.*	*d.*	EXPENDITURE.	£	*s.*	*d.*
Wages	1	5	0	Rent (*nil*)			
				Household expenses	0	6	6
				Club subscription	0	0	6
				Balance for food	0	18	0
	£1	5	0		£1	5	0

No. in family, in terms of men . . . . 3·0
Approximate weekly sum available for food per man . 6*s.*

FINANCIAL CONDITION OF PATIENT AND HIS FAMILY DURING HIS STAY IN THE SANATORIUM.

The only private resources which this patient had in case of sickness was 10*s.* a week from a sick club, which fell to 5*s.* a week after six months. When he had to give up his work, he lost his house as well as his income, and in consequence had but 10*s.* a week with which to provide his consumptive wife and three children with food and lodging. The only thing for him to do under these circumstances was to apply to the Guardians for relief, who then took charge of his family, the patient contributing to their support his club money allowance. The C.O.S. meanwhile paid his fee of 17*s.* a week for his treatment at the Mundesley Cottage Sanatorium. After his discharge from the sanatorium he was only out of work for a week, and during this time he received from the C.O.S. a quart of milk daily and an allowance of 6*s.* for food. During his stay at the sanatorium one child died, which reduced his family after discharge to a wife and two children.

CONDITION ON DISCHARGE FROM THE COTTAGE SANATORIUM IN JUNE, 1903.

Lung Disease.—Quiescent and considerably arrested, adventitious sounds being few in number.

General health.—Very largely restored, walking seven miles per day.

Normal working capacity.—Probably quite restored.

AFTER HISTORY.

June, 1903.—On his discharge from the Cottage Sanatorium this patient returned to London and looked for another situation as coachman, as this appeared to be the best thing he could do. Being a man with a good character, he had no difficulty in obtaining a situation.

April, 1904.—In April, 1904, in answer to our letter, this patient sent the following account of himself: "I am pleased to say that I am keeping well and strong since I left Mundesley. I have got a very comfortable place as coachman, and have been in it about five months. I have been at work since the week after I left Mundesley, and have never lost a day; in fact, I feel as well as ever I did in my life, and my weight is about the same as when I left Mundesley. I always sleep with my windows open. My wages are 29*s.* per week, with rooms, gas, and coal. I have all my meals at home. I generally have eggs and bacon or fish for breakfast, and beef or mutton for dinner, with sometimes a milk pudding, jam roll, or suet pudding. For supper I have cold meat and bread and cheese. I don't drink much milk, as I don't much care for it. I have a glass or two of beer sometimes, and occasionally a glass of whisky; in fact, I don't find that anything hurts me. As to how many hours a day I work—well, you know what a coachman's work is; sometimes I'm out late at night. I quite believe that the treatment at Mundesley has set me up again."

November, 1904.—In November, 1904, this patient was visited in his own home.

SOCIAL AND ECONOMIC CONDITIONS IN NOVEMBER, 1904.

Work.—He is a private coachman to a gentleman, and lives in a mews in Kensington. In addition to the driving, which entails irregular and sometimes late hours, he has to clean the carriage, which means a certain amount of muscular exertion. However, he finds the work well within his powers, and has not lost a day's work since his return from Mundesley. The patient expressed himself as being in excellent health, and looked so.

Home conditions.—He lives in a mews in Kensington, having three rooms, rent free. The rooms are not very airy, the

windows being small, but they gave one the impression of their occupants being in comfortable circumstances.

Wages.—He earns 29*s.* per week, with free quarters and gas, and, in addition, gets two suits of clothes a year. He is now, in fact, in better financial circumstances than he was before he went to Mundesley, and has been able to save a little money.

The following is an approximate statement of his income and expenditure:

Income.	£	*s.*	*d.*	Expenditure.	£	*s.*	*d.*
Wages . . .	1	9	0	Rent (*nil*) .			
				Household expenses	0	8	0
				Subscription to club	0	0	6
				Balance for food .	1	0	6
	£1	9	0		£1	9	0

No. of family, in terms of men . . . . 2·6

Approximate weekly sum available for food per man . 7*s.* to 8*s.*

The following is a sample week's dietary taken by this patient at the present time:

	Breakfast.	Dinner.	Tea.	Supper.
Sunday	Bloater, bread, butter, tea	Rabbit pie, potatoes, cauliflower, blancmange, apples, bread	Tea, bread, butter, cake	Cold beef, cold beans, potatoes, beetroot, bread, stout.
Monday	Cold rabbit pie, bread and butter, tea	Stewed rabbit, carrots, turnips, potatoes, bread	Tea, bread, and butter	Bread, butter, fresh herring, stout.
Tuesday	Egg and bacon, bread, butter, tea	Stewed rabbit, potatoes, ground rice, bread	Toast, butter, tea	Haddock, bread, butter, stout.
Wednesday	Kippers, bread, butter, tea	Lamb, potatoes, cauliflower, onion sauce, bread	Bread, butter, and tea	Cold mutton and cauliflower, bread, stout.
Thursday	Bread, butter, bacon, egg, tea	Mashed turnip, cold mutton, potatoes, jam, suet pudding, and bread	Tea, bread, and butter	Cold mutton, bread, stout.
Friday	Kippers, bread, butter, tea	Stewed steak, carrots, turnips, bread	Bread, butter, tea	Mackerel, bread and butter, stout.
Saturday	Pressed pork, bread, butter, tea	Stewed steak, turnips, potatoes, carrots, bread	Bread, butter, tea	Fish, bread, butter, and stout.

Patient always drinks half a pint of stout for lunch, and at night a half pint of the same. In addition to this he usually has a glass or two of stout during the day, and occasionally he takes spirits.

He drinks practically no milk whatever, except that in tea, puddings, etc.

CASE 7.—*Age.*—19.
Occupation.—Apprenticed to the cabinet-making trade.
Type of disease.—Early disease (infiltration) of one apex.
Duration of disease.—Indefinite; probably twelve months, with long period of complete arrest. Recent illness of three months' duration.
No. of weeks in San.—10.

	Condition on admission.	*Condition on discharge.*
Fever	98·6 a.m., 100·4 p.m.	*Nil.*
Digestion	Normal.	Normal.
General health	Very poor.	Excellent.
Weight	8 st. 12 lbs.	11 st.
Relation of weight to highest known weight before illness	?	Unknown, but certainly well above.

SOCIAL AND ECONOMIC CONDITIONS AT DATE OF CONTRACTION OF TUBERCULOSIS.

Family.—This patient was unmarried and lived at home with his father and mother, a married sister and her two children.
Work.—As a cabinet-maker's apprentice he had to work in a very close and dusty shop, his hours being from 7.30 a.m. to 6.30 p.m., with one hour off for dinner. He lived three-quarters of an hour from his work.
Home conditions.—His home was comfortable, Personally he had been rather careless in his habits; he kept late hours, often not being in bed till between 1 and 2 p.m., and he had to rise again at 6. His meals, too, had been somewhat inadequate, although there had been no financial necessity for this. His daily dietary had been roughly as follows: Breakfast, bread and butter and tea; dinner (usually in a cook shop), potatoes, greens, and pudding, and usually meat, on the average costing 8*d.* per day; tea at home consisted of bread and butter and tea; supper, bread and cheese with butter, sometimes also fish and potatoes. On Sunday he always had better meals.
Wages.—His wages were 12*s.* 6*d.* weekly.

The following is an approximate statement of his income and expenditure at this date:

Income.	£	s.	d.	Expenditure.	£	s.	d.
Wages . . .	0	12	6	Fares . . .	0	1	0
				Dinners . . .	0	3	0
				Kept for himself .	0	2	6
				Gave to his family .	0	6	0
	£0	12	6		£0	12	6

The family was independent of the patient's wages, the father having an income of 63*s.* per week, which just about covered their expenses.

Condition on Discharge from the Sanatorium in September, 1903.

Lung disease.—Arrest apparently complete, physical signs being limited to impaired resonance, and no adventitious sounds being audible.

General health.—Excellent—better, in fact, than it had ever been in his life; walking ten to twelve miles a day.

Normal working capacity.—Very largely restored; a tendency to slight hæmoptysis, however, rendered him unfit for anything but light work.

After History.

1903.—After discharge this patient went to live at Henley-on-Thames, to which place his family had moved during patient's illness. Upon his leaving Mundesley he saw Dr. Fowler, at the Middlesex Hospital, in whose wards he had been treated previous to his coming to Mundesley, who advised him not to commence work, but to continue the treatment at home, there being no urgent financial necessity for his doing so.

In November, 1904.—When visited twelve months later this patient was in excellent health, and except for a slight light colour in his sputum on one or two occasions he had kept quite well since his discharge from Mundesley. He is walking twelve miles a day simply for exercise, and seems quite fit for light work. He had not seen Dr. Fowler for four months past. He had done no work since his return home, though he had done a little gardening and carpentering for his own people. As soon as he can obtain Dr. Fowler's consent he will start looking for some outdoor employment.

Social and Economic Conditions in November, 1904.

Family.—His family consists of father and mother, himself, married sister, and her two children.

Home conditions.—His conditions at home are still quite com-

fortable, the house being an airy one. The patient has a room to himself and always keeps the window open. The patient, not doing any work, is kept by his father, but tries to be as little expense to him as possible. Since his return from Mundesley he has completely changed his habits, and he now keeps early hours and is very careful about his meals. He feels he could do a light job provided the hours were not too long.

The following is an approximate statement of the income and expenditure of the family at the present time:

Income.	£	s.	d.	Expenditure.	£	s.	d.
Father's pension .	3	3	0	Rent, taxes, etc. .	0	14	0
				House expenses .	0	7	0
				Clothes, etc. . .	0	12	0
				Balance for food .	1	10	0
	£3	3	0		£3	3	0

No. in family in terms of men . . . . 4·6
Approximate weekly sum available for food per man . 5*s.* 3*d.*

The following is a sample week's dietary taken by the patient and his family:

	Breakfast.	Dinner.	Tea.	Supper.
Sunday	Quaker oats, egg, tea, toast and butter	Beef, haricot beans, potatoes, sprouts, bread, rice pudding, fruit	Bread, butter, jam, tea	Quaker oats, egg, bread, butter, and ½ pint skim milk.
Monday	Bacon, bread and butter, tea, Quaker oats	Beef, potatoes, greens, beet-root, tapioca, bread	Bread, butter, jam, tea	Quaker oats, egg, bread, butter, ½ pint skim milk.
Tuesday	Quaker oats, bloater, bread, butter, tea	Steak pudding, potatoes, greens, haricot beans, bread	Bread, butter, jam, tea	Quaker oats, rice pudding, bread and butter, ½ pint skim milk.
Wednesday	Quaker oats, egg, toast and butter, tea	Boiled bacon, haricot beans, greens, potatoes, jam tart, bread	Toast, butter, tea	Quaker oats, beef sausage, bread and butter, ½ pint skim milk.
Thursday	Quaker oats, bloater, bread and butter, tea	Potatoes, greens, cucumber, haricot beans, apple pudding, bread, butter, fruit	Bread, butter, jam and tea	Egg, bread, butter, and ½ pint skim milk.

Week's dietary (*continued*):—

	Breakfast.	Dinner.	Tea.	Supper.
Friday	Quaker oats, toast, butter, egg, tea	Fish, butter, sauce, potatoes, greens, haricot beans, bread, rice pudding	Bread, butter, jam, tea	Quaker oats, egg, bread, butter, ½ pint skim milk.
Saturday	Quaker oats, bread, butter, bacon, tea	Haricot beans, potatoes, greens, jam tart, bread, fruit	Toast, butter, and tea	Quaker oats, egg, bread, butter, ½ pint skim milk.

In addition to the above, the patient drinks 1½ pints of skim milk during the day, making his total allowance of skim milk one quart daily. Three days a week he has in addition half-pint of new milk. It is of interest to add that this patient says that he preferred the meat-free diet upon which he was treated at Mundesley to the food he has at the present time.

Case 8.—*Age.*—22.
Occupation.—Labourer.
Type of disease.—Early disease of two lobes, considerably arrested.
Duration of disease.—12 months.
Number of weeks in Cottage San.—12.

	Condition on admission.	*Condition on discharge.*
Fever	*Nil.*	*Nil.*
Digestion	Impaired.	Improved.
General health	Poor.	Very fair.
Weight	10 st. 3 lbs.	11 st. 6 lbs.
Relation of weight to highest known weight	− 15 lbs.	+ 2 lbs.

Social and Economic Conditions at date of Contraction of Tuberculosis.

Family.—His family consisted of himself, his mother, and a grown-up brother.
Work.—His work was that of chaff-cutting in one of the Depôts of the London General Omnibus Company. This work is distinctly heavy, and is done in a room which, though large and airy, is always dusty, and fifteen other men are also employed at the same work in the same place. His hours were nine hours daily except Saturday, when he worked only four hours—forty-nine hours per week. He had been employed as a chaff-cutter for eighteen months. Previous to this he had

worked at "humping" wood off barges for part of the day, and for the rest of it he picked up blocks of wood as they fell from a wood-cutting machine. This kind of work is paid for at the rate of 6*d.* per hour. Wood-humping is very heavy work.

Home conditions.—He and his mother and brother lived in three rooms in Chelsea, and were quite comfortably off. He was, however, careless about his meals, and apparently did not take quite so good a diet as he might have done.

Wages.—He earned 24*s.* per week.

The following is an approximate statement of his income and expenditure at this date. The weekly budget of his family is given, since they share their income and expenses.

Income.	£	*s.*	*d.*	Expenditure.	£	*s.*	*d.*
Wages of patient .	1	4	0	Rent . . .	0	8	0
Wages of brother .	1	4	0	Patient kept for himself . .	0	8	0
In addition mother occasionally earned a few shillings.				Brother kept for himself . .	0	8	0
				Balance for food, household expenses, etc. . . .	1	4	0
	£2	8	0		£2	8	0

No. in family in terms of men . . . 2·8

Approximate weekly sum available for food per man 6*s.*

Financial Condition of Patient and his Family during his stay in the Sanatorium.

The patient's fee of 17*s.* per week was paid by the Charity Organisation Society, and his home, mother, etc., were maintained by the brother who was still at work.

Condition on Discharge from the Cottage Sanatorium in April, 1903.

Lung disease.—Arrest fairly complete, very few adventitious sounds being audible.

General health.—Good, walking six miles per day.

Normal working capacity.—Incompletely restored, quite fit for work not entailing very much exertion. This patient was handicapped by having double mitral disease, which though well compensated, prejudiced his prognosis.

After History.

On his discharge from Mundesley patient returned to London, and tried to find some suitable employment. This he found extremely difficult to do. In reference to this the Charity Organisation Society wrote to me as follows: "The Chelsea Committee on the patient's return from Mundesley tried in

all possible ways to get him employment in the country, which they were advised was the only thing likely to be of real use to the man, but owing to his condition, with both lungs and heart wrong, and also that he was unskilled and not very adaptable, they quite failed in their object. Of course the case passed from the Committee's hands when it was found impossible to obtain employment for him in the country." Patient also failed to get regular employment in London, but got odd jobs from the parish, which is paid for at the rate of 6*d.* per hour. This work consists of road-cleaning, and though he does this whenever he has the opportunity, it only averages about two days a week, which brings in an income of 7*s.* 6*d.* weekly. In March, 1904, he wrote to say that his health was failing again, and that he was attending the Brompton Hospital again as an out-patient. He added that he thought he should have kept well if he had got proper food, but owing to the loss from his income, he and his family had been unable to live as well as formerly. The family income was, in fact, reduced by some 14*s.* per week, owing to the patient's inability to return to his former employment, and the weekly amount available for food per man was reduced to probably some 4*s.*, which meant that their diet became an inefficient one. Under these conditions it was only to be expected that his health would deteriorate.

November, 1904.—When visited in November, 1904, this patient was still out of regular work, the parish giving him employment on the average two days per week.

SOCIAL AND ECONOMIC CONDITIONS IN NOVEMBER, 1904.

Work.—He gets up every morning at 5.30, and walks down to the vestry yard to inquire for a job. If successful, he starts work either at 6 a.m., leaving off at 5.30 p.m., or 8.30 a.m., leaving off at 7.30 p.m. Out of these hours there is an allowance of half an hour for breakfast, and one hour for dinner. He always goes home to his meals. Patient says that he could easily get indoor work again, but will not take it on account of his health. Regular outdoor work of a suitable nature he finds quite impossible to obtain.

Home conditions.—At the present time he lives in a fair-sized house in Chelsea containing nine rooms. He shares this house with his mother, his brother, a married sister and her husband, and their two children. He always keeps his bedroom window open. The patient appeared to be in very fair health and quite fit for light work if he could find it.

Wages.—His average weekly earnings are 7*s.* 6*d.* per week.

This patient's loss of income meant material reduction in the income of his family, and the patient himself is now to some extent

dependent upon them instead of being of some assistance to them. Apparently, the married sister and her husband, although living at the same house, do not make any contribution to the income of the patient's family, but from a financial point of view are quite independent. We can, therefore, omit their income and expenditure from the weekly budget of patient and his family.

The following is an approximate statement of income and expenditure of patient and his family at the present time:

Income.	£	s.	d.	Expenditure.	£	s.	d.
Patient's wages	0	7	6	Rent	0	7	6
Brother's ,,	1	4	0	Household expenses	0	4	6
				Food	0	11	6
				Brother keeps for himself	0	8	0
	£1	11	6		£1	11	6

No. in family in terms of men . . . . 2·8.
Approximate weekly sum available for food per man . 4*s.*

When the patient is out of work altogether, as he is for a week or two at a time now and again, his family is distinctly poor and certainly not in a position to keep the patient's standard of living up to what it should be. We notice from the budget that no allowance is made for expenditure upon clothes, the patient trusting entirely to charity for his clothing. The following is approximately the weekly dietary taken by patient when in work.

	Breakfast.	Dinner.	Tea.	Supper.
Sunday	Bacon and eggs, bread and butter, tea	Roast mutton, greens, and potatoes	Bread and butter, and tea	Cold mutton, bread and butter, 1½ pint of milk.
Monday	1 egg, bread and butter, and tea	Cold mutton, potatoes, and bread	Bread and butter, marmalade, tea	1½ pint of milk.
Tuesday	Haddock, bread and butter, tea	Chop and potatoes, bread	Bread and butter, tea	1½ pint of milk.
Wednesday	1 egg, bread and butter, tea	Chop, potatoes, greens, bread	Bread, butter, marmalade	*Nil.*
Thursday	Bread and dripping, tea	Chop, potatoes, bread	Tea, bread, butter	*Nil.*
Friday	Haddock, bread, butter, tea	Steak, potatoes, and bread	Bread, butter, herrings, tea	*Nil.*
Saturday	Haddock, bread, butter, tea	Chop, potatoes, bread	Bread, butter, herring, tea	1½ pints of milk.

CASE 9.[1]—*Age.*—39.
Occupation.—Gardener.
Type of disease.—Fairly extensive disease of three lobes, partially arrested.
Duration of disease.—6 months.
No. of weeks under treatment.—26 weeks.

	Condition on admission.	*Condition on discharge.*
Fever .	Slight.	*Nil.*
Digestion .	Good.	Good.
General health	Fair.	Good, walking 10 miles daily.
Weight .	11 st. 7 lbs.	13 st. 7 lbs.
Relation of weight to highest known weight	—2 st.	= to

SOCIAL AND ECONOMIC CONDITIONS AT DATE OF CONTRACTION OF TUBERCULOSIS.

Family.—His family consisted of himself, his wife, and five children, aged from 5 to 13.
Work.—His work consisted of the ordinary routine work of a gardener to a private family. The grounds were only small and he also assisted in the stable.
Home conditions.—He lived rent free in a five-roomed cottage. The patient himself is a steady, intelligent man. As a lad he had worked on a farm until he was 20. He was then for some years in the police force. After leaving the police he for a time had a small farm of 20 acres, but this not proving very profitable, he took to gardening. Patient's wife also is a sensible, capable person.
Wages.—He earned 18*s.* per week. In addition, he had his cottage rent free, and made a certain amount of money in tips.

The following is an approximate statement of his income and expenditure at this time:

INCOME.	£	s.	d.	EXPENDITURE.	£	s.	d.
Wages . . .	0	18	0	Household expenses	0	6	0
Extra in tips (average weekly) .	0	4	0	Club . . .	0	0	6
				Balance for food .	0	15	6
	£1	2	0		£1	2	0

No. in family, in terms of men . . . . 4·4
Approximate weekly sum available for food per man . 3*s.* 6*d.*

This sum was obviously inadequate to provide this patient

[1] This patient was treated in rooms in Mundesley village.

and his family with an efficient diet, and the man's breakdown must, at least in part, be attributed to this cause. The following is a rough idea of his daily dietary:

Breakfast.—Porridge, bacon or egg, and bread and butter.

Dinner.—Generally meat, vegetables, bread, and occasionally a milk pudding.

Tea.—Bread and butter, and tea.

Supper.—Usually none.

Financial Conditon of Patient and his Family during his Stay in the Sanatorium.

The patient himself was treated gratuitously. His wife and family, during patient's illness, were maintained by a lady who was interested in the family. The family, in addition, had 10*s.* per week which was paid to the patient throughout the six months by the Oddfellows' Sick and Benefit Society.

Condition on Discharge in March, 1904.

Lung disease.—Very largely arrested, adventitious sounds being few and only audible on cough.

General health.—Very largely restored; walking ten miles daily. He suffered, however, a good deal from dyspnœa on exertion.

Normal working capacity.—Incompletely restored, fit only for moderately light work.

After History.

March, 1904.—This patient, on his discharge, at once obtained a situation as gardener on a small property in the country, where he is at the present time.

Social and Economic Conditions in December, 1904.

Work.—He is now gardener on a fair-sized property, where an assistant gardener is kept and additional labour usually taken on when there is much digging to be done. The patient himself is head gardener, and is thus able to avoid much of the heaviest gardening work which a man has to do when single-handed, *e. g.* digging, wheeling heavy barrows, etc. His hours are from 8.30 a.m. to 5 p.m. The patient says he is quite able to do the work he has to do, but could not do a really hard day's work. He has enjoyed good health since his return to work, his only trouble being dyspnœa on exertion. During his first four months' work he lost some 14 lbs. in weight, but since then his weight has remained stationary. Examination of his lungs at this

date showed further improvement since his discharge, no moist sounds being audible.

Home conditions.—He lives rent free in a well-built eight-roomed cottage in a very healthy situation.

Wages.—He earns 20*s.* per week, lives rent free, and in addition to this he still receives 5*s.* per week from the lady who helped the family during his illness.

The following is an approximate statement of his income and expenditure at the present time:

Income.	£	s.	d.	Expenditure.	£	s.	d.
Wages . . .	1	0	0	Flour and bread .	0	5	0
Allowance . .	0	5	0	Groceries . .	0	7	6
				Meat . . .	0	4	0
				Milk, 4 pints daily .	0	3	6
				Eggs . . .	0	1	3
				Coal . . .	0	1	11
				Oil and candles .	0	0	6
				Club subscription .	0	0	6
				Clothes, etc. . .	0	0	10
	£1	5	0		£1	5	0

No. of family in terms of men . . . . 4·4
Approximate weekly sum available for food per man . 4*s.* 10*d.*

The weekly expenditure upon food has already been given in the weekly income and expenditure given above. In addition, the patient has his dinner five days a week at the house where he works, which is a material saving in his wife's housekeeping. This has not been allowed for in estimating the amount available for food for the family, so the figures are rather better in this respect than they appear. The patient's meals, which are now appreciably better than they were formerly, are approximately as follows:—

Breakfast (7.30).—Bread and milk, with bacon or egg.

Lunch (11.30).—Cake, biscuits, etc.

Dinner (1.30).—Meat, two vegetables, bread, milk or suet pudding, and one pint of milk.

Tea (6.0).—Bread and butter, meat, milk pudding, and milk.

In all he has 2½ pints of milk per day.

Case 10.—*Age.*—34.

Occupation.—Workhouse hospital attendant.

Type of disease.—Recent active disease of upper and lower lobes of right lung.

Duration of disease.—4 months.

No. of weeks under treatment.—14.

	Condition on admission.	*Condition on discharge.*
Fever . .	Slight.	*Nil.*
Digestion. .	Poor.	Excellent.
General health.	Poor.	Excellent.
Weight .	9 st. 7 lbs.	10 st. 10 lbs.
Relation to highest known weight before illness	— 21 lbs.	— 4 lbs.

SOCIAL AND ECONOMIC CONDITIONS AT DATE OF CONTRACTION OF TUBERCULOSIS.

Family.—His family consisted of himself, his wife, and five children, whose ages ranged from one to eleven.

Work.—As attendant in the wards at the workhouse infirmary he worked twelve hours a day, and was on duty for a fortnight at a time during day and during the night alternately. During the fortnight on day duty he spent one week in the sick-room with a lot of helpless and dirty cases—some of them usually tuberculous, and during the other week he was in charge of the day-room. Of the fortnight's night duty he spent one week in the sick-room, and during the other week he was in charge of a room of forty-eight epileptics.

Home conditions.—Patient lived in a five-roomed cottage about half a mile from the workhouse. It is situated in a recently built working class district on the outskirts of the city. The house itself is quite comfortable. The patient is an old soldier and a very sensible man, who thoroughly appreciated his position.

Wages.—He earned 30*s.* 8*d.* per week.

The following is an approximate statement of his income and expenditure at this date:

INCOME.	£	*s.*	*d.*	EXPENDITURE.	£	*s.*	*d.*
Wages . . .	1	10	8	Rent . . .	0	4	9
				Clothes, etc. . .	0	5	0
				Fuel, light, etc. .	0	2	0
				Superannuation sub. and benefit club .	0	2	3
				Food . . .	0	15	0
				Sundries . .	0	1	8
	£1	10	8		£1	10	8

No. in family in terms of men . . . 3·4.
Weekly sum available for food per man . . 4*s.* 4*d.*

Financial Condition of Patient and his Family during Illness.

This patient left off work on July 29th, 1899, and returned to work in December, 1899.

During the first ten weeks he had the following income:

	£	s.	d.
From the workhouse authorities .	1	2	0
From his benefit club . . .	0	18	0
Total per week	2	0	0

During the next three weeks he had the following income:

	£	s.	d.
From the workhouse authorities .	1	2	0
From the benefit club . . .	0	12	0
Total per week	£1	14	0

During the remaining seven weeks he had from the benefit club 12*s*.

The patient thus had on the average an income of 29*s*. per week throughout his illness for the support of his wife and family. This proved quite sufficient for their proper maintenance, and the fact that the patient himself was independent of this income for fourteen weeks made the family income still better than at first sight appears.

Condition on return to Work in December, 1899.

Lung disease.—Arrest almost complete, a few adventitious sounds only being heard on cough.

General health.—Excellent.

Normal working capacity.—Completely restored.

After History.

December, 1899.—He returned to work at the Fir Vale Workhouse, an institution situated on the outskirts of Sheffield, the authorities having found him employment looking after cattle on the workhouse farm. His working hours were from 6 a.m. to 6 p.m., and he continued to live at his own house, which, though only half a mile from the workhouse, is three miles from the farm. This work, though not involving any manual labour, was tiring, since he was standing about practically the whole day in addition to his three miles' walk every morning and evening. This routine did not prove quite satisfactory, as he felt very tired after his day's work, and after a time his cough returned.

January, 1900.—The workhouse management, in consequence of this tendency to relapse, took him off the farm and found him

work in the workhouse itself. His new duties consisted in looking after the lunatic inmates. This work was much lighter, and took him a good deal out of doors; *e.g.* five hours a day he was in charge of the inmates working in the garden, and the rest of the time was spent in the day-room and the dining-room. His hours were from 7 a.m. to 7 p.m., and instead of being three miles away he is now again only half a mile distant from his home. Under these new conditions of work he rapidly improved in health.

September, 1900.—At this date he was feeling very well indeed, and looking in excellent health. He was still at the same work.

February, 1901.—He was still in excellent health, and had not missed a day's work since his return.

October, 1901.—He wrote saying he was as well or even better than when he left the infirmary two years ago.

January, 1902.—He was visited in his own home by Mr. Chapman. His home was not very airy, and beyond sleeping with his window open, he did not go in for much open air at home. His conditions of life were, however, at the workhouse quite satisfactory. The disease in the lungs was completely arrested, his temperature was normal (97° a.m. to 98·4° p.m.) and his morning pulse 70. His general health was excellent, and his weight 11 st., at which he had for long been stationary. His appetite and digestion were normal. Examination of his blood gave the following result: Hæmoglobin 98 %, red corpuscles 4,800,000, and was another proof of the excellence of his general health.

August, 1902.—Another good report was received from him at this date: Temperature and pulse normal, weight 10 st. 12 lbs., general health very good indeed, and he is still doing his work with ease. Dr. Bellamy, the house surgeon to the workhouse, had recently examined his sputum, and found no bacilli present; the sputum, one might add, consisted of traces only now and again.

February, 1903.—He was again visited by Mr. Chapman in Sheffield. He had been at work and in good health since the last report. He had rather a severe cold on him at the time, and, probably on account of this, there were some adventitious sounds audible in the right lung. With a possibility, however, of these sounds indicating fresh tuberculous activity, he was advised to get leave of absence and come for a time to the Cottage Sanatorium at Mundesley, which had just been started. The workhouse committee agreed to the proposition, and in

March, 1903, he came to Mundesley. His condition on arrival was distinctly better than it had been ten days previously, when he was seen in Sheffield, the cold, and with it most of the

moist sounds, having cleared up. His temperature was normal, and also his appetite and digestion, but he was a trifle (3½ lbs.) below his highest known weight of 11 st. After a stay of six weeks his general health was completely restored in every respect. He weighed 11 st. 1 lb., and he was walking eight to ten miles daily without the least fatigue. The lung disease appeared to be completely arrested, some impairment of resonance being the only physical sign which was to be noticed in his chest.

January, 1904.—Patient reported himself as being still at work and in good health.

January, 1905.—In answer to a letter, the patient wrote saying he was still in good health, and at work, and was good enough to kindly give me the following account of his social and economic conditions at the present time. This description, as a matter of fact, applies equally well to the past five years, with the exception that his family's income is now increased by 5*s*. per week—the wages of his eldest boy, who has recently started work.

Social and Economic Conditions in January, 1905.

Family.—This family still consists of himself and wife and their five children, aged now from 6 to 15.

Work and home conditions.—Both his work and his home conditions have already been described, and, as already stated, his conditions of life in these two important respects are quite satisfactory.

Wages—His wages are 32*s*. 8*d*. per week. The following is an approximate statement of the weekly income and expenditure at the present time:

Income.	£	*s.*	*d.*	Expenditure.	£	*s.*	*d.*
Patient's wages	1	12	8	Rent	0	4	9
Son's wages	0	5	0	Lights and coals	0	2	0
				Clothes	0	5	0
				Household sundries	0	3	8
				Club and superannuation	0	2	3
				Food	1	0	0
	£1	17	8		£1	17	8

No. in family, in terms of men . . . . 4·7
Weekly sum available for food per man . . 4*s*. 4*d*.

As a matter of fact, this patient gets most of his meals at the workhouse infirmary; the cost of these forms part of his salary and in the budget has been included in his salary. The patient

actually receives 24*s*. in cash, the extra 8*s*. 8*d*. being received in the shape of food and clothes.

COMMENTARY.

Review of the Cases from a Clinical Standpoint.

When attempting to form an estimate of the prognosis of any consumptive, the clinical condition of the patient when he first comes under treatment is probably the factor to which one attaches most importance: for though experience has shown one that patients with the best possible clinical prognosis may prove most disappointing failures if compelled by various circumstances to return after treatment to unfavourable conditions of life and work, and that, conversely, patients with somewhat advanced disease sometimes do unexpectedly well, if they are fortunate enough to find healthy employment which brings them in a living wage, the fact remains that the smaller and less active the area of disease the better is the outlook, both for the immediate present and for the future. I propose, then, to first briefly review the *ten patients* from the point of view of their several clinical conditions when they first came under treatment. Such a review is shown in Table No. 1, which also includes data as to the length of time each patient was under treatment, and his condition on discharge and at the present time. I have purposely not given a detailed description of the clinical condition of each case before and after treatment, since for my present purpose it would be of no great value; moreover, the reaction of favourable cases of consumption to sanatorium treatment—viz. gain of weight and strength and improvement in physical signs—are now well known. It will be sufficient for me, after having noted the type of consumption to which each patient belongs, to say that the progress of them all under treatment was, typically, satisfactory. The clinical condition of the patients when discharged is a more important point to state clearly, but a difficult one to do briefly.

The approximate condition of the lung disease, the state of the general health, and the degree to which normal working capacity has been restored, are, I think, the chief points to be noted, and for the sake of brevity I have, in this paper, made use of various terms to express them. I have to thank my friend Dr. Fowler for his assistance in framing the following definitions indicative of the conditions of patients on discharge from treatment:—

(1) *Complete arrest of the disease.*—General health completely restored in every respect, and lung disease completely arrested (apparent cure), there being no physical signs present, or only such as are compatible with a completely healed lesion.

(2) *Incomplete arrest of the disease.*—(A) General health completely restored but physical signs of lung disease, though much improved, not entirely cleared up—*e.g.* perhaps limited to a few moist sounds on cough. (B) General health only imperfectly restored and physical signs of the disease—*e. g.* moist sounds, etc.—still well marked.

It will be seen from the Table No. 1 that six of the ten patients were [really] early cases, the disease in five of them being apparently limited to infiltration of the apex of one lobe, and in the case of the sixth of the apices of the upper and lower lobes of one lung, the average duration of the disease in these six being some three and a half months only.

In all these six there was absence, moreover, of any serious constitutional impairment, although two of them (Nos. 2 and 7) had slight fever; in short, they belonged to the type of consumption from which by far the largest number of successes are obtained. Cases Nos. 6 and 8, with two and three lobes affected, and with disease of one and two years' duration respectively, though chronic, when judged by the length of time they had been consumptive, were, in fact, still only early cases, pathologically speaking. Both of these patients had enjoyed long periods of more or less complete arrest of their disease,

TABLE No. 1.—*Clinical summary.*

No. of case.	Type of disease.	Duration of disease.	Period under treatment.	On discharge.		Condition at the present time.
				Condition of lungs.	Normal working capacity.	
1	Infiltration of one lobe	3 months	12 weeks	Disease completely arrested	Completely restored	Health excellent; at full work after 1 year and 8 months.
2*	Infiltration of one lobe	3 months	11 weeks	Disease completely arrested	Completely restored	Health excellent; at full work after 11 months.
3	Infiltration of two lobes	4 months	14 weeks	Disease completely arrested	Completely restored	Health excellent; at full work after 1 year and 8 months.
4	Infiltration of one lobe	3 months	13 weeks	Disease completely arrested	Completely restored	Health excellent; at full work after 1 year and 9 months.
5	Infiltration of one lobe	4 months	16 weeks	Disease completely arrested	Completely restored	Health excellent; at full work after 5 years.
6	Disease of three lobes considerably arrested	2 years	15 weeks	Disease incompletely arrested A	Completely restored	Health excellent; at full work after 18 months.
7*†	Infiltration of one lobe	3 months	10 weeks	Disease completely arrested	Normal working capacity *not* completely restored, but quite fit for light work	In good health, but doing no regular work: living at home in the country.
8	Infiltration of two lobes considerably arrested	12 months	12 weeks	Disease incompletely arrested A		In fair health; doing light work (casual) after 1 year and 9 months.
9*	Active disease of three lobes	6 months	46 weeks	Disease incompletely arrested A		Health excellent; at full work after 9 months.
10*	Active disease of two lobes	3 months	13 weeks	Disease incompletely arrested A		Health excellent; at full work after 5 years.

* These patients had some degree of fever and other symptoms of active disease.
† This patient's unfitness for work is due to a history of recurrent hæmoptysis.

so that in neither case had it made much progress nor general health been much damaged. The prognosis in the case of these two was not, then, much worse than in the case of the preceding six, and, indeed, they reacted to treatment just as well. The last two patients, viz. 9 and 10, both had active disease of a more extensive character, with some fever and other symptoms of active tuberculosis. Their prognoses were, in consequence, distinctly less favourable than in the other eight, though by no means unfavourable, certainly, so far as the immediate present was concerned. Taking the ten together, one may safely say that, clinically speaking, they were on admission a distinctly favourable lot of patients from whom a fair proportion of permanent successes could be looked for if the sanatorium treatment for the working classes is indeed of lasting value. Their conditions on discharge were equally satisfactory, no less than eight being restored to their normal capacity for work, and the remaining two sufficiently restored for work of a fairly light nature.

Review of the Social and Economic Conditions of the Ten Patients.

As I have already remarked, when reviewing the prospects of these ten patients, from a clinical standpoint, the conditions of life and work to which any consumptive returns after treatment is in the long run probably almost as important a factor as any in determining his subsequent life history. In the case of the consumptive working man this is especially true, and to fully appreciate this fact and the difficulties, often insuperable, which confront many of them when they become tuberculous, some knowledge of the conditions which govern the life of the working class is necessary.

The study of "the *social and financial conditions*" of these ten patients serves to illustrate the importance of this subject, and I propose next to briefly describe them, the most important points to be noted being:—

(1) Work and wages.

(2) Financial obligations, viz. the number of people dependent upon them.

(3) Their ability to obtain a diet of nutritive value adequate for the maintenance of tuberculosis in arrest.

(4) Their financial resources when out of work through illness, trade depression, or any other cause.

As a matter of experience one finds that these "factors" are all intimately associated and depend one upon the other; *e. g.* any specific income will prove to be adequate or inadequate for any individual in proportion to the amount he has to do with it. A single man earning 18*s.* per week will often be found to be taking a proper diet and to be living under suitable conditions of life and work—he can afford to do so; whereas a man with a wife and family with similar work and income is found to be living in absolute poverty. The division, then, of the subject of social conditions into various heads is somewhat arbitrary although convenient, and in reality they can all be included under one head, viz. the capacity of consumptive patients to earn a living wage; by which one means an adequate sum for the maintenance of the physical efficiency of himself, and in the case of married men, of his wife and family as well. If a consumptive returns to the most suitable employment possible, from a health point of view, but one at which he cannot earn an income adequate for his requirements, he fails, his home conditions and his diet both becoming unsatisfactory. On the other hand, a return to sufficiently remunerative but not very healthy work frequently also ends in failure, a period of good health, adequate income, and with it satisfactory home conditions and an efficient diet being followed by one of reduced income, due to failure to maintain health; and with this reduction in income are associated loss of home comforts, inadequacy of diet, etc.—in short, conditions of poverty. In both these examples failure is due to inability to earn a living wage. A living wage, then, is only a relative term. With these preliminary remarks I will

now discuss the social conditions of the ten men recorded in this paper.

Review of the Social and Economic Conditions of the Ten Patients.

(1) *Occupation and Wages.*

In Table No. 2 are shown the occupations and weekly wages of these ten patients at the date of their contracting tuberculosis, and the employment and wages they returned to after discharge from treatment. I have in the table grouped the patients under two headings, viz. those who have only themselves to keep, and those who are the bread-winners of families, since the weekly income of any man loses much of its significance if his financial obligations are not also shown.

Of the occupations of these ten men when they became tuberculous, those of the unmarried men were all unsuitable for a case of arrested consumption, viz. brewer's drayman in London, restaurant cook (in a shop in a side street off the Strand), coal-miner and cabinet-maker. These four all gave up their employments when discharged, and three of them were fortunate enough to obtain work of a much more suitable character, viz. as coachman in the country, omnibus conductor, and outdoor worker for a telephone company. The two former lost respectively 4*s*. a week and 6*s*. a week by this change; but having no one dependent upon them, they could afford to make this financial sacrifice in the interest of their health; their incomes, moreover, were adequate for their requirements. The coal miner, on the other hand, gained financially by the change to the extent of 4*s*. per week. The cook had considerable difficulty in getting a situation as 'bus conductor, but the brewer's carman who, as an ex-trooper in a Hussar regiment, and subsequently a driver of one of Shoolbred's vans, was thoroughly used to horses, and in addition a smart-looking man with

TABLE NO. 2.—*Showing the occupations and wages of the patients at the date of contraction of tuberculosis and subsequent to their discharge.*

	No. of case.	At the date of contraction of tuberculosis.		After discharge.		
		Employment.	Wages.	Employment.	Wages.	Weekly loss, or gain, in income.
Unmarried and with no one dependent on them.	2	Brewer's carman (London)	27*s.* per week	Coachman (country)	18*s.* per week and lodging = 23*s.* weekly	− 4*s.*
	4	Cook in restaurant (London)	20*s.* with board and lodging = 36*s.* weekly	Omnibus conductor	30*s.* per week	− 6*s.*
	5	Coal-miner	16*s.* to 18*s.* per week	Telephone company's labourer	21*s.* per week	+ 4*s.*
	7	Cabinet-maker	12*s.* 6*d.* per week	Light work at home	*Nil*	− 12*s.* 6*d.*
Married, with families or others dependent upon them.	3 (*a*)	Wood-sawyer	32*s.* per week	Wood-sawyer	30*s.* per week	− 2*s.*
	1 (*b*)	Salvation army officer	28*s.* to 30*s.* per week	Insurance agent for Salvation Army	30*s.* per week	Same.
	6 (*c*)	Coachman (London)	25*s.* and house = 30*s.* weekly	Coachman (London)	29*s.* and house = 34*s.* weekly	+ 4*s.*
	9 (*d*)	Gardener	22*s.* and cottage = 27*s.* per week	Gardener	25*s.* and cottage and dinner = 32*s.* weekly	+ 5*s.*
	10 (*e*)	Workhouse attendant (indoor)	30*s.* 8*d.* per week	Workhouse attendant (outdoor)	30*s.* 8*d.* per week	Same.
	8 (*f*)	Chaff-cutter	24*s.* per week	Casual parish laborer (outdoor)	7*s.* 6*d.* per week	− 16*s.* 6*d.*

(*a*) Has wife and 3 children dependent upon him. (*b*) Has one child dependent upon him (patient is a widower), and servant to take charge of child. (*c*) Has wife and 3 children dependent upon him. (*d*) Has wife and 5 children dependent upon him. (*e*) Has wife and 5 children dependent upon him. (*f*) This patient and his brother are jointly responsible for the support of their mother and their home in addition to themselves.

a good character, found employment as coachman almost at once. The coal-miner (a young man of 22) had no difficulty in getting work with the telephone company, the labour being unskilled. The cabinet-maker lives with his people in the country, who, fortunately, can just afford to keep him. He is in good health at the present time and on the look-out for some suitable work. The social and economic conditions, then, of these four subsequent to their discharge have distinctly favoured their chances of remaining in good health. Turning now to the consideration of the six men who were the bread-winners of families, we see that four of them had unsuitable occupations, viz. the wood-sawyer, the Salvation Army officer, the workhouse attendant (indoor, much night work), and the chaff-cutter. The remaining two fortunately belonged to very good occupations, viz. gardener and coachman. These two, though they both lost their situations when they left home to undergo treatment, very soon obtained regular work again after their discharge, both being steady men with good characters and well qualified in their respective callings. In both instances they gained somewhat by their change of situation—an important matter as they both had a wife and a "fair-sized" family, and could not have possibly afforded to return to less remunerative employment however suitable from a health point of view; indeed, their incomes of 32*s*. and 34*s*. per week—66*s*. in all—are none too much for the maintenance of four adults and eight children, the size of their joint families. The fact that these two men had the worst prognosis on clinical grounds of the ten patients, and are at the present time in as good a position as any of them, illustrates the great value a suitable occupation is to the consumptive working man. Two of the remaining four were also very fortunate in getting suitable work after their discharge, viz. the Salvation Army officer and the workhouse attendant; for in both instances their former employers retained their services, but changed the nature of their work to something more suitable to their present condition. As an insurance agent in the

country and an attendant upon paupers, for the most part in the workhouse gardens, these men returned to about as satisfactory conditions of work as could be wished for without any loss of weekly wage. In addition they were able to go straight back to work after their discharge, a matter of importance where there are wives and families to be supported. The wood-sawyer was not so lucky, and his experience is, unfortunately, more usual. With the help of club money and a grant from the C.O.S. for the support of himself and his family, he spent the first few months after his discharge looking for a healthy kind of employment which would bring him in an income adequate for his family requirements (viz. himself, his wife and five children) —at least 30*s*. per week. This he failed to do in spite of the active co-operation of the C.O.S. on his behalf, and he was ultimately compelled to return to his original occupation in the saw-mills. That he has remained at this work for the past fifteen months and maintained his health is evidence of what can be done by consumptives even under unfavourable conditions, when their disease has been thoroughly arrested. The chaff-cutter, as in the last case, was an unskilled labourer, and he also found it impossible to procure suitable employment. Since his discharge he has as yet done nothing more than casual outdoor work provided by the parish of Chelsea, such as road-sweeping, etc., and his income has in consequence dropped from 24*s*. per week to an average of 7*s*. 6*d*. per week. This loss of 16*s*. 6*d*. per week has been a very serious thing, both for himself and his mother and brother, with whom he lives, the gross weekly income of the family being now inadequate for their requirements.

This case is, however, the only one of the ten in which a change of employment has resulted in sufficient financial loss to reduce any individual or family to a condition of poverty. It may be noted that the total income of these ten men after their discharge fell short of their total income before becoming tuberculous by 28*s*. per week, or an average weekly loss of nearly 3*s*. per head. Taking

them together there is no doubt that these ten men have been unusually fortunate in the way of obtaining work of a suitable nature after their discharge, financially and from a health aspect, and when estimating the part played by various factors in their so far satisfactory "after histories," that of occupation must be given a prominent position.

(2) *Dietetic Efficiency.*

The provision of a diet of high nutritive value is generally accepted as being one of the essentials for the successful treatment of consumption, but it is not so widely appreciated that for a consumptive to maintain his disease in a condition of arrest he must continue to live at a high nutritive level for many months after his discharge from a sanatorium. The provision of adequate diets for the working-class consumptives—frequently a difficulty—is a matter of the highest importance, and one directly depending on the social and economic conditions to which they return after treatment. Ability to earn a living wage means, as a rule, the taking of a proper diet; and equally so any serious loss of normal working or wage-earning capacity results in the diet becoming inefficient—a sure precursor of relapse. Anyone familiar with the way in which the working classes live will have noticed that food is considered by them to be the least essential item of what we may term unavoidable expenditure, and, consequently, the first direction in which economy can be effected. Certain forms of expenditure cannot be got rid of or even materially reduced—*e. g.* rent, light, firing, etc.—and these for the most part remain constant, even with a falling income; but the expenditure upon food in any family is found (after allowing for the number in the family) to be directly proportionate to its total weekly income, the nutritive efficiency or inefficiency of the diet varying with the weekly sum available for food per head. From the point of view of the treatment of consumption, the composition

of the diets taken by the working classes with various incomes forms, perhaps, the most important aspect of social economy to inquire into, but inasmuch as the weekly sum available for food in any case depends upon the total income of the family, the number to be maintained on it, the amount paid for rent, etc., and for other unavoidable expenditure, any inquiry upon this particular point must needs consider these various factors as well, and has to be, in consequence, of a rather comprehensive character.

Method of determining the Efficiency of Diets taken by the Working Classes.

The best method of determining the efficiency of diets taken by working class families is to get a detailed return from the wife of all foods bought during a definite period with a note as to their cost. From this data, and with a knowledge of the number in the family, the average diet taken per head can be ascertained, and its nutritive value calculated in terms of P., F., C.H., and Cals. This, in practice, is found to be a difficult matter to manage, unless the work can be frequently supervised. Another method which gives a good approximate idea of the efficiency of the diet, and is one more easily carried out, is that of deducting from the total weekly income of the family the amounts spent weekly upon necessary expenditure—*e. g.* rent, benefit clubs, light, coals, clothes, etc.—thus leaving a maximum balance available for the purchase of food. This information, supplemented by a detailed description of an average daily menu, allows one to form a very fair estimate of its nutritive efficiency. Weekly budgets of income and expenditure compiled in this way give one, moreover, valuable information as to the minimum necessary income that is required for the efficient maintenance of families of various sizes, thus allowing one to form an opinion as to the relative economic values of different occupations for married working men. It is upon these

lines that the inquiry into the social conditions of the patients who form the subject of this paper have been investigated and the weekly sum available for food in each case been determined.

Table 3 shows the weekly sum which was available for food per head in each case before becoming tuberculous and after discharge, and also the amount which was available for the same purpose for their families during the enforced absence of the bread-winner. Before criticising these various sums it is necessary to establish some standard of dietetic efficiency for comparison, viz. a sum representing the minimum expenditure which in the case of the working classes is found to represent the taking of a physiologically sound diet. To give all the data which have helped one to form such a standard would be too long a matter for the present paper; but I may say here that the cost of the very cheap diets, physiologically efficient, which can be easily constructed by any one with a knowledge of the nutritive value and economics of different foods must not be taken as a standard, since the working classes have not this special knowledge, and very often in consequence habitually take inadequate diets when the weekly sum available for food is quite large enough for the purchase of a dietary efficient in every respect if the money were laid out to better advantage. The adequacy or inadequacy of a given sum for the purchase of food is, then, a relative matter, depending upon the capabilities of the buyer. My own experience leads me to think that an expenditure per head of about 9*d.* per day or 5*s.* per week on food represents an efficient diet, and that any sum appreciably less than this is usually associated with some degree of dietetic inefficiency, especially so for a consumptive. One other point must be noted when dealing with this subject, viz. the fallacy arising from people of different sex and age requiring varying amounts of food and, in consequence, a varying weekly sum for the purpose. This fallacy is partly got rid of by reducing all families to equivalents in terms of men, and for this

purpose I have made use of the following table used by Rowntree in his work upon the efficiency of diets taken by the working classes in York ('Poverty: A Study of Town Life.')

The dietary requirements of women and children may be stated as follows :—

Woman,	equivalent	to	0·8	of a man at	moderate work.
Boy,	14 to 16 „	to	0·8	„	„
Girl,	14 to 16 „	to	0·7	„	„
Child,	10 to 13 „	to	0·6	„	„
„	6 to 9 „	to	0·5	„	„
„	2 to 5 „	to	0·4	„	„
„	under 2 „	to	0·3	„	„

On reference to Table 3 it will be seen that three of the patients (Nos. 5, 9, and 10) before admission had less than 5*s*. per week per head to spend upon food, the exact sums being 3*s*., 3*s*. 6*d*., and 4*s*. 4*d*. These three men, then, had diets which were inadequate for their physiological requirements, and this no doubt was a contributory factor in the incidence of their disease. The large families of the two latter, in each case a wife and five children, accounted for their being in somewhat straightened circumstances, for their incomes, viz. 27*s*. and 30*s*. 8*d*. per week, were quite up to the average of the working man's wages. The third patient was a single man who kept himself, his mother, his brother and a sister upon 17*s*. per week, a sum obviously incompatible with physical efficiency. To arrest tuberculous disease in these three patients, and to send them back to their old conditions of dietetic inefficiency, could only end in disaster; but, fortunately for them, their economic conditions after discharge improved in each case, and to this fact they owe to a very large extent their immunity from relapse and the good health which they enjoy at the present time. The other seven patients had all an adequate weekly sum for the purchase of food at the time of their contracting tuberculosis—on an average 6*s*. to 7*s*. per week. Of more immediate interest, perhaps, is an analysis of the budgets of these

TABLE No. 3.—*Showing the size of family, the total weekly income, and the weekly sum available for food per head of each case before treatment and after discharge, also of the families of the married men during their illness.*

No. of case.	At date of contracting tuberculosis.					After discharge.			Of family during patient's illness.		
	Family.										
	Adults.	Children.	Family = to in terms of men.	Total income from all sources (weekly).	Weekly sum available for food per man.	Family = to in terms of men.	Total income from all sources (weekly).	Weekly sum available for food per man.	Family = to in terms of men.	Total income from all sources (weekly).	Weekly sum available for food per man.
1	2	1	2·1	30*s.*	10*s.* 6*d.*	2·1	35*s.*	10*s.* 6*d.*	1·1	Child and	nurse supported by the Salvation Army.
2	2	—	1·8	33*s.*	7*s.* 6*d.*	1·0	23*s.*	11*s.*	—	—	—
3	2	2	3·3	32*s.*	5*s.* to 6*s.*	3·6	30*s.*	5*s.* 4*d.*	2·3	16*s.*	3*s.* 2*d.*
4	1	—	1·0	36*s.*	10*s.*	1·0	30*s.*	10*s.*	—	—	—
5	2	2	2·8	17*s.*	3*s.*	1·0	21*s.*	7*s.*	—	—	—
6	2	3	3·0	30*s.*	6*s.*	3·0	34*s.*	7*s.*	Wife and	children	supported by the Parish.
7	4	2	4·6	75*s.* 6*d.*	6*s.*	4·6	63*s.*	5*s.* 3*d.*	3·6	63*s.*	5*s.* 3*d.*
8	3	—	2·8	48*s.*	6*s.*	2·8	31*s.* 6*d.*	4*s.*	1·8	24*s.*	5*s.* to 6*s.*
9	2	5	4·4	27*s.*	3*s.* 6*d.*	4·4	32*s.*	4*s.* 10*d.*	3·4	20*s.*	2*s.* 4*d.*
10	2	5	3·4	30*s.* 8*d.*	4*s.* 4*d.*	3·4	30*s.* 8*d.*	4*s.* 4*d.*	2·4	29*s.*	6*s.* to 7*s.*

ten patients after their return to work, several of them to new employment. On reference to Table No. 3 it will be seen that the minimum sum which any of the patients had for the purchase of food was 4*s*. per week, though two of the others, viz. those with the large families, had, respectively, only 4*s*. 4*d*. and 4*s*. 10*d*. These last two sums were probably just sufficient; for the man with only 4*s*. 4*d*. was really better off than the figures indicate, since he had most of his meals at the workhouse, and these were quite satisfactory. (The value of his workhouse diet formed a part of his weekly salary, and in the table has been included in it.) The patient with only 4*s*. per week for food is the only one of the ten whose illness has resulted in himself and his family drifting into a condition approaching poverty; his inability to earn more than 7*s*. 6*d*. per week after discharge, compared with his former income of 24*s*. per week, reducing the total weekly sum for the maintenance of himself, his mother and brother from 48*s*. per week to 31*s*. 6*d*., and the amount available per head for food from 6*s*. to 4*s*. This means that the patient since his discharge has often had an unsatisfactory diet, and this does not improve his chance of regaining his former working capacity. With this single exception, then, all the ten patients have large enough incomes to enable them, after paying for the necessary current expenses, to provide themselves with physiological diets, the actual weekly sums available for the purpose varying from 5*s*. to 11*s*. per week. This, no doubt, has been another contributory factor in their success. It is of interest to note that two of the largest weekly sums available for food, viz. 11*s*. and 7*s*., were obtained from the two smallest weekly incomes, viz. 23*s*. and 21*s*. This is due to the men with these incomes being unmarried and with no one dependent upon them, and is a good illustration of the advantages in this respect enjoyed by consumptive single men compared with men with wives and families. The weekly sum available for the provision of food for the wives and families of the

married men while under treatment is also of interest. In two instances (Cases 3 and 9) the loss of the income of the bread-winner resulted in families living in conditions approaching poverty, in spite of charitable assistance, the sum available for food per head being 3*s.* 2*d.* and 2*s.* 4*d.* per week. The significance of two growing families living for several months on diets physiologically inefficient is obvious. This will be referred to again later when discussing the financial resources of the ten patients in case of sickness, but it may be noted here that but for help from the charitable the conditions of three of the married men's families would have been desperate during the absence of the bread-winner, with no alternative but parochial relief or a life of absolute poverty.

The Financial Resources of the Ten Patients during Illness.

There yet remains another most important aspect of the social economy of these patients for our consideration, viz. the financial resources available for the maintenance of themselves and their families during their illness. In the next table (No. 4) I show their several conditions in this respect, their incomes being noted under the two heads Private Resources—*e. g.* money from benefit clubs, etc., and charity,—viz. financial help from employers, private philanthropy, etc. The first thing that strikes one when looking through these records is the hopeless financial outlook that the majority of the patients had to face when overtaken by illness. In fact, when out of work the total income available from all sources for the maintenance of ten men, five women, and sixteen children amounted to but 35*s.* per week. Only one of the five single men was in a club,[1] and having no money saved, they had nothing whatever to fall back upon either for the payment of treatment or for their maintenance after

[1] This patient had only 10s. per week for twelve weeks.

TABLE No. 4.

No. of case.	Dependent upon patient.	Financial resources during illness.	
		Private.	Charity.
1 (*e*)	1 child and a servant	*Nil*	Kept at Cottage Sanatorium by Salvation Army and child provided for.
2 (*d*)	*Nil*	*Nil*	£10 collected for patient's maintenance after discharge.
3 (*c*)	Wife and 2 children	10*s*. weekly from sick club	23*s*. weekly from C.O.S. (Charity Organisation Society, London).
4 (*c*)	*Nil*	*Nil*	17*s*. per week from C.O.S.
5 (*a*)	*Nil*	*Nil*	*Nil*.
6 (*c*)	Wife and 3 children	10*s*. a week from sick club	17*s*. per week from C.O.S.
7 (*d*)	*Nil*	*Nil*, but was able to live at home	*Nil*.
8 (*c*)	*Nil*	*Nil*	17*s*. per week from C.O.S.
9 (*f*)	Wife and 5 children	10*s*. weekly from benefit society	35*s*. per week from private philanthropy.
10 (*b*)	Wife and 5 children	Averaged 15*s*. weekly from benefit society	22*s*. per week from employers during 13 weeks of patient's illness.

(*a*) and (*b*).—These two patients were treated in the special open-air wards of the Sheffield Royal Infirmary.

(*c*).—Treated at the Mundesley Cottage Sanatorium for an inclusive fee of 17*s*. a week, paid by the Charity Organisation Society.

(*d*).—Treated gratuitously at the Mundesley Sanatorium, being two of a series observed on an entirely meat-free diet.

(*e*).—Treated at the Mundesley Cottage Sanatorium for an inclusive fee of 17*s*. a week, paid by the Salvation Army.

(*f*).—Treated in rooms in Mundesley village.

discharge whilst on the look out for work, and only one of these five had a home where he could go to and live free of cost. The outlook of the five married men was no better, one of them having no financial resources at all, whilst of the other four, three had club money to the extent of 10*s*. a week, the fourth patient having 18*s*. a

week (the last sum falling to 12*s.* after ten weeks) to provide for a wife and two children, a wife and five children, and a wife and five children. Not one of these ten patients, then, was in a position to pay even the smallest fee for treatment, and in the case of the married men there was not one who could in the least way provide for his wife and family whilst in the sanatorium. In other words, but for the help that was given them either by charitable bodies—*e. g.* the Charity Organisation Society—or philanthropic persons, none of the latter could have gone through their course of treatment without at the same time plunging their families into a condition of hopeless poverty or compelling them to go on to the poor rate. To avoid such a contingency all of them would have preferred to remain at work till utterly unfit to do anything—a course which could only have meant a postponement of the inevitable poverty and a hopeless case of consumption to treat in the place of one with a fair prospect of recovery. The financial conditions of these patients is typical of a large majority of the consumptive working men, but the good fortune which happily enabled them to meet their financial difficulties is, unfortunately, by no means so commonly experienced. Taking them *seriatim*, we see that the cost of treatment of Case 1 (17*s.* a week) and of the maintenance of his child and servant was paid for throughout by his employer—the Salvation Army. Case 2 was treated gratuitously and his sole capital (£10) subscribed for him on his discharge sufficed to keep him until he found work. Case 3 had the cost of treatment (17*s.* a week) and maintenance of his wife and two children—apart from 10*s.* a week he received from a benefit club—defrayed throughout his illness by the Charity Organisation Society, who also continued to allow him a guinea a week after his discharge while out of work. The cost of treatment of Case 4 (17*s.* a week) was also entirely paid for by the Charity Organisation Society. Case 5 was treated gratuitously at the Royal Infirmary, Sheffield, and found work immediately after his discharge.

TABLE No. 5.—*Showing the cost to charity of restoring the ten men to a working capacity, the total cost including that of patients' treatment and of the maintenance meantime of those dependent on them. (Any income derived from club money has been deducted from the cost of any patient who received it.)*

No. of case.	Cost of treatment and maintenance of families.				Total cost to charity.	Weekly incomes earned by patients at the present time.	
	Patient's treatment.		Maintenance of family.				
		£ s. d.		£ s. d.	£ s. d.		£ s. d.
1	Cost of 12 weeks' treatment at 17s. per week .	10 4 0	30s. per week for 13 weeks from the Salvation Army	19 10 0	29 14 0	As insurance agent . . .	1 10 0
2	Cost of 11 weeks' treatment at 20s. per week .	11 0 0	Collected for patient's maintenance while he was looking for work .	10 0 0	21 0 0	As coachman .	1 3 0
3	Cost of 14 weeks' treatment at 17s. per week .	11 18 0	Total from C.O.S. for the period during which patient was out of work.	22 1 0	33 19 0	As wood-sawyer .	1 10 0
4	Cost of 13 weeks' treatment at 17s. per week .	11 1 0	*Nil*		11 1 0	As restaurant cook	1 15 0
5	Cost of 16 weeks' treatment at 20s. per week .	16 0 0	*Nil*		16 0 0	As engineer. .	1 6 0
6	Cost of 15 weeks' treatment at 17s. per week .	12 15 0	10s. per week paid by the parish for 15 weeks .	7 10 0	20 5 0	As coachman .	1 14 0
7	Cost of 10 weeks' treatment at 20s. per week .	10 0 0	*Nil*		10 0 0	Out of employment . . .	0 0 0
8	Cost of 12 weeks' treatment at 17s. per week .	10 4 0	*Nil*		10 4 0	As casual outdoor labourer . .	0 7 6
9	Cost of 46 weeks' treatment at 25s. per week .	57 10 0	10s. per week for 46 weeks from private philanthropy	23 0 0	80 10 0	As gardener. .	1 12 0
10	Cost of 13 weeks' treatment at 20s. per week .	13 0 0	22s. per week for 13 weeks from patient's employers	14 6 0	27 6 0	As workhouse infirmary attendant	1 10 8
	Total . . .	£163 12 0	Total . . .	£96 7 0	£259 19 0	Total weekly earnings .	£12 8 2

£12 8s. 2d. × 52 gives total amount of wages earned in one year, viz. £645 4s. 8d.

Case 6 was given 17*s*. a week by the Charity Organisation Society to pay for his treatment; the parish kept his wife and three children during the three months he was in the sanatorium, the patient paying 10*s*. a week, received from his club, towards their cost. The cost of treatment of Case 8 (17*s*. a week) was also paid entirely by the Charity Organisation Society. Case 7 was treated gratuitously. The cost of treatment of Case 9 and the maintenance of his wife and five children during his six months' absence from home was, with the exception of 10*s*. a week received from a sick club, defrayed entirely by a charitable lady interested in the family, and in the case of the tenth patient, also with a wife and five children, his employer gave him 22*s*. a week for thirteen weeks towards the support of his family. In addition he had from 12*s*. to 18*s*. a week from his club, and was treated gratuitously at the Sheffield Royal Infirmary. The very great benefit that has resulted from this well-directed charity in the case of these ten patients is clearly evidenced by their several conditions at the present day, and but for the timely assistance they received, the life histories, both of the men themselves and of their families, would doubtless have given one a much less favourable view of the prospects of the consumptive working classes. Apart, however, from any question of philanthropy, the economic soundness of the expense incurred in treating these patients and supporting meanwhile their wives and families is, at the present time at least, well shown by the incomes they are able to earn. The approximate estimate of the cost to charity of these patients and statement of their present incomes given on opposite page shows the economic soundness of the lines adopted in dealing with them.

The figures in the above table show that the total cost of restoring these ten patients to health and to a working capacity, including that of maintaining the wives and families of five married men, amounted to £259 19*s*. This sum was entirely subscribed by charity, and wherever any income was derived from the patient's own resources, such

as club money, etc., this amount has been deducted from the total cost of the patient, and thus does not appear in the table. In return for this outlay of £259 19*s*. the ten men are now bringing in an income of £12 8*s*. 2*d*. a week, equivalent to an annual sum of over £600 a year. This return cannot, I think, be considered otherwise than most satisfactory, and but for the capital outlay this income would be much smaller and several families by this time living on charity or the poor rate.

Conclusions.

The information obtained from this inquiry into the social conditions of the consumptive working classes, though perhaps it has not brought to light any facts hitherto unknown to those interested in the subject, is at least of value as representing a faithful picture of some of the difficulties that must attend any effort to help the consumptive working classes. This report fully confirms the opinions that have often been expressed by writers on this subject as to the necessity for increased accommodation for the provision of very cheap or gratuitous sanatorium treatment, and of the utmost value of early diagnosis of the disease. I propose to make no further comment on these two points, but with regard to the first—viz. the provision of cheap treatment—I would draw attention to the fact that the cost of restoring five of the patients (Nos. 1, 3, 4, 6 and 8) to their normal health and capacity for work was only 18*s*. per week a head. These were five patients treated by Mr. Chapman and myself in an ordinary house in Mundesley village, supervised by a landlady with whom we had arranged, for a fixed charge of 8*s*. per week a head, for lodging, cooking and attendance. The remaining 10*s*. per week a head provided each man with a most satisfactory diet, the composition and nutritive value of which had been carefully worked out by Mr. Chapman. Medical advice was, of course, given

gratuitously. Such a method of treating early cases of consumption amongst the poorer classes has the great advantage of requiring no outlay of capital expenditure, and I think might be more generally adopted in the absence of sufficient sanatorium accommodation. It would mean, in short, the establishment of small cottage sanatoria run on very similar lines as cottage hospitals. Two other most important problems associated with the question of the treatment of the consumptive poorer classes which I think this paper serves to illustrate are:

(1) The question of maintenance of the wives and families of consumptive working men whilst under treatment in a sanatorium.

(2) The provision of an adequate living wage for consumptives after their disease has been arrested.

This latter question is, of course, intimately associated with that of the provision of suitable employment, and the two may be dealt with together.

(1) *Maintenance of Wives and Families.*

Compulsory insurance against sickness would be a great help towards the maintenance of wives and families, though, in the large majority of cases, the income derived from this source, viz. sick and benefit clubs (usually about 10*s.* per week), is quite insufficient for the purpose. In the absence of any such source of income—the usual state of affairs—there remains the alternative of parochial relief or living in poverty, and the latter of these two alternatives is in my experience the one usually chosen. A comparatively small weekly allowance, say from 15*s.* to 20*s.* per week, to a wife with several children is often sufficient to keep them going, especially when there is in addition a small weekly income from a benefit club. This was the case in three of the families recorded in this paper. A charitable fund, either local or connected with a sanatorium for the poor, for the purpose of assisting those dependent upon the income of patients, if carefully

administered on the lines adopted by the Charity Organisation Society, would be of the greatest benefit, and can be recommended both on economic and philanthropic grounds.

(2) *Provision of a Living Wage, or, in other words, Suitable Employment for Cases of Arrested Tuberculosis.*

This is a good deal more difficult and complicated question, especially in the case of married men. To begin with, one must bear in mind that no employment, however healthy or open-air its nature, is suitable unless the income the man can earn at it brings him in a sufficient weekly sum to maintain himself and his family in a condition of physical efficiency. This fact is one, I think, which is not sufficiently appreciated. Personally, rather than send a man back to such conditions of work, I would advise him to return to indoor or to even an unhealthy occupation with which he is well conversant and at which he is certain to earn a living wage. In the case of unmarried men a change of employment, entailing even a considerable loss of income, is often quite advisable, since physical efficiency can usually be maintained by a single man on a wage of from 15*s.* to 20*s.* per week—a sum quite inadequate for the needs of a man with a wife and family. Judging from my own experience, we may assume that change of employment means some loss of weekly income; on the other hand, the return to unhealthy employment means grave risk of subsequent relapse. A method of meeting this difficulty would be to give a man whose occupation is to be either unhealthy or unprofitable some financial assistance, for a time at least, after his discharge. If this was done, he could afford to return to the better, but less remunerative, employment; or, again, he could return to his original employment but with shortened hours. I have seen both these methods work very well in practice, the financial help in both cases being provided by private charity. The grant would cease with the man's ability to earn a living wage. Of course, if all

cases of consumption were diagnosed at a really early stage, and could be kept under conditions of sanatorium life for a long enough period to make permanent arrest as far as possible a certainty, this difficult question of employment after discharge would disappear; but such a happy state of affairs is a long way off. In my opinion a committee attached to a charitable sanatorium, with a fund to draw upon, would do a great deal of good if they devoted themselves entirely to the investigation of the social conditions of the patients admitted, and actively assisted them to return to conditions of life and work in which they would be likely to remain in good health. Such a committee would get into touch with large employers of what we may term healthy labour, *e. g.* tram and omnibus companies, insurance companies, house agents (for caretakers), etc., and supply them with employees whose credentials had been thoroughly investigated. This committee could also give financial help to patients after their discharge when it seemed advisable for them to return to employments at which, either from the size of their families or the poorly paid nature of the work, they would not earn a living wage. One has seen many instances in which financial help of from 10*s.* to 15*s.* per week for the first six months or so after discharge has made all the difference between ultimate success or failure. As an appendix to this paper I have prepared a table showing the income required for families of various sizes. Such a table would help any committee to determine the adequacy of the income to be returned to in any particular case. There is another way in which considerable practical assistance can be given to the working classes on leaving sanatoria—viz. advice as to the cheapest way they can supply themselves with a proper diet. In cases where there is no possibility of the weekly sum available for food being more than 3*s.* or 4*s.* per week the only thing left to do is for the man or family concerned to sacrifice to some extent the palatability of his diet, and to buy cheap foodstuffs of high nutritive value—*e. g.* pulses, beans, and other

forms of vegetable, proteid, tinned meats, separated milk, etc. A printed form containing this information might well be given to the patients' wives on their return home.

Lastly, before leaving this question of employment, I should like to say a few words upon the subject of the establishment of farm colonies in connection with sanatoria, since in the opinion of many such a scheme would help us to solve the difficulty of employment after discharge. In the absence of any such colony "in being" at the present time in this country, there are no certain grounds upon which to base an opinion as to its probable value, and the best idea of its possibilities are, I think, to be obtained from the experience of those in charge of colonies run on somewhat similar lines to those proposed for the treatment of consumptives. The only colony on any scale that I know of is the one managed by the Salvation Army at Hadleigh in Essex for the unemployed, and the conclusions I formed as the result of a short visit to this excellently managed place as to the practical value such a colony would prove as part of a sanatorium's work are perhaps worth recording. The colony at Hadleigh consists of 3000 acres of agricultural land, upon which are carried out the following industries: ordinary mixed farming and dairy work, market and fruit gardening, horticulture and flower-growing, poultry-rearing and brick-making. At these various occupations all sorts and conditions of labour, skilled and unskilled, are at work, the officer in command of the colony putting each new arrival to the work he seems to be most fitted for, although in most cases it is absolutely unfamiliar to him. In charge of these several departments of industry are a number of permanent officials, highly skilled in their several callings, whose duty it is to oversee the work and instruct newcomers. The men admitted to the colony, excluding the permanent staff, are housed in large airy dormitories which can be erected at a cost of £40 a bed, and the cost of their maintenance works out at 10*s*. per week a head, viz. 1*s*. 6*d*. per week for lodging, 6*d*. for washing, and 8*s*. for

food. The dining-hall, which sits three hundred, with kitchen adjoining, cost £750. In return for the board and lodging, men are expected to do a good day's work, viz. 8½ hours. The foreman of each department estimates the value of the work done by each man under his charge, and this is put to the credit of the man as wages. The wages are calculated on a scale prevalent in the district, and whatever sum remains to the man's credit, after deduction of 10*s*. per week for his maintenance, he is allowed to keep. A third of any such surplus, however, he has to bank, and he also has to refund the cost of any clothes advanced to him. New-comers, of course, engaged at unfamiliar work frequently fail to pay their way even if willing enough workers, and lazy men of course, also. Men on the land for the first time in their lives, so an official told me, cannot get through a third of the work of that done by a skilled labourer, and as he remarked "it is not possible to turn a decayed clerk or a drunken doctor into a skilled agricultural labourer in a few months." When a man cannot pay his way the loss, of course, falls on the colony, and when a man earns more than his keep he is allowed to take the balance, thus encouraging thrift and industry. This principle is not favourable to the financial success of the colony, which, though run on essentially business lines, is fundamentally philanthropic rather than commercial in character. Another factor which does not tend to commercial success is that of the constant changing of colonists, for as soon as the man's work becomes of some real use he leaves. The colony passes through its hands some 500 men a year, the average stay being about four months, and the work results in the loss of about £3,000 a year. In answer to my query as to the proportion of the men who return to work on the land, the officer in command of the colony told me that the majority of the men do so; but as they are all unmarried, many of them young, and willing to go back to a wage of 10*s*. a week or so, the significance of this fact is of less value than it at first appears. The very brief outline I have given of this

most interesting work allows us to form some opinion as to the probable value such a colony would prove for the treatment of consumption. A consumptive colony would have several things in common with Hadleigh; *e. g.* it would suffer from the same disadvantage of having to employ men at occupations at which many would have no knowledge, and they would also lose their men as soon as they had learnt enough about their occupation to make their labour of any real commercial value. For these two reasons alone it is manifestly difficult for such a colony to successfully compete in the open markets, commercially speaking, or even to be self-supporting, but the financial loss, as in the case of the Salvation Army colony, would be more than compensated by its social and philanthropic value. A consumptive colony would moreover suffer from the additional disadvantage of having tuberculous labour instead of sound labour. As slight compensation, however, the class of colonist would be better than at Hadleigh, whose labour is recruited from the submerged tenth. The work done at Hadleigh would, for the most part, be suitable employment for consumptives, with the exception of the brick-making, but additional sound labour would be necessary in most of the departments to do the heavier work—*e. g.* to do the digging in the market garden, etc. The necessity for having permanent skilled heads of departments would be the same in both cases. With regard to the very important point, viz. the cost of maintenance of consumptives on such a colony, quite apart from the profit or loss made on the various industries, I see no reason why it should be very much greater than at Hadleigh, where board and lodging per man costs 10*s.* per week. The housing accommodation in dormitory form as it exists at Hadleigh could be retained, though that for consumptives would have to be on a somewhat better scale. This would raise the cost of lodging somewhat, and the weekly expenditure per head on lodging would certainly exceed 1*s.* 6*d.*, the cost of the same at Hadleigh. The cost of washing at Hadleigh, viz. 6*d.* per head per week, would

also be exceeded. The weekly cost per head at Hadleigh for food, viz. 8*s*. per week, should be quite enough to supply convalescent working men with an efficient diet. Of this I am quite confident, as Mr. Chapman and myself have demonstrated this fact by practical experience. The actual dietary given at Hadleigh would, I believe, be quite satisfactory for convalescent consumptives, and judging from my personal experience of meals at Hadleigh, the food supplied is of good quality and given generously.[1] That new-comers to the colony invariably gain weight rapidly tends to support this opinion, and is additional evidence that an efficient diet can be provided at the small weekly cost of 8*s*. per head.

Making allowance for the extra cost for housing and washing compared with the Hadleigh figures, I think that a number of convalescent working class consumptives could be kept for from 16*s*. to 18*s*. per week. The practical value of such a colony run in connection with a sanatorium would be two-fold. In the first place a certain proportion of patients, made up almost entirely of young unmarried men with no one dependent upon them, would learn enough of some open-air employment to enable them to return to that work after discharge, though probably at a small wage, say from 10*s*. to 15*s*. per week, but this latter figure would be sufficient for their requirements. Such patients would in all probability permanently return to country life, and eventually make fair incomes—at least, this is the Hadleigh experience in many cases. Apart from this number who would learn new employment—and they, I think, would be the minority—a month or two on the colony would be an excellent thing for many convalescents, even though they had no intention of returning to the work they would learn there; married men with families, for instance, could rarely afford to do so. The removal of such convalescents, however, to the colony would make room for others in the sanatorium itself, and by living at the colony on less expensive lines—probably

[1] A sample diet taken at Hadleigh will be found in an appendix.

at a cost of something under £1 per week—and, in addition, doing a certain amount of work, the total length of their treatment could be appreciably lengthened at a comparatively small cost. This would be a great gain, in view of the necessity for many patients to return to somewhat risky employments, in which case a long course of treatment and as complete arrest of the disease as possible is essential for the maintenance of health. The colony would, in short, be in the nature of a finishing school, where men would gain confidence in themselves and in their ability to do work. They would also be living under ideal conditions at a comparatively small cost to the sanatorium, and under conditions moreover, in the matter of accommodation, diet, and possibly work, more allied to those to which they would be shortly returning. A consumptive colony on the lines that I have briefly sketched attached to the sanatorium would, I feel sure, prove a success and is well worth a trial. In conclusion, I wish to thank the Salvation Army officials for the opportunity they kindly afforded me of visiting the Hadleigh Colony, and especially I am indebted to their officer in charge there, Brigadier Iliffe, for his hospitality and courtesy during my stay there, and for all the detailed information he kindly gave me in regard to its management. I must also thank Mr. Chapman for his assistance in getting the after histories of Cases Nos. 5 and 10.

Appendix I.

Suggested Sliding Scale of necessary Weekly Income to provide Cases of Arrested Tuberculosis with a Living Wage.

This table is based upon figures in a table prepared by Rowntree, giving the minimum necessary expenditure per week for families of various sizes.

It allows no margin for luxuries, such as alcohol, or expenses incurred by sickness.

Family.	Food.	Rent.	Household sundries.	Total.
1 man	6s.	3s.	3s. 6d.	12s. 6d.
1 man and 1 woman	12s.	4s.	4s.	20s.
1 man, 1 woman, and 1 child	15s.	4s.	4s. 6d.	23s.
1 man, 1 woman, and 2 children	18s.	4s.	5s.	27s.
1 man, 1 woman, and 3 children	20s.	4s.	5s. 6d.	29s. 6d.
1 man, 1 woman, and 4 children	22s.	5s. 6d.	6s.	33s. 6d.
1 man, 1 woman, and 5 children	24s.	5s. 6d.	6s. 6d.	36s.
1 man, 1 woman, and 6 children	26s.	5s. 6d.	7s.	38s. 6d.

APPENDIX II.

Some Details of the Hadleigh Colony.

Farm of 950 *acres,* 200 of this arable land, remainder permanent pasture. Work done, mainly fattening cattle.

Dairy work.—45 cows kept. The milk is sold locally, and proves a profitable undertaking.

Poultry farming.—30 acres, on which are kept 3000 birds in 100 separate houses and 64 coops. This work consists almost entirely of breeding first-class birds. Their live birds and settings of eggs are in great demand for stock purposes, and a good deal of export trade is done in this line. No birds are reared for killing. This department is in charge of a particularly able man.

Market gardening, 400 *acres.*—Fruit growing (200 acres)—*e.g.* plums, apples, currants, gooseberries, etc.—pays well, the market being Southend. Flower-growing (2 acres) pays its way, and the growing of rhubarb and strawberries is also profitable.

On the whole colony there are from 50 to 60 permanent workers who can overlook and direct the work of from 200 to 300 colonists.

Extract from Balance-Sheet of the Darkest England Fund

(the Salvation Army Social Work).

Dr.	£	s.	d.	£	s.	d.	Cr.	£	s.	d.	£	s.	d.
The Farm and Dairy Section—							*The Farm and Dairy Section—*						
To Stock, Sept. 30th, 1903	5138	15	0				By Sales (including supplies to other sections) ...	4907	12	4½			
„ Purchases, Wages and Expenses	5343	0	6½				„ Stock as per valuation, Sept. 30th, 1904 ...	4951	17	8			
				10,481	15	6½					9859	10	0½
The Market Garden and Nursery Section—							*The Market Garden and Nursery Section—*						
To Stock, Sept. 30th, 1903	4042	12	3				By Sales (including supplies to other sections) ...	3490	0	11			
„ Purchases, Wages and Expenses	4753	2	6				„ Stock as per valuation, Sept. 30th, 1904 ...	4288	16	0			
				8795	14	9					7778	16	11
The Poultry Section—							*The Poultry Section—*						
To Stock, Sept. 30th, 1903	828	13	0				By Sales (including supplies to other sections) ...	956	7	1			
„ Purchases, Wages and Expenses	1534	19	10				„ Stock as per valuation, Sept. 30th, 1904 ...	1028	10	9			
				2363	12	10					1984	17	10

Sample diets supplied to the men at the Hadleigh Colony at a cost of 8s. per week per head.

Monday	Breakfast	Tea, bread and butter, porridge.
	Dinner	Roast meat and stew, potatoes and cabbage, bread, tart.
	Tea	Bread and butter and tinned salmon.
Tuesday	Breakfast	Tea, bread and butter, cold bacon.
	Dinner	Roast meat and stew, potatoes and beans, bread, plum pudding and jam.
	Tea	Bread and butter and German sausage.
Wednesday	Breakfast	Tea, bread and butter, porridge, and an egg.
	Dinner	Roast meat and stew, potatoes and peas, bread, and currant roll.
	Tea	Bread and butter and corned beef.

ABDOMINAL TUBERCULOSIS IN CHILDHOOD

A CLINICAL STUDY

BY

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THIS paper is concerned with tuberculosis appearing, clinically at all events, as a primary lesion of the abdomen; that is to say, I have taken no account of cases in which a terminal infection has incidentally involved the belly, nor of those in which a known pulmonary lesion has been complicated by abdominal infection. The data are drawn from forty cases at the East London Hospital for Children, in whom the diagnosis was considered conclusive; but it is to be observed that patients offering good evidence of the identity of the disease are those in whom the lesions are somewhat advanced, and this fact affects unfavourably the prognosis to be drawn from the series.

ELEMENTARY LESIONS.

The elementary lesions constituting primary abdominal tuberculosis as a clinical condition are these: lesions of the gut, lesions of the mesenteric lymphatic glands, and lesions

of the peritoneum. Any one of these may achieve an importance clinically paramount, but they generally occur together, though in varying degrees of prominence.

The lesion of the gut is ulceration, usually of the ileum, and usually transverse to the axis of the gut. The serous face of such a tuberculous ulcer is almost invariably the seat of grey tubercles, and consequently adhesion to neighbouring coils of bowel is almost constant. Some degree of mesenteric tuberculosis of the appropriate glands is also probably constant. I have never failed to find it after death.

The lesion of the mesenteric glands is caseation. The glands may form massive tumours, and grey tubercles on the serous face of such masses are fairly common. In consequence adhesions of these tumours to the anterior wall of the belly are frequent, and when they become purulent they tend to point at or about the navel. Whether mesenteric tuberculosis postulates a previous lesion of the gut is doubtful; at all events the lesion, if essential, may be insignificant, for it is not very uncommon to find one or two caseous or calcareous glands in the mesentery of subjects with no sign of tuberculosis elsewhere in the body, and no evidence of past lesions of the intestine. None the less, mesenteric caseation does generally connote tuberculous ulceration of the gut.

The lesions of the peritoneum are of two main varieties: (1) A general miliary tuberculosis; (2) adhesive peritonitis. This distinction, though clinically sound, is not pathologically defensible. The miliary variety is commonly accompanied by ascites, while the adhesive one is generally dry; but all degrees of combination may be seen in the dead-house. The plastic or adhesive form is by far the more frequent, the relation borne by it to the ascitic form in the series being as 4·7 to 1.

The combined effect of deep ulceration and adhesion produces in some cases, where perforation takes place, fæcal abscesses of varying dimensions; but general septic peritonitis from this cause is infrequent, for, by the time

perforation occurs, the cavity of the belly is generally obliterated.

Past adhesive peritonitis may leave a legacy of fibrous bands, which are a not uncommon source of intestinal obstruction, while dense adhesion, by kinking the gut, may, and in fact often does, produce a similar condition to a modified degree.

It may be mentioned here that genito-urinary tuberculosis as seen in adults is extremely rare in children, and as a factor in primary abdominal tuberculosis is, for practical purposes, negligible.

In brief, then, the fundamental lesion of this disease in childhood is ulceration of the gut, though the secondary involvement of the peritoneum and lymphatic glands may eclipse it in clinical prominence. Unless the results of autopsies are profoundly misleading, this dogma is true of a large majority of all cases.

Symptomatic Expression of Lesions.

In symptomatic importance the lesions of the peritoneum commonly transcend the others to such a degree that tuberculous peritonitis has become the generic title of cases of this class, though the enteric lesions are probably the fundamental ones.

Tuberculous enteritis.—So much is this the case that it is hard to say what is the expression of pure tuberculous enteritis uncomplicated by gross peritoneal lesions. Only one case of the kind appears in the series, and in it the symptoms were progressive wasting with the passage of large offensive stools, no distension of the belly, and no diarrhœa.

Mesenteric tuberculosis.—Similarly cases where large mesenteric glands can be palpated without the discovery of doughy masses formed by the matted guts are quite uncommon. That they might be found more frequently than they are I have no doubt, for they are not very infrequent as post-mortem curiosities. But in such cases

the process is latent, and attracts no attention to the belly, though it is far from unimportant, since it may determine a general infection at any moment. This is emphasised by the results of autopsies on cases of tuberculous meningitis in children previously believed to be healthy. Of thirty-one such autopsies, seven, or 22·5 per cent., showed no old lesion of tuberculosis elsewhere than in the intestines or mesenteric glands. With regard to diagnosis it may almost be laid down that hard movable tumours in the belly of a child, which are not fæcal, are caseous mesenteric glands.

Tuberculous peritonitis. — Having thus cleared the ground of the clinical rarities, I propose to deal with the peritonitis in a general review of the disease as it is most commonly met with, viz., with the peritoneal symptoms paramount. The onset may be properly divided into two periods:

(1) The period preceding, and
(2) The period following, the local symptoms and signs in the belly.

First period.—This is a period of vague ill-health. Its only constant feature is loss of flesh to some extent. Irregularity of the bowels is common, as often in the direction of constipation as of diarrhœa, which may alternate. There may be an insignificant amount of abdominal pain, and the child becomes pallid. This period is, properly speaking, the period of tuberculous enteritis, and may antedate by some months the advent of local manifestations.

Second period.—This is the period of definite peritonitis, and is heralded by distension of the belly. In the miliary form this distension depends to a variable degree upon serous effusion free in the belly; in the plastic form it is due to interference by adhesions with the free peristalsis of the gut—that is, to a partial obstruction. Not that adhesions only at this stage begin to exist, for inter-intestinal adhesion is probably an early feature; but parietal adhesion of the guts is a different matter, and one much more liable to limit their freedom. I suspect, though it is

hard to prove, that parietal adhesion is the real starting-point of the second period in plastic cases; for with the distension there appear pain, often paroxysmal and severe, and very often vomiting, and these three are cardinal signs of intestinal obstruction.

The typical variants in the history of onset are well indicated by the following three, taken from the series:

(1) "A child aged 3½ has been ailing since whooping-cough five months ago. She has had diarrhœa off and on for three months, and for nine days swelling of the belly and vomiting."

Sometimes the first symptoms recorded are those of the second period, the earlier ones having been overlooked, thus:

(2) "A child aged 3½, previously well (*sic*), was seized a week ago with great abdominal pain. The belly became swollen, and she has vomited frequently during the week." But, as is always the case, except occasionally in ascitic forms, this child showed marked evidence of wasting and chronic ill-health.

Sometimes, again, the case may actually present itself for the first time as one of acute obstruction of the bowel. For instance (3), "A child of seven was seized three days ago with acute pain in the belly and vomiting. Since then she has had great pain, constant vomiting, and absolute constipation." Fæcal vomiting ensued, and laparotomy revealed obstructive adhesions in the pelvis, with a general miliary tuberculosis of the peritoneum, free fluid, and large masses of caseous mesenteric glands.

With the distension, the superficial veins often enlarge, and in plastic cases palpation finds tenderness, and physical evidences of lesion. The belly takes on a doughy character, well described by Dr. Goodhart as giving the idea that "the whole abdomen is packed with some semi-solid material." This represents the matted intestines, but in addition definite irregular masses and hard tumours may generally be felt; the latter are caseous glands, the former, most often the great omentum, puckered and in-

filtrated with tuberculous material. The omental mass generally takes the form of a sausage-shaped horizontal tumour at a level just above the navel, and is characteristic of the disease. It is easy to mistake this tumour for the liver in some instances. When the mesenteric glands become purulent they protrude the region of the navel in a way quite peculiar to this malady, and as the abscess proceeds to its discharge, this region becomes dark red and boggy to the touch.

In ascitic cases the obstructive symptoms are often lacking, and the shifting dulness in the flanks indicative of the effusion may be the only discoverable abnormality. But removal or absorption of the fluid in a good number of such cases reveals the characteristic features of the plastic form.

Action of the bowels.—This is as a rule abnormal, but not in any fixed direction. In fatal cases diarrhœa towards the end of life is almost invariable.

Vomiting.—This is a very common incident at the onset of the second period. It is, as has been said, obstructive, and thus depends for its appearance on accidents of adhesion.

Fever.—A few cases, particularly ascitic ones, are afebrile throughout; but an occasional rise is frequent even in mild cases, while high hectic fever is the rule towards the termination of a fatal instance.

Course of the Disease.

Ascitic cases.—I have retained this division because it represents a well-known clinical type, but in discussing the course of the illness it must be remembered that it should more properly be called the "ascitic stage," for many of these cases pass later into the ordinary plastic variety. Thus considered, the variety is seldom marked by severe constitutional symptoms. The fluid, if not removed, is absorbed in the course of a week or two, and

the child, for the time at least, recovers. The ultimate outlook for these cases is commented upon below.

Plastic cases.—Retrogression of the disease in these cases is marked by gain in weight, with the subsidence of such grave symptoms as high fever or diarrhœa. Unfortunately, most of these patients become progressively worse and extremely emaciated. Pain is frequent and severe, but still paroxysmal, and in the intervals the child appears listlessly comfortable. It is surprising how the appetite survives in many cases; such children will sometimes spend almost all their intervals from pain in crying out for inadmissible articles of food, but if the wish be gratified small advantage is generally taken of it. Perforation of an ulcer may produce a fæcal abscess, usually in one or other iliac fossæ, while the red and pouting umbilicus may discharge the caseous remains of mesenteric glands, or even fæcal products in cases where gut in the vicinity has been eroded. These umbilical abscesses seldom discharge, for death commonly anticipates their rupture. But they may do so, either on the surface, or into the bowel, and the relief of symptoms occasionally following such rupture suggests the propriety of incision for this purpose even in cases apparently hopeless.

Termination of fatal cases.—Life commonly ends from exhaustion, in which the severe diarrhœa of the later stages no doubt plays an important part, and the conjunction of this diarrhœa with almost constant pain makes the death-bed of tuberculous peritonitis a very sad and painful one. In some cases death is due to a generalised miliary infection.

Age of incidence.—Of the forty cases there were in their second year, 5; in their third year, 14; in their fourth year, 6. The first year supplied none, and the remaining fifteen were unevenly distributed among the other years of childhood. The third year, therefore, is the year of choice for primary abdominal tuberculosis as a clinical manifestation.

The Path of Infection.

Eleven autopsies were made on cases in the series. In six the infection was limited to the abdomen, and the rest of the body was absolutely free of macroscopic tuberculous lesions, except in one of the six, where a few miliary tubercles, obviously recent, were scattered about the pleuræ. It seems idle, therefore, to insist with Koch that primary abdominal tuberculosis is a great rarity. In this country, at all events, it is far from infrequent, and the temptation to regard it as alimentary in origin is irresistible.

Diagnosis.

The diagnosis in the first period is full of doubt and difficulty. It appears to me that sufficient advantage is not taken of the hard enlargement of the mesenteric glands which is an almost constant feature of the disease. Unaccountable loss of weight with irregularities of the bowels should always demand a careful examination of the belly, under an anæsthetic and bimanually as well as abdominally. By this means moderate enlargement of the mesenteric glands is perceptible, and palpable enlargement of these glands is practically always a sign of tuberculosis. It is true that in chronic non-tuberculous gastro-enteritis (the likeliest alternative) these glands are always found enlarged after death, but they are then always soft, and could not be identified during life.

Apart from this sign and the tuberculin test, of which I have no experience, I know no means of making an early diagnosis.

In the second period, when the belly is distended, the disease has to be distinguished: (*a*) in the case of the ascitic form from cirrhosis of the liver; (*b*) in the case of the plastic form from (1) chronic gastro-enteritis; (2) peritoneal sarcoma.

Cirrhosis of the liver and sarcomatosis of the peritoneum are both rare in children. In the few cases of cirrhosis

that I have seen there was little loss of weight, and the liver was hard. In tuberculosis, on the other hand, the liver is generally large and soft from fatty infiltration; lardaceous disease in abdominal tuberculosis I have not come across.

I have once seen a diffuse peritoneal sarcoma in a child mistaken for tuberculous peritonitis, and know of no means of identifying it, except the tuberculin test.

It is with chronic non-tuberculous gastro-enteritis that the tuberculous disease is most likely to be confounded, especially that variety described by Dr. Eustace Smith as "mucous disease." In either case there is tympanitic distension of the belly, with unhealthy motions and loss of weight and health; but if under an anæsthetic nothing abnormal is to be discovered by palpation of the belly, it is probable that the case is not tuberculous. This is especially likely if the onset of the disease can be traced to an attack of acute gastro-enteritis, and if palpation during consciousness elicits no signs of pain. When the disease presents itself as one of acute obstruction, an accurate previous history, with the presence or absence of fluid, and the general bodily nutrition, afford the best guides. But in such cases the diagnosis of the underlying condition is not of so high an importance, for the resulting laparotomy will reveal the facts.

Prognosis.

As I have said, a prognosis based upon the cases of this series may err upon the side of pessimism, since the identity of the disease is so obscure in early stages. At the same time, it has value as showing the probable fatality of the declared disease.

Of the 40 cases 16 died in the hospital; 2 were taken out in a moribund condition, and 1 when steadily going downhill: it is almost certain that these may be held for dead. Of the remaining 21 I have traced 14, and of them 5 are dead. Assuming that none of the untraceable 7 have died, the mortality of declared tuber-

culous peritonitis appears as 24 out of 40, or 60 per cent. at the lowest possible computation.

As regards the relative gravity of the ascitic and plastic cases respectively, it will be remembered that their frequency was as 1 to 4·7. Of the 7 ascitic cases, 6 are accounted for, and of them 3 are dead, a mortality in known cases of 50 per cent. It is worth remark that none of these deaths occurred in hospital, the ascitic stage not being of immediate gravity.

Of the plastic cases, on the other hand, 27 have been accounted for; 21 of these have died, but 1 fell to an intercurrent atack of diphtheria, and has been omitted from consideration. Thus of the plastic cases 76 per cent. have died, and a large proportion of them died in hospital.

In tabular form the conclusion is as follows:—

Total mortality of diagnosed abdominal tuberculosis	71·8 per cent.
Mortality of plastic cases alone	76 „
Mortality of ascitic cases alone	50 „

Prognosis from symptoms.—Of the incidental symptoms diarrhœa, if persistent, is the most ominous. It is noticeable that of the nine survivors not one had severe diarrhœa while in the hospital, though the condition was the rule in cases proving fatal. This diarrhœa is sometimes due to a late involvement of the large bowel in tuberculous ulceration, but not by any means always.

The red and pouting navel is a grave sign as indicative of advanced mesenteric disease, and therefore, presumptively, of severe ulceration. But discharge of pus, either by the navel or the rectum, may result in recovery. One of the survivors began to mend immediately upon the discharge of a quantity of pus by the bowel. The development of a fæcal abscess is probably a fatal complication.

Condition of survivors.—Of the 9 known survivors I have seen 8, at the following intervals since the illness: After five years, 1; after three years, 1; after two years, 3; after eighteen months, 2; after ten months, 1.

The 3 survivors of the ascitic form of the disease, seen at the expiry of three years, two years, and ten months respectively, are all in robust health and free from symptoms. In 2 of them, however, the belly has distinctly lost elasticity.

The 5 survivors of the plastic form, seen at intervals varying from eighteen months to five years, appear as follows: 2, though in fair bodily health, are profoundly tuberculous, 1 having numbers of large glands on both sides of the neck, the other a large nodular mass in the belly. The remaining 3 seem to be in excellent health.

It is noteworthy that 2 of these survivors are girls, now aged 16, and that though both appear in the best of health, neither of them has yet menstruated. If this be constant, another element of gravity is added to tuberculous peritonitis.

Treatment.

It is clear, then, that we have to deal with a malady of the greatest gravity, under existing circumstances at all events. A mortality of over 70 per cent. is a reproach to medicine and will justify even drastic measures that offer a reasonable likelihood of success.

In this, as in all forms of tuberculosis, early treatment is the essence of success, and therein early diagnosis. I submit that in this disease, alone perhaps among all the lesions of tuberculosis, the urgency of early diagnosis is conspicuously overlooked, and to this neglect may be traced a large measure of its fatality. The means of achieving early diagnosis I have described above, and believe they are worthy of trial. Once diagnosed in time, there is no reason to suppose that this variety of the disease is not amenable to the recognised hygienic remedies already established in popular appreciation,—that is, fresh air and a generous though careful dietary; careful, because in the critical condition of the gut a grave diarrhœa is readily induced. But to wait until universal adhesion of the intestines has taken place, connoting, as it generally

does, advanced lesions of the gut and mesenteric glands, and then to practise mercurial inunction, is surely a counsel of despair.

There is little doubt that in children of the better classes this early hygienic treatment will meet with a fair measure of success, for these abdominal infections are not, in the main, remarkable for virulence.

But among the children of the poor this scheme of therapeutics is often impracticable, and with this in view it may be permitted to invite the opinion of surgeons as to the practicability of removing the disease in early stages with the knife. Pathological experience prompts the hope that this might be done, for 50 per cent. of autopsies on cases of this class show no old infection of the thorax, and it is fair to premise that in an earlier stage a larger proportion is only locally infected. Moreover, though after death the intestinal ulceration is often generalised, there is no doubt that the ileum, and particularly the lower part of it, is the first to suffer, and it should be possible to attack the disease while it is thus limited. I recognise the gravity of resection of the gut in children, but the risk has to be balanced against a 70 per cent. chance of a painful death if the disease becomes an established tuberculous peritonitis; and apart from this establishment, the risk of tuberculous meningitis is demonstrably considerable if nothing is done; for, excepting only the bronchial glands, caseation of the mesenteric glands is the most frequent promoter of tuberculous meningitis. If nothing more radical is held to be feasible, the removal of these softened glands from the mesentery would remove a standing menace to life, and I submit this alternative to the judgment of practical surgeons. Finally, I find no evidence of benefit from mere incision. This has mostly been practised in ascitic cases, which are inherently the most hopeful. I suspect that paracentesis, if called for by the bulk of the effusion, is equally efficacious, and at the same time evades the tuberculous granulation of the wound, which is so common an incident of laparotomy.

DISCUSSION.

Dr. J. WALTER CARR agreed that abdominal tuberculosis was practically an unknown disease in the first year of life. The tabes mesenterica of very young children, as recorded in death certificates, was generally marasmus from improper feeding. This fact had, he considered, a bearing on the source of infection, throwing doubt, as it did, on the supposed origin from milk. Children were not insusceptible to tuberculosis, for it was quite common in the lungs even in the early months of life. Moreover the number of infants reared on cows' milk was now very large, and yet abdominal tuberculosis was common later on than the first year when, in many cases, comparatively little milk was given among the poor. One factor to be considered was the crawling stage of existence, which favoured direct infection. It was not uncommon to find tuberculosis of the abdominal glands without any local intestinal lesion. If a primary tuberculous enteritis were always the precedent of tuberculous peritonitis, the prognosis would be much worse even than it was. The acute ascitic cases were, he believed, often due to a blood infection, and the chance of recovery in these cases was much greater than in the chronic dry form, which undoubtedly was usually of local origin.

Dr. SIDNEY PHILLIPS also thought that tuberculosis of the peritoneum often occurred without primary intestinal infection. The presence of enlarged abdominal glands was generally accepted as the chief diagnostic point of tuberculous peritonitis. Suppurating mesenteric glands did not necessarily discharge through the umbilicus as the paper seemed to imply. Parietal adhesions were not the only cause of intestinal obstruction in these cases. He had not seen the speedy absorption of ascitic fluid in tuberculous peritonitis that Dr. Branson had described. The pains might be violent and paroxysmal in tuberculous peritonitis. The results of operation, as at present performed, were, in his experience, much better than was implied in the paper.

Mr. STEPHEN PAGET believed that any attempt to resect the gut or remove the enlarged glands could only end in the death of the child. Incision or paracentesis seemed to be the only chance of surgical relief.

Dr. A. E. GARROD, from his own pathological experience, emphasised the enormous preponderance of thoracic over abdominal tuberculosis in young infants. In the peritonitic cases the glands had failed to discharge their normal function of intercepting the tubercle bacilli.

Mr. J. D. MALCOLM referred to the presence of calcareous mesenteric glands that he had found in the course of operations on grown-up and sometimes elderly people. These masses

appeared to be the remains of inflammatory mischief which was cured. The author of the paper was asked whether he thought such conditions might arise from tubercular mischief in early childhood.

Dr. BRANSON, in reply, said that the fact that the third year of life was the year of maximum liability to primary abdominal tuberculosis might be explained by the chronicity at this age of gastro-intestinal disorders, these troubles during earlier life being mostly of an acute nature. He thought his suggestion as to the possibility of early operation had been misunderstood by some speakers. In autopsies on cases of apparently primary tuberculous meningitis it was not uncommon to find that the only old lesions were one or two ulcers in the lower part of the ileum, or a few caseous glands in the mesentery. He thought that this was the stage of the disease to which, if at all, surgical interference should be applied.

ON A GROUP OF ASSOCIATED CONGENITAL MALFORMATIONS,

INCLUDING ALMOST COMPLETE ABSENCE OF THE MUSCLES OF THE ABDOMINAL WALL, AND ABNORMALITIES OF THE GENITO-URINARY APPARATUS

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THE co-existence of several distinct congenital deformities in the same individual is common enough, but the case which forms the subject of this communication offers an example of a group of malformations which may lay claim to a special interest.

Congenital non-development of the muscles of the abdominal wall is itself a sufficiently rare abnormality, and when met with it is wont to be associated with such structural defects of the genito-urinary organs as were present in this instance. The association is clearly no fortuitous one, and a study of the few recorded examples, some of which have been observed in this country, leaves

no doubt in the mind that either the several items of the malformation-complex are dependent upon a common cause, or that, as is far more probable, some are primary and others are secondary to these.

The recorded cases have been collected together, and the whole subject has been discussed in a masterly manner by E. G. Stumme (1) in a monograph which was published in 1903, but the examples are still so few that further records are called for, and Dr. F. E. Batten's report on the condition of the spinal cord of our patient fills in a gap in our knowledge to which Stumme refers with regret.

G. K—, a male infant, aged 8 weeks, was admitted to the Hospital for Sick Children, Great Ormond Street, on May 27th, 1903.

A bulging forward of the chest wall and a sinking in of the abdomen were noticed immediately after the infant's birth, and a few days later the abdomen began to enlarge, whilst the protrusion of the sternum persisted.

The patient, the youngest of a family of three, was born at full term. The eldest child died with convulsions at the age of fifteen months, the second was alive and in good health.

On admission the infant was well nourished, the mouth was conspicuously large, and there was some asymmetry of the lower jaw. The pinnæ of the ears were abnormally large, and the nose was rather flattened. The palpebral fissures were somewhat oblique. There was some asymmetry of the skull, with prominence of the right frontal and parietal eminences.

The thorax was obviously asymmetrical. The sternum protruded, and was tilted forwards and upwards, a peculiarity which has been observed in other cases of the kind, and which results from the lack of anchorage by the recti and other abdominal muscles.

The circumference at the level of the nipples was thirteen and a half inches, and there was an expansion of

Photograph (taken after death) of the infant G. K—, showing the furrows in the abdominal wall and the deformity of the thorax.

only a quarter of an inch on inspiration. The pectoral muscles were well developed, as also were the muscles of the back of the thorax.

Physical examination of the chest revealed nothing abnormal save that a soft systolic murmur was heard in the first and second intercostal spaces to the left of the sternum.

The appearance of the abdomen was very remarkable, and exactly recalled that seen in Guthrie's case, presently to be referred to.

The abdomen was large, flabby, and bulged in the flanks. Its surface was deeply scored by a number of longitudinal furrows, nine of which could be counted. A deep central furrow extended from the tip of the ensiform cartilage to the pubes, and in it was included the umbilicus which, as in other cases of the kind, was represented by a mere linear scar. A second deep furrow, running immediately to the right of the central one, formed a V by uniting with it below the umbilical scar. When the sides of the furrows were held apart lines of cicatricial tissue were exposed, and the grooves could not be permanently smoothed out. Besides the longitudinal furrows there were a few short and almost horizontal ones.

The distance from the tip of the ensiform cartilage to the pubes was five and a half inches, and the girth, at the level of the umbilicus, was fifteen and a half inches.

There appeared to be no muscular substance, capable of contraction, in the abdominal wall. The walls acquired no firmness when the child cried, and passing backwards the first developed muscle which could be felt was the erector spinæ. Moreover there was no response to Faradic or galvanic stimulation in the regions which were apparently devoid of muscular coating.

In either flank the contours of coils of intestine were plainly visible. During life there was some doubt whether these coils might not have been distended and tortuous ureters, as in Guthrie's case.

The liver could be plainly felt, lying in the right hypo-

chondrium, and with its lower border three fingers' breadths below the costal margin.

The left kidney, also, was readily palpable, but the right kidney could not be felt during life.

The spleen descended to one finger's breadth below the costal border, and was smooth. Neither testicle was descended, but that on the right could be made out by palpation, lying just below the region of the kidney.

The contour of the transverse colon could be seen crossing the abdomen just above the umbilicus, and the stomach, when full, stood out prominently, and measured three and a half to four inches in its long, and two inches in its transverse axis.

When the bladder was full its outlines could be clearly mapped out. It reached upwards to within a finger's breadth of the umbilicus, to which it appeared to be anchored by its upper pole. It was distinctly spindle-shaped.

The very easy palpability of the several abdominal viscera was due to the absence of any resistance from the abdominal walls.

Per rectum.—When once the sphincter ani was passed the bowel proved to be fairly capacious. The bladder was easily palpable, as an abdominal viscus, and could be felt to contract when distended with urine. No dilatation of the ureters could be felt by the examining finger. The testes were palpable, lying below the lower poles of the kidneys.

There was double talipes varus.

The urine was of low specific gravity, was turbid, alkaline in reaction, and had an ammoniacal odour. The motions were greenish and contained undigested curd.

The child was discharged from the hospital on June 10th, but was readmitted on August 4th with a temperature of 101° F. In the lungs were small scattered patches of broncho-pneumonia.

On August 5th the temperature rose to 103° F., and a bright red area of inflammation was observed, which

extended in front from the root of the neck up to the face, and was bounded by a brighter red line, which ran parallel with the alveolar process of the upper jaw.

The erysipelatous blush persisted, and the child died on August 13th. The photograph was taken after death.

The report of the post-mortem examination, which was carried out by Dr. E. P. Baumann, is as follows :

Length of body 22½ inches, weight 7½ pounds.

Abdomen.—The anterior abdominal wall was soft and flabby, and through it the viscera could be palpated. On dissecting away the skin, which was in most places unduly adherent to the underlying fascia, there was found to be a general failure of development of the muscles of the anterior abdominal wall. There were seen in the fascia, here and there, pinkish strands, which probably represented the remains of the obliqui, transversales, and recti muscles. Posteriorly the erectores spinæ appeared to be normally developed, whereas the quadrati lumborum were represented by thin imperfect sheets of muscle-fibres.

The muscles of the perinæum appeared to be normally developed. The diaphragm, intercostals (external and internal), the pectorales, and other thoracic muscles were also macroscopically normal. There was a boss over the right parietal bone, which was found, on removing the calvarium, to be an outward dent of the bone without any thickening.

The bladder was enlarged and thick-walled; it extended up into the abdomen, and from its apex a urachus, of the size of a thin lead pencil, extended to the umbilicus.

Both testicles lay loose in the pelvis. The right kidney was very small, of about the size of a large bean. The corresponding ureter appeared normal. The left kidney was perhaps rather larger than is usual in a child of four months. Its ureter was of normal size above, but in the lower half became dilated to the [illegible] small

pencil. There was otherwise no marked enlargement and no tortuosity of the ureters.

The capsule of the spleen was white and opaque, but on section the organ was natural. The capsule of the liver was also thickened and unduly adherent. The liver was hard, and its appearance suggested interstitial change.

Intestines natural; some congestion of mesenteric glands.

Thorax.—Nothing abnormal was found in the heart. The lungs showed changes associated with bronchitis. The bronchial glands were not enlarged.

Cranium.—The brain and membranes appeared natural.

Spinal cord.—No macroscopic changes.

We are greatly indebted to Dr. F. E. Batten, who was good enough to examine the spinal cord and the rudiments of muscular substance in the abdominal wall, and who has furnished us with the following report:

"To the naked eye the spinal cord and nerve-roots appeared normal. On section of the cord at various levels the diameter was equal to that of a normal cord from a child of the same age, with which it was compared.

"On microscopical examination no group of cells could be seen to be absent or diminished in number or size.

"The number of cells in the anterior horns of five consecutive sections at each level of the thoracic region were counted.

"The difficulty of counting cells is known to all who have undertaken such work. The personal factor plays an important part in such counts. The two counts were made at the same time and under similar conditions of light and staining. Drawings of several sections were made under the Zeiss projection apparatus.

"The following table gives the relative numbers of cells in five consecutive sections, at various levels, compared with those in the spinal cord of another child, of the same age, which showed no morbid changes.

	Spinal cord of normal child.			Spinal cord of G. K.		
	Large cells.	Small cells.	Total.	Large cells.	Small cells.	Total.
1st thoracic segment	72	31	103	59	70	129
2nd „ „	33	19	52	46	29	75
3rd „ „	35	30	65	33	41	74
4th „ „	29	36	65	47	34	81
5th „ „	41	42	83	49	41	90
6th „ „	33	40	73	55	29	84
7th „ „	53	37	90	36	51	87
8th „ „	26	21	47	32	51	83
9th „ „	35	28	63	40	41	81
10th „ „	44	34	78	31	61	92
11th „ „	36	32	68	21	111	132
12th „ „	51	43	94	15	103	118
	488	393		464	662	

"The number of large cells is approximately the same in the two counts. The number of small cells is larger in the case in which the abdominal muscles were absent."

These observations clearly lend no support to the idea that the condition of the abdominal muscles in such cases may be due to a poliomyelitis occurring during intra-uterine life.

On the scantily distributed and rudimentary muscle-fibres in the abdominal walls Dr. F. E. Batten reports as follows:

"Sections were made of the entire thickness of the abdominal wall, the sections being cut in a horizontal direction. They were stained by van Giessen's method. Specimens were also prepared from a normal child for comparison with them, and these were cut in the same way, and stained by the same method.

"The total thickness of the abdominal wall from skin surface to peritoneum is as great in the abnormal as in the normal specimens.

"In the abnormal child rather more than two-thirds of the entire thickness of the abdominal wall is occupied by the epidermis, cutis, and subcutaneous tissue. The glandular elements in the skin are greatly in excess of the normal, and the true skin is of three times the normal thickness.

"The subcutaneous tissue also is greatly in excess, and is composed of a considerable layer of fat and connective tissue, which is not present in the specimens from the normal child."

"The 'muscular layer,' instead of forming about two-thirds of the total thickness of the wall as in the normal child, occupies but one-third of the total thickness. Small bundles of what appear to be undeveloped muscle-fibres are seen in this region in transverse section, and in close proximity to these some fibres cut in a horizontal direction.

"The fibres of this layer are irregularly arranged, for there are two sets of fibres cut in a transverse direction, the superposed being considerably thicker than the deeper layer, and between these a thin layer of fibres cut longitudinally.

"Except for the shape of the bundles and the arrangement of the fibres it is quite impossible to identify them as muscle-fibres, as they have neither the shape nor the striation of the normal muscle-fibres. The nuclei are elongated and rod-shaped, and, as far as can be ascertained, occupy the centres of the fibres.

"The fibres of the aponeuroses of the muscles, which form a very striking feature in the normal sections, are almost entirely absent in the sections of the abnormal muscles. In appearance the muscular fibres resemble unstriped muscle-fibres, or fibres in the early stage of development, and it would appear that arrest of development had occurred in the abdominal muscles at an early stage of their fœtal life. There is nothing to suggest that atrophy of a normally developed muscle has taken place.

"The fact that no alteration can be found in the spinal cord is only in accordance with our present pathological knowledge of the condition of the nerve centres in cases of primary muscular atrophy."

Of the malformations which were present in this case

the following constituted the group of deformities which calls for special consideration:

1. The almost complete defect of the muscles of the abdominal wall, the recti, obliqui, and tranversales, and the very imperfect development of the quadrati lumborum.
2. The fœtal situation of the urinary bladder, its fusiform shape, hypertrophy and attachment to the umbilical scar by a short urachus.
3. The linear scar-like umbilicus.
4. The imperfect development of the right kidney.
5. The undescended testicles.
6. The carinated thorax and tilted sternum.

The remaining deformities, viz.:

7. The double talipes varus;
8. The asymmetry of the skull; and
9. The abnormal size of the pinnæ of the ears;

may be looked upon as mere concomitant malformations, having no obvious connection with those of the first group.

Before proceeding to speak of similar cases, of which accounts have been published, we have Dr. F. E. Batten's permission to refer to a case recently under his care as an out-patient at the Hospital for Sick Children, which one of us had an opportunity of examining.

This patient also was a male child, born at full term. At birth the anterior wall of the abdomen appeared "plastered down" to the spine, and the thorax was drawn upwards and tilted forwards. When seen, at the age of nine months, the appearance of the abdomen closely resembled that observed in our patient. The skin showed similar grooves and furrows, and no trace of functionally active muscles in the abdominal wall could be made out. The abdominal viscera were easily palpable, including the bladder, which occupied the fœtal position and appeared to be attached by a urachus to the umbilical scar.

The child afterwards died at its home of some intercurrent disease, and no post-mortem examination was

made. The case obviously belonged to the same group as that which we have described and others presently to be referred to.

Stumme has collected seven cases from the literature in which there was a failure of development of the abdominal muscles, and adds to these an eighth observed by himself. Five of these cases resemble those which we have described more or less closely, whereas in the three remaining cases the resemblance is much less striking, and the published accounts of some of them are very brief. We have not been able to find any cases to add to Stumme's list, nor do we know of any recorded since his paper appeared.

Three of the cases may be dismissed in a few words.

In 1839 Fröhlich (2), in a thesis which has not been accessible to us, described a male child who presented a defect of the lateral abdominal muscles and a condition of pigeon-breast. However, the recti abdominis were developed, and, as the condition of the bladder is not mentioned, even in the account of the post-mortem examination, we may conclude that it was normal. The testicles, moreover, lay in the scrotum.

In a case described by F. A. von Ammon (3) in 1842 there was a defect of the lower segment of the recti and of other muscles of the lower part of the abdominal wall. The patient was a male child. The bladder is not mentioned. The defective muscles are stated to have become developed at about the time of puberty.

In 1890 B. Henderson (4) recorded the case of a man, aged 60, who had a similar defect of the abdominal muscles, including the lower segments of the recti. The testicles were not descended, but there is no mention, in the brief account of the case, of any abnormality of the urinary bladder.

The main features of the remaining five cases we have arranged in tabular form, which is best calculated to bring out the points of resemblance in and differences between them.

Name of observer.	Sex of patient.	Age.	Shape of chest.	Appearance of abdomen.	Umbilicus.
I. W. B. Platt (5), 1898. (As we have been unable to consult the original paper the details are given from Stumme's abstract)	Male	2 years	Not stated, but shown in the figures to be deformed	Skin doughy and irregular; horizontal furrows seen in the pictures	A vertical slit; beneath it a hard, flat scar
II. R. W. Parker (6), 1895. Fatal case. Post-mortem examination	Male	Newly born	Thoracic cavity small	Large and very flaccid; skin over it wrinkled and inelastic; outline of coils of intestine clearly seen	Not depressed; normal in position; had the appearance of a surface scar
III. L. Guthrie (7), 1896. Fatal case. Post-mortem examination.	Male	9 weeks	Pigeon-breast, and costal arch unusually wide	Flaccid, bulging laterally; deep vertical furrows of skin, which could not be smoothed out; what appeared to be coils of intestine, but were really coils of ureter, seen in both flanks	Represented by a white linear cicatrix; at the bottom of a furrow; normal in position
IV. W. Osler (8), 1901	Male	6 years	Harrison's sulcus well marked; lower part of sternum thrust forward, almost at a right angle with the xiphoid cartilage	Flattened out, bulging in flanks in recumbent position; coils of intestine seen in peristalsis; furrows of skin, "crows' feet," below the umbilicus	Linear, forming a furrow about an inch in length
V. E. G. Stumme (1), 1903. Laparotomy; vertex of bladder was freed, and the dilated left ureter was connected with the bladder	Male	17 years	Broad and very flat; sternum depressed at level of sixth rib, and coming forward again at xiphoid cartilage	Protuberant when standing; bulging in flanks in recumbent position; between xiphoid cartilage and umbilicus a number of horizontal folds of skin; a vertical furrow in middle line from xiphoid cartilage to umbilicus, forking above umbilicus; at bottom of this groove a linear scar.	A flat depression the size of a two-mark piece, with a central scar

Abdominal muscles.	Bladder.	Kidneys and ureters.	Testicles.
Electrical examination; recti developed in their entire length, but very imperfectly; fibres of both external obliques present; presence of internal obliques uncertain; no sign of transversales	Bladder could not be made out either by palpation or by percussion. Prepuce easily retracted	—	Not descended.
P.M.—Latissimus dorsi slightly developed; a band of fibres represented external oblique; erector spinæ well developed; quadratus lumborum normal; rudiments of internal oblique; transversalis quite absent; a thin layer of fibres represented upper segment of rectus; lower segments wholly wanting, or represented by mere traces of muscular fibres	Bladder greatly hypertrophied; abdominal in position; at the apex of trigone bladder wall closely adherent to rectum; no signs of urethral obstruction. Prepuce easily retracted. No mention of any attachment of the bladder to the umbilicus	Pelves of kidneys and ureters greatly dilated and hypertrophied; orifices of ureters free; kidneys large and yellowish-white in section	Undescended. Both lay in iliac fossæ, quite free from the internal rings.
P.M.—Only the two upper segments of the recti show any muscular fibres; the costal margins of the obliqui and transversales showed muscle fibres for about two inches below the ribs; such fibres reappeared in the iliac regions; latissimi dorsi well developed, also erectores spinæ; quadrati lumborum rudimentary	Bladder much hypertrophied, lying wholly in abdominal cavity; its apex firmly adherent to the back of the umbilical scar; no trace of urachus. No obstruction of urethra; no phimosis	Kidneys not enlarged, but much inflamed; ureters dilated to size of small intestine of an adult; very tortuous; orifices free	Not mentioned.
Attachments of upper segments of recti to costal margin and sternum clearly seen. Examination showed that the boy had practically no abdominal muscles	Bladder felt as a firm ovoid body, reaching almost to the umbilicus	No information could be obtained	Not descended; not felt in groins.
Electrical examination:—Recti present above the umbilicus. Of the lateral muscles, the transversales gave the best response. The external obliques responded, but very feebly; the internal obliques gave no response; quadrati lumborum responded well	Bladder very large, fusiform; attached to the umbilical scar by a band as thick as a pencil, and 3—4 cm. in length	Right kidney movable, enlarged; left kidney very small. Left ureter as large as a lamp chimney; right ureter of the size of a little finger	Not descended; could not be palpated externally; left testicle lay near the internal ring.

A comparison of these cases, numbered I to V, and of our own case, and Dr. Batten's, which will be referred to as VI and VII respectively, brings out the following points:

α. All the patients were of the male sex.

β. In none was there any evidence of existing obstruction to the outflow of urine, either from phimosis or any blocking of the ureters or urethra.

γ. In several cases conspicuous deformity of the thorax was noted, but the form varied considerably. In several cases there was a tilting of the sternum forwards and upwards, III, IV, VI, VII.

δ. Furrowing of the skin of the abdomen was noted in several cases. In some it was very conspicuous, III, VI, VII, in others comparatively trifling, IV, V.

ε. In all the umbilicus resembled a scar, and was in most cases linear, I, III, IV, VI.

ς. In all there was a conspicuous lack of development of the abdominal muscles, but in none were all traces absent. The upper segments of the recti were more or less fully represented in almost every instance.

η. In all cases except I the bladder lay wholly in the abdomen in the fœtal position. In Case I there was no evidence of its presence there, but the umbilicus was linear and scar-like.

θ. In all cases in which the point could be verified, except Cases I and II, the bladder was attached to the umbilicus either directly or by a urachus.

ι. In Cases II, III, and V there was conspicuous dilatation of the ureters. In VI there was only very slight dilatation of the lower part of one ureter. In the other cases no information on this point could be obtained.

κ. In Cases V and VI there was conspicuous inequality of the sizes of the two kidneys.

λ. In all cases (except III, in connection with which the testicles are not mentioned) the testicles had not descended.

The chief points in which our case differed from the

rest is in the absence of any noteworthy dilatation of the ureters, and in the presence of other malformations, such as talipes varus.

Let us now pass on to consider briefly the relationship to each other of the several abnormalities which these cases present.

Dr. Bardeen, to whom Professor Osler referred the problem, and who is entitled to speak with special authority on the development of muscular structures, suggested two possible explanations, which may be quoted in his own words:

"1. It is possible that the lack of resistance normally met with in the abdominal wall by the bladder, at the time when the kidneys begin to secrete urine, may cause the bladder to expand rather than to empty secretions into the amniotic cavity through the urethra.

"2. Under normal conditions the growth of the abdominal musculature into the *membrana reuniens*, the early covering of the abdominal cavity, is preceded by the formation of a vascular plexus, supplied from above from the internal mammary, from below by the epigastric artery. It is possible that an abnormal arrangement of the blood-vessels in the embryo prevented the formation of this plexus, and impeded the growth of the abdominal musculature, and that, at the same time, circulatory disturbances gave rise to the abnormal conditions found in the bladder and ureters."

Besides the possibilities here suggested, viz. that the bladder condition is secondary to the muscular, or that both result from a common cause, there is a third possible explanation which is favoured by Stumme.

Stumme, after discussing fully the embryological questions involved, summarises his conclusions in a passage, of which the following is a translation:

"As the result of an occlusion of the urethra, occurring at a comparatively late period of embryonic life, retention of urine occurred, to which the bladder, and afterwards the ureters, responded by hypertrophy and dilatation.

By pressure of the much dilated bladder atrophy of the greater part of the rectus abdominis, or at least of the entire sub-umbilical portion of that muscle, was brought about; again the stretching, aided by pressure from the dilated ureters, led to a similar symmetrical atrophy of the lateral muscles of the abdominal wall.

"The enlargement of the bladder led to fusion of its vertex with the umbilicus, and later to the formation of a urachus. Moreover, by pressure upon the umbilical vessels, it caused a diminution of the blood-supply on the one hand, resulting in deficient nutrition of the embryo; and, on the other hand, by interfering with the return of blood from the embryo, it rendered possible the development of ascites, and a resulting further increase of the abdominal extension. Lastly by obstructing the entry to the inguinal canal, it hindered the entry of the testicle into the *processus vaginalis*, and its descent into the scrotum.

"Later on the cause of the retention of urine disappeared. The abdominal walls, which had been stretched to a greater or less extent, became thrown into folds and contracted, owing to the increase of amniotic fluid which resulted from the outflow of urine into it. The bladder also shrank, but was unable to sink down into the lesser pelvic cavity, on account of its being fixed to the umbilicus, and of the altered conditions in the pelvic flood."

We are inclined to accept the view, here put forward, that the condition of the abdominal musculature is secondary to dilatation of the bladder, and for the following reasons:

The appearance of the abdomen in such cases suggests that at some period it has been greatly distended by pressure from within, and the linear cicatrices at the bottom of the deep cutaneous furrows, observed in some cases, may have resulted from splitting of the deeper layers of the much stretched skin. On the other hand there can be no doubt that any such distension had already disappeared before the patients were born, for in more

than one case the abdomen was said to have appeared peculiarly flattened at the time of birth.

It is noteworthy that in no instance were the abdominal muscles completely absent, and in the case of the recti the defect of the lower segments was much more pronounced than that of the upper. This strongly suggests that the development of the muscles was not primarily at fault, but that either their development was interfered with by pressure from within the abdomen or that the muscles had originally been formed in the normal manner, but had undergone atrophy from a similar cause. That such atrophy was due to a poliomyelitis can hardly be supposed in the light of Dr. Batten's report on the spinal cord of our patient, and the very careful dissection carried out in Guthrie's case showed that the muscles were comparatively well developed near their peripheral attachment, whereas the central and anterior portions were much more defective. This is just what might be expected if their condition were due to the pressure from within exerted by a central viscus, such as a distended bladder. However, as we have seen, Dr. Batten considers that the microscopical appearance of the rudiments of muscles present in the abdominal wall suggest a failure of development rather than an atrophy.

The temporary obstruction during intra-uterine life, which Stumme's theory involves, is not very easy to explain, and it must be remembered that in no instance was any condition observed which would account for urethral obstruction, not even a tight prepuce.

The fact that all the patients have been males tempts one to ascribe the obstruction to some kinking or constriction of the penile urethra, but as Stumme points out, there is another possible explanation. In his case a cystoscopic examination, made before the laparotomy at which the apex of the bladder was freed from the umbilicus, showed a deep fold in the anterior wall of the bladder and a displacement of the orifices of the ureters, which lay obliquely and almost in a line with each other;

after the operation the anterior fold had disappeared, and the orifices of the ureters had assumed their normal relations. He therefore suggests that the tugging of the urachus may have caused some obstruction of the intramural portions of the ureters, and of the vesical orifice of the urethra also. However, as Stumme points out, this necessitates an attachment of the upper pole of the bladder to the umbilicus as the prime factor, whereas such attachment has not been shown to be present in all cases.

Stumme is inclined to agree with Guthrie in attributing the abnormality of the umbilicus to the drag of the distended bladder upon it, but it is noteworthy that in Platt's case, in which no evidence of abnormal situation or size of the bladder was forthcoming, and in Parker's case, in which no attachment of the bladder to the umbilicus is mentioned in the report of the autopsy, similar abnormalities of the navel were observed.

The condition of the thorax in our case, and the similar deformities observed in other cases, seems to be sufficiently explained by the want of the anchorage which under ordinary circumstances is supplied by the abdominal muscles.

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DISCUSSION.

Dr. LEONARD G. GUTHRIE said that in his case he believed the testicles were in the scrotum. He disagreed with the author's explanation of these cases as being due to some kinking or constriction of the penis which left no traces, but assuming that the faulty development of the abdominal muscles was primary, he thought that emptying of the fœtal bladder when surrounded with the liquor amnii might be difficult, and so lead to hypertrophy of the bladder. The dilatation of the ureters might be secondary to attachment of the summit of the bladder to the anterior abdominal wall leading to mechanical obstruction of the ureters. There was no direct evidence of any temporary obstruction to the urethra during intra-uterine life, and such would have to be in operation a very long time to bring about these effects. The case described in the paper was almost identical with his own.

Dr. E. FARQUHAR BUZZARD was inclined to agree with Dr. Guthrie, and supported the view that arrested development of the abdominal muscles might be held to explain the other abnormalities in these cases. Before hypertrophy of the bladder had time to arise, dilatation of the bladder and ureters would take place owing to the accumulation of secretion which was not properly expelled in the absence of abdominal muscular contraction. He cited other instances of congenital symmetrical absence of certain muscles which he had observed.

Dr. G. NEWTON PITT asked whether the hypertrophy of the bladder wall was uniformly distributed. Usually in a hypertrophied bladder the anterior wall is much the thicker, because the abdominal wall, holding up the bladder, flattens the anterior surface and reduces the curvature of that part. In the absence of the abdominal muscle the bladder should be spherical and the thickness of the wall uniform.

Dr. GARROD, in reply, said that he fully recognised the difficulty of explaining the theoretical urinary obstruction during fœtal life, but it appeared to him that the evidence for the view that the urinary condition was primary was stronger than that opposed to it. He could not answer the question as to the relative hypertrophy of the bladder on its anterior and posterior walls, as he was himself away when the autopsy was made, and the point was not mentioned in the notes.

ANKYLOSTOMIASIS,

PRINCIPALLY AS AFFECTING COAL AND METALLIFEROUS MINERS

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SINCE the opening out of the St. Gothard tunnel in 1892, the malady known as ankylostomiasis, or the miner's worm disease, has played havoc in the mining districts of Central Europe. Having, through the kindness of the mine manager, Mr. Thomas, previously seen something of the disease as it exists in the Dolcoath mine in Cornwall, I visited in June last Westphalia and Hungary, in order to become personally acquainted with the conditions under which ankylostomiasis flourishes there. It is this experience which I wish to place before you. Although ankylostomiasis has not so far become in Britain the scourge it is on the Continent, the importance attached to the disease may be gauged by the fact that the malady has already been made the subject of very able Reports to the Home Office by Dr. J. S. Haldane, of Oxford, and Dr. A. E. Boycott, of Guy's Hospital, and has also been made the basis of an inquiry by the British Association for the Advancement of Science. It is generally stated that, except in the case of some of the deep mines, there is not much likelihood of the disease becoming epidemic in this country, for the temperature is too low for the hatching of ova to occur.

In the North of England the number of coal miners working underground is for Newcastle, Northumberland, and part of Durham, 71,458 ; Durham, 68,967; Cumberland, 7,022; and Yorkshire and North Lincoln, 87,049. To these may be added the miners working in the Manchester and Liverpool districts, who number 36,331 and 51,074 respectively. Of the total number of men employed in the coal mines of Great Britain, it is estimated that 650,000 work underground, many of whom might become infected were the disease to be imported into the mines and the conditions therein prove favourable to the development of the larvæ. It is fortunate for this country that the coal mines, generally speaking, have a lower temperature, are drier and are better ventilated than many of those on the Continent. In some of the deep metalliferous mines in Cornwall there is a high temperature, and there is moisture, and it is in these mines that ankylostomiasis has already appeared.

As my remarks deal specially with ankylostomiasis as affecting miners, I shall only incidentally refer to the disease as met with among the Egyptians, the coolies of East and West Indies, the agricultural populations of South America, and the brick-makers of Cologne. Many of the workers in the brick-fields around Cologne follow the vocation of coal mining in winter and take to brick-making only in summer.

The disease is due to the entrance into the body of a larval parasite, which ultimately finds its way into the intestine, where it becomes sexually mature, and although the name ankylostomum duodenale has been given to the parasite, the fully developed worm does not limit itself to the small portion of the alimentary canal which its designation suggests.

It was the opening out of the St. Gothard tunnel that drew European attention afresh to the subject of ankylostomiasis, for at first it was thought that the anæmia the men were suffering from was a form of bloodlessness peculiar to miners and due to breathing vitiated air, hence the name given to the disease of "*la maladie de tonelles.*"

Further investigation, however, not only established the parasitic nature of the malady but demonstrated it to be exactly the same disease as Egyptian chlorosis, mal d'estomac of the negroes of the French West Indies, etc., and in addition, that far from being as was at first supposed a disease peculiar to Italian miners, the malady existed elsewhere, and was known by various names in India, Ceylon, in the tropical and subtropical countries of South America, and in North and South Queensland, Australia, and the islands of the Pacific. For much that we know of the anatomy and the life history of the worm, we are indebted to Dubini (Milan) (1841), Perroncito (Turin), Tenholt (Westphalia), Lambinet (Liege), Goldman (Hungary), Looss (Cairo), Manson, Haldane and Boycott, of this country.

The ankylostomes are male and female. They are usually present in the alimentary canal of man in the proportion of one male to three females. The male worm measures $\frac{1}{4}$ to $\frac{1}{2}$ inch in length by $\frac{1}{2\frac{1}{4}}$ in breadth, while the female measures $\frac{3}{4}$ of an inch and is slightly broader than the male. Possibly fully developed ankylostomes vary somewhat in measurement in different countries. The worms are bluntly filiform in shape. If examined when dejected after an anthelmintic many of them will be found to be red and filled with blood, while others are of a pale grey or white colour. "In both sexes[1] the posterior end is the broadest part, whence the body tapers forward to a narrow neck ending in a powerfully armed bulging and distinct mouth capsule. The margin of this remarkable organ is furnished with four strong claw-like hooks—two on each side of the ventral line—and two conical teeth—one on each side of the dorsal line.[2] The tail of the female is conical and ends in a short delicate spine, the anus being subterminal and the vagina opening on the ventral surface at the commencement of the posterior third of the body.

[1] 'Tropical Diseases,' by Sir Patrick Manson, revised edition, p. 647.

[2] Briançon and other writers speak of two additional teeth dorsal to these.

The tail of the male is provided with a large umbrella-like trilobate bursa possessing eleven ribs. Two long and very delicate spicules project from the cloaca which opens at the bottom of the bursa. Owing to the relative positions of the sexual openings the worms in conjugation look like the Greek letter Υ."

The female ankylostomes produce and keep throwing off an enormous number of ova, which are constantly being discharged in the fæces. The ova, which measure $\frac{1}{400}$ by $\frac{1}{700}$ inch in diameter, do not undergo any further development in the human intestine, partly owing to the temperature of the alimentary canal being too high and partly owing to an insufficient supply of oxygen, but if the fæces are deposited where there are moisture and a fairly warm temperature, say 65° F. to 90° F., the ova develop into larvæ, which at first are very active and extremely voracious. The larvæ are about $\frac{1}{112}$ inch in length and $\frac{1}{2000}$ inch in breadth. The best nidus for hatching the ova is the fæcal mass itself. Within a few days (three to four), under suitable conditions, the ova may develop into active larvæ, to which by some writers the name "rhabdites" is erroneously given. These grow rapidly, and after moulting two or three times they become torpid and cease to eat, when further growth is suspended. The ova of the miner's worm are extremely resistent to the action of disinfectants. In the Westphalian coal mines I found the larvæ in the muddy water and sludge along the sides of the main ways. Here they may live for several months (nine to twelve), or they enter the wooden props in the mine that are supporting the roof, and they reach upwards at least three feet, causing the props to become moist and rotten, and thus a source of danger. Should the larvæ become transferred to the alimentary canal of man they lose their external covering and acquire the sexual characters of the fully developed worm.[1]

[1] Major F. Smith, Army Medical Corps, in the 'Journal of the Royal Army Medical Corps,' January and February, 1905, states that when in Sierra Leone he found in a small insect which haunts fæces, and which

Mode of entrance into the human body.—While the *larvæ* may reach the intestine by persons *drinking* contaminated water or by *eating* food with unwashed hands, it is to Dr. Looss of Cairo, who accidently infected himself by a culture coming into contact with his hand, that we are indebted for the knowledge we now possess of the less direct route which the larvæ take to gain the alimentary canal. Through the kindness of Dr. Looss, and with his permission, I am able to throw upon the screen several slides made by Dr. Looss from his experiments upon dogs. He rubbed a culture rich in larvæ into the skin of a dog, and in 2½ hours he found the larvæ in the deeper portions of the dermis and extremely active. From the subcutaneous tissue Dr. Looss found that they penetrated a vein, and by the blood-stream were carried to the right side of the heart, and by the pulmonary artery into the lungs. From the pulmonary capillaries they escaped through the epithelial lining of the alveoli into the air-cells of the lungs. Proceeding up the bronchi they reached the trachea. Having reached the upper limits of the respiratory passages, they made their way into the œsophagus, and by this tube reached the stomach. Escaping from this viscus by the pyloric orifice they reached the duodenum and the upper part of the small intestine, where they became arrested and finally clung by their hooklets to the mucous membrane. This is one of the routes pursued by these larvæ which Dr. Looss' experiments have completely demonstrated. Many experimenters have tried to obtain similar results and have failed. Within the last few weeks Dr. Hermann, of Mons, and one of Dr. Tenholt's assistants have succeeded in infecting themselves through the skin. That the larvæ of the ankylostoma have the power of penetrating the skin had long been suspected. When Dr. Looss infected himself, the skin around the

he named the "grey fly," the embryo of what is possibly the ankylostoma, and he therefore raises the question whether the worm is simply the parasite of the "grey fly" or whether the "grey fly" may not be an alternative host of the ankylostoma of man or of a lower animal.

hair-follicles at the site of the inoculation became red and irritable; in 3 to 7 hours afterwards the part affected was painful and swollen. Subsequently symptoms developed which confirmed his suspicions of his having accidentally become the subject of ankylostomiasis. Nine weeks after the inoculation he found the ova in his fæces. Sir Patrick Manson tells us in his book that the skin of a human leg one hour before amputation was similarly inoculated, and that when sections of the skin were made, numerous embryos were not only found in the hair follicles, but many had penetrated into the surrounding connective tissue. The dermatitis which affects the feet of the coolies working on plantations in Assam and the West Indies, and which is known as ground itch, water pox, etc., has been shown by Bentley to be due to the ankylostoma larvæ having penetrated the skin. The coolies work barefoot upon soil polluted by fæcal matter, and as during the warm weather the ova become hatched in the moist earth into larvæ, these readily penetrate the skin. In several of the Cornish miners, Dr. Haldane found a skin affection known as "bunches," which he was led to regard as the local irritative results of the entrance of ankylostoma embryos into the skin.

While ankylostomiasis occurs in man, there is a similar disease met with in the dog, wolf, fox, and sheep: the two diseases are not intercommunicable. Schaudinn, of Berlin, has succeeded in infecting a monkey with human ankylostomiasis, but the human form of the disease cannot be transferred to the dog nor that of the dog to man. It is interesting to note, however, that the ova, larvæ, and fully developed ankylostomes of the dog are in nearly all points comparable with those of man. The differences that exist are observed at the mouth of the fully-developed worm, and refer to the size and direction of the teeth. The fully-developed worms resemble each other closely in their internal structure. Lambinet, of Liege, has infected dogs by hypodermically injecting larvæ, and in 16 to 21 days the fully-developed worms were found in the intestine,

having reached the alimentary canal by the circuitous route mentioned by Looss. It required the same length of time for the worms to become sexually mature in the intestine, when the larvæ were given by the mouth.

Stiles has described another form of the ankylostoma, which is known as the American uncinaria. This is met with in man in Texas, Brazil, and the islands of the Pacific, etc. Since the fully-developed worm is found in the chimpanzee it is believed that the parasite belonged originally to this animal.

Causes of the anæmia in ankylostomiasis.—It is since the tunnelling of the St. Gothard that Europe has awakened to the reality of ankylostomiasis and its power for harm. During one year when work was in progress at the St. Gothard, there were registered between October 1st, 1880, and September 30th, 1881, 186 cases of anæmia in miners. At this date, although Professors Perroncito and Concato of Turin, had demonstrated at the autopsy of a miner the presence of a very large number of ankylostomes in the intestine, Dr. Sonderegger of St. Gall, and Professors Pozzolo and Pagliani of Airolo still held that miner's anæmia generally was a consequence of the poor food consumed by the workmen, breathing of the vitiated air of the tunnel, and want of proper care, and that the presence of ankylostomes in the intestine was simply a coincidence and not the cause of the anæmia. In a paper recently contributed to the Paris Academy of Medicine, Manouvriez of Valenciennes has again raised the question as to whether miner's anæmia is not the result of an intoxication arising from inhalation of deleterious gases or bad air. The epidemic of anæmia that broke out, between 1778—1792, in the silver mines of Schemnitz in Hungary, was at the time considered to be the result of lead poisoning, and at the Anzin mines in France a few months ago, Manouvriez was at first disposed to attribute the malady to impure gases. In the absence of more extensive knowledge it was natural in past years to attribute the anæmia of the workers to unhealthy conditions in the mines, poverty of food, and bad

housing, but the discovery of the ankylostoma in the intestine has changed all this. Besides, as Dr. Haldane has shown, in the mines in Cornwall at least there is no impurity of the air, and yet there is miners' anæmia. The usual habitat of the fully developed worm is the upper part of the small intestine. Here the ankylostome attaches itself by its six claw-like hooks to the lining membrane of the alimentary canal from which it draws its nourishment. It was formerly held that the parasite caused anæmia by sucking the blood of its host, and certainly when ankylostomes are passed in the dejecta many of them, especially the females, are seen to contain blood. The question, therefore, is how far the presence of blood in the worm is an accident, or how far blood is essential to the nutrition of the parasite. The late Dr. Beaven Rake was of the opinion that the anæmia of affected miners was due to the abstraction of blood by the parasite. Dr. Looss, on the other hand, maintains that the worm lives upon the mucus and the material secreted by the large cells lining the alimentary canal. As it is only some of the ankylostomes that contain blood, the probability is that miner's anæmia in the chronic form is caused not so much by what the parasite abstracts from the blood as by what it imparts to it. Ankylostomes secrete materials of a toxic nature, and it is the absorption and circulation of these that probably produce anæmia. Not only is the blood thin and watery, and its colouring matter diminished, there is also observed on microscopical examination an increase in the eosinophile corpuscles.

Two main theories therefore are before us to explain the anæmia of ankylostomiasis. Against the "abstraction of blood" theory may be placed the fact that the amount of anæmia is said to be not always proportional to the number of ankylostomes present in the intestinal canal. It is not possible to estimate the number of worms present in the alimentary canal, nor do we exactly know how many ankylostomes it requires to cause symptoms. Dr. Tenholt is of the opinion that the parasite can remain in the

intestine five or six years, and it is maintained by some writers that it requires 500 worms to cause symptoms. Experimental research on the other hand suggests that miner's anæmia is not due to loss of blood, but is the result either of toxins formed by the ankylostomes and absorbed into the system, or that it is a consequence of some deranged intestinal chemistry, and therefore akin to an intoxication. Alessandri ('Policlinico,' December, 1904, p. 541) found that an extract obtained from the worms is toxic. On examining microscopically the head of a fully developed ankylostome there are observed glands at the level of the mouth. Looss tells us that the toxin is thrown out through the outermost ventral teeth into the lymph or blood stream. The secretions of these glands have a distinct hæmolytic action upon human blood *in vitro*. It may, therefore, be that this glandular secretion by inoculating the duodenal mucous membrane is a cause of the anæmia. Opinions, however, are divided upon this point. In a paper presented to the Academy of Sciences, Paris, 1904, MM. Jammes et Mandoul, of Toulouse, gave the results of their experiments, which went to prove that intestinal parasites do not secrete toxins. Their experiments were made by taking the juices obtained not from ankylostoma but from tænia and ascarides. As opposed to these we have the experiments performed by Isaac and Van der Velden ('Deutsch Medicin. Wochens.'), also of Fleckseder and Stejskal ('Wiener klin. Wochens.'), whose conclusions are that all intestinal parasites secrete toxic substances. In favour of the loss of blood theory, however, is the fact alluded to by Professor Stockman[1] that in the liver and spleen of five persons who had died of ankylostomiasis in Fiji and Ceylon he found a marked absence of iron, a circumstance which rather points to actual loss of blood from the small vessels in the intestinal wall than to destruction of red-blood cells by a poison secreted by the parasite, and circulating afterwards in the blood of the host. From the contradictory results obtained by experi-

[1] 'Brit. Med. Journ.,' 1896, vol. ii, p. 189.

ments and from opposing clinical opinions, it is clear that the last word upon this important subject has not yet been said. If anything were required to support this statement, it is the results of the experiments made by Lambinet, and published by him in the 'Bulletin de l'Académie Royale de Médecine de Belgique' of January 28th, 1905, a few weeks after this paper was written. By infecting dogs with large doses of ankylostomal larvæ several of the animals died, and at the autopsy numerous sexually matured worms were found in the ileum, not the duodenum; the intestinal canal contained a good deal of blood, the mucous membrane was softened, and many of the worms were gorged with blood. It would seem, therefore, as if in Lambinet's dogs the severity of the symptoms depended largely upon the number of ankylostomes present in the intestine and the acute character of the infection. It may be therefore only in the more chronic forms of the disease that toxins are passed by the parasite into the mucous membrane of the host.

Westphalian coal fields.—I entered the Westphalian coal fields from Bochum. This is the most important centre. Here, too, Dr. Tenholt lives, who is not only the principal medical officer of the Knappschaftsverein or Miners' Union, but is the recognised authority on ankylostomiasis in Germany. With him I visited the Elizabeth Hospital in Bochum (400 beds). A wing of the hospital, shut off from the main building, is set aside for the treatment of patients suffering from ankylostomiasis. On the occasion of my visit I found nineteen men the subject of miners' worm disease. In the hospital is a specially equipped bacteriological laboratory superintended by Dr. Nagel. Here 300 young medical men have been specially trained by Drs. Tenholt and Nagel, all of whom are now holding appointments at the various mines throughout Germany. Although coal mining in Westphalia originated about the year 1760 it was not until 1840 that the impetus was given to the industry which has made Westphalia so prosperous to-day. Some idea of the enormous demand for coal may

be formed when it is stated that in one year in very recent times 20,000 miners were introduced into Westphalia. These men came from Posen in Prussia, from Poland, Hungary, and Italy. At the present time Italian miners are taken on at German mines if they have satisfactorily passed a medical examination, but on no account is a miner from Hungary accepted. It is not known how ankylostomiasis found its way into the Westphalian mines. Some authorities state that it was introduced by brick-makers from Cologne, others that it was brought by Italian miners who had worked in the St. Gothard tunnel, but the fact that Italian workmen are received, and on no account Hungarian, rather points to the Hungarians as the people upon whom the blame is thrown. While the cause of the outbreak of ankylostomiasis in Westphalia is not known there is an almost general consensus of opinion that the disease has spread in the mines through the use of water-sprays compulsorily ordered by the Government on account of the fiery character of the coal dust and the numerous explosions that had taken place attended with loss of life. Water-spraying became general in 1900. In the valley of the Ruhr, where most of the coal mines are situated, the number of cases of ankylostomiasis has been as follows:

1896	=	107	1900	=	275
1897	=	113	1901	=	1030
1898	=	99	1902	=	1355

Is the enormous increase in the number of cases of miner's worm disease since 1900 only a coincidence, or is it a consequence of the introduction of water-spraying into the mines? On this point it is interesting to remark that some months ago permission was granted by the Government to suspend the water-spraying so as to ascertain whether the drying of the roadways would diminish the number of cases of ankylostomiasis. As the results have not been satisfactory the permission has been cancelled in at least three of the mines. It is just possible, of course, that relaxation of the rules as to sprinkling with water came too late and that the damage had

already been done, but as the experiments are still in progress it is well to reserve an opinion upon this point.

The epidemic of ankylostomiasis has been a serious financial matter for Westphalia. In attempting to grapple with the disease the Miners' Union and the colliery owners have conjointly spent £100,000 within the last two years.

With Dr. Tenholt I went to the Lothringen mine, about three miles from Bochum. Of the 2000 men employed in this pit 1600 work underground. Ankylostomiasis broke out in the Lothringen mine in 1885, but it was not until 1894 that Dr. Tenholt was appointed medical head of the Miners' Union. Since that date he has had from this pit alone 4000 miners through his hands suffering from ankylostomiasis. Three years ago 72 per cent. of the underground men were affected, at the time of my visit only 8 per cent. Descending to a level 1000 feet below the surface, I noticed here and there along the sides of the main ways the covered iron boxes with concealed iron pails, which are the closets used by the miners. At places these receptacles were stationed in recesses in the rock and screened off by sacking. The soil and broken stone around these boxes were often saturated with slaked lime so as to disinfect the boots of the miners and any fæcal matter that might be deposited thereon. The pails are removed to the surface every day, emptied, and rinsed with boiling water. In the muddy water and sludge that lie here and there by the side of a roadway myriads of the larvæ of the ankylostoma are found. They feed upon any organic matter that may be present, and they attack the wooden props which support the roof of the mine. As these larvæ and other parasites known as rhabdites cause the props to become wet and rotten, they become a source of danger to the workers and entail a heavy financial expenditure upon the owners.

On the road from the Lothringen to the Erin mine, two or three miles further away, we passed several coal miners. They looked so clean and were dressed so appropriately that

one would have thought they were going to, instead of, as was the case, returning from work. In this part of Westphalia you never meet on the road a begrimed coal miner, as in Britain, simply because every miner on coming out of the pit has a warm bath and general wash, and he puts on his walking clothes, leaving his wet clothes in the dressing-room, where he finds them next morning, dried and warm. A finer set of baths, wash-houses, dressing-rooms, and lavatories than those at the Erin mine it would be impossible to find anywhere.

At most of the large pits there is a small hospital, which is set aside for the treatment of patients suffering from ankylostomiasis. The Erin hospital has fifty beds, of which on the date of my visit twenty-five were occupied. Dr. Perner, who is in charge of the hospital, tried to ascertain how long larvæ could live in a disused part of the mine, and he found eight months afterwards that they were just as brisk and active as ever. It is sometimes stated that the ankylostoma may develop from an ovum through a larva into a fully developed and sexually mature worm outside the body of a mammal, but there is no proof of this. It would therefore appear that for the larva to develop into a worm the organism must reach the alimentary canal of a host.

Hungary.—At Sopron-Brennberg I was the guest of Dr. Goldman, the medical officer to the mines. Seven years ago, when he received his appointment, fully 70 per cent. of the men in the colliery were suffering from ankylostomiasis; to-day there is only 30 per cent. A few years ago, when the epidemic was more widely spread, the affected miners suffered severely in their health.* They were extremely anæmic and languid; they were so tired and their feet so swollen that they could scarcely walk. By degrees the virulence of the outbreak subsided, so that now the symptoms are milder than they used to be. The temperature of the Sopron colliery is extremely high; in some places it runs up to 104° to 106° F., and at other places where the ventilation is not good, and where water-spraying is

not in use, it may rise to 170° F. The coal is a kind of pitch coal and very fiery.

It is not known how ankylostomiasis got into the Sopron mine. Hither, as to Westphalia, Italian miners came on the completion of the St. Gothard tunnel (1890-92), and they are blamed for having brought it; but Mr. Rudolf, the manager of the mine, informed me that to his knowledge ankylostomiasis existed in Hungary as far back as 1864. During 1903 Dr. Goldman treated 400 cases of ankylostomiasis at Sopron.

Bohemia.—Until recent years Bohemian mines were free from ankylostomiasis. The first cases were detected in March, 1903, in the persons of three miners, neither of whom was strikingly anæmic, but in whom eosinophilia was very pronounced—the proporton of eosinophiles being respectively 10 per cent., 13·7 and 20·3 per cent., a circumstance which suggests that the toxins which cause hæmolysis are different from those which bring about an increase in the number of eosinophiles.

Belgium.—Ankylostomiasis was first detected in Belgium, in the coal fields of Liége, by Firket, in 1884. In the following year it was ascertained that many of the patients were miners who had worked in the St. Gothard. Between 1884 and 1895, 92 cases of ankylostomiasis were treated in the hospital at Liége, but shortly after this the number of cases throughout the country became so large that a Commission was appointed to deal with the subject. Although certain sanitary recommendations were made by this body, the miners themselves refused to allow them to be put in force; and as a consequence the disease spread, with the result that in November, 1903, there were 7000 miners around Liége who were suffering from ankylostomiasis. At Liége there is now a miners' dispensary with a well-equipped bacteriological laboratory, where Dr. Lambinet has carried out several interesting and useful experiments.

France.—Miner's anæmia has long been recognised in France. At Anzin, where it was prevalent at the com-

mencement of last century, it was attributed to defective ventilation of the mines and to the inhalation of harmful gases, to the hard character of the men's work, and the absence of sunlight. In 1882, Perroncito, of Turin, recognised the parasitic nature of the anæmia of the miners at St. Etienne. Ankylostomiasis is known to have recently existed in the mining districts of the North of France, the Pas-de-Calais, etc., where 90,000 men are employed; also at St. Etienne and Lens, Anzin, Escarpelle and Bruay. So far as the valley of the Loire is concerned, the mines of Rive-de-Gier and of La Peronnière seem to have been the worst. The subject has been frequently reported upon to the French Government by several commissions. In France, as in Germany, it has been noticed that improved ventilation of the pits and diminished humidity have been followed by a reduction in the number of cases of ankylostomiasis.

South Africa.—Miners have been drawn to South Africa from all the world over. The latest importation in bulk is that from China. The native population of British Central Africa has been known to suffer from ankylostomiasis for many years back. Daniels found ova in the fæces of 25 per cent. of the people. There is no epidemic of ankylostomiasis in South Africa at the present, but the disease is known to have existed a few months ago in some of the mines near Kimberley. Wherever the supply of local labour is difficult to obtain in any large mining centre there is a risk of the malady breaking out, as occurred in Westphalia ten to fifteen years ago. The importation of Chinese labour therefore into South Africa is not without its danger from an ankylostomiasis point of view, and ought to be met by continued watchfulness and examination on the part of the medical authorities. No coolie known to be infected should be allowed to descend into a mine.

Great Britain.—For much that we know of ankylostomiasis in Britain we are indebted to Dr. J. S. Haldane and Dr. A. E. Boycott. In their paper on "Ankylostomiasis in

England,"[1] mention is made of the outbreak of the disease in Cornwall, and particularly in the Dolcoath mine. Incidentally it may be mentioned that it is only in the deep metalliferous mines of this country, where the temperature is high, that ankylostomiasis has been detected. For many years before it was recognised at Dolcoath it had been usual to attribute the anæmia from which the men were suffering to the heat of the mines, the exertion at work, and inhalation of impure gases. Dr. Haldane has shown that on the whole the air in the mine is extremely pure. So far as can be ascertained the disease broke out about nine years ago, and at the present time it is not making progress. There are 700 men employed at Dolcoath. Dr. Haldane gives a list of 57 men suffering from miner's anæmia, in the fæces of nearly all of whom the ova of anklyostoma were found. Out of Cornwall the malady is not known to have any existence in mines. Professor Stockman, of Glasgow University, has reported the occurrence of the disease in a hospital patient, a young miner, but as he had but recently returned from India, where he had been serving in the army, the probability is he contracted the disease abroad.

Symptomatology.—The term miner's anæmia suggests that anæmia is a well-marked physical sign, and that in all persons who harbour the worm bloodlessness is a striking feature. Such is not always the case. A miner may be the subject of ankylostomiasis and yet have neither signs nor symptoms. This is a point to be remembered; for although such an individual may not exhibit symptoms and may apparently not be suffering in his health, he is yet extremely harmful, since it is possible for one person harbouring the worms to infect a whole mine. It is probably when an epidemic breaks out in a mine for the first time that its effects upon health are most pronounced. Either the parasite afterwards becomes less virulent or the individual becomes more resistent or immune to it. Besides, as the worm cannot reproduce

[1] The Journal of Hygiene,' January, 1903, p. 95.

itself in the alimentary canal, it will disappear in the course of time and the patient recover, provided there is no reinfection. When symptoms develop they are such as are usually met with in aggravated anæmia, viz. shortness of breath on exertion, palpitation, extreme tiredness and a sense of weakness in the limbs, œdema of the feet and legs, loss of appetite and distaste for food, pain after eating, and irregularity of the bowels. It is seldom that blood is passed with the fæces. Serous effusion may take place into pleural and peritoneal cavities. Apart from complications ankylostomiasis is rarely a fatal disease. Its baneful effects are to be measured, not so much by the number of deaths it directly causes, as by the amount of general distress and the complete and lengthened unfitness for work which it induces.

Diagnosis.—Short of the detection of ova in the fæces or the presence of worms therein after the administration of an anthelmintic no absolute diagnosis is possible. Considerable assistance, however, may be obtained, as Dr. Boycott has shown, by an examination of the blood. Not only is there a deficiency of hæmoglobin with a diminution in the number of red blood corpuscles, but there is a notable increase in the number of eosinophile corpuscles. In the case of a suspected miner the blood should be examined microscopically and stained, and in the event of there being eosinophilia the fæces ought to be examined for ova. In a healthy man the percentage of eosinophiles among the white corpuscles of the blood varies from 0·5 to 5, but in infected miners Dr. Boycott frequently found that the percentage rose to 18.

Treatment: preventive and curative.—The disease is due to the presence of a parasite in the intestine. Now that the life history of this parasite is pretty well known, ankylostomiasis ought to be prevented. Possibly one infected workman, as already stated, can infect a mine. Since the ova of ankylostoma are killed by cold or rather do not hatch readily in low temperatures, and also require plenty of oxygen, the transformation

of ova into larvæ in mines can to a large extent be retarded or prevented by reducing the temperature by free ventilation, and by keeping the atmosphere and the pit as dry as possible. Temperatures in a mine ranging from 68° F. to 90° F. are extremely favourable to the hatching of the ova, and these ova, as already stated, must have moisture. Low temperatures do not always prevent hatching of the ova, for Lambinet of Liége found that the ova of ankylostomiasis could be hatched at lower temperatures than 60° F., and Haldane states even as low as 48° F. One reason why the ova of ankylostoma do not become transformed into larvæ in the human intestine is that the temperature therein is too high and oxygen is not present in sufficient quantity. It is the dryness of British mines and their low temperature, compared with those on the Continent, that to a large extent explains why the disease has not got an extensive hold in this country. Some of the deep metalliferous mines of Cornwall, to and from which miners are coming and going from all parts of the world, have become infected, and it remains to be seen whether the malady can be prevented spreading from these to other mines or can be exterminated altogether. The ova of ankylostoma exhibit considerable resistance to the action of antiseptics, so too do the larvæ. In Westphalia I had the opportunity of seeing Dr. Tenholt carrying out in one of the disused galleries of the Lothringen pit some experiments, viz. the sprinkling of the floor and the sides of the gallery, also sprinkling of the wooden props upwards to the extent of three feet, with strong solution of quicklime. In his hands the results had proved satisfactory.

Formerly it was held that the larvæ reached the duodenum by miners eating with unwashed hands. It is desirable that as regards taking food the miners should preserve the greatest cleanliness, and that there should be here and there in the mines filtered water for drinking, and plenty of water from a healthy source for washing purposes. Dr. Looss' experience and experiments have shown that the larvæ can penetrate the skin and by a

circuitous route reach the intestine. The naked handling therefore of polluted soil and of infected wooden props should be guarded against as far as possible.

Time will not allow of me doing more than briefly indicating the lines that should be followed, and of alluding to some of the anthelminthics and intestinal antiseptics that have been employed. The one thing above all others is to clear the intestine, if possible, of any ankylostomes that may be attached to its mucous membrane. This is not always an easy matter. If left alone in a patient who is of fairly good health, not markedly anæmic, and who is taking his food well, the worms, since they cannot reproduce themselves in the human alimentary canal, will, if there is no further infection, die off in course of time. Dr. Tenholt, however, holds that the worms can live in the intestine for five or six years, and as during that period they can do a considerable amount of harm, it is well to get rid of them. Calomel and aperients must be given. In Cornwall large doses of thymol have been administered, with results of a more encouraging nature than have been obtained either in Westphalia or Hungary. On the Continent all sorts of anthelminthics have been tried, but no absolute reliance can be placed on any particular drug. Filix mas has been pushed, and with varying success. In a large number of cases its administration has been followed by very unfortunate results. In this country we are not familiar with the toxic effects of Filix mas, but in Germany and Hungary the drug has caused blindness, which in many instances has been permanent. Goldman, of Sopron-Brennberg, found in some of his patients that the amblyopia lasted only for eight days and then disappeared, but a few of Tenholt's patients have remained blind. Sidler-Huguenin reports that of seventy-eight patients that were poisoned by Filix mas twelve died. Katayama and Okamoto found that 32·5 per cent. of their cases of ankylostomiasis treated by Filix mas suffered from amblyopia. Opinions are divided as to what the cause of the blindness is. It may be purely toxic, and therefore

central and temporary; there may be a neuro-retinitis, as some maintain, or thrombosis of the central artery of the retina, the effects of which are permanent. Alcoholic and rheumatic subjects are said to stand Filix mas badly. So far as we know there is no specific remedy for ankylostomiasis.

DISCUSSION.

Sir WILLIAM BROADBENT, the Vice-President in the Chair, congratulated Dr. Oliver on the interest and thoroughness of the paper he had communicated. It threw many important side-lights on medico-social problems. The question of the production of the anæmia in ankylostomiasis seemed to be still unsettled, and its solution might yield valuable information on the causation of anæmia in general. In view of the fact that quite a large number of the parasites were required to produce symptoms, it seemed to him not impossible that the anæmia might in this case be due to the abstraction of blood by multitudes of the ankylostomes.

Sir PATRICK MANSON considered the paper was a very timely as well as interesting one. Epidemic ankylostomiasis was as likely to be prevalent in the future in England as it is at present in Germany. The paper helped to show that the healthiness of a country was not a function of climate but of the presence of parasites. A manipulated ovum of ascaris lumbricoides was very much like the ankylostomum ovum. The route for the introduction of the parasite was very elaborate; the purpose of the roundabout route was probably to confer on the embryo powers of resisting the action of the gastric juice. Looss had pointed out that the mature worms were developed only from those embryos that did not pass direct into the stomach. The most important means of preventing the disease was the education of the miners in the life history of the ankylostome, thereby ensuring their intelligent co-operation. The ovum, unless freely supplied with oxygen, perishes; the Chinese, unconsciously acting on this principle, stored excreta in cemented tanks until they fermented and decomposed, thus securing destruction of the ova. Eosinophilia, as an evidence of ankylostomiasis, was of little value, as the infection by intestinal parasites was usually multiple. The only certain diagnostic was the microscopic examination of the stool. His experience of thymol in large repeated doses such as a half drachm every two or three hours for three or four doses, the last dose with castor oil, had been altogether favourable, but no thymol solvent such as alcohol must be given at the same time. Areca nut, which he had not himself used, would probably be a useful anthelminthic against this parasite, and should be tried. The cases were not so rare as was supposed; he had himself seen four or five of late years in Europeans who had lived abroad, some of which had been confounded with pernicious anæmia.

Dr. OLIVER, in reply, said that the worms frequently shifted their position in the intestine from place to place, for in

Lambinet's experiments on dogs blood had been seen oozing from points of previous attachment. There was no certainty that Britain would remain free from an outbreak of the disease, hence education of the miners as to the nature of ankylostomiasis and how it spreads should be a matter of the first moment. In Germany and Hungary less importance was attached to eosinophilia as a sign of the disease than in this country. Although observed in patients the subjects of intestinal parasites other than the ankylostoma duodenale, and in maladies not of intestinal origin, Dr. Oliver was of the opinion that eosinophilia is a sign of considerable importance in miner's worm disease. In suspicious cases the discovery of eosinophilia should be followed by a microscopical examination of the fæces for ova.

SOME CHANGES WHICH TAKE PLACE IN THE LUNGS AND PLEURA

CHIEFLY IN

PULMONARY TUBERCULOSIS

AS SHOWN BY SKIAGRAPHY

BY

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For two reasons it has been decided to exclude the evidence afforded by radioscopy as to the changes which take place in the progress of lung disease. In the first place, the field covered by radioscopy is so large that it cannot conveniently be brought within the scope of this paper. And in the second place, its incorporation would entail the use of a large amount of evidence which is incapable of demonstration at a meeting such as this. Except, therefore, on those very few occasions on which a reference to the appearance seen on screening is absolutely necessary for the elucidation of the text, radioscopy will not be referred to.

It is convenient to begin by referring to the radiographic appearances presented by those pathological

conditions of the pleura more commonly found associated with pulmonary tuberculosis. In presenting to you a picture showing the following simple experiment, I trust I shall not be held to be dealing with a matter of so elementary a nature as to be unworthy of your attention. Two ice-bags were taken, and into each was poured an equal quantity of serous fluid which had shortly before been obtained by aspiration from a case of fluid pleurisy. From one of the bags air was afterwards, as far as possible, extracted, but in the case of the other it was allowed to remain. The bags were then examined by screening, and afterwards the skiagrams now shown (1) were taken. The different appearance presented by those bags is most interesting. Your attention is arrested by the fact that whilst that portion of the bag (A) which contained fluid only is represented by a dark shadow, whose upper border gradually merges off into a clear area above, the appearance is different in the case of (B), which contained not only fluid but air in addition; for there you will observe that, unlike the appearance obtaining in the case of (A), a clear line of demarcation exists between the shaded area below, corresponding to fluid, and the clear portion above, in which only air is present. And again, whilst the upper level of the fluid in (A) shows a somewhat irregularly curved form, it is not so in (B). There the line of demarcation is absolutely horizontal. Moreover, the edges of the shadow in (A) are seen to be drawn up to a considerable distance above the central portion of the curve. It is not so in (B), whose clean-cut horizontal line extends across from edge to edge. *In short,* in the first bag, where the upper portion is not distended by air, the force of capillarity has full play and tends to draw the fluid up in the form of a wedge-shaped column, which eventually tapers off and disappears. This accounts alike for the curve at the edges and also for the gradual diminution in the intensity of the shadow from below upwards. In the second bag, which contains in addition to fluid, air at atmospheric pressure, owing to the sides of

the bag being kept apart by the air, the action of capillarity is reduced to a minimum, and, in consequence, the fluid is allowed to remain in accordance with the laws of gravity in a horizontal position.

Analogy with Pleural Conditions.

The analogy existing between a rubber bag containing fluid, and a closed serous sac, such as the pleura, with an imprisoned effusion, was considered sufficiently close to warrant the inference that the phenomena observed in the case of the bag (A) were not unlike those which one might reasonably expect to find in a case of fluid pleurisy; and that those present in the case of bag (B) might be found to closely resemble the appearance present in the case of hydro- or pyo-pneumothorax.

The skiagram now shown is that of a young naval cadet, whom I saw for the first time in September, 1903. Your attention is drawn to the presence on the left side of a shadow whose upper border is at once level and fairly clearly defined. This shadow may be due to one of two conditions. In the first place, its presence may be accounted for by consolidation in the lung, as in the case of basal pneumonia or actinomycosis, which not unfrequently has this distribution; in the second place, it may correspond to fluid in the pleura. The fact that the right border of the heart, as shown, is displaced towards the right side suggests the presence of fluid.

As a means of discrimination between those two conditions, the position of the patient was altered and the body inclined from the vertical laterally until it stood at an angle of forty-five degrees. The chest was again examined. The appearance then noted is shown in the *skiagram* (2) now before you. Your attention is particularly directed to the fact that notwithstanding the alteration in the position of the patient from the vertical line, not only is the upper border of the shadow plain and distinct, but it remains perfectly horizontal. The heart now appears

to be displaced towards the right side to the extent of over two inches. If any *further evidence* were required to convince one that the shadow observed was due to the presence of fluid in the pleural cavity, which also contained air, it was forthcoming when, on screening the patient, the presence of quick rhythmical wavy movements at the upper border of the shadow was at once recognised, movements which on closer observation were seen to correspond to the movements of the heart. The case being, then, plainly one of hydro- or pyo-pneumothorax, operation was decided on. On opening the chest next morning, I was rewarded by finding muco-purulent fluid, a pint and a half of which was freely evacuated. The pleural cavity was washed out with a solution of peroxide of hydrogen, and efficient drainage established. Twenty-one days after operation the wound was allowed to close, and the patient made an uninterrupted recovery. He returned to the Navy fourteen weeks after operation apparently quite well. Immediately before doing so his chest was again X-rayed, and the next *skiagram* (3) shows you the appearance then present. Its principal interest for us lies in the following points. Firstly, the shadow present, although dark, is no longer a uniform one, but is relieved at points by clear areas, due probably to the readmission of air into the lung at those parts. Next, that the distance separating the ribs from each other upon the affected side, is generally less than that between those on the sound side. No doubt this is due to the presence of an imperfectly expanded lung upon the one side, contrasted with the compensatorily dilated lung upon the other side. And lastly, that the distance from the centre line to the periphery on the left side is less than the corresponding distance on the right side, shown in the diagram to be about half an inch, and in all probability is due to the same cause.

After having undergone six months' service at sea, the patient returned on furlough in June, 1904, when I was fortunate in again having an opportunity of examining his chest. Two skiagrams were taken—one from the front

and one from the back. The one here shown (4) was taken from the front. By comparing the breadth of the two sides of the chest, which are seen to be practically identical, and the absence of shadow in the vicinity of the diaphragm upon both sides, it will at once be seen that complete absorption of fluid and perfect re-expansion of the lung has taken place. Such, then, are the appearances shown by a case of pyo-pneumothorax which has run a favourable course.

The next two skiagrams shown were also taken from a case of pyo-pneumothorax. But here the result was altogether different. It is true that in this instance the patient also recovered his health and strength; but, unlike the previous case, in this one the condition of the lung left upon the affected side was altogether different from that upon the sound side, for in it re-expansion of the compressed lung never took place. This slide shows the condition present two years after operation and fourteen months after the wound in the chest-wall had closed. Whilst upon the sound side, as evidenced by the unusually light appearance which it presents, considerable compensatory emphysema is present, that collapse of the other lung is "absolute" and "complete" is evidenced by the very intense shadow shown.

We pass now to consider the appearances shown in two cases of *simple fluid pleurisy*. In both instances the diagnosis, based upon the X-ray appearances, was afterwards confirmed by the withdrawal of fluid. These cases will be found to possess certain points in common, and certain points of contrast. In the skiagram (5) now before you attention is directed to the former, namely, to those points which they share in common:

Firstly.—The *heart-shadow* appears upon the right instead of upon the left side. The amount of displacement in this case is nearly three inches.

Secondly.—The upper border of the shadow upon the left side caused by the presence of fluid differs from that of the case of pyo-pneumothorax already shown, *firstly*, in

not being horizontal but on the contrary curved, the curve running upwards at the outer and inner sides, so as to produce a concavity in the centre; and, *secondly*, in not being clearly defined, but, on the contrary, vague and indefinite in outline.

Thirdly.—As in the case of pyo-pneumothorax, there is in both cases a relatively light area above the shadow.

A diagnosis of left-sided fluid pleurisy was made and confirmed by the withdrawal next day of thirty ounces of straw-coloured fluid. Before this slide is removed, you are asked to give particular attention to the appearance present in the upper half of the left lung above the level of the fluid. For, as will hereafter be shown, it is an appearance which is not always present in cases of fluid pleurisy. I refer to the relative clearness shown in that part when compared with the portion below corresponding to the fluid area. By comparison with the lower, which is dark, the upper part of the chest is practically clear.

The next *skiagram* (6), taken from the same case, after fluid had been withdrawn, furnishes two interesting points of contrast to that shown by the skiagram taken before paracentesis was performed. In the first place, the shadow running perpendicular to the spine already referred to no longer occupies one half of the right side of the chest. At the level of the tenth rib, instead of being about three inches it is only one inch and a quarter from the middle line, and on the left side the semi-level indefinite shadow has disappeared from view, and its place has been taken by one which runs abruptly downward and outward, corresponding to the left border of the heart. In fine, the fluid has practically disappeared, and the heart, from being displaced towards the right, has returned to the left side and now occupies approximately its normal position.

The next case, also one of fluid pleurisy, has been chosen for the purpose of illustrating an appearance sometimes present in a case of fluid pleurisy and altogether different

from that already described. Whilst the heart is again shown to be displaced towards the right side, the absence of any upper limit to the shadow on the left side leaves one quite at a loss to say how far it owes its existence to the presence of fluid and how far to other factors. At first sight one might be disposed to infer that the whole of the left pleural cavity is filled with fluid, but experience of the relative amount of cardiac displacement present in cases of very large effusions leads one to the conclusion that the effusion here is not a large one. If it had been, the heart would undoubtedly have been displaced a much greater distance towards the right side than is here shown to be the case. The uniform dark shadow present upon the left side in all probability owes its existence to *two* factors. In the *first* place there is undoubtedly fluid present, but it is inconsiderable in amount. In the *second* place a certain amount of collapse has taken place, but it is limited to the area of the fluid. The mottled shadow present above the level of the fluid is due to the presence of an apical lung lesion which was present before the pleurisy appeared. This then represents the appearance so commonly met with where a fluid pleurisy of moderate amount has supervened upon the same side as an apical tubercular lesion and which is apt to be erroneously interpreted as indicating the presence of a very large effusion. In this as in the previous case about twenty ounces of clear straw-coloured fluid were withdrawn and the patient made an uninterrupted recovery. A skiagram taken soon after paracentesis had been performed has this special point of interest for us: it shows by the clearing which has already occurred the re-expansion of the lung taking place, and as might have reasonably been expected is doing so from above downwards. The next *slide* from a skiagram, taken three months later, shows these points even more clearly.

I have purposely chosen these two cases of fluid pleurisy representing distinct types of appearance commonly met with in chest skiagraphy. My reason for

doing so is twofold. In the first place I wish to draw attention to the fact that where the presence of fluid is suspected on the same side as an apical tubercular lesion we ought to look for a shadow which is not delimited by the level of the fluid, but one which extends almost if not quite to the extreme summit of the chest; and when a shadow of this extent is recognised it must not forthwith be inferred that a very large quantity of fluid is present until we have, amongst other things, ascertained the extent to which the displacement of the heart towards the opposite side has taken place. My other reason for exhibiting examples of these contrasted types is to raise the question—for I understand it is still a question—of what is the behaviour of that portion of the healthy lung which lies above the level of fluid present in a surrounding pleura. I have put myself to the trouble of consulting the works of twelve of the best known writers on lung diseases with the object of ascertaining if anything approaching unanimity obtains in the opinion held by these authorities upon this point, and I find that no such harmony exists. Some hold that the tension in this portion of the lung stands at a lower level, and that it contains less air than is present in it in normal health. Others that the lung becomes partially compressed, and that the space previously occupied by air comes to be taken up by patches of congestion; and there is still a third group who maintain that total collapse of the whole lung takes place, and therefore that that portion lying above the level of the fluid contains no air whatever. Where such diversity of opinion exists it may be assumed that any evidence which a study of the X-ray appearances, which is present in such cases, may be able to contribute will be welcome.

It is, I take it, fairly well settled amongst men who devote a large portion of their time to the practical application of chest skiagraphy that the lightness of an appearance present is in direct proportion to the amount of air contained in the area under observation. Thus

hyperaerated conditions, such as emphysema and pneumothorax, are found to be represented by an extremely light appearance on the screen, and areas devoid of air, such as are present in collapse, are always represented by a very dense shadow. Now, if it were the case that that portion of a lung which lies above the level of a moderate sized pleural effusion were in a state of collapse, then not only that portion of the print corresponding to the fluid, but that representing the portion of lung above the fluid would be represented by darkly shaded areas practically undistinguishable from each other. Now cases such as the former of the two shown—cases of uncomplicated fluid pleurisy—in my experience never show this shadow at the summit of the chest; indeed, not only is the area above a moderate sized effusion not represented by a darker appearance, but in many cases it is represented by a lighter appearance than that presented by the healthy lung upon the other side. And if one is to be guided by the evidence which skiagraphy affords in such cases, not only is the condition of the lung above the level of the fluid *not* that of collapse, but it is one in which this portion of the lung contains as much, if not more, air than normal, and which closely resembles compensatory emphysema. I have not found amongst writers who have contributed to the literature of this subject any who have suggested that this condition is present. Nevertheless, in view of the very suggestive evidence which this method of examination affords, evidence which leads one to a conclusion at variance with that which has been previously accepted as sound, I venture to submit that the premises from which that conclusion has been drawn merit reconsideration.

There is one other point in this connection which may be profitably considered. We are told that in cases of fluid pleurisy, *firstly*, that the intercostal spaces are largely increased in size, and *secondly*, that cyrtometric measurements invariably show the circumference to be greater upon the side containing the fluid than it is upon the healthy side. If this view is accurate,—and where prac-

tically all authorities agree as they do here upon its correctness, its accuracy may be assumed,—then it seems at once to follow *firstly*, that the spaces separating adjoining ribs should be greater on the affected side than they are upon the sound side, and *secondly*, that the measurements from the mid-sternal line to the outer border of the ribs should be greater than the corresponding measurements upon the other side. This skiagram (7) is one of left-sided fluid pleurisy of moderate amount. In view of what has been said it is particularly worthy of note that the spaces here separating adjacent ribs upon the affected side *are* NOT greater, but are less than those separating adjoining ribs upon the sound side. And further that the measurement from the mid-sternal line to the edge of the chest upon the affected side is not greater, but is less than that upon the sound side. It is easy to conceive that intra-pleural pressure by pushing the soft intercostal tissues outwards furthers the approximation of the ribs to each other; but how a state of affairs whereby an increase of the circumference measurements is accompanied by an apparent diminution in the radial measurements is produced is not quite so plain. It is held by some that the lower portion of the sternum moves over toward the healthy side. In so far as the central point in our skiagram is that of the middle of the spine, on this hypothesis, a diminution in the apparent radial diameter is at once explained. If, however, it is not the case that a pushing over of the lower portion of the sternum takes place, then the phenomenon may be explained by the establishment of compensatory emphysema with consequent upward and outward rotation of the ribs on the sound side causing an increase to take place in the transverse measurements upon that side. The disparity of the measurements would, upon this hypothesis, be explained, not by a diminution having taken place in the width of the side containing fluid, but by an increase having accrued in the diameter of the healthy side. A *third* suggestion is that the lower part of the sternum is moved

forwards and upwards by the compensatory process already alluded to, and thereby operates by converting the shape of the chest from that of an ellipse to that of a sphere. This explains the establishment of an increase of the circumferential alongside of a diminution in the diametric measurement as shown in this skiagram. Whatever the correct explanation is, and about that no doubt diversity of opinion obtains, the *practical point* to be noted is this, that in skiagraphing a case of fluid pleurisy of moderate amount, uncomplicated by an apical lesion, one must be prepared to find an apparent diminution present alike in the measurement of the intercostal spaces and in the transverse measurement of the affected, as compared with the sound side of the chest.

I have already shown skiagrams of two cases in which, as the result of intra-pleural pressure, pulmonary collapse has taken place. In both of the cases shown the shadow was a very dark one. I need hardly recall the fact that there is another condition of intra-pleural pressure which may produce collapse, and in that condition the appearance produced is altogether different from that caused by the presence of fluid in the pleura. I refer to collapse produced by pneumothorax. In this skiagram (8) you will readily recognise in the extremely light appearance upon the left side, the unusual depth of the intercostal spaces and the presence of complete dextrocardia, the typical appearance of a left-sided pneumothorax. The position of the collapsed lung is represented by a shadow present upon the left side, a shadow whose form and size are not unlike those produced by the heart upon the right side. From those already seen, the shadow differs in not being uniform, but, on the contrary, in presenting a mottled appearance, and instead of being almost black, it differs but little in density from its immediate surroundings. No doubt the presence of a thick column of air intervening between the chest-wall and the lung tends to relieve the darkness of the shadow which would otherwise be present. In the cases of collapse due to fluid, no such air is present

between the lung and the chest-wall; hence its relative density compared with that here shown.

Dry Pleurisies.

The skiagram which is now before you is that of a case which was believed to be one of right-sided dry pleurisy. The history pointed strongly to that view being the correct one, and the complete absence of sputum throughout supported that belief. Its appearance affords one an opportunity of alluding to the outstanding points of difference between the skiagraphic appearances produced by intra-pulmonary infiltration and by dry pleurisy respectively. *Firstly*, the appearance of the ribs here is symmetrical, and there is a complete absence of anything of the nature of "roof-tile" arrangement present upon the affected side. The reverse would undoubtedly have been the case had a shadow of this extent been due to intra-pulmonary disease. *Secondly*, the heart is seen to be practically in its normal position, maintained there by two equally expanded lungs. Had the condition present been that of an intra-pulmonary infiltration, there would have been a marked tendency to dextrocardia. *Thirdly*, the breadth of the right chest as measured from the middle line to the outer border of the ribs is as great as that of the left chest measured in the same way. Intra-pulmonary tuberculous disease of the extent corresponding to the distribution of the shadow would be revealed by the presence of narrowing of the affected as compared with the non-affected side. Such, briefly, are the leading points, outside the physical signs and the clinical history, that enable one to state with confidence that the condition here represented is one of dry pleurisy, and not one of intra-pulmonary disease. But there is another point which is worthy of notice in this connection, namely, the difference in the appearance shown at a later date after resolution has taken place, compared with what is usually seen in a lung lesion of similar extent. This is well shown in the skiagram now before you, taken from the same case. The interval which elapsed between

the taking of the previous and the taking of this skiagram was twelve months, during the latter half of which the patient had followed his usual occupation, to all appearance in enjoyment of perfect health. The special interest this skiagram has for us lies in the facts—*firstly*, that the shaded area corresponding to the thickened pleura on the right side has largely cleared up; *secondly*, that in doing so it has not appreciably disturbed the position of the heart; *thirdly*, that the position of the ribs has remained unaffected; *fourthly*, nor has the disappearance of the shadow been followed by any signs of the establishment of compensatory emphysema of the other lung. By way of bringing out these points of contrast more clearly, the two skiagrams, one showing the condition twelve months before and the other showing it twelve months afterwards, are presented upon the same slide. As in the case already shown, it is to be noted that here, again, clearing takes place more rapidly towards the base and apex of the lung than it does in the centre. These have been shown as a type of what is commonly seen in cases of dry pleurisy and as illustrating the value of skiagraphy in helping one to accurately diagnose the presence of a thickened and inflamed pleura as distinguished from an infiltrated lung. But they also point to the fact that in certain cases during convalescence the pleura does not necessarily remain permanently thickened to any great extent, but, on the contrary, tends to resume its normal consistency. I am fortunate in being able in the next two slides to show you the appearances presented by a case of infiltration of the right lung in its upper half accompanied by thickening of the left pleura, taken at an interval of slightly over twelve months. In this (9) case you will notice that there is a shadow present upon both sides, and that that upon the left side is more extensive than the one which is shown on the right side. If these shadows had been due to the same cause—for example, to intra-pulmonary infiltration—in both instances, then the behaviour of the ribs on the two sides would have been similar in nature, though differing in degree. Thus we

would in that case expect to find a small amount of "roof-tile" arrangement obtaining in the ribs over the smaller lesion on the right side and a much more marked amount of "roof-tiling" in the ribs over the more extensive lesion upon the left side. In this, however, we are disappointed, for on examining the picture closely we find the contrary to be the case. Whilst a definite amount of approximation of the ribs towards each other has taken place upon the right side, on the left side their relationship has not suffered any disturbance from normal. In regard to the position of the heart in this skiagram the right border is seen to occupy a position as measured on the skiagram of two inches from the middle line (*a*). The next *skiagram* (10), taken twelve months afterwards, shows *firstly*, that the shadow on the left side, believed to be due to thickened pleura, has to a very large extent cleared up—in this case from below upwards; *secondly*, that some clearing, but to a very slight degree, has taken place in the shadow—believed to be due mainly to intra-pulmonary lesion upon the right side; *thirdly*, that the tendency to "roof-tiling" of the ribs over this lesion has been increased rather than diminished; *fourthly*, that the cardiac displacement towards the right side—that is, towards a contracting lung lesion—has been increased according to measurements on the skiagram from 2 to $2\frac{3}{4}$ inches approximately. For purposes of rapid comparison the left sides are now shown on this slide and the right side on the next one.

Before leaving this section of the subject I venture to place before you a slide which at first sight may be mistaken for a case of extensive left-sided disease with a right pleural effusion. The case is one of actinomycosis involving the left lung and pleura, mainly on the right side.

The outstanding point of interest is that, unlike tubercular disease, the disease has at first attacked the base and is apparently progressing from below upwards.

It is next proposed to deal with some purely lung con-

ditions, and before doing so it has been thought advisable to refer, and that in the briefest possible manner, to some of the points, in addition to those already alluded to, which enable one when in doubt to distinguish between shadows cast by pulmonary and by pleural conditions respectively.

Firstly.—Cardiac movements communicated to adjoining fluid within the pleura when air is also present produce at its surface a rippling wave-like motion which moves synchronously with the heart-beat and is easily seen on screening the patient. This phenomenon is never seen in a case of pure lung disease.

Secondly.—Cardiac displacement when due to fluid always takes place towards the opposite side of the chest to that on which the fluid is present, but when due to a lung lesion it is, with few exceptions, found on the same side as that on which the lesion is placed. This is well shown in the case of dextrocardia now before you, caused by a contracting lung lesion on the right side.

Thirdly.—When changes take place in the position of the ribs due to the presence of fluid or air in the pleura the space between the ribs is invariably increased. If we except the case of hypertrophic emphysema, it is not so where the lesion is present in the lung itself, for there the intercostal spaces, instead of being increased, are diminished, and, as in the *case shown*, roof-tile arrangement of the ribs takes place.

Fourthly.—Again, if a line be drawn down the centre of a skiagram and measurements taken from it outwards towards the lateral borders of the ribs, where the shadow is due to fluid, the measurements upon the shaded side either remain unaltered or are less than those upon the unaffected side.

The following six slides are presented as examples of the more common types of tubercular lesions:

One lobe confined to the right apex.

Two lobes unilateral (showing involvement of the upper and lower lobes on the right side).

Two lobes (bilateral).

Three lobes.

Four lobes (right side older standing).

Five lobes.

This skiagram (13) has a special interest in so far as it is that of a man whose disease was declared to be arrested two years ago, and who has since that time been working in the office of a factory of which he is part proprietor. This skiagram was taken at the end of two years' work, when the patient stated he felt fairly well. It is interesting as showing how efficient working capacity may be associated with the possession of a very small proportion of healthy lung.

I have reserved to this point the case of a special type of lesion which, if it has been alluded to by others, which is not clear, has certainly not received that amount of attention which its importance merits. I refer to the case of *primary* dense infiltration of the middle or upper third of a lower lobe, either, apparently, unaccompanied by disease in the upper lobe, or if disease is present in the upper lobe, then it is at a much earlier stage there than it is in the lower lobe. These cases are characterised by very distinct features which mark them out for special consideration. One of these I have now watched for over two and a half years, and others have been under observation for periods varying from six to eighteen months. It is not within the province of this paper to deal with them from a clinical standpoint, but I may be pardoned for digressing for a moment in order to mention a few outstanding points which they seem to share in common. *Firstly*, they have in each case occurred in patients between the ages of 21 and 24. *Secondly*, the patients, previous to the outbreak of the disease, have been unusually robust, and, with one exception, showed no hereditary burden of tuberculosis. *Thirdly*, the proportion of males to females is as five is to one, but, as during the period under review three times as many males as females have passed under observation, the proportion I have stated should not be taken as an

accurate one. *Fourthly,* they have for the most part exhibited a fairly rapid pulse rate—from 90 to 100 in the morning, of good volume and well-maintained. *Fifthly,* sweating, by no means severe, has usually been present. *Sixthly,* although high temperatures have been the rule, in no instance has the diazo reaction been detected in the urine. In no point have they resembled each other so closely as in their extreme intractability to treatment; for in no instance has a favourable result been obtained. Two have died, two have passed on to chronic disease complicated by laryngeal tuberculosis, and in the case of the remaining two there is every indication that the disease will run an unfavourable course, and terminate probably within eighteen months from the time the disease was first recognised. The point of special practical interest in connection with these cases appears to be the matter of prognosis. When this type of lesion is recognised as being present then, it seems, one will do well to give, if not an altogether unfavourable opinion to the friends, certainly an extremely guarded one, especially so having respect to the apparently excellent general condition of health such patients enjoy when they come under observation for the first time, and to the comparatively limited distribution of the disease present recognised by physical examination. So clearly defined are these lesions that where only the upper portion of the lung at the front and back has been examined their existence has escaped observation.

In one well-known book on the subject of diseases of the lung and pleura there appears the following passage: "It is very rare, except in cases of crossed lesions, to meet with a case, either during life or on the post-mortem table, in which the lower lobe is affected whilst the apex of the lung is free from disease."

This, it may be assumed, is the generally accepted view with regard to the rarity of unilateral lesions entirely or mainly confined to the lower lobe of one lung. The fact that six such cases have come under the notice of a

single observer in so short a period as three years seems to point to the probability that their occurrence is by no means so rare as has hitherto been supposed. In three of the cases which I have cited, so far as could be ascertained from a careful study of the physical signs taken in conjunction with the X-ray appearances present at the time of their first coming under observation, whilst there was no doubt about the presence of consolidation in the lower lobe, the existence of an upper lobe lesion could not be recognised. In these instances, however, lesions were soon afterwards recognised in the upper lobe. Whilst, therefore, one is not disposed to claim that the infiltration in this type takes place in the lower to the exclusion of the upper lobe, it certainly occurs at a much earlier date there than it does above (11, 12). (Five skiagrams shown.) From the point of view of the various opinions which are held as to the reason of the selective affinity shown by the apices of the upper lobe for the tubercular processes this lesion has a peculiar interest. For it seems to favour above others the view of Hirschfeld that the tuberculous process starts in the mucous membrane of the bronchi and works outwards. Upon this assumption the tubercle in these cases finds a nidus in one of two bronchi, viz. either the first dorsal hyparterial bronchus shown in this diagram (14) or in the cardiac bronchus, which passes off the main bronchus between the first and second dorsal hyparterial branches, and goes to supply the azygos lobe when it is present. The distribution of the former vessel, it will be recalled, is somewhat peculiar, for although it enters the lower lobe high up it does not supply the apex, but only the middle third of that lobe. The cardiac bronchus, distributed as it is throughout the azygos lobe when present, supplies the inner or cardiac aspect of the middle of the lower lobe. That the tubercular process starts thus in a few and not in the majority of cases may be accounted for by the presence of the anatomical irregularity of the larger bronchi recognised by Aeby as being present in certain lungs. In those cases the right bron-

chus, instead of standing approximately at right angles to the left bronchus, occupies a different position. The right bronchus by passing more directly downwards than usual forms an angle with the left bronchus, which is considerably less than a right angle. This naturally facilitates the deposition of foreign materials and infective organisms in the cardiac and hyparterial bronchi upon the right side more readily than under normal conditions. The tubercle having been deposited in this nidus sets up a process which, commencing in the mucous membrane of the bronchus, rapidly communicates itself by direct extension to the lung immediately surrounding it. The conditions of work in a sanatorium do not afford one many opportunities of confirming theoretical views by the incontestable evidence only obtainable in the post-mortem room; and it has not yet been my fortune to examine after death a lung in which during life this type of lesion had been recognised as being present. Now, however, that attention has been directed to the subject it is hoped that others more favourably situated will, as time goes on, be able to confirm or reject views which, however plausable they appear to be at present, nevertheless are largely based upon conjecture.

That this anatomical peculiarity in certain cases explains the existence of this type of lesion is not asserted. The view is merely advanced as a possible explanation of a relatively uncommon, and consequently exceedingly interesting, variation in the manifestation of the tubercular process in lung disease.

Changes in an Advancing Lesion.

The skiagram which is now before you shows the appearance presented by the chest of a lad aged 15, who first came under observation eighteen months ago. The case is one of extensive tubercular infiltration of the upper and lower left lobe, accompanied by collapse at the base of the lung, said to be caused by an old fluid pleurisy.

During the next four months the patient undoubtedly lost ground, and a *skiagram*, which was then taken, revealed two interesting points. That excavation had at this time already commenced is indicated by the light area here shown at the apex of the left lung; and the presence of the shaded area at the top of the right lung closely confirms the opinion formed as to the presence of infiltration diagnosed by physical examination as being there. In observing the condition present at the right apex you are particularly requested to note two points. In the *first* place, the shadow is denser at the upper than it is at the lower half; and *secondly*, the shadow is not limited at its lower border by any clear line of demarcation. On the contrary, it merges imperceptibly into the clear area below. The importance of this allusion will be appreciated when you come to consider the appearance presented by the next skiagram taken from the same case. Throughout the next three months the process steadily advanced, and the outstanding changes which had taken place during that time are readily seen by reference to this *skiagram*. In it you at once recognise the characteristic appearance shown in excavation of the left lung.

The apex of the right lung, no longer solid, reveals the presence of a clearly defined cavity; below it the indefinite merging of the shadow into the clear area below has disappeared, only to give place to a definite line of demarcation. No doubt what has occurred is as follows: The infiltration has steadily proceeded downwards until it reached the septum separating the upper from the lower lobe. This it failed to cross, and for the time being the disease seemed to have been arrested. Meanwhile in the upper section of this lesion a disintegrating process was at work. Excavation was taking place, and the area which but three months before was known to be occupied by infiltrated lung gave place to the cavity which is here seen.

During the next four months the clinical record pointed to a distinct diminution in the activity of the process, and to an attempt at arrest. The temperature fell and re-

sumed a more regular form, the patient put on weight and manifestly gained strength. It was hoped that the skiagram next taken would bear out the expectations so formed. However, that was not the case. Although some contraction is recognisable in the cavity, the appearance here shown leaves no room for doubt that the disease has crossed the confines of the septum as shown by the speckled areas having displaced the transparent lung, and that infiltration is now taking place in patches throughout the lower lobe. At this stage the patient passed from under my care, so that I am unable to present to you any further evidence as to the changes which have taken place since that time.

It is proposed to conclude by submitting two slides representing another case, one made from a skiagram taken shortly before death, and the other made from a photograph of the lung obtained after death. The skiagram (15) seems to clearly point to the existence of four cavities in the left lung, marked respectively *a*, *b*, *c*, *d*. The view thus formed received remarkable confirmation in the post-mortem room. The lung was divided in such a manner that the anterior half was removed. The photograph (16) here shown is that of the surface, which remained. True, it does not reveal the presence of four cavities, but it shows three, one of which is a very large one, and which is almost completely divided into two by a constriction which made it somewhat hour-glass in form. At the lower end of the lower lobe is clearly seen a fairly large cavity (*d*) in the skiagram, and at its upper end one which it may fairly be assumed corresponds to cavity (*c*) of the skiagram. The whole of the upper lobe is occupied by a cavity which is divided into an upper and lower portion by a constriction, which is faintly shown. The fact that the specimen had been subjected to the action of formalin before being photographed somewhat distorted and contracted the cavities. Still, one may reasonably claim that for practical purposes the evidence obtained by X rays as to the condition present in this case was substantially correct.

DISCUSSION.

Dr. HUGH WALSHAM referred to the skiagraphic appearance of a case of pyopneumothorax, in which the succussion ripples from the splashing of fluid appeared on the fluorescent screen. The great use of the X-rays in pneumothorax and pyopneumothorax was in the doubtful cases. The upper border of the shadow of serum in the closed pleural cavity was always concave. There was still doubt as to the relative opacity of pus and serum; he himself was confident that pus was much more opaque than serum. The skiagram was often of use in doubtful cases of pleural effusion. The position of the heart, as revealed in the skiagram, in cases of pleural effusion was not of the same value in children as in adults, as the heart in childhood was placed more to the left than in the adult. He had never found any shadow from a dry pleurisy. In regard to diagnosis of pulmonary tuberculosis, the X-rays ought to make it possible earlier than by the ordinary methods; the shadow in cases recognisable by ordinary methods extended beyond the limit of physical signs. The delayed lighting of the lung with the screen on deep inspiration, and the deficient excursions of the diaphragm on the affected side, were confirmatory of tuberculosis.

Dr. J. E. SQUIRE thought the value of X-rays in chest work was to confirm by sight what had been detected by other methods, and to give a permanent record of the conditions present. They might also be of value in revealing fluid in a certain proportion of difficult cases. He was doubtful if they could enable tuberculosis of the lung to be diagnosed earlier than by physical examination. Cases in which tubercle began in the lower lobe were not so infrequent as was supposed, but it was difficult to say whether a shadow seen with the X-rays was due to lesions in the lower part of the upper lobe or in the upper part of the lower lobe. His experience made him doubt whether the prognosis in these basic cases was so universally bad as Dr. Lawson's cases suggested.

Dr. C. R. C. LYSTER had not succeeded in diagnosing tubercle with the X-rays, which was not clearly revealed by the stethoscope. Their best use would he thought, be in the charting. The movements of the ribs and diaphragm revealed by the screen might be a useful indication.

Dr. KINGSTON FOWLER considered the chief importance in regard to the X-rays was the question of diagnosis. He had six years ago examined with the screen six early cases of tubercle. Four, which he had previously examined by ordinary methods, showed signs comparable with the physical signs, and two, which he had not then examined, showed little or nothing. Probably

in nearly every case there was about three times as much disease as the physical signs indicated; hence the shadow might, on this explanation, stretch beyond the adventitious sounds. There was need for training in interpreting skiagrams. His experience was rather disappointing in diagnosis from using the X-rays.

Dr. RUFENACHT WALTERS referred to cases in which the lesion was supposed to have been very limited, but which soon proved to be more extensive, and in such he thought skiagraphy should be of value.

Dr. G. NEWTON PITT regretted that no skiagram was shown to demonstrate whether the lung was more translucent than normal above the level of fluid, as the author suggested was the case.

Dr. LAWSON, in reply, and in allusion to the level of the shadow being lower than the physical signs, said that skiagraphy was not a matter of simple photography, the deflection of shadows came in. With the lamp placed directly over the sternum opposite the second rib the apex of the heart appeared, for example, to be in the tenth space. In this case the lateral shadows are practically reliable, but those in the vertical axis are entirely fallacious. The skiagrapher rarely, if ever, obtains cases for observation in which tubercle had not previously been diagnosed by physical signs, and therefore an exact opinion as to the relative reliability of the two methods of examination is not available. The shadow of pus, in his experience, is, generally speaking, denser than that of serum, but pus varies in character. The view that the shadows shown in the cases of the special lesion were due to lesions in the lower, and not in the upper, lobe was based upon the relative position of the solid object to the screen; the shadow appeared much denser by the posterior than by the anterior method of examination. The view was supported by a careful study of the physical signs.

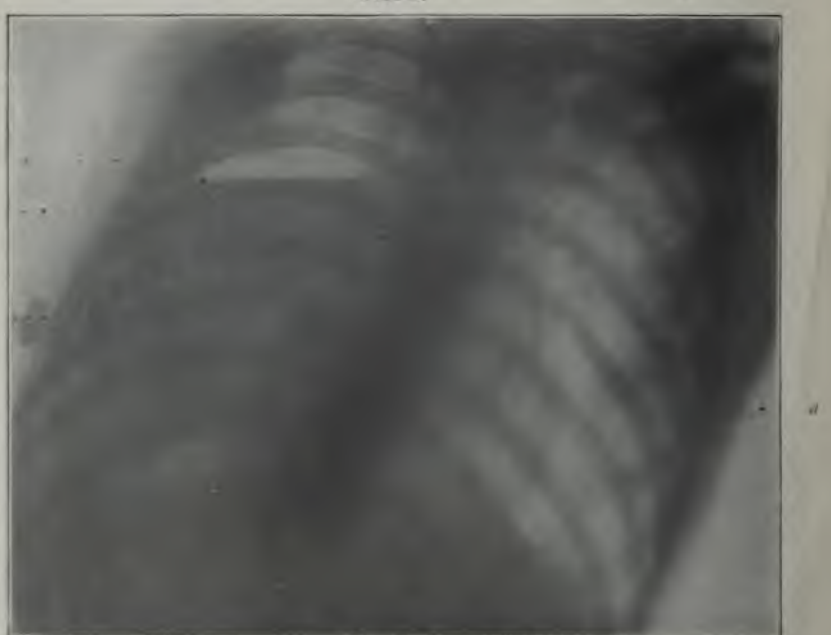
FIG. 1.

Fluid only. Fluid and air.

Bag. Experiment.

a. No line of demarcation. *b*. Line of demarcation.

FIG. 2.



Pyo-pneumothorax. Patient at angle of 45° before operation.

a. Air. *b*. Fluid. *c*. Site of operation. *d*. Right border of heart.

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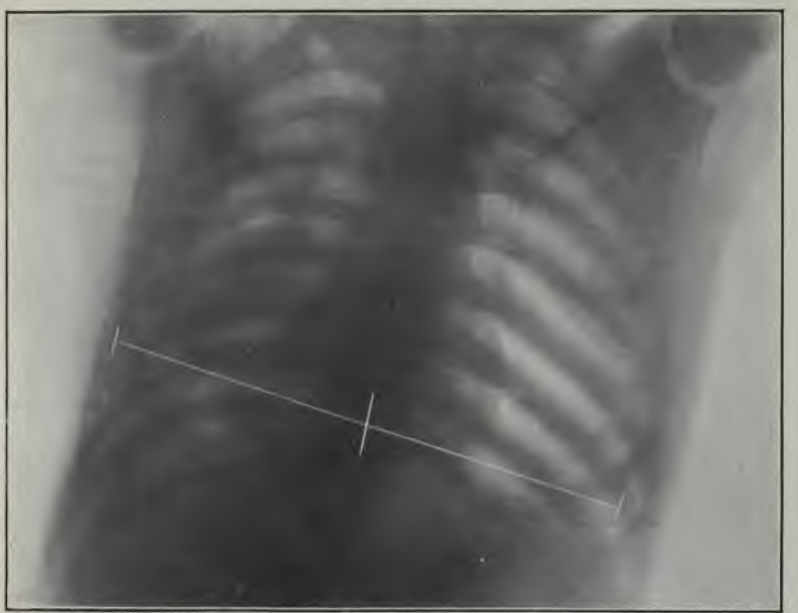
FIG. 3.



Same case 12 weeks after operation.

FIG. 4.

Same case 6 months after operation. Complete recovery.

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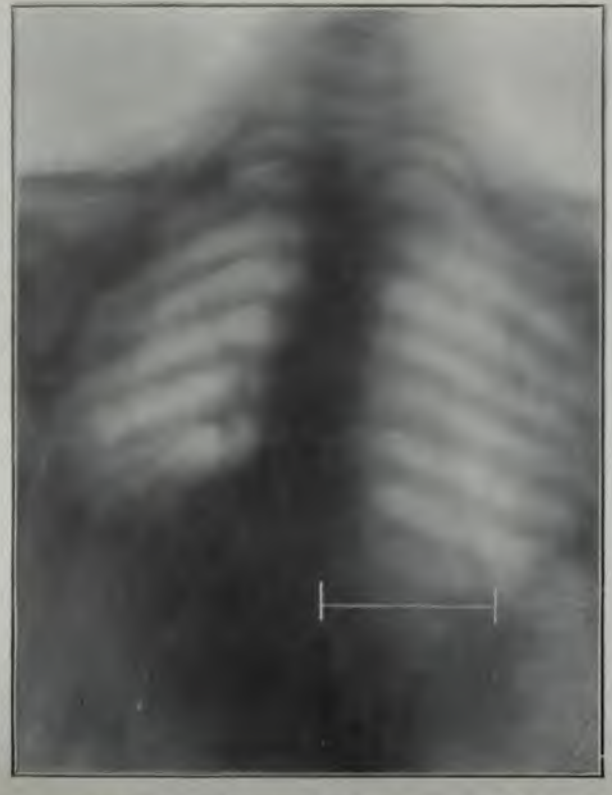
Fig. 5.



Typical left-sided pleurisy before paracentesis.

Fig. 6.

Same case, after paracentesis.

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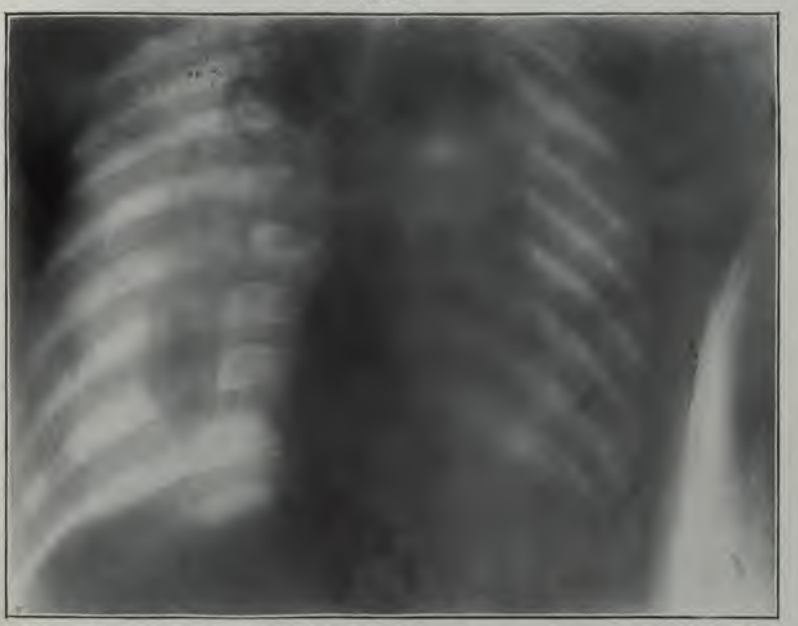
FIG. 7.

Fluid pleurisy, showing:—1. Narrowing of intercostal spaces. 2. Diminution in the diametric measurement on the affected side.

FIG. 8.



Pneumothorax showing heart on the right and collapsed lung on the left side.

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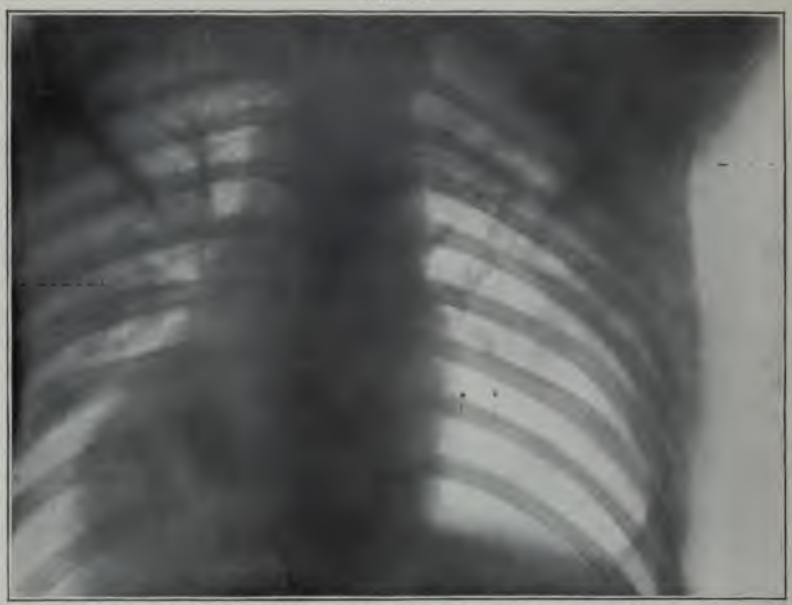
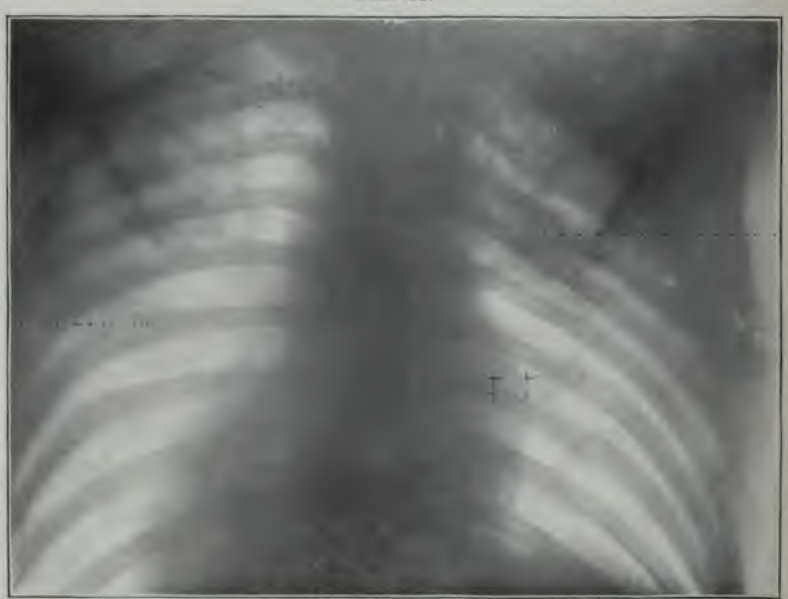
FIG. 9.



Dry pleurisy on the left with pulmonary lesion on the right side.

FIG. 10.



Case No. 9. Appearance shown 12 months afterwards.

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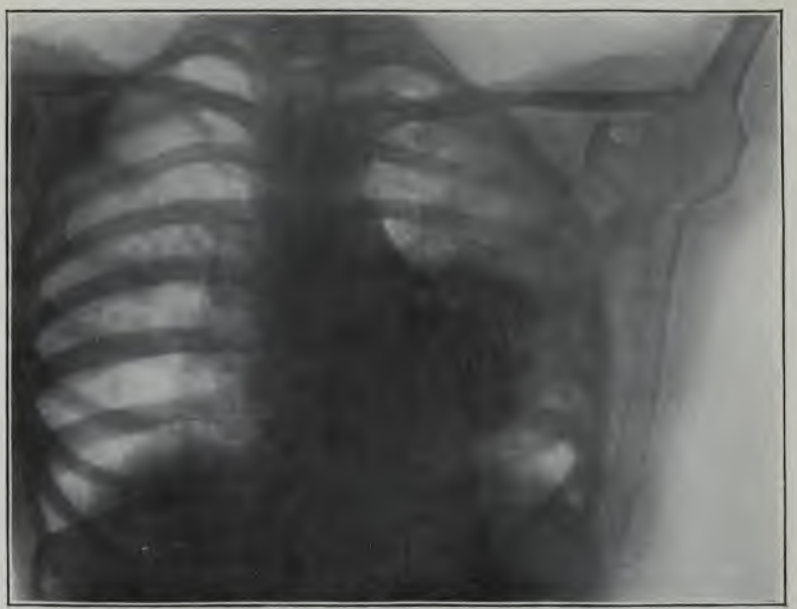
Fig. 11.



Special lesion, left lower lobe, female (patient lying on face).

Fig. 12.

Special lesion, right lower lobe, male.

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Fig. 13.

Five-lobed lesion with full working capacity. Appearance two years after discharge from a sanatorium.

Fig. 14.

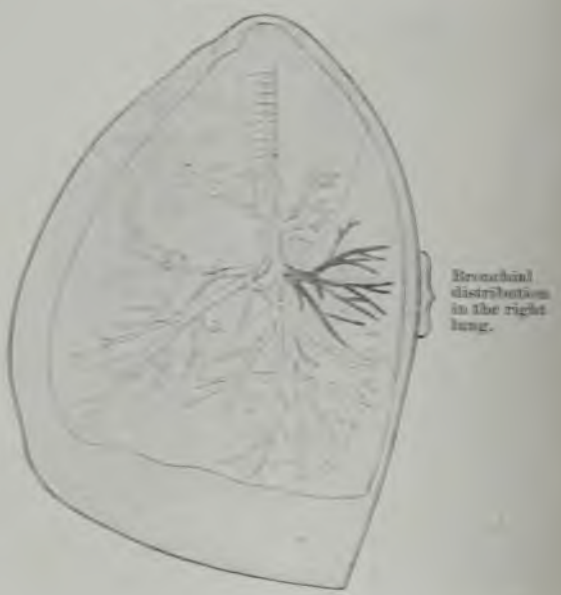

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Fig. 15.

Multiple cavities. Skiagraphic appearance during life.

Fig. 16.

Post-mortem appearance of the lung on section of Case 15 (photograph).

Adlard & Son, lmpr.

DISCUSSION

ON THE

SUBSEQUENT COURSE AND LATER HISTORY

OF CASES OF

APPENDICITIS AFTER OPERATION

OPENED BY SIR FREDERICK TREVES

OTHER SPEAKERS.

February 28th—
Sir WILLIAM BROADBENT.
Mr. CHARTERS J. SYMONDS.
Sir LAUDER BRUNTON.
Mr. PEARCE GOULD.

March 7th :
Sir RICHARD DOUGLAS POWELL.
Dr. H. P. HAWKINS.
Sir WILLIAM BENNETT.
Mr. C. B. LOCKWOOD.
Mr. W. H. BATTLE.
Mr. G. R. TURNER.
Mr. H. F. WATERHOUSE.
Mr. P. L. MUMMERY.

March 14th—
Mr. HARRISON CRIPPS.
Dr. SAMUEL WEST.
Mr. BRUCE CLARKE.
Mr. W. G. SPENCER.
Mr. MALCOLM.
Mr. F. C. WALLIS.
Dr. G. NEWTON PITT.
Mr. MAYO ROBSON.
Sir RICHARD DOUGLAS POWELL.

TABLES AND STATISTICS ILLUSTRATING THE SUBJECT OF DISCUSSION;

THE PROSPECTS AND VICISSITUDES OF APPENDICITIS AFTER OPERATION.

BY

SIR FREDERICK TREVES, BART., G.C.V.O., C.B., LL.D.

SERJEANT-SURGEON TO HIS MAJESTY THE KING.

THE matters, Mr. President, which you have done me the honour to ask me to discuss arrange themselves under two heads:

1. The degree of imperfect relief, or of imperfect recovery after operation.
2. The complications which may attend operation, and which may be regarded as accidental or independent of the direct surgical results of the case.

I have based the comments upon these two topics, for the most part, on the records in my private case-books. The lamentable deficiencies in these records are, I hope, compensated for by the admirable series of statistics very kindly prepared for me by Mr. Hugh Lett from the registers of the London Hospital. These statistics cover a consecutive period and embrace 1000 cases of operation

for appendicitis. In addition to the colossal labour of transcribing the notes of these cases, Mr. Lett has addressed no less than 797 letters to discharged patients in order that the after-history should be, in each instance, complete.

I believe that these particulars, collected with so much patience and care, will form the most important contribution which has yet been made to the statistics of this common malady.

In the account which follows I have kept the records of my own cases distinct from those collected by Mr. Lett, which latter are set out in a coherent whole.

I. Imperfect Relief or Imperfect Recovery after Operation.

In order that the issues may not be scattered over too wide an area it will be well to consider this question from the point of view of the operation performed during the quiescent period and that carried out for the relief of suppuration during the acute or active stage.

1. *Imperfect Results After the Operation of Removing the Appendix During the Quiescent Period.*

It is as long ago as 1887 that I ventured to suggest —in a paper read before this Society—that cases of recurrent appendicitis should be treated by removal of the appendix during the period of quiescence. My proposal was not very enthusiastically received at the time, but of later years I have no ground for complaint on this head. The procedure is one of the most common of abdominal operations, and certainly one of the most satisfactory. It is attended with but trifling risk and with but little distress to the patient, while in the vast majority of instances it is followed by a complete and unconditional cure.

I find, however, in my case-books the records of 45 patients who consulted me, and in whom the operation had—from their point of view—more or less completely failed. Had I been the operator in all these examples it would be possible to express the dissatisfied patient in the form of a percentage, but I am responsible for but the minority of them. The collection, therefore, from a statistical point of view, is almost valueless. It is natural that a patient who feels that an operation has failed should pour his woes into the ears of other than the operator. Thus it is that we all have to search for many of our failures without the walls of our own consulting rooms.

The relative frequency of these cases of failure may be gathered from the London Hospital statistics. From these it would appear that among 231 patients in whom the appendix was removed during the quiescent period, no less than 11 complained that since the operation they had had "attacks like those they had before it."

I have excluded from my 45 cases all examples of trouble in the healing of the wound and of ventral hernia. Ventral hernia is comparatively rare, but I have noticed that it does not exempt cases performed by methods which are considered to render a yielding of the scar impossible. I have met with only two instances of persisting sinus after this operation. In both cases the sinus had been open from eight to nine months when I first saw the patients, and in both fragments of silk ligature had escaped. Of one of these operations I have no knowledge, but in the other case it was reported that the procedure had been very difficult, and that a gauze drain was necessary.

TABLE A.

Patients who complained of Imperfect Relief after the Removal of the Appendix in the Quiescent Period.

	No. of Cases.
Appendix imperfectly removed	2
Ovarian trouble coexisting	9
Persisting or relapsing colitis...	8
Persisting local pain	7
Neurasthenia or hypochrondriasis	5
Continued attacks due to gall stones	3
„ „ colic	2
„ „ movable kidney	2
„ „ stone in kidney	1
„ „ an unexplained cause ...	1
Tender mass in the right iliac fossa	5
	45

The 45 cases of which I have notes represent patients who came complaining that "they were no better for the operation," or that "they were nearly as bad as, or perhaps even worse than, they were before." They also include examples in which "attacks" have continued unabated after the removal of the appendix.

As will be seen from the following summary, the degree of failure claimed to be manifest varies greatly.

A man of 60 from South America consulted me for persisting sinuses in the right iliac region with continued pain and discomfort in that part. The trouble had followed upon a large perityphlitic abscess which had been opened five years previously. The sinuses had been dealt with and counter openings made, but without benefit. Finally, the abdomen had been opened and the appendix removed. A well-healed scar in the usual situation indicated the site of this operation. Still no improvement followed. Thinking that a loose concretion may have been overlooked I reopened the abdomen. Buried amongst many adhesions I came upon an undisturbed but diseased appendix, which I removed. In seven months all the sinuses were healed. I have no explanation to offer for this case, beyond recalling the

fact that the statements of patients, even when given emphatically and in perfect faith, are not always to be relied upon.

A second case of this kind was in the person of a youth of 20. His appendix had been removed by an eminent surgeon, during the quiescent period, after the third attack. The appendix is said to have been strictured, to have contained no pus, and to have been surrounded by few adhesions. The operation was in August. In the following October the patient had another attack of appendicitis with fever, and was in bed ten days. In December he had another attack of some gravity. He had hardly recovered from this when he was again laid up for the third time after the operation. The attacks which followed the operation differed in no essential from those which had preceded it, except in this, that in the second of the early attacks a small abscess was evacuated, which gave no further trouble, nor did suppuration supervene in any of the subsequent outbreaks. As soon as the last attack had subsided—now two years ago—I opened the abdomen in the iliac fossa. The stump of the amputated appendix was swollen, hard and tense. It measured ¾ inch. Its distal end was well closed in by sutures which were still in evidence. The little tube was distended by muco-pus, and it was strictured at the very point where it opened into the cæcum. It was removed, and the patient has had no trouble since. This case suggests the wisdom of always removing the appendix close to the cæcum. A stricture at the actual cæcal orifice is not common. It would appear to be always so placed in the mucocele of the appendix—in those strange examples where the organ is translucent and is distended with a perfectly clear white jelly. In connection with this matter I may mention that in one operation after I had removed the appendix—as I thought—close to the cæcum I found that the proximal end of the tube had been invaginated

into the caput coli. The little intussusception was therefore at once reduced and the appendix re-excised.

Two cases, allied to the above, were found by Mr. Lett among the London Hospital records. In one the appendix was removed after the third attack. It was described as "half an inch long and very adherent." The patient subsequently had two more attacks. After the second outbreak the remainder of the appendix was discovered and removed. In the other instance an abscess was opened and "the appendix removed." The patient had another attack with suppuration, when a considerable portion of appendix was found and excised.

I find among the series now under notice no less than nine cases in which continued trouble after the operation was due to *mischief in the right ovary*. In some of these examples an inflamed, prolapsed, or adherent ovary had been noted at the time of the operation, but had not been removed. In others a second abdominal section was performed for continued distress, and a diseased ovary discovered and removed. In nearly all these cases the condition before operation would be described as chronic appendicitis supervening on acute or subacute attacks.

The following is a typical example: A married lady, aged 30, had an attack of appendicitis in 1893, for which she was confined to bed for ten days, and a second attack in 1896, which laid her up for three weeks. After this attack she was never well. There was continued pain in the right iliac fossa, tenderness there, occasional fever, irregular bowels, dyspepsia, wasting, and a condition of chronic invalidism. In 1897 her appendix was removed; it was free, was much thickened, and was full of mucopus. The state of the ovary was not noted. She was practically none the better for the operation; her old symptoms persisted, and she remained a chronic invalid. A year later a second abdominal section was made, and an adherent, chronically-inflamed ovary discovered and removed, to the patient's complete relief.

It is needless to point out how close is the anatomical

association between the vermiform appendix and the right ovary, especially in the matter of their lymphatic vessels. It is very common indeed to find both organs simultaneously inflamed, and the evidence most usually suggests that the ovary was infected from the appendix. In many instances, after the appendix has been removed, the ovary recovers, or at least gives the patient no further trouble. The surgeon's prejudice will be, naturally, in favour of leaving the ovary unremoved, but I am sure most operators will agree that there is often great difficulty in deciding when this can be done with the confidence that no further inconvenience will follow.

I hope I am not uttering a heresy when I say that it is often almost impossible to distinguish clinically between chronic appendicitis and chronic ovaritis. I have found the appendix sound and the ovary diseased in instances in which competent authorities have stated beforehand that the ovary had no part in the matter, and I have, on the other hand, found a perfectly healthy ovary accredited with mischief-making for which a diseased appendix was in reality answerable.

In operations upon women I think it is very desirable that the right ovary should be systematically examined when the appendix is being removed. If it be found to be entirely healthy, the knowledge is useful should the patient complain—after the operation—of any persistence of her pains.

The small opening made in the abdomen in that operation in which the muscular fibres are split and drawn aside has much to commend it, but it has the objection that it is not always easy to examine the ovary through such a gap, nor to remove the organ should it be found to be diseased, adherent, and deeply placed.

Eight patients out of this collection of those who expressed themselves as unbenefited by the operation were the subjects of *colitis*. The following is a typical instance :—A married lady, aged twenty-seven, developed severe colitis during residence in India. This was

followed in due course—and after her return to England—by an attack of appendicitis, for which the appendix was removed. The colitis much improved for a while. Some little time after the operation this catarrh of the colon returned. The patient became nervous and despondent, and more or less of an invalid. She had persisting discomfort in the right iliac fossa, tenderness in that region, occasional slight rise of temperature, irregular bowels, flatulence, and an inability to do anything. She maintained that she was in no way better for the operation. After many months the trouble in the colon subsided, and the patient made a good recovery.

The association of colitis and appendicitis is common. In certain cases—especially in those which develop in the tropics—I am under the impression that it is the trouble in the colon that leads, by extension, to the inflammation of the appendix. In other cases the relation is reversed, and the colitis seems to be due to chronic appendicitis. The appendix in such instances is little more than a culture tube for bacteria, the contents of which are emptied from time to time into the cæcum. Those individuals who have not had really acute attacks of appendicitis are often justified in maintaining that, for a time at least, they appear to be unrelieved by the operation.

Seven patients in the present series complained of the partial failure of the operation on the grounds of *persistent pain in the right iliac fossa*. For this pain there was no apparent cause. Of the seven patients, three were men and four women, and the ages were between twenty and thirty-three. The following cases will serve as illustrations. A lady, aged thirty-three, had her appendix removed during the quiescent period by a distinguished provincial surgeon. I saw her one year and eight months after the operation. She maintained that she was no better. She complained of abiding and severe pain in the right iliac fossa, and was more or less of an invalid. She was nervous, irritable, and full of troubles. The wound was

perfect, and nothing abnormal could be found in the abdomen. She had very defective teeth, ate much meat, and suffered greatly from dyspepsia and constipation. It is customary to ascribe all obscure abdominal pains after operation to adhesions, but in this case I was assured that there were no adhesions, that the procedure was most simple, and that the ovaries were normal.

The second case is that of a military officer, aged thirty-two. He was the reverse of neurotic, was active, and most eager to be well. His appendix had been removed two and a half years previously. The wound was sound, the abdomen revealed nothing abnormal, and the patient had the aspect of perfect health. Ever since the operation, however, he had been troubled with pain in the iliac fossa, which varied in intensity, was of neuralgic type, and occasionally rendered him unfit to do his duty. I could suggest no explanation of the trouble, and sought refuge in those vague neuralgias which occasionally follow any operation. After many months the pain entirely left him.

In five cases out of the series the only explanation of the patient's persisting trouble was the inadequate one that they were *neurotic*. This, I am aware, is no explanation, for the term "neurotic" is little more than a cloak to cover ignorance. Three of the patients were men and two were women. Their ages ranged from thirty-two to forty-nine. They all were chronic invalids, and all maintained that they were none the better for the operation. The chief complaint was of persisting pain in the appendix area and a sense of abiding illness. There was not lacking the environment of sympathetic relatives and much pampering. One patient was haunted by the dread of cancer, and was sure that his pain was due to it. Another—a gentleman of forty-three—was exceedingly gouty, and to gout some of his trouble may have been due. One patient had had his abdomen opened twice already, and was searching for a surgeon who would open it a third time.

As a typical example may be mentioned the case of a lady of thirty-two, whose appendix had been removed for chronic appendicitis with subacute attacks. Four years later she maintained that she was "as bad as ever." She was severe in her judgment of the surgeon who had operated upon her. She had constant dyspepsia, with flatulence and irregularity of the bowels, constant pain in the appendix region, where she could feel swellings and tumours which were apparent to no other fingers than her own. She was quite an invalid, and had moved from one health resort to another without benefit. Medical treatment, as a rule, made her worse. The simple measures I suggested did her much harm. Finally she fell in with a "Nature cure," which led to her speedy and complete recovery and to the deepening of her contempt for orthodox medicine.

In 9 instances in the present series *attacks* were reported to have continued without improvement after the operation. The patient in each instance maintained that the attacks which followed the removal of the appendix were identical with those which preceded it. In 3 examples the attacks were proved, in course of time, to be due to gall stones, in 2 to colic, in 2 to movable kidney, and in 1 to renal calculus. In the remaining instance no explanation of the attacks was forthcoming. In this case, during the first five months after the operation, the patient, a spinster of 37, had four attacks of pain, with a temperature of 100° to 103° F. These attacks laid her up for from three to seven days. They ceased and never reappeared again. From the patient's account it would appear that the operation wound had suppurated.

In the cases of hepatic colic there is no doubt but that gall stones and a diseased appendix coexisted. On two occasions I have removed the appendix and evacuated the gall bladder at one operation, but through two incisions. It is not always easy to diagnose hepatic colic from certain acute disturbances in the appendix. That an operation in certain of these instances may fail to relieve

the patient is not improbable. One must remember, also, in connection with these cases what extensive disease may be found in an appendix which has never given the patient the least trouble.

It is easy to imagine a case in which the attacks are due to gall stones, but in which marked tenderness in the iliac fossa leads to the diagnosis of mischief in the appendix. The appendix is removed, its walls are thickened, and its mucous membrame is ulcerated. It has caused no symptoms except the one of local tenderness, and the patient continues to have attacks as badly as before.

The case associated with renal calculus was as follows: A young man of twenty-two had for two years been liable to attacks of pain in the right iliac fossa, with vomiting and fever. On the last attack, which was one of definite appendicitis, a tender swelling developed in the region of the appendix. The appendix was removed. It was adherent, was bent acutely upon itself, and was full of muco-pus. After the operation, the patient continued to have precisely similar attacks to the number of eight or nine in the year. In these outbreaks there was no fever and no iliac swelling. It was not until these attacks had persisted for four years that the kidney was suspected to be the seat of the trouble. The gland was cut down upon, a calculus discovered and removed. The attacks, which had now lasted for seven years, at once ceased. The two operations were therefore unavoidable. A case, given by Messrs. Battle and Corner,[1] may here be quoted in illustration of this point: "A boy was said to have had twenty attacks of appendicitis, and when operated on the appendix was normal, but there was an oxalate of lime calculus, the size of a marble, in the pelvis of the right kidney."

In the two examples in which the "attacks" were of the nature of colic, there is little doubt but that the paroxysms of pain were due to adhesions.

[1] 'The Surgery of the Diseases of the Appendix Vermiformis and their Complications." Battle and Corner. 1904.

In the present series are five cases in which a *tender mass appeared in the right iliac fossa* some time after the operation. It occasioned great alarm, and led to the complaint that the patient was none the better for the removal of the appendix. In one instance the mass was simply inflammatory and appeared as a very tender lump, the size of a hen's egg. It was supposed to be due to thickening about a ligature or ligatures. After a few weeks it vanished, but while it lasted the patient was quite an invalid. In three instances the tender lump was a fæcal mass, and the patients were so troubled, at the same time, with colic, flatulence, and dyspepsia that they maintained that they were no better for the operation. They were both confined to bed for some days. In the fifth example the mass was due to tuberculous glands and there followed upon their appearance tuberculous disease within the abdomen, of which the patient finally died.

B. Imperfect Results after the Evacuation of a Perityphlitic Abscess.

After these operations ventral hernias are quite common, as are also instances of tardy, imperfect, and irregular healing of the wound. Such cases need not now be considered, as they present features of no especial interest.

I find from the records of my case-books that the following imperfect results may follow upon the evacuation of a perityphlitic abscess. They are arranged in percentages, but the figures are of little value, since they do not represent the experience in practice of any one operating surgeon:

TABLE B.

Persistent sinuses	40 per cent.
Recurring abscesses	24 „
Recurring attacks of appendicitis	16 „
Fæcal Fistulæ	12 „
Inflammatory deposits in the iliac fossa ...	9 „
	100

With regard to the *sinuses*, the persistence in the minority of the examples was due to the ordinary conditions which render a sinus obstinate. In some the suppurating tract was long and irregular, or there were many openings, or the main collection of pus was so placed as to be incapable of efficient drainage. In other instances the canal passed through a narrowed strait in a deep fascia or was so placed that it was exposed to constant movement. Many of these sinuses healed spontaneously after many months or yielded to simple treatment. The persisting sinus, in the majority of the examples, is due to a diseased appendix, or, as commonly, to a retained concretion.

With the removal of the offending substance the sinus closes. I have seen one instance in which the sinus closed spontaneously, without operation, after it had discharged for seven years; others have closed, under like conditions, after two or three years. To effectually treat the persisting sinus which will not yield to simple measures the area of the appendix must be exposed, that structure removed, and a careful search made for an escaped concretion.

The *recurring abscesses* show great variation in the manner of their appearing. The original collection of pus is evacuated and drained; the wound heals; after a varying period of time, the patient has pain and tenderness in the part with fever and constipation; a second abscess appears, and is promptly opened.

In a few examples the trouble entirely ends with the healing of the second abscess. In the majority of instances the abscesses are frequently repeated. I have known the abscess appear ten times before the patient was dealt with by a radical operation. The second abscess may appear within a week or so of the first, or, on the other hand, it may not become evident for months.

The following example will show the uncertain progress of these cases. A gentleman, aged forty-six, had his first attack of appendicitis in June, 1895. An abscess formed

and was incised. The wound closed in five months, and the patient made a good recovery. In June, 1896, a second abscess appeared. It was opened, and the wound closed in fourteen days. A third abscess appeared in July, 1897, and here again the incision closed in fourteen days. On no occasion was the appendix or a concretion met with. With the exception of a ventral hernia the patient now remained well, and, so far as I know, had no further trouble in the iliac fossa.

There can be little doubt but that the relapsing abscess is due, almost without exception, to the retention of a diseased appendix or a concretion, and that the condition can only be satisfactorily treated by removing these causes of offence.

In a few instances there is no apparent abscess, but in its place a troublesome *inflammatory mass in the iliac fossa* or pelvis. One example of this will suffice :—A man, aged twenty-eight, had his first attack of appendicitis when he was twenty. During his second attack, eight years later, a large abscess formed and burst into the rectum. The patient made a good recovery, and remained perfectly well for five months. He then began to complain of a dull pain in the right iliac fossa and rectum, with fever and constipation. He became very ill and wasted. The pelvis was found to be almost filled with a hard inflammatory mass. In four weeks this slowly subsided, and with it the fever vanished. Although a careful look-out was kept, no matter was known to escape *per rectum*. Three months after the disappearance of the swelling I removed the appendix. It was behind the cæcum in the iliac fossa, was very adherent, was bent upon itself, and full of pus. Nothing abnormal was to be discovered in the pelvis or about the rectum.

Into the very wide subject of *fæcal fistula* after the evacuation of a perityphlitic abscess it is impossible to enter on this occasion. I may be allowed to mention the following points which have impressed themselves upon my mind :—(1) That while the fæcal fistula exists another

attack of appendicitis is exceedingly uncommon. (2) That the fistula, unless due to an actual cutting or tearing of the bowel, has a tendency to close spontaneously. This process may involve months, sometimes many months, but healing takes place in the end. (3) That those fistulæ which appear some days after the evacuation of the abscess do better than those which are evident at the time of the operation. (4) That a still diseased appendix or a retained concretion is often the cause of the persistence of the fistula.

From the London Hospital statistics it would appear that a fæcal fistula may be expected in a little less than 6 per cent. of those cases of abscess which are treated by operation, and that the great majority of such fistulæ close spontaneously.

In connection with the sequelæ now under discussion the greatest interest attaches to the occurrence of *fresh attacks of appendicitis after the abscess has healed.* These outbreaks are definite attacks of appendicitis which must be clearly distinguished from those relapsing troubles which may attend an imperfectly-healed abscess cavity. These attacks need not be attended by suppuration, and, indeed, usually are not so associated.

I was at one time disposed to think that the patient who had had a perityphlitic abscess was, *ipso facto,* cured of his malady, and that although he might have further trouble with the abscess, he need fear no other attack of definite appendicitis. Longer experience has proved that this assumption is not correct. I am of opinion, however, that the number of patients who have definite attacks of appendicitis after a perityphlitic abscess has been evacuated are very few, and that the examples of such relapse in cases in which a concretion is evacuated with the discharge are quite rare. The escape of a concretion is an evidence that the appendix is extensively ruptured. As a result of this rupture it does not necessarily shrink up, as some suppose, but it seldom gives further trouble. Unfortunately, there are cases in which there are two or

more concretions, one of which only may be discharged. The patient then has "attacks" until the remaining substance is evacuated, and an apparent exception is made to what is very nearly a rule.

The interest in this matter centres around the question whether the appendix should be removed during the quiescent period in any case in which an abscess has been opened, but in which the appendix has not been dealt with. Mr. Battle, in his able and interesting work on the 'Surgery of the Diseases of the Vermiform Appendix,' answers this question in the affirmative.

In each such case as has been named he would advise the removal of the appendix. His opinion would appear to be largely based upon the case of a young woman who had an appendix abscess evacuated. Six months later she died of diffused septic peritonitis. The appendix was found to be in a state of acute inflammation and sloughing. There was no concretion present.

Mr. Battle further contends that, even if there be no second attack, the retained appendix may be the seat of chronic trouble and so greatly damage the patient's health.

The first point to be determined in this discussion is the frequency with which further attacks of appendicitis occur in cases of local abscess in which the appendix has not been removed. Dr. Miles Porter considers that such relapses may be expected in 13 per cent. of the cases. Mr. Lett, in his London Hospital statistics, places the number of such relapses at 17·2 per cent. (see Table F). I think that this percentage may be unduly high, since the decision that "the attack since the operation is like that the patient had before the operation" depends, in most cases, solely upon the patient himself. Some of such "attacks" might well be due to trouble associated with an imperfectly-healed abscess.

The appearance of the subsequent attack in these abscess cases shows remarkable variations. For example, among my own cases I notice that in one instance the

second attack appeared four weeks after the abscess was evacuated, while in another the patient did not have an attack until two and a half years had elapsed. One individual had only one further attack, while another had five outbreaks in the first eighteen months after the operation. In a third example there were several attacks during the first twelve months, and after that they ceased —or, at least, the patient, when he saw me, had been free from trouble for five years.

While I think that the appendix should be, without doubt, removed in any case in which the least trouble is experienced, it appears to me that the following arguments may be urged against the establishment of a rule that in every example of abscess (in which the appendix has been unremoved) it should be excised at the first convenient opportunity.

1. Upon the most liberal estimate it is evident that eighty-three patients out of every hundred will never have another attack.

2. The risk of a second attack is comparatively small. Thus, Mr. Battle, while he places the mortality of a first attack at 25 per cent., gives the mortality of a second attack as 7 per cent., and that of a further attack as 2 per cent.

3. In those cases in which an abscess has formed, although the operation is often unexpectedly easy, it is often not only very difficult but distinctly dangerous. In certain cases in which the adhesions have proved to be very extensive and dense, and in which the appendix is buried deep in the pelvis, I have failed to remove the offending body or even to find it. I cannot help thinking, therefore, that if Mr. Battle's rule became absolute the surgeon would find himself engaged in not a few operations which were attended with considerable risk to life. In the face of the facts above stated—and keeping in mind the fatal case Mr. Battle mentions—I do not consider that such risk is justified.

4. It may not be unfair to state the belief that the

evidence that an abscess has burst into the bowel is not always conclusive. The passage of a quantity of decomposed and long-retained mucus might well give a nurse the impression that pus is being evacuated.

II. The Complications which may Attend Operations for Appendicitis.

The length to which this paper has already extended will forbid any but a very superficial discussion of this subject.

With the exception of fæcal fistula, intestinal obstruction, and the persisting or extending abscess, the principal complications are those only of septic infection. They are such complications as may occur in connection with any septic wound. The fact that the wound is within the range of the portal system permits of the limited blood infection known as pylephlebitis.

With this exception, the most conspicuous complications differ in no essential from those which may attend a suppurating stump left after an amputation of the thigh.

Certain of the pleurisies and of the empyemata are due to direct local extension of inflammation from the original seat of infection.

The parotitis is probably due to that septic condition of the mouth which is common in this and in other abdominal disorders.

The common thrombosis of the left femoral vein is not open to a ready explanation. If movement be in any way concerned in this localisation it may be noted that while the right thigh is kept at rest the left lower limb is much, and often severely, used in the necessary movements of the patient in bed.

Mr. Lett's statistics (Table E) show the complications which have occurred in 1000 consecutive cases of operation. In the detailed appendix to that table will be found particulars of the circumstances under which these

complications have occurred, as well as of the period at which they have become manifest. It cannot be claimed that these details reveal any novelty in the familiar history of the infected wound. (See Table C.)

If a patient has several attacks of appendicitis the later attacks are less severe, and less likely to be accompanied by the formation of an abscess than the earlier ones. This is confirmed by Table E, which includes all the cases of abscess and of general peritonitis in this series. Cases of abscess with general peritonitis are included under the heading of abscess.

Fæcal Fistula.

A fæcal fistula developed in forty-nine cases, or in a little less than 6 per cent. of the cases which recovered. In thirty-five the fistula closed spontaneously. In two cases the fistula was closed by operation after it had persisted for seven months in one case, and two and three-quarter years in the other, and in two it still exists. Of the other ten cases, five died soon after the operation, four in twelve days or less, and one in three weeks with pylephlebitis. In the majority of the cases in which spontaneous healing took place the sinus closed within two months. In one case the fistula closed spontaneously after eight months. Two fistulæ are still open two years and eighteen months respectively after their appearance.

Three of the fistulæ appeared in ten, fifteen, and twenty-four days respectively after the operation; sixteen others appeared within a week of the operation, and twelve of the number appeared within three days.

Thrombosis of the Femoral Vein.

[illegible] patients who suffered from this complication [illegible] men and five were women. The [illegible] eleven cases, the right in one

In eleven of the cases the average date at which evidence of thrombosis appeared was twenty-four days after the beginning of the illness. In the remaining case the appendix was removed thirty-six hours after the onset of the attack, and thrombosis took place eleven days later.

With regard to the circumstances of the operation, the appendix had been removed during a quiescent period in three cases. Two of these healed by first intention, but in the third a fæcal fistula developed. Of the remaining cases there was in one general peritonitis, while in all the others an abscess was present.

The above particulars precisely coincide with my own experience of this complication. I have noticed no common factor in the circumstances of the patients who have become the subjects of thrombosis, nor have I any explanation to offer of the fact that the trouble nearly always occurs in the left vein and not in the right.

Acute Intestinal Obstruction.

Ten patients who were the subjects of appendicitis were operated upon for acute intestinal obstruction; four recovered, six died.

One patient had been ill for five days, and had had symptoms of obstruction for two days. At the operation the appendix was inflamed and adherent to the mesentery, forming a band which caused the obstruction; four inches of gangrenous gut was resected.

Another patient had been ill for ten days, and had had symptoms of obstruction for four days. At the operation, in addition to an abscess in the right iliac fossa and general peritonitis, fourteen inches of paralysed gut were found and resected.

In the other patients the obstruction was due to adhesions or to kinking of the gut, while in one there was a volvulus of the small intestine.

Pulmonary Complications.

There were forty-five cases in which pulmonary complications supervened. In seventeen there was broncho-pneumonia, in fourteen pleurisy with effusion, in two pleurisy without effusion, in seven there was an empyema, in four acute bronchitis, and in one pulmonary embolism.

All the examples of broncho-pneumonia occurred in cases of abscess or of general peritonitis, with the exception of two. In these two the appendix had been removed after the attack had subsided. As these were aseptic cases, and as signs of broncho-pneumonia were present on the day following the operation, they were probably examples of ether-pneumonia.

Pleurisy with effusion occurred in fourteen cases. In two instances there was pleurisy without effusion. In eleven the right side was affected, in five the left. In one case only in this series was the appendix removed during a quiescent period.

Of the seven cases of empyema six were on the right side and one on the left. The one on the left occurred with a left subdiaphragmatic abscess. Three of those on the right side were associated with a right subdiaphragmatic abscess, two with general peritonitis (in one of which the empyema communicated with the general peritoneal cavity through a hole in the diaphragm one inch in diameter), and the remaining case appeared four weeks after an abscess in the right iliac fossa had been opened.

There were four cases of acute bronchitis. In one instance the patient was admitted with acute bronchitis; in one case it appeared six days after an operation for an appendix abscess, and in two cases it came on after an interim operation.

Parotitis.

Four cases are noted, in none of which did the gland ɔurate. The right side was affected in three patients, in one. Two cases occurred one and two days

respectively after an interim operation, the third case after a large abscess had been opened, while the fourth case followed a laparotomy for general peritonitis two weeks after an abscess had been evacuated.

Pylephlebitis.

There were four instances of pylephlebitis among the thousand cases. One patient, a man aged thirty, had been ill for seventeen days before the operation; he had had no previous attack. The temperature was irregular and varied between 100° and 98°, with very occasional rises to 102° or 103°. The local signs were slight, but he had several rigors. At the operation the appendix was found to be ulcerated, inflamed, considerably thickened and occupied by an opaque fluid. It was adherent to the cæcum and was not perforated. Surgical emphysema developed round the wound. At the *post-mortem* there was pylephlebitis, with many abscesses in the pancreas, and broncho-pneumonia in the right lung.

The second case was a woman twenty-five years old, who was admitted with an abscess in the right iliac fossa. She had had no previous appendix attacks. The abscess was evacuated, and ten days later it was necessary to open up the wound again to let out more pus. An opening was then found in the cæcum, from which fæces escaped. Twelve days after the first operation she had an attack of acute intestinal obstruction, for the relief of which an artificial anus was made. Twelve weeks after the first operation the fistula leading to the cæcum was explored, and an attempt was made to close it. The artificial anus ceased to discharge on several occasions, but was still open at the time of her death, twenty-one weeks after the primary operation. At the *post-mortem* examination there were signs of early lardaceous disease, pylephlebitis with many abscesses in the liver, and pelvic peritonitis with pus between the coils of the small intestine.

The third case was that of a [illegible] who had

been ill four days. The appendix, which was gangrenous, was removed. He died three weeks later. At the *post-mortem* examination there was a little local peritonitis round the cæcum, otherwise the peritoneum was healthy. The superior mesenteric and portal veins were thrombosed and filled with pus; the splenic vein contained blood and pus.

The fourth case was that of a man, aged twenty-six. He was admitted with jaundice and an enlarged liver. He had had frequent rigors. At the *post-mortem* examination a loculated perityphlitic abscess was found, together with pylephlebitis and abscesses in the liver.

It is to be noted that none of these patients had had a previous attack of appendicitis.

Abscess.

After an appendix abscess has been opened and drained, other abscesses immediately or remotely connected with the appendix may form in various parts of the abdomen. These arrange themselves into three groups:

1. The residual abscess, in which a reaccumulation of pus takes place underneath or close to the operation scar, in the position of the original abscess. Such reaccumulation of pus is not accompanied by signs of another attack of appendicitis.

2. The secondary abscess, which is the result of the direct extension of the inflammatory process from the primary abscess to other parts of the abdomen.

3. The abscess which accompanies subsequent attacks of appendicitis.

Among the 499 cases of appendix abscess there were eleven cases of residual abscess which were opened at intervals after the operation varying from ten days to seven weeks.

There were twelve secondary abscess. Six of four being on the right two were opened three

weeks after the primary abscess had been drained, three two weeks after, and one nine days after.

Five of the secondary abscesses were situated in the pelvis while the remaining one pointed at the external abdominal ring. These were opened from twelve to twenty-one days after the primary operation, with the exception of one pelvic abscess which was opened seven days after.

Finally, in fourteen of the cases in which there were further attacks of appendicitis, the attacks were again accompanied by abscess formation. Three of them returned a third time with another attack of appendicitis and another abscess.

Pregnancy.

Among the 1000 cases of operation were six women who were pregnant. Of three patients who were operated upon for general peritonitis, two were six months pregnant, and one was four months pregnant. Abortion took place three to five days after the operation in each case, and all the patients died; one on the fifth day, one on the thirteenth, and the third on the eighteenth day (after an attack of secondary hæmorrhage from a vessel in the pelvis).

Of three cases who were admitted with an appendix abscess two were six months pregnant, and one was four months pregnant. One patient aborted ten days after the operation, and another four and a half weeks after the operation; they both recovered. In the third case pregnancy was undisturbed.

Table F shows the result of the investigation into the frequency of further attacks of reputed appendicitis after abscess formation or general peritonitis. It also gives the number of patients who stated that they had attacks of pain subsequently to the removal of the appendix during the quiescent period, similar to those they had before the operation.

Letters were sent to 797 patients, and replies were received from 506.

Of 264 cases of abscess or general peritonitis, further "attacks" were complained of in twenty-seven cases, or 10·2 per cent.

Of 242 cases of operation during the quiescent period, further "attacks" were complained of in 4·5 per cent.

It will be seen from the table that, of 107 cases of abscess or general peritonitis in which the appendix was said to have been removed, only six complained of further "attacks."

In 122 cases in which the appendix was not removed, twenty-one patients complained of further "attacks."

DISCUSSION.

Sir WILLIAM BROADBENT said that the paper by Sir Frederick Treves turned so entirely on surgical questions that he did not see where the physician came in. The consulting physician was in a particularly unfavourable position in regard to this subject. Patients were brought to him, and perhaps the existence of recurrent appendicitis was recognised, whereupon the patient was handed over to some surgeon, and the consulting physician heard later on whether the result was favourable or unfavourable, but was not supplied with any details. At other times the consulting physician was called in to assist in deciding whether an operation for appendicitis was necessary or not. The best advice was given, and the probability was that a surgeon was called in, operation performed, and the consulting physician heard nothing more about the case. For those reasons Sir William Broadbent thought it would be absurd in him to attempt to contribute in any way to the discussion.

Mr. CHARTERS SYMONDS said that in a series of seventy-two [illegible] cases, as far as he had been able to obtain the information, the following after-results had been observed. In one [illegible] was thrombosis of the left femoral vein; in one case [illegible] embolism with death on the fourth day; in [illegible] mild pyrexia for fourteen days followed by [illegible] in one case there was a pelvic abscess which

discharged into the rectum. Of these seventy-two cases, seventy-one were known at the present time to remain in perfect health. In another series of fifty cases from his hospital records there was no case of thrombosis. Concerning the after-history of these fifty cases, he had been able to trace four cases with after-results. In one the attacks diminished in severity, but still continued, the pain probably being due to a renal cause; in another case a surgical operation for fixation of the kidney gave relief, and in another recurrent attacks occurred, but he had not been able to secure the subsequent history. In another case death occurred from acute peritonitis. In addition to the four cases he had mentioned, he had had three other cases under his care in which operations had been performed by other surgeons. In one of these three cases the attack recurred before the patient left the nursing home. Subsequently fixation of the kidney was performed, and he hoped the pain would be cured. The second case was a man of thirty, in whom the pain continued after the appendix had been removed; the pain became localised to the region of the loin, and relief was obtained by removing a stone from the kidney. In the third case the patient was neurotic. In his experience, when recurrent abscess occurred, some part of the appendix had been left, or there was some concretion at the bottom of the abscess. In regard to the question of thrombosis, he agreed with Sir Frederick Treves's statement that it was more common on the left side. It occurred in some cases where the wound ran a perfectly healthy course. In looking over his cases other than appendicitis, Mr. Symonds said that he found it had occurred after radical cure of hernia in a woman, after suturing a patella, after compound fracture of an ankle in the opposite leg, and after fracture of the femur. Thrombosis had taken place in those cases after perfect union had occurred, and some explanation of the disaster must be sought for. In a certain number of cases where there were septic changes in the wound, thrombosis was no doubt the result of the septic process. When thrombosis occurred while the wound was running a healthy course, he thought there must be a degree of septic poisoning present, although there were no means of measuring it; in other words, a condition of the blood must exist which tended towards thrombosis. Thrombosis after operations in the upper part of the body he had never seen, nor had a case of thrombosis of the femoral vein come under his notice in a child. Some common factor must exist in these conditions, and he submitted that that common factor was the enforced rest which followed all operations on the abdomen. He advocated movement in his abdominal cases, and he dispensed with pillows placed under the knees; in fact, he directed his patients to move their legs after abdominal operations quite freely. In

his hospital cases there was no record of thrombosis after appendicitis; it was more common in private practice because of the greater care and the more stringent rest imposed. Referring to the removal of the appendix in acute conditions, he advocated one rule which was perfectly clear, that when the patient was septic, when the temperature was raised, the appendix must be removed, otherwise the source of the infection was left. In cases where there was no septic infection or elevation of temperature it was justifiable to lay open the abscess, clean it carefully, and drain. The best results were obtained if flushing out was avoided and the operator was content with dry mopping.

Sir LAUDER BRUNTON observed that in a certain number of cases where pain recurred after the removal of the appendix it was found that the patients were suffering from chronic colitis. In these cases the chronic colitis continued after the removal of the appendix, and the symptoms of discomfort and the pain were so like those produced by appendicitis that the patient usually said that the operation had done little or no good. Many of those cases were benefited, and recovery took place, simply by subjecting them to irrigation carried out either at home or at Plombières. Many suffered from a condition of the nervous system which was found commonly associated with colitis; they complained of aches and pains when no definite evidence of anything wrong in the appendix or in the colon could be found. It seemed as if the pain were fixed in their sensorium.

Mr. PEARCE GOULD said that it was impossible to deal adequately with the mass of statistical matter that had been furnished. He had looked up the records of just over 300 consecutive cases of appendicitis, about half being cases in his private practice and about half his hospital patients. In the main they conformed very closely indeed to the statements made by Sir Frederick Treves. He could confirm what Sir Frederick Treves said about the difficulty in some cases of removing the appendix, and also about the importance of removing the whole of the appendix, and how disastrous it might be to leave even a small fragment behind. As to the best course to follow after the patient had recovered from an appendix abscess, whether a second operation should necessarily be advised, he found in his own cases that of forty-one hospital cases there was a subsequent return of the disease necessitating an operation in four. Out of thirty private cases he had only known one. If he took the hospital cases, which he thought afforded safer ground on which to make a statement, they showed that just 10 per cent. of cases of abscess that recover after operation were liable to have a return of the disease. Therefore it was better to wait and see if mischief returned

before deciding the question of a second operation. In regard to complications, he mentioned that out of over 300 cases there were seven with sinuses, four with fæcal fistula, one with femoral thrombosis which affected both veins, and one with thrombosis of the internal saphena vein. Another complication was the formation of a second abscess before the patient had recovered from the first. He had seen eight cases in which that had occurred. He thought that it was an important complication. In one case cerebral softening occurred, the patient being hemiplegic the day after the operation. In one case there was intestinal obstruction, and in another both ovaries had to be removed for suppuration. In two patients he found and dealt with double pyosalpinx at the time of the operation. There were two cases of parotid bubo, one case of acute swelling of the thyroid, three of colitis, and two cases of marked neurasthenia. One point that had struck him was the glib use of the words "the operation for appendicitis," as if there were but one single and simple operation for appendicitis, whereas there were still three distinct procedures. One of these was the removal of the appendix, as in 214 of his cases. The second was that concerned with the treatment of a localised abscess, of which there were seventy examples in his list. The third was concerned with the treatment of perforation of the appendix, general infection of the peritoneum, and peritonitis without limiting adhesions. Studying these three groups separately, he found that among the 214 operations of removal of the appendix—either in the quiescent stage, or in the early stages of acute appendicitis—there were three deaths. One death was due to cardiac disease in a patient who, it was known, had grave and serious cardiac trouble; he had had three attacks of appendicitis, and still had a swelling over the cæcum, and it seemed better to those in charge of the case that he should run the risk of having the appendix removed. That patient, however, died from cardiac disease. The second death was in a patient who was suffering from septicæmia at the time of the operation. In the third case acute peritonitis ensued shortly after the operation. In these 214 cases there was a mortality of under $1\frac{1}{2}$ per cent. In the cases of abscess and general peritonitis the mortality was 26 per cent. The statistics of the London Hospital, dealing with a much larger series of cases, were almost identical, for the mortality in cases in which the surgical treatment consisted in removal of the appendix only was about 2 per cent., while in the cases of abscess and general peritonitis the mortality was 27 per cent. This seemed to show that his personal experience had not been peculiar. He wished to emphasise the point that if the case was such that it could be treated by simple removal of the appendix, the mortality was 2 per cent. But if patients were allowed to reach the point where an abscess had formed, or

where there was general peritonitis, a mortality of 26 or 27 per cent. was reached. Sir Frederick Treves had referred to the fact that, when in 1887 he first of all before that Society had suggested the removal of the appendix during the quiescent stage as a preventive of further attacks, that proposal was not well received, but it had been amply justified by experience. Mr. Pearce Gould submitted that the time had come when they ought to advance a step further and declare that if in acute appendicitis a surgeon was not called in till there was a general peritoneal toxæmia or an abscess, it ought to be regarded as a reflection on whomsoever was in charge. Now they were debating whether an abscess around the appendix should be drained, or flushed, or wiped out, and whether the appendix should be removed then or later, but he looked forward to the time when abscess would be prevented, and all would be interested in the more precise diagnosis of appendicitis in the early stages. There was hardly a parallel, so far as the pathological and operative conditions were concerned, with what was seen in appendicitis. All were agreed that the mischief started in the appendix, and for a time was limited to the appendix, and that the appendix could be removed without any detriment to the patient. They were not mutilating their patients by removing the appendix. But if the acute disease was once allowed to spread beyond the appendix it was impossible to set a limit to the mischief that might ensue, and operative measures for dealing with the consequences were serious and often unsuccessful. With the exception of the chronic ill-health that sometimes resulted from absorption of the contents of a diseased appendix all the grave consequences of appendicitis resulted from the extension of the disease beyond the appendix, and could be prevented by the timely excision of this organ. He did not think it was an exaggeration to say that it was as much an error for a surgeon to allow an abscess to develop about a diseased appendix as it was for a dentist to allow an abscess to develop about the fang of a tooth. The mortality of 27 per cent. in the cases of abscess and peritonitis ought to be reduced to the minimum of 2 per cent. by early operation.

Resumed Discussion, March 7th, 1905.

The PRESIDENT (Sir R. Douglas Powell).—The physician sees in consultation from time to time cases of pulmonary infarction secondary to injury or to surgical operation. I have only notes of a few of these cases, and could give no statistical statement about them—indeed my individual experience would be much [illegible] any statistics. I may certainly say, however, [illegible] the cases within my experience have been

cases after operation for appendicitis. As regards liability to this occurrence after appendicitis we must, I think, separate the chronic cases from those acute cases attended with suppuration in which the operation is imperatively immediate, and the risks of subsequent complications must be accepted as subsidiary to those urgently present. If there be ways of minimising such risks they are purely of surgical procedure, upon which I shall not venture to touch. These cases give the larger percentage of this complication of pulmonary embolism. Thus Oppenheimer[1] in his statistics of 217 cases of appendix operations gives eighteen as for perityphlitic abscess, of which three developed pulmonary embolism, *i. e.* 16 per cent. Of the remaining 189 chronic cases four developed pulmonary embolism, or under 2 per cent. I confess that the results with regard to the frequency of this complication recorded by Oppenheimer are more nearly in accordance with what I had gathered from my own experience than the, so far as I can make out, much more favourable statistics before us. It is, I think, of some importance to observe that in these statistics, although the number of cases of pulmonary embolism came far below those of Oppenheimer, yet in Sir F. Treves's and in the St. Thomas's statistics a certain number of cases of pleurisy and empyema appear which probably have their source in embolic infarction. I have always advocated the importance in all chronic cases of appendicitis, with the view of avoiding preventible complications, of carefully preparing the patient before operation by a few days' rest and careful diet, with the use of small doses of calomel and other intestinal disinfectants. For I believe these and some other allied complications to be traceable to local septic incidents leading to thrombosis, spreading from veins entangled in the adhesions, or to abnormal blood conditions favouring thrombosis consequent upon the bowel disorder. The septic dose cannot, however, be very strong, for most of these cases recover. My chief object, however, for intervening in this discussion is to give a brief note of a particular case of pulmonary embolism presenting a special feature of interest.

I saw, in consultation with Dr. Hartley, of Bishop's Stortford, early in October, 1903, a gentleman whose history was as follows:—In October, 1900, he had a severe attack of abdominal colic, and other intermittent abdominal symptoms followed, resulting, in January, 1901, in more definite suppurative appendicitis. Dr. Hartley, to whom I am indebted for the notes of the case, writes with regard to the operation on: "The onset of the attack, which was considered to be due to appendi-

[1] "Pulmonary Embolism after Surgical Operation, with special reference to Cases after Operation for Appendicitis." Oppenheimer. 'Berl. klin

citis, dates from January 4th, 1901. Sir F. Treves, early in February, cut down over the site of the appendix into a very large abscess then bulging into the rectum, through which, as

CHART 1.

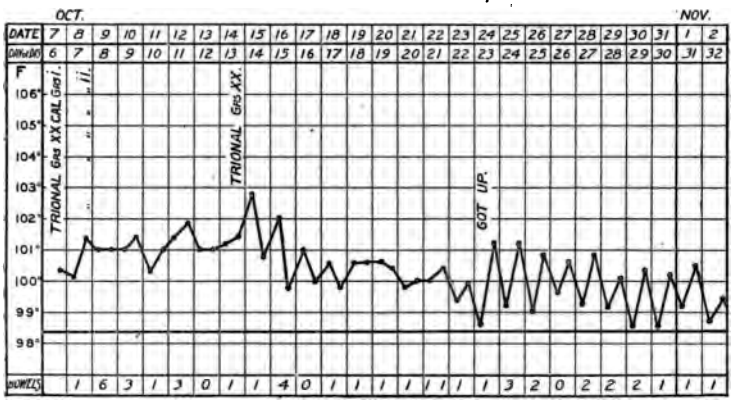


CHART 2.

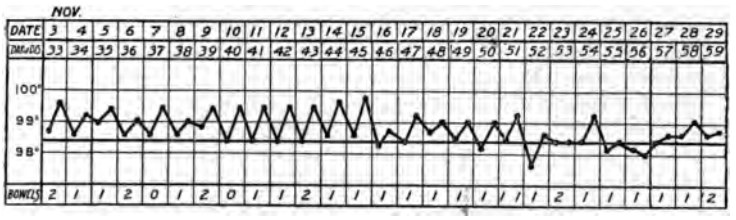
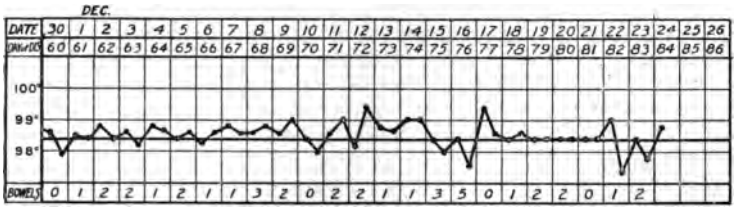


CHART 3.



well as by the abdominal incision, it was drained. The sac of the abscess, or rather the resulting sinus, was not soundly healed until August of last year (1902) after a sea voyage." In 1903 this gentleman hunted and played polo. On October

1st, in the late afternoon, whilst quietly playing cards, he spat up ʒss of blood. I saw him a few days later. He was expectorating dark blood-stained mucus and some pure semi-coagulated blood. The temperature ranged between 100° and 102° (see chart).

There was some impaired movement at right lateral base, and over a small area in the lower axillary region there were some impaired resonance, small crepitations, and feeble breath-sound. The lungs were elsewhere free. The diagnosis was infarction of the lung from some clot loosened from blocked veins in the adhesions. I took away a specimen of sputum which was carefully examined by Dr. Young on October 6th, and found to consist chiefly of blood-cells and to contain numerous micrococci but no tubercle bacilli and no elastic-tissue elements. A specimen was again examined on October 26th: "Viscid, yellowish, with a few streaks of pus, containing pus-cells; no tubercle bacilli; many cocci, etc." I saw the patient again in December. He was better, but had not gained much. There was still some evening fever, 99°. The cough continued; there had been slight return of blood. Râles were heard scattered through the right base in patches. Some more sputum examined December 16th contained pus cocci and *a few tubercle bacilli very sparsely and solitarily scattered over the film.* Again, December 22nd, a very few bacilli. He got about a little, and after Christmas went to Bournemouth, whence he returned on February 1st thin and rather breathless. Physical examination showed the right lower lobe to present scattered areas of crepitation with weakened breath-sounds, and an area of weak bronchial breathing, and crepitation was also observed at the apex of that lobe. Here and there, on deep inspiration, one could hear a single clicking sound. He subsequently went to the Riviera, and ultimately to Nordrach, and his health has been to a great extent restored. Dr. Hartley reports, "He is still at Nordrach and appears to be doing right well. His weight has steadily increased, he is walking from seven to ten miles a day, and Dr. Walther reports most favourably of his lung condition." I interpret this case as being probably an example of suppurative appendicitis due to, or complicated by, tubercle, followed many months afterwards by detachment of a clot contaminated by tubercle, which infected the base of the right lung, causing a gradually extending tuberculosis there. The definite signs and symptoms pointing to infarction of the base of the right lung, the absence of tubercle bacilli from the sputum examined one week and four weeks after the time of attack (October 6th and 26th respectively), and their discovery in small numbers about ten weeks later, December 16th, are the facts in support of this view.

Dr. H. P. HAWKINS said that the point which struck him in regard to complications was the lateness of their onset and the probability that they could be obviated by earlier operation. He drew attention to the fact that in spite of increased earliness of operation the mortality from appendicitis showed no fall and in some cases a slight rise as compared with a period of ten years ago. He accounted for this by a gradual rise in the severity of the disease which was occurring *pari passu* with its increased frequency. He agreed that a great reduction in the mortality was to be anticipated by continual insistence on early operation. As regards imperfect relief or recurrence of symptoms after excision he thought cases were few. Putting on one side cases where confusion occurred between appendicitis, floating kidney, and renal and biliary colic (the occasional difficulty of differential diagnosis being allowed), he believed that the rest of the cases of imperfect relief should be attributed to a colon-neurosis. Of this group mucous colitis, a non-inflammatory motor and secretory neurosis, was the best known example, but this shaded off into minor grades of pure enterospasm of which a clinical description could be given. In all degrees of the neurosis, if the trouble occurred in the ascending colon, with attendant neurasthenia and secondary hypochondriasis, the appendix could be excised without necessarily giving relief.

Sir WILLIAM BENNETT said it was obvious that any speaker engaging in a discussion concerning a condition which was so much a matter of common knowledge as that now under consideration was unlikely to be able to add anything to what had been already said upon the subject by such experienced persons as those who had already spoken. His only excuse, therefore, for encroaching upon the time of the meeting must be that unavoidable circumstances prevented his taking advantage of the opportunity of speaking earlier which was kindly offered to him, and that he rather felt it was the duty of those to whom the mere accident of seniority had provided an experience which justified their formulating definite views to give them for what they were worth. Under some circumstances the real value of statistics must rather depend upon a relative than an absolute application of the information which they provided, and that was especially apt to be so in statistics dealing with appendicitis, a condition which was met with in practice in very variable relations to the collateral circumstances of the patient and to the conditions under which its treatment had to be undertaken. Taking for an example the admirable statistics relating to the London Hospital series at their face value, they would, large as the numbers were, in his opinion give a somewhat too serious aspect to the prospect generally upon operation in that disease; on the same precise details of a similar number of from the higher-middle and upper classes

of society only were provided, the result would, so far as his experience enabled him to judge, give in a general way too favourable an impression; for the value of the information in either instance was, as he had said, relative rather than absolute so far as the general question was concerned. The truth of this could be easily demonstrated by taking the series of cases at St. George's Hospital during the years 1900–4 inclusive, and comparing them with a similar series from the better classes of patients. The total number in the St. George's series is 442. In 28 there was acute appendicitis with general peritonitis, in 178 ditto with abscess, in 106 acute appendicitis without abscess, in 130 the appendicitis was "subacute, chronic, or quiescent"; this latter class including, he supposed, cases of the recurrent or relapsing kind. In the same number of cases derived from the higher classes but extending, of course, over a much longer period, the following results were obtained:

Acute appendicitis with general peritonitis	5	cases
,, ,, with abscess . .	122	,,
,, ,, without abscess . .	83	,,
Subacute, chronic, and quiescent . .	232	,,

The preponderance of the more severe cases amongst the hospital patients was very marked—in consequence the death-rate was naturally higher after operation, reaching 11 per cent. on the whole series in the hospital cases against something like 6½ per cent. in the other series. In neither series did any death occur after operation in the quiescent stage. The disadvantage on the side of the hospital patients was primary neglect owing to their inability or disinclination to obtain proper treatment immediately upon the onset of symptoms, contrasted in the case of the well-to-do with easy accessibility to the best of advice and an intelligent appreciation of the necessity of securing it at once. One of the most important points had always appeared to him to be, in relation to future complications, that concerned with the treatment of the appendix in abscess cases—should it be removed as a routine measure, or should it not? Personally he thought not. According to the St. George's Hospital cases the mortality was practically the same whether it was removed or not, but this was not his personal experience. He had never had reason to regret having left the appendix untouched in cases of that kind; he could not say the same with regard to its removal. Sir Frederick Treves had observed that about 83 per cent. of patients were free from all subsequent trouble after mere evacuation of the abscess; his experience would place the circumstances even more favourably than that. Although he followed as quickly as he could upon Sir Frederick Treves's heels in freely operating upon cases of appendicitis, he had only once had to remove the appendix which had been left untouched after the evacuation of an abscess. On two occasions

he had scooped concretions out of recurring sinuses with a perfect result in cases in which he had no doubt some people would have done, or endeavoured to do, a formal resection. So far as the original abscess was concerned, if it was small and the situation of the appendix was obvious, he removed the appendix; but, having regard to the good results of mere evacuation, he doubted the wisdom of a prolonged search if the cavity were large and the patient's condition bad. With regard to the routine removal of the appendix after the healing of an evacuated abscess, his experience led him to be strongly opposed to it unless, of course, signs of trouble developed. As to the complications following upon operation, setting aside those which arose from the septic condition of the patient at the time of operation or septic conditions arising from unexplained reasons later, he could not help feeling that a large percentage were due to inadequate operation. The question then arose as to what an adequate operation was. Until he recently read in the Journals some letters and papers dealing with that matter he was under the impression that the first essential in an ordinary case of appendicitis was the removal of the appendix flush with the cæcum; it seemed, however, that that was not the universal view, since it was recommended by some that an interval should be left between the cæcum and the point of removal of the appendix varying from $\frac{1}{3}$ inch to even 1 inch. It was not, therefore, remarkable that the relief at times given by the operation was disappointing, seeing that any diverticulum left must have potentialities of recurrent trouble, whether the stump was "buried" or not. He had himself removed from the cæcal wall the stump of an appendix distended to the size of a cherry by muco-pus which had accumulated on the distal side of a stricture at the point of junction of the appendix with the cæcum. But the mere removal of the appendix did not complete the perfect operation; there still remained the examination of the ovary and tube in the female, and the ureter in all cases—a point to which, so far as he knew, no reference had yet been made in the discussion, although it was one which had a very definite relation to certain cases in which the removal of an appendix had been followed by no relief. The following instance would afford an example. A middle-aged man had suffered from eight attacks of pain, presumably due to appendicitis, with the characteristic symptoms. The appendix was removed, but attacks occurred subsequently of an absolutely identical kind. During one of the attacks the symptoms subsided very suddenly and on the following day a small calculus was passed by the urethra. No further attack had occurred. Stone in the ureter was more common than some of them supposed, and although a stone might rest for a long time in the ureter without giving trouble at any point between the renal pelvis and the pelvic brim, yet when it reached the latter point it might cause symptoms

identical with those of certain cases of appendicitis. An X-ray photograph in some of the obscurer cases of pain, etc., would sometimes prevent errors—a fact of which he had had personal experience. His memoranda of patients complaining of imperfect relief after removal by other surgeons of the appendix in the quiescent state gave the following result: In a total number of 18 cases there was irritation about the stump of the appendix in 3, and co-existing ovarian disease in 5; relapsing attacks indistinguishable from those before operation occurred in 2 cases of colitis, in 1 case of stone in the ureter, in 2 cases of movable kidney (one with calculus), in 1 with band-like adhesions, in 2 with an inflammatory mass behind the cæcum, and in 2 cases of persistent local pain, of which the cause was not discovered. In addition to these there were 3 very interesting cases in which he had removed the appendix, which was obviously and grossly diseased, without any immediate benefit at all. Attacks of constipation, followed by the usual fever, etc., occurred after the operation as they had before. In two cases the attacks became less frequent, and finally disappeared within eighteen months; the other patient still remained incapacitated. In each of those cases there was old-standing malaria, and in two there had been dysentery. The cæcum in each case was thinned and of huge dimensions. The recurrent attacks were clearly due to the faulty condition of the bowel. A very distressing pain about the region of the operation, frequently continuing for six or seven weeks afterwards, which he at one time occasionally met, he had seen nothing of since he had ceased applying ligatures round undivided peritoneum. With regard to fæcal fistulæ after abscess, he found in his own cases it had occurred in 5 per cent. In the St. George's series it was 11·2, in the London 6. He thought the term "fæcal fistulæ" was too loosely used in those cases. "Fæcal discharge" would be much more appropriate for those cases healed in less than two months, retaining the term "fistula" for the more permanent cases. He had had experience of only one fæcal fistula which did not heal spontaneously, either in his own cases or in those of other surgeons which had come under his observation. His experience of persistent or recurring sinuses was comparatively small, excluding those which came under the head of fæcal fistulæ, and such cases as he had seen had been after operation by other surgeons. One was worth recording: A female patient, aged about 30, had the appendix removed for the usual train of symptoms without relief; the ovary was [illegible] by another surgeon, again without [illegible] subject of a relapsing [illegible] opened up, and through [illegible] quarter of the [illegible] the remainder [illegible] expressed [illegible]

appendicitis were worse than the disease. With regard to femoral thrombosis he had never met with a case occurring in the absence of suppuration in or around the appendix; in two, which occurred in the series referred to, the thrombosis was, contrary to what seems to be the ordinary experience, on the right side. He did not think the occurrence of thrombosis had anything to do with keeping the patient too still, as had been recently suggested, but depended rather upon the condition of infectivity of the subject.

Mr. LOCKWOOD said that the time allotted was brief, and therefore he would not give any of it to mere criticism of what had been said by others. He believed that one of the main objects of that discussion was to learn what happened to those who had had the appendix removed. As he continued, it would be clear that the method of proceeding by clinical statistics alone was incomplete and fallacious, mainly because so many possibilities were ignored and so much was omitted. The future of the patient must in a large measure depend upon the disease which existed in the appendix itself at the time of its removal. Many of its conditions were favourable, but others most unfavourable. For instance, some were tuberculous, some cancerous, some actinomycotic. He ventured to say that although they could in ninety-five out of one hundred cases correctly infer from the clinical history and signs that the appendix was diseased, yet they could not infer the nature of that disease. Next, it was certain that correct conclusions could not be arrived at by examining the appendix with the naked eye. Some which looked perfectly healthy were dangerously ulcerated or infected, and he would narrate instances in which a naked-eye inspection had failed to detect carcinoma of the appendix, and had also failed in cases of tubercle, both being diseases which had a profound influence upon the subsequent fate of the patient. For those and other reasons it was clear that clinical evidence ought, in a scientific inquiry such as that, to be supported by microscopical evidence, and, if possible, by bacteriological. Some of those requirements had been fulfilled in the synopsis of 200 cases which he had provided, and which were all operated upon and afterwards examined by himself. He regretted not to have had sufficient time to work up additional ones of which he possessed microscopical specimens, or, indeed, to mention a tithe of what they showed. He would not pause to comment upon one particular case in which the part removed could not be microscopically identified as an appendix. But the possibility of the operator being deluded by a sham appendix might explain cases such as the one mentioned by the opener of the discussion. Doubtless in that the first operator honestly thought that he ndix, although that organ was actually ion. He possessed another specimen

which could not be identified as an appendix. He showed them on the screen a microscopical specimen of carcinoma of the appendix, probably primary columnar-celled carcinoma. The patient had the ordinary clinical signs of appendicitis. The disease was unrecognisable by the naked eye. Although the cæcum and end of the ileum were removed the disease continued to grow slowly in the pelvic glands, and ultimately killed the patient. He showed on the screen another variety of carcinoma of the appendix, namely, columnar-celled carcinoma undergoing colloid change. The appendix had been removed four years before he performed exploratory laparotomy, and had seen a tumour behind the right colon and mesocolon, with nodules in the peritoneum and enlargement of lymphatic glands. The disease of the appendix had not been recognised at the time of its removal. Finally, he placed another specimen before them of what was probably the ordinary type of carcinoma of the appendix. It was given to him by his colleague, Mr. McAdam Eccles, who told him that when he removed the appendix he thought the two nodules on it were tuberculous. The patient was a gentleman, aged 18, and had the ordinary symptoms of appendicitis. He ought to mention that an appendix in his list of cases looked very like a cancerous one. Opinions were divided as to its real nature. The patient was well a year after its removal. He did not think there was anything exceptional about his own experience, for cases of carcinoma of the appendix had not infrequently been recorded by others who had realised the necessity of microscopical examinations, and a certain proportion—say 1 in 250—were likely to be met with when every appendix was systematically examined. Next, as regards tubercle: since he began that investigation, about nine years ago, he had always looked for tubercle and expected to find it frequently. Fortunately, it was rare, and only four instances were included in the list. He said fortunately because the subsequent history was unfavourable. In a boy the wall of the empyema of the appendix was tuberculous when microscopically examined. The patient died about a year later of tubercle. In another case the patient was known to be tuberculous, and a tuberculous appendix was removed, but the disease progressed within the abdomen. Five years after they could detect no actual sign of tubercle in a third case, although the patient had a cough and was not well. The ultimate fate of one who went to Australia was unknown. Once he diagnosed tubercle of the appendix with the naked eye, when the microscope showed that the nodules were merely inflammatory. The patient was well more than two years after. Next, the microscopical examination had revealed another and undescribed condition which had a profound influence upon the subsequent fate of the patient, and which had not yet been included in clinical statistics. The question had been raised of

the propriety of attempting to remove the appendix after an appendicular abscess had been evacuated. He was accustomed to decide each case on its merits, and after the abscess had been opened, but his bias was strongly in favour of removing the appendix, provided the general and local conditions did not forbid. He had not time to go into all the reasons by which he was guided, but he showed them on the screen the condition of an appendix six months after an abscess containing a fæcal concretion had been opened. The mucous membrane was extruded through the perforation so as to form an intra-abdominal appendicular fistula. No wonder a second abscess formed. In three other cases of delayed removal of the appendix after abscess, one exhibited slight inflammatory changes, but two others were in a state of active inflammation and ulceration. Besides those conditions, the appendix left after the opening of an abscess might, as he had seen himself, contain concretions or foreign bodies. The sinus which persisted after an appendicular abscess, and which was often due to intra-abdominal appendicular fistula, had hardly been mentioned, although it might necessitate an operation quite apart from a fresh attack of appendicitis. Fæcal fistulæ were another complication, but fortunately got well unless tuberculous or carcinomatous. He next showed the commonest disease of the appendix (123 in 200), namely, ulceration of the mucosa, with the penetration of bacteria into the tissues of the mucosa and into the lymphatics and onwards through the hiatus muscularis into the peritoneum. That dangerous class was the one which gave such perfect results after operation. The ulceration of the mucosa was associated with septic, muco-purulent, or fæcal contents, and the latter with concretion (23 in 200). As regards the latter, a point in practice arose. When operating for other abdominal diseases the appendix was sometimes found to have a concretion in its interior. When that was the case the condition of the appendix was one of danger, and it ought to be removed. In the specimen shown the mucosa was ulcerated; the fæcal and bacterial contents of the lumen had penetrated the lymphoid tissue, and could be seen making their way along the lymphatics. Almost the same condition as that was seen in an appendix which he had taken away whilst engaged in removing an inflamed ovary and Fallopian tube of the left side. The appendix was removed simply because it contained a concretion. It was not known to have caused any trouble. Such contents as glass, hair, threadworms, and so forth, had been met with during microscopical examination, but had no particular bearing upon the ultimate fate of the patient. The immediate clinical complications of appendicitis had been given so fully by others that he would pass them over, merely calling their attention to two of mesenteric thrombosis. The patients were suffering from acute intestinal obstruction, but he examined the

appendix because he had long thought that appendicitis might be one of the causes of the thrombosis. Lastly, as regards eight cases which were not cured, one had an appendix distended with fæces. Distension was a cause of appendicular pain. But she had also a Riedel's lobe of the liver, which he afterwards removed. Another had muco-membranous colitis; another mucous colitis, and might have got well ultimately; two had very large movable cæca which fell into the pelvis. One had prolapse of the hepatic flexure of the colon; in one he could never find a reason for the non-relief of discomfort; and finally, a chronic invalid had an extraordinary melanosis of the appendix. He showed the specimen on the screen. Were they in the presence of a new disease of the intestines similar to the bronzing of the skin in Addison's disease? The mucosa was intact, with columns of deeply pigmented cells in the tissues between the tubular glands. The ring of pigment was easily visible to the naked eye. He had met with similar pigmentation in a less degree in four or five other cases, and began to associate it with a chronic abdominal disease, and one which might not be cured by removal of the appendix. In conclusion, two more patients had spurious relapse about six months after removal of the appendix. Each had enormous dilatation of the cæcum, which was also unduly movable. In spite of warnings, they had both been careless about their diet and the action of their bowels, and in consequence their cæcums had become loaded with fæces. They were quickly cured by enemata. It was not without interest to speculate as to what would have happened had they gone to one who was unfamiliar with their anatomical peculiarity. That brought to an end a brief reference to the causes within the appendix and some of the causes outside the appendix which militated against the success of the operation. He might seem to have placed appendicitis in an unfavourable light as regarded its dangers and complications, for he had not alluded to the question of cure. In that an obvious difficulty arose, because they were not yet agreed upon a time when a cure should be supposed to have become established. Inasmuch as some of the patients with dilatation of the cæcum, and those with colitis, were slow in deriving the full benefit of the operation, he would suggest that a year's probation should be adopted. If that were accepted the curative results of appendicectomy would compare most favourably with all the other major operations in surgery.

Mr. W. H. Battle asked permission to deal only with one part of the subject under discussion, that of appendix abscess, and submitted a list of sixty-one cases, of which 50 presented some point of importance. Whenever possible, the condition of the appendix was stated. He referred to five cases in which there was a return of symptoms after an abscess had been

evacuated with success. In all of these the appendices were removed later and showed marked pathological changes. In five other cases a secondary abscess formed, the appendix not having been removed at the first operation. Sinus formation occurred in six cases after a localised abscess had been opened and drained. In two other cases operation was performed for commencing peritonitis where there had been previous localised suppuration. The appendix had been removed in seven cases after an abscess had been evacuated and all inflammatory symptoms had subsided. In several of these cases concretions were found, and in all there were evidences of pathological conditions which might give rise to further trouble. Similar conditions were found in the appendices removed after evacuation of an abscess into the lower part of the bowel. The appendix had been successfully extirpated in seven cases where localised suppuration was present, but in one of these cases the patient had died from a cardiac seizure four days after the operation. The condition of the appendix was also described in some fatal cases. He was glad to find that Sir F. Treves now regarded the condition of the appendix after the formation of a localised abscess as a source of possible danger. For his own part, he did not consider a patient safe from further attack even if a concretion came away; there might be another concretion or irreparable damage, as shown in cases in his list. He submitted the following propositions regarding the treatment of localised suppuration in this region and the treatment of the appendix. An operation for the evacuation of the original abscess should be done as soon as possible, unless the abscess was deeply seated, the abdomen much distended, and the patient an unfavourable subject. He considered that an appendix which was so seriously diseased that it could cause suppuration would, in the majority of instances, not recover, and would be a source of anxiety and danger to its possessor, and should be removed. Surgeons should not be deterred from doing this by the possibility of a difficult operation, for the results were satisfactory and the mortality very small. The best time, in his opinion, for operation was soon after the closure of the abscess, when the adhesions of the appendix to surrounding parts were comparatively recent, and yet the tone of the bowel wall had been regained. At that time also any recent intestinal adhesions could be rectified, and obstruction prevented.

Mr. G. R. TURNER said that unless an acute case was going on for convalescence he operated as soon as he could, and the statistics he had laid before the Society showed a mortality of only 8 per cent. in cases operated on "during attacks." He hoped with Mr. Pearce Gould that the mortality might be reduced to low as 2 per cent. in acute cases if it became the rule to rly. His friend Mr. Pendlebury had put together ppendicitis at St. George's Hospital between the

years 1892 and 1898. Out of the 156 cases, 51 had been operated upon; of the 16 cases operated on in the quiescent stage all recovered; taking those operated upon in the acute stage, the percentage mortality during those years was almost 30 per cent. The mortality in acute cases in the tables presented by Mr. Lawrence Jones during the past four years at St. George's Hospital was only 15 per cent. He attributed this marked decrease to the fact that operation was now performed there much earlier than formerly. With regard to complications and sequelæ, he thought that they would hear less of them if the cases were subjected to operation earlier. He trusted that when Sir Frederick Treves replied he would add to the great benefits he had bestowed on surgery by avoiding saying anything too crushing against early operation in acute appendicitis.

Mr. H. F. Waterhouse referred to the fact that, although appendicitis was three times more common in the male than in the female sex, complications were more frequent in women. The appendix should be removed flush with the cæcum, and the absence of this precaution was responsible for recurrent trouble in some cases. Ventral hernia did not occur when the muscle-fibres were separated and not divided—a method which when combined, if necessary, with transverse division of the sheath of the right rectus abdominis and forcible retraction of this muscle gave room enough in all cases. The debate had emphasised the necessity for carefully examining the right ovary and tube at the time of operation. Pyonephrotic kidneys and ureteral calculi were possible sources of errors in diagnosis. Early operation was a very important factor in reducing the mortality of the disease, and in 19 cases which had been operated on within twenty-four hours of the onset of symptoms there had been no deaths, in spite of the fact that perforation had been present in three instances. The appendix should not be removed as a rule when evacuating an abscess.

Mr. P. Lockhart Mummery said that in his own analysis of 100 consecutive cases the percentage of complications occurring after the operation was 37 per cent. He had worked out the percentage of complications occurring soon after the operation from the statistics which had been provided; and, taking the total of 3225 operations, the percentage of complications was, with deaths included, 27 per cent., but leaving out the deaths, it was 18 per cent. He believed these figures were too low, and that if all the complications were included the percentage would be higher. The percentage of cases in which symptoms due to adhesions were present some considerable time after the operation was about 12 per cent. out of 100 cases he had written to. The symptoms in those cases were great difficulty in getting the bowels to act, difficulty in holding the urine for any length of time, painful micturition, and pain in

the abdomen after standing or walking, relieved by lying down. He thought that this high percentage of complications was an indication for greater care in the after-treatment of these cases. And he believed that the percentage of complications could be considerably reduced by careful attention to after-treatment. In regard to the time after the operation at which the patient was able to resume his ordinary occupation, it was found on the average to be thirteen weeks. This was, he believed, much longer than was generally supposed. It might be partly accounted for by residence in convalescent homes and by payment from sick clubs. Further statistics on this point would be of interest.

Adjourned discussion, March 14th, 1905.

Mr. Harrison Cripps said that the title of the original introduction was "The After-Effects of the Operation for Appendicitis." He did not quite understand what the operation was; there were innumerable operations for the condition, and the after-results necessarily depended in great measure on the kind of operation performed, the time at which it was performed, and even upon the individuality of the surgeon who performed it. He thought surgeons could fairly divide their cases into two classes: (1) Those seen between attacks of appendicitis, what was called recurrent appendicitis; and (2) those seen during the acute attack. He had been much struck by the unanimous opinion which seemed to exist with regard to the treatment of recurrent appendicitis. For his own part, if a patient had had one attack and he was asked to see the case a few weeks afterwards, and the patient was found to have practically recovered, if he found there was no swelling, no pain, and no tumour to be felt, he assumed, and he thought often rightly, that that attack was a pure accident, and that the patient might never have another, and therefore he did not advise operation. But when a patient had had two or three or more attacks the fact of recurrence showed that there was some abiding cause. Therefore, whether he felt a tumour or not, he liked to advise operation during the quiescent period. There seemed to be an almost universal opinion in the profession, and one which he endorsed, that those cases which were operated upon between attacks yielded some of the most satisfactory results in surgery. As with every other surgical operation, accidents would occasionally occur, but it would be found that an ordinary surgeon using antiseptic precautions would have a very small mortality, perhaps not more than 1 per cent. But the whole character of the class of acute or fulminating attacks was different. Those attacks might go to a satisfactory conclusion and might end in resolution. Many of them ended in abscess, and some which

were operated on, and others which were not, ended in death. Much had been said in the discussion about collections of micrococci in the appendix, the appendix becoming a sort of test-tube, and that those micrococci escaped and produced an attack of appendicitis. That was a view which he did not share. He believed that every person at the present moment had a certain quantity of bacteria in his appendix; therefore, why did not everyone have an attack of appendicitis? It was known that a certain number of those attacks were due to a concretion. How that concretion acted—whether by producing an ulceration and then by extra movement getting protruded and causing inflammation he could not say, but in a certain number of the cases a concretion was found at the operation. In other cases there was no concretion, but everything was found much matted together, the omentum generally adherent over the appendix, the small intestine covering it, so that there was considerable difficulty before the appendix could be exposed at all. Sometimes it was tucked away in the most extraordinary positions, sometimes it had got under the cæcum, sometimes into the pelvis, and, of course, occasionally on the surface. He desired to know what produced that curious distortion, and would remind all surgeons accustomed to operate on such cases how frequent it was to find the appendix doubled on itself. He held that a far larger number of cases than was generally imagined were due to twisting of the mesenteric attachment of the appendix, and he was not aware that anyone had called attention to the point before. He exhibited a small model, consisting of an indiarubber tube, from which depended a sheet of wash-leather, and demonstrated the method in which the twisting occurred, showing that if the twist was below the appendix got tucked underneath the cæcum, and if the twist were above it was found superficially. Many attacks of acute appendicitis would come on unexpectedly, the patient having previously been perfectly well. They were attributed to strain or to sprain, but it was difficult to understand why a sprain or strain should suddenly produce those violent symptoms. It must be remembered that when the operation was performed late in such a case everything was disguised; there were adhesions everywhere, the structures were difficult to see, and consequently what were supposed to be adhesions were often part of the twisted mesentery. He recently had two cases bearing on that point. One was that of a young man who, while straining at stool, was suddenly seized with a very violent pain in the region of the appendix. A few hours afterwards vomiting occurred, and the patient went to bed very ill, and remained so during the night. When the patient was seen next day at four o'clock the pain had gone, and he seemed well, except for just a little tenderness in the abdomen. No operation was done. Six months afterwards the same patient,

while practising a young horse at some hurdles, was suddenly seized with an exactly similar pain in the lower part of the abdomen, and was unable to ride home. He was assisted home, and vomited violently during the night, and was very ill the next day. A surgeon was not sent for, because the patient said he had had a similar attack before, and had got over it. The patient was seen on the third day, and he was then very ill with a fulminating attack of appendicitis of a typical character—a brawny swelling over the appendix region, with some redness and œdema. At the operation the appendix was found to be absolutely twisted on itself and almost black. Undoubtedly the first attack was due to the same cause, but the twisting undid itself, or it was only partial. In another recent case the patient, who had had two previous attacks, had apparently been well for many months before. He was playing tennis, and in reaching back he was seized with the same pain which he had had before. He saw the patient the next day, and was able to operate in forty-eight hours from the commencement. The symptoms were not so acute as in the first case, but the appendix was very swollen and œdematous and doubled on itself. Without the slightest difficulty he was able to undo the twist, and then the appendix, which at first looked swollen, came out straight. He held strongly that in no inconsiderable number of those acute cases the attack commenced in that way. All his fatal cases had been those in which an abscess was present. A few years ago he regarded it as his duty, abscess or no abscess, to dissect out the appendix. But with subsequent experience he had changed his views. He believed now that if in acute abscess the surgeon satisfied himself by letting the matter out by a free incision a better result followed. Of course if the appendix was seen in front it would be difficult to resist the temptation to put a ligature round it and remove it. But in many of these cases the appendix was not seen, but only an abscess cavity, of which it was assumed the appendix formed part. If a formal surgical operation was performed in such a case, it was necessary to do a great deal of dissection, and at what cost? There were broken-down adhesions, and a contaminating cavity, and some of the cases ended fatally. He believed the best treatment was to make an incision, not attempting to remove the appendix unless it was readily seen, evacuate the pus, and put in a gauze drain. Seeing that operation where there was no abscess caused very little danger to life, that if operation were delayed until an abscess had formed it became more hazardous, and that if no operation were done many of the cases were fatal on account of the abscess bursting into the peritoneal cavity, what was the proper course for the surgeon to follow? He was not referring to a very slight attack of appendicitis, but to a fulminating attack. In his opinion the

surgeon should operate as soon as the case was seen. Such cases were very analogous to those of twisted ovarian tumours. The patient was suddenly attacked with illness in both cases, and if operation was done as soon as the condition was diagnosed, the cases almost invariably did well. On the other hand, if no operation was done, how many of those cases got acute sepsis and acute peritonitis or dense adhesions round the part! Any one who was called in to a case of strangulated ovarian cyst which was easily diagnosed, and who did not operate, behaved in a way which was almost criminal, and he would go almost as far in speaking of the appendix in fulminating cases. He felt that if he operated at once the procedure was fraught with very little danger. The case looked formidable, the parts were brawny and swollen, etc., but everything could be identified, and it was possible to operate before the abscess had really formed. With regard to after-treatment, he thought the real question was whether the patient lived or died from the operation, and he believed that those who operated at once when they saw a case of acute fulminative appendicitis before the abscess broke would have better results than if they waited for something to turn up, with the result that only pus turned up; and they would then have to operate under very unfavourable conditions.

Dr. Samuel West trusted it would not seem out of place for a physician to take part in a discussion which had become so purely surgical. Yet only a few years ago typhlitis or perityphlitis was regarded as a medical disease and treated by the physician; of recent years, under the name of appendicitis, it had been transferred bodily to the surgeon. The experiences of both physician and surgeon were necessary in order to arrive at a full understanding of the disease, and that experience naturally presented the subject to each from a somewhat different point of view. Certain questions suggested themselves for consideration. The first was one which had not been referred to in the discussion. Was it a fact that the appendix was an obsolete organ and of no value to the body? They thought this once of the supra-renal capsule, thyroid gland, and pituitary body. They did not think so now. It seemed hardly wise to assume that an organ of complex structure which had triumphed so far in the struggle for existence had no special function to discharge. They might find hereafter that its removal led to symptoms of a kind they did not now anticipate. Anyway, the probabilities were in favour of the view that on the whole the human body was better with an appendix than without it. If the appendix were diseased, the question was different. He was only now protesting against the assumption that the appendix was an obsolete structure, and that it was a matter of indifference to the body whether it were there or not. That might be so, but they did not know

it for a fact, and they could not safely assume it. The next questions concerned the statistics in which this discussion had been so prolific. Statistics were often misleading in medicine and surgery. They could at the most only establish averages or general rules. Skill and experience would still be necessary in order to apply these general rules correctly to individual cases. Had appendicitis become a more common affection than it was? When surgeon after surgeon rose and referred to 100, 200, or 300 cases which had been under his own observation within a few years, the numbers reached such enormous totals that some explanation seemed necessary. The statistics from St. Bartholomew's Hospital were instructive in this respect. The returns for the ten years 1894 to 1903 showed for the first half of that period an average of seventy or eighty cases a year, and for the second half 200 or more, *i. e.* three times as many. There was no reason to suppose that during the last five years the affection had become in any way more common than it was before, nor that there was any difference in the power of diagnosis. The increase was, no doubt, due to the large number of operations for appendix trouble during the quiescent period. These cases ought really to be excluded, for they were not cases of appendicitis at all, but of post-appendicitis laparotomy. They confused and diluted the real statistics and rendered the conclusions inaccurate. Finally, the same case might easily appear twice—first as acute appendicitis, and secondly as an operation in the quiescent stage. The increased frequency of appendicitis of recent years was therefore only apparent and not real. Before the statistics of operation for appendicitis could be appraised at their proper value, it was necessary to compare them with the statistics of appendicitis without operation; they required, in fact, to know the natural history of the disease when left to itself and treated medically. For these statistics they must go back some years. Speaking generally, the cases of appendicitis, typhlitis, or perityphlitis, whichever name they used, fell into three groups, mostly distinguished from one another in old and recent statistics alike—(1) those associated with general peritonitis, often the result of perforation; (2) those in which the peritonitis was local and ended in suppuration or abscess; (3) those in which the local inflammation subsided without suppuration. They required to know the sizes of these groups—*i. e.* the number of cases each contained and their proportion one to another, and lastly the mortality in each group with and without operation. There did not appear to be many statistics available of the kind they wanted. He trusted one of the results of this discussion would be to draw attention to this want and lead to the deficiency being supplied. For the present purpose he might refer to those contained in the excellent monograph of Dr. Hawkins. Of his 264 cases 190 recovered

without operation. Of the remaining 74 cases there was suppuration in 38, of which 10 died; while 36 had general peritonitis, and of these 27 died. If they reduced these figures to percentages they got the following table:

100 Cases

	Without operation, 72.	With suppuration, 15.	With general peritonitis, 13.
Recovery	72	11	3
Death	0	4	10
Mortality rate	0	26%	77%

Recovery	83	87
Death	4	
Mortality rate	4·6%	

In respect of the last two columns it was to be borne in mind that all the severe cases would find their way into the hospital, while some of the slighter ones would not. But making no allowance for this, the figures showed that 72 per cent. of all the cases recovered from the acute attack without operation. This he thought would approximately represent the experience of most of the older physicians who could remember the results of typhlitis when treated medically. Treves himself stated that the majority of cases of appendicitis got well without any operative interference, and Fitz placed the mortality of cases treated medically at 11 per cent. Hawkins' figures yielded a total mortality rate of 14 per cent. If we excluded from Hawkins' figures the cases of general peritonitis, four out of five of which, at least, were fatal, and added the other two groups together, we arrived at a death-rate of 4·6 per cent.; and as in his series the abscess cases had been treated on the lines of modern surgery, this would lead to the conclusion that the death rate of these two groups of appendicitis treated with the minimum of surgery was under 5 per cent. The only risk we had to add to that was that of perforation and general peritonitis, a risk which Treves himself said has been greatly exaggerated. If this were the truth or anywhere near the truth, it should make them pause before recommending indiscriminate operation. One fact seemed to have been conclusively established by this discussion, that the risk of removing the appendix in the quiescent stage was very small, probably hardly more than 1 per cent. In the cases quoted recurrent attacks formed nearly 20 per cent., and as with every recurrence the risk of dangerous complications seemed to increase, the elimination of these cases by operation in the quiescent stage would still further reduce the general risk and mortality of the disease. He did not wish to lay too much stress upon the actual figures

he had given, but if they were even approximately correct, as he thought they were, they should be borne in mind in this discussion. He trusted no one would think he was opposing surgery. He was not afraid of prudent surgery, but he did not like indiscriminate operation, and the opening of the abdomen was not the trifling procedure it was often assumed to be. The art of the physician and surgeon alike lay in determining when operation was necessary and when it could be avoided. Each could learn much from the other, and in this question of appendicitis it was most important that they should be in accord. He did not wish to be misunderstood, and he would like in conclusion to express his own attitude to this subject in the following propositions: (1) The great majority of cases of acute appendicitis got well more or less completely without operation. (2) Some recurred, and with each recurrence the existence of some gross lesion became more probable and the risk of serious complication greater; these were the cases for operation in the quiescent stage, the risk of which was very small, 1 or 2 per cent. (3) Where suppuration was manifest, or from the character of the symptoms even probable, the case should be treated at once on general surgical principles. (4) Cases of perforation or septic peritonitis were frightfully fatal, so that hardly one case out of ten survived. Still, judging from the results of operation for perforated gastric or typhoid ulcer, the mortality of which increased so rapidly with delay that minutes were of vital importance, they might hope that with more immediate operation the results might become less unfavourable than they now were. Unfortunately, many cases came under observation in a moribund state, when no operation was possible. *Per contra*, the risk of perforation occurring in any given case of appendicitis had been exaggerated, and fortunately was not as great as it is often assumed to be. (5) In the acute stage, considering that 70 to 80 per cent. of the cases recovered without operation, it was not necessary to be in a hurry or to operate upon all cases indiscriminately. Each case must be considered on its own merits and dealt with accordingly. (6) The successful results obtained by the surgeons who had taken part in that discussion were not such as the less experienced and less skilful had any right to expect. In estimating the results of operation, the personal factor of the operator must be taken into consideration. The conclusions seemed to be these: that each case must be specially studied and the *pros* and *cons* of operation in each case carefully considered, that general rules admitted only of general application and must be modified as each case demanded, and finally that the most skilful, careful, and experienced would make the fewest mistakes.

Mr. Bruce Clarke said that it had been assumed that there were only two varieties of appendicitis, acute and chronic. He

would not enter into the question of operating in the quiescent stage as he believed all were agreed upon that, but two classes of cases remained—the very acute and the moderately acute cases. Many speakers said they operated in the acute stage, but without saying what that term really meant. The point he thought surgeons must bear in mind was that when the case began to get acute, provided the operation was done early enough, it was then done with almost as much advantage as if it were undertaken in the quiescent stage; in other words, the surgeon operated before the outside of the appendix became thoroughly septic. He had looked over the cases which to him were most instructive, namely, those which he had lost, and he asked himself why he had lost them. He had come to the conclusion the reason was that in some way or other the peritoneum had been fouled. An operation could be undertaken with a reasonable degree of safety on even a very acute case if it could be done without fouling the peritoneum. He therefore felt that it was not advisable to attempt to remove the appendix when suppurating, unless it was under the surgeon's eyes; if it could not be seen or easily found he contented himself with letting out the pus. He remembered two cases which were very instructive; in one the abscess was in the pelvis, in the other case the appendix lay on the outer side of the colon. In the first case he made an incision into the abscess and let out the pus, and the patient made an almost uneventful recovery. In the second he could not get at the pus to let it out as easily, and the consequence was that he had to get the appendix up from below. He tried to prevent the peritoneum from being contaminated, but he supposed that he failed. The same micro-organisms were found in both cases, but one case, the second, died in eighteen hours, while the other recovered satisfactorily. Those two cases made a great impression on his mind, and since then he had contented himself with letting out the pus by the route by which it could be evacuated most easily. If it could be let out through the vagina readily, he made an incision there. In some cases where the suppurating appendix was deep in the pelvis he had made an opening by the side of the coccyx through the ischio-rectal fossa, and in several instances with complete success. His ordinary plan was to have every case submitted to microscopical examination. The conclusions he had come to were briefly the following: he had examined upwards of 200 cases, and found that in 191 there was inflammation present; the epithelium was denuded from the inside of the appendix, and, therefore, the septic material could get through and invade the deeper parts. Two of the cases were tuberculous, leaving 7 in which no denuded surface was found, and in which he assumed he might have been wrong in removing the appendix at all. The appendix was not opened

from end to end when it was examined microscopically, so that the denuded portion might well have been missed. Five of them, so far as he knew, remained well, and two of them came back afterwards with recurrence of the symptoms. One of those two had gall-stones, which he removed subsequently, and the other had renal calculus. Another point which had been raised was as to what happened to some of the cases which were assumed to be appendicitis, but which turned out not to be so. A patient of his had the appendix removed for assumed appendicitis. The patient was not under his care at the time. Six months later he came under his care with a recurrent attack, apparently of appendicitis, stating that he had had more than one attack since the removal of the appendix. Mr. Clarke, having satisfied himself that the appendix had really been removed, kept the patient under observation for a week, during which time he found a swelling just above the region of the appendix. Thinking it was connected with the kidney, he had a separator put into the bladder, and the water from one side of the bladder was in excess of that from the other, scarcely any coming from the right kidney. He thought it probable that the swelling in the right kidney had been mistaken for appendicitis. He opened the abdomen on the right side, but found the kidney healthy, and that the swelling was inside the peritoneum. There was an ileo-colic intussusception; and having with some considerable trouble got that undone, there appeared to be some swelling in the cæcum, which turned out to be a polypus close to the end of the cæcum. He resected the intestine. The patient had practically recovered, and would leave the hospital in a day or two. Were such a case to be met with again, it would very likely deceive the surgeon unless he were aware that such a case had been met with before.

Mr. W. G. Spencer remarked that he had put in a table of 100 consecutive recent operations, mainly for the year 1904, but also for 1903. Some of the other tables put in extended over several years, so that an objection might be raised that all sorts of improvements had been introduced since the earlier years. His own table showed what happened when operation was carried out immediately, and the appendix removed in all cases. Practically all those cases were seen by others before him, and the diagnosis of appendicitis made. Absolute uncertainty prevailed with regard to prognosis in individual cases, and no additional experience afforded him any added power of making a prognosis. Prognosis resting on statistics gave cold comfort to the individual, because he could not be assured that he did not form one of the unfortunate minority. In cases with abscess and peritonitis he believed that considerable improvement might be made by much freer operation, and the filling of abscess cavities with iodoform gauze, as was done by Professor Bergmann in 1886. Richardson, of Newcastle, published four cases following of diffuse general peritonitis in which he stuffed the peritoneum

with gauze and afterwards employed secondary suture. In a case since the table, in addition to the abscess, the peritoneal cavity was designedly opened, and a large pool of septic material was found in the pelvis. The nurses had recorded a pulse of 130; one hour after operation it fell to 104, and three hours afterwards to 84. The wound was filled with gauze and sewn up on the tenth day, and the whole cavity was healing well. He showed one of the most extensive cases at the Clinical Society some time ago. The man had an abscess for the fourth time, but had not been operated upon except for the opening of the abscess. The abscess extended from the last rib to the internal abdominal ring. The cavity was filled with gauze and sewn up on the tenth day, and when exhibited the man had a firm scar. It had been said that in that class the mortality was nine out of ten, but in his opinion, unless there were tympanites in advanced degree and the streptococcal variety of infection, with the employment of freer operation and sponging-out, recovery would be more likely to ensue in nine out of ten cases than in one out of ten.

Mr. MALCOLM said one of the most important points brought out by Sir Frederick Treves was that, with certain exceptions, the principal complications of appendicitis were "those only of septic infection." They were "such complications as may occur in connection with any septic wound." The exceptions mentioned were fæcal fistula, intestinal obstructions, persisting or extending abscess, and pylephlebitis. These, however, were not peculiar to appendicitis, and all except intestinal obstruction, and perhaps some cases of fistula, were due to septic changes. The septic conditions which might arise in connection with appendicitis were, therefore, extremely various and they might tax the ingenuity and skill of the surgeon, as well as the powers of endurance of the patient, to the uttermost. There seemed to be no doubt that, as in other diseases, the surgeon's efforts should be directed chiefly to the prevention of septic complications. Just as complete removal of a diseased appendix was necessary to obviate the possibility of future attacks of inflammation in it, so an early diagnosis and early treatment were necessary to prevent, with certainty, the onset of septic troubles. In regard to this matter the speaker was in complete accord with the views expressed by Mr. Gould, Dr. Hawkins, Mr. Turner, and Mr. Waterhouse. There was a form of mischief in the appendix which had not been mentioned in the debate, but which seemed to throw much light on the pathology and on the indications for treatment of disease of that part of the bowel. In the cases referred to the onset of symptoms was extremely insidious and deceptive, yet the patients died with certainty if they were not operated on, and if an operation was performed a gangrenous condition of part of the wall of the appendix was revealed. A typical case of this kind

was related. The patient, a lady, aged 71, complained of pain in the right side, and in the course of about thirty hours her temperature gradually rose to 100°, whilst the pulse remained below 90. The bowels did not move, but the patient's habit was very constipated at all times. During the following twelve hours the abdomen rapidly distended, vomiting commenced and was soon continuous and the patient became greatly exhausted. The pulse rose from below 90 to 130; the temperature only rose to 100·6°. The abdomen was opened forty-four hours after the illness began. The vermiform appendix lay outside the ascending colon, above the crest of the ilium and in contact with the small intestines. It had three separate gangrenous patches in its wall. Around it, in the peritoneal cavity, there was some flaky lymph, and there was a collection of serous fluid lying in the loin pouch, but not in any way shut off from the general peritoneal sac. The serous membrane at a little distance appeared to be healthy, but the intestines were over-distended. The patient died on the day after the operation. The sloughs in such cases were usually single, round, and not more than a quarter or three eighths of an inch in diameter. In two cases in which the disease began with the very insidious symptoms referred to, the gangrenous patches remained in contact with the living tissue and there was no rupture of the appendix. The speaker had seen several cases of limited gangrene, but in the tables of statistics which had been distributed such conditions had for the most part been ignored or included with other cases under the heading "gangrene" or "sloughing." The condition to which attention was directed was altogether different from a gangrene of the whole appendix such as had been described by Mr. Harrison Cripps. No. 190 in Mr. Lockwood's list was the only case of the kind noticed. On thinking over these cases, and especially the one shortly related, the suggestion arose that the gangrenous patches did not depend on inflammatory changes. An inflammation terminating in gangrene was a very strenuous affair. It was attended by an increasing pulse rate. The pulse beats were full and bounding at first, but became weak and feeble later. The temperature rose to 103°, 104°, or higher. The tongue was furred, perspiration might be profuse, but more often the skin was hot, dry, and harsh. The inflamed area was tender, and if the parts were exposed there was unusual vascularity and obvious swelling of the tissues in the neighbourhood. The symptoms of such a sthenic inflammation progressed in severity for about three days, before the process terminated in recovery, in pus-formation, in sloughing, or in death of the patient. This series of symptoms was very different from that observed in the case of the lady whose surgical history had been briefly stated. In her case the symptoms were those of a very slight inflammation, the acute symptoms were all due to irritation and paralysis

of the intestines, and at the end of forty-four hours three well-defined sloughs were found in the wall of the appendix. In Mr. Lockwood's case to which reference had been made a gangrenous patch was found after sixteen hours. The evidence seemed to show that these gangrenous patches formed without pain or symptoms of any kind. There were other conditions in the human body under which a spontaneous, painless, localised gangrene took place; for example, this occurred now and then in the walls of ovarian tumours, and senile gangrene was sometimes painless. But in connection with the subject under consideration it was more important to note that a sloughing without pain or other symptom occurred not very infrequently in the wall of the stomach or of the duodenum. The first sign of sloughing in these organs might be a profuse hæmorrhage or the onset of symptoms of perforation. It had been said that the appendix was allied in its structure and functions to the principal digestive organs, and Sir William Macewen laid much stress on this view in his address in opening the session at Charing Cross Hospital in October, 1904. It seemed that the resemblance between them extended to their pathology, and that both were liable to a spontaneous, painless, localised, non-inflammatory sloughing of their walls. In both, these sloughs might be simple or multiple. The spontaneous occurrence of a painless gangrene in the wall of the appendix, if separation of the slough was delayed, would satisfactorily account for all the symptoms and conditions found in the cases in which the illness began very insidiously. After the slough had formed without symptoms a filtration of infective material took place through it, and gave rise to a slight septic diffusing peritonitis of an asthenic character. When this affected the small intestine with sufficient intensity, a paralysis of the gut and abdominal distension were induced, and if the intestine could not recover its tone death was certain whether an operation was performed or not. Another class of cases might be accounted for by this spontaneous sloughing. If there was any obstruction to the exit of the contents of the appendix into the cæcum, the intra-mural pressure thus induced, when combined with the spontaneous formation of a gangrenous patch, would be very apt to produce a rupture at the area of sloughing, and in this way some of the rapidly fatal cases of so-called appendicitis might be intelligibly explained. Mr. Lockwood's case, which he happily saved, seemed to illustrate this condition well. A gangrenous patch in the appendix and pus in the iliac fossa and pelvis were found sixteen hours after the attack began. Such cases did not exhibit the symptoms of an acute inflammation terminating in gangrene. The [illegible] to be due to an inflammatory [illegible] of the stomach, [illegible] a gangrenous patch [illegible]

if these views were new, but he had not seen or heard of them except in conversation with his friend Dr. Andrew Elliot, to whom the same idea had occurred. He was quite sure that this explanation of some of these conditions deserved attention. It was obvious that in some of these cases an immediate operation was as urgently necessary as it was in the case of a perforating gastric ulcer. Moreover, the mortality from operations for such conditions in the appendix should be exceedingly low, if operative treatment was undertaken sufficiently early, before the small intestine was paralysed. But the symptoms might be of the most insidious or of the most acute character, and it was well-nigh impossible to differentiate these cases with certainty from those of true inflammatory mischief. Hence the conclusion was forced upon them that if these cases of spontaneous gangrene were to be saved, it might be necessary to make it a rule to operate as soon as possible, whenever appendix mischief was diagnosed. It was, therefore, of the greatest importance to determine whether such early operative treatment would prove harmful to the other and more numerous cases of true inflammation of the appendix. As the inflammation in these cases began in the mucous membrane, it seemed that by operating sufficiently early, it must be possible to remove the whole disease, and so to clear away the source of septic mischief, just as an ovarian tumour strangulated by twisting of its pedicle might be cleared away. But to do this with the appendix the operation must be performed very early. By waiting until it was certain that an operation was necessary, although many patients might be saved from surgical interference altogether or for a time, it was impossible to be sure that the only chance of helping them was not being lost, or that some dangerous complication would not arise. Moreover, if an unsuccessful attempt was made to tide the patient over the acute condition, those cases in which surgical treatment became necessary would be subjected to it at the worst possible time—that is, when the evil effects of the initial inflammatory mischief had fully developed. Individual cases must, of course, be treated on their merits; but it seemed that the only cases in which it was quite safe to delay operative treatment were those in which there was a small, well-defined swelling, the intestines were not affected, the symptoms were those of a slight but definite inflammation, and the patient was evidently already recovering when first seen. In advancing and especially in doubtful cases very early interference was the safer practice.

Mr. F. C. WALLIS referred to the condition of the omentum in cases of acute appendi is, whether associated with suppuration or not. As a rule, t mentum quickly became attached, and formed a safeguard peritoneal cavity, but in some cases the omentum, althou infiltrated with inflammatory the operation, formed any

adhesions. It was important to either fix the omentum, or better still, to remove it in such cases, otherwise it was liable to form later attachments to the intestine, and might cause intestinal obstruction. He related a case in which, after an abscess had been evacuated and the appendix removed, signs of incomplete obstruction arose, and it was necessary to open the abdomen again, when it was found that the small intestine had become adherent to the omentum in two places; these were separated and the patient then made a good recovery. There was a danger that it might become a fashion to allow patients to think themselves well ten to fourteen days after the operation. This was true enough as far as the healing of the wound and any fear of ventral hernia was concerned, but much had been said about mucous colitis, and other intestinal troubles which were frequently associated with appendicitis. If patients were indiscriminately allowed to walk and take exercise too early, only harm would come of it in such cases. Moreover, all patients, however well they might do if kept quiet, were liable to have a nervous breakdown if allowed to do too much too soon after a severe operation.

Dr. NEWTON PITT remarked that Dr. Samuel West's conclusion that appendicitis was not now more frequent than formerly did not accord with the Guy's Hospital figures or with his own experience. When he took out the statistics some years ago, they showed that the disease was much more frequent than twenty years ago and the total mortality greater in spite of improved treatment. There was also a marked increase in the year 1889 or 1890, when influenza was very rife. He did not believe that those increased figures merely represented the increased activity of the surgeon.

[Owing to unavoidable absence, Mr. Mayo Robson, who had been invited to take part in the discussion, sent the following communication, which was read by Mr. STEPHEN PAGET:]

DEAR MR. PRESIDENT,—I thank you for your invitation to take part in this important discussion on the complications following appendicitis, and am sorry that, owing to the shortness of the time between your invitation and the time of the meeting, it was impossible for me to write to all my patients and ascertain their condition subsequent to operation. It seems to me that in considering the results and the complications subsequent to operation, it is convenient to classify the cases into (*a*) fulminating cases associated with general peritonitis, speedily ending in death within two or three days, unless operated on; (*b*) acute cases associated with suppuration or gangrene; (*c*) chronic or

interval cases. Sir Frederick Treves has taken the last class of cases first. In operations undertaken in the interval between the acute seizures, since rigid asepsis can be followed out and the operation can be undertaken in a limited area, there should be no mortality unless from some exceptional complications, such as intestinal obstruction from pre-existing adhesions, or from some unavoidable heart or lung complication. This is also shown by Sir Frederick Treves's experience and that of others who have spoken, as well as by the experience of some of the American surgeons. I think, therefore, that the estimate suggested by one of the speakers, which places the mortality of appendicectomy in the quiescent cases at 2 per cent., is excessive. Of the complications following operations in the quiescent period, intestinal obstruction, thrombosis, and hernia seem to be the most important. I have only once seen intestinal obstruction the result of old adhesions come on after the wound had healed, and necessitate an enterotomy. With regard to thrombosis, this complication, which figures largely in the London hospital statistics, must, I think, be due to keeping the patient too long in one position. I have certainly not seen it oftener after appendix operations than after any other abdominal section, which is very seldom. I always allow my patients to be daily turned on the right side, and I think this may have something to do with the prevention of thrombosis, as Mr. Charters Symonds has also suggested to be the case in his practice. With regard to hernia, this is also a preventible complication, and I have made inquiries of my house-surgeons and of the resident surgical officers, who have seen my hospital patients subsequently, and they tell me that they do not see herniæ after the operation performed without division of muscular tissue. This has also been my experience in my private cases, though I cannot say the same for those cases in which, some years ago, I performed the operation by dividing the muscles. From the number of times that I have been consulted for ventral herniæ after the division of muscles, even by very capable operators, I feel sure that hernia must occur much more frequently than those surgeons are aware who regularly practise division of muscles. Contrary to what has been said by some of the speakers, I know that it is quite feasible to remove the appendix and to explore the uterine appendages, the right kidney, and the gall-bladder through an opening made without division of the muscular fibres, either by means of the McBurney incision, or by one through the rectus sheath, with displacement of the rectus inwards and division of the posterior part of the sheath separately. Through this incision I have on several occasions not only removed the vermiform appendix, but also the right uterine appendage, and on other occasions I have at the same time that I removed the appendix performed chole-

cystotomy. After opening the abdomen without division of muscles the patient can conveniently be turned on the right side, and is able to be up on the sofa within a fortnight. With regard to symptoms not being relieved after operation, this may arise from three conditions: (1) Mistaken diagnosis, as, for instance, mistaking cholelithiasis, chronic pancreatitis, duodenal ulcer, renal calculus, right ovaritis, and colitis for appendicitis. This may usually, but not always, be avoided by more care in diagnosis before operation. (2) The double disease where the appendicitis exists along with one or the other condition. This difficulty may usually be overcome by carefully examining the uterine appendages, the kidney, and gall-bladder region at the time the appendix is removed. (3) The persistence of nervous and other general symptoms associated with neuralgic pains which may be dependent on adhesions following operation, or on the division of nerves, or on the persistence of nervous symptoms subsequently, all these being conditions which will usually yield to general treatment without further operation. I very thoroughly agree with what Mr. Pearce Gould has said concerning acute appendicitis generally, that if operation were undertaken for these conditions at the earliest possible moment we should hear much less of the very many serious septic complications which follow operations for removal of the appendix in acute cases. These complications, with the exception of fæcal fistula and thrombosis of the femoral vein, are probably all dependent on septic absorption present before operation, and are not dependent on the operation itself. I refer to such conditions as pylephlebitis, subphrenic abscess, liver abscesses, residual secondary abscesses, and septic thrombosis and embolism. The adoption of this early treatment gives so much better results, as shown by some of the American statistics—for instance, in Dr. Ochsner's practice at the Augustana Hospital, Chicago; out of 255 acute appendicitis cases without perforation there were 5 deaths, a mortality of 1·9 per cent.; of 86 cases of acute appendicitis, perforation occurring without abscess, there was no death; of 117 [illegible] perforated with abscess [illegible] or not; but of 35 [illegible] there were [illegible] very little [illegible] operation, [illegible] fistula has [illegible] removal of [illegible] If when the [illegible] out before [illegible] will often be found [illegible] I have on several [illegible] under these circumstances [illegible]

collections of pus before I reached the gangrenous appendix. In all these cases, had I left the appendix, secondary abscesses would certainly have formed, and there would all along have been a danger of the peritoneum being invaded or of other septic complications. With regard to fæcal fistula, if the root of the appendix be crushed close to the cæcum and the stump ligatured, fæcal fistula will seldom occur, and much more rarely if at the same time the stump can be buried by a purse-string suture, which, however, is not always possible, when the parts are rigid from inflammation. Of the fulminating cases, where there are no barriers of lymph limiting the inflammation, and where the general peritoneum is involved, it is both useless and dangerous to wait, and if the patient is seen within twenty-four hours of the attack, and the abdomen is opened and thoroughly flushed with hot saline solution after the appendix has been removed, these cases are not so hopeless as they appear to be and a very fair proportion may be saved. If the peritonitis is still localised to the right side, though not bounded by a barrier of lymph, wiping out the cavity instead of flushing is, I think, the better practice. In this condition it is important to clear the peritoneum before the system has absorbed a lethal dose of poison, and should the peritonitis have been present for more than forty-eight hours, and paralytic distension of the intestines have already supervened, the mortality, with or without operation, will be very great indeed. These fulminating cases may be likened in some respects to perforating gastric and duodenal ulcer, when every hour's delay before operation adds to the risk. I recently operated on two cases of general peritonitis due to perforative appendicitis within twenty-four hours of operation, one in a patient aged 70, and another in a boy aged 15. In both cases removal of the appendix and lavage of the abdomen resulted in recovery, whereas in two cases operated on some days after the onset of general peritonitis operation merely expedited the fatal termination.

The PRESIDENT (Sir RICHARD DOUGLAS POWELL). I much regret that Sir Frederick Treves is prevented from attending this evening to conclude the discussion, and I am, therefore, obliged to make a few concluding remarks. I think all will agree that this debate has been of extreme value. The subject of it has ripened through many years of experience, discussion, and criticism since ... first propounded at this Society by Sir Frederick ... in 1887; and it was particularly appropriate, therefore, ... should have introduced and reviewed it on this occasion. ... been gratifying to him and to many other surgeons, ... spoken in this debate, to have watched the ... domain of surgery to its present state of ... value of a discussion such as we have ... prolonged and well-attended meetings,

is that it has for that time at least concentrated the attention of both surgeons and physicians upon one particular problem, in this case as to the best way of rendering the results of operations for appendicitis most satisfactory, and of avoiding untoward complications. Many minds will have been settled as to the best modes of procedure, many points decided in the course of the debate, and with the aid of the large statistical experience with which it has been enriched. (1) One point, for instance, insisted upon by Sir F. Treves, and strongly advocated by Mr. Pearce Gould, Sir W. Bennett, Mr. H. Cripps, and Mr. Bruce Clarke, was that in cases of perityphlitic abscess it is best as a rule not to try to remove the appendix when imbedded, as it generally is, in entangled adhesions. (2) Another point which seemed to me to come out very strongly in the debate, and was referred to by Sir Frederick and, I think, especially alluded to by Dr. Hawkins and Mr. Battle, was the fact that failures and subsequent complication of the operation are sometimes attributable to its not having been adequately performed—*i. e.* to the appendix not having been excised from its very origin at the cæcum, but only a portion cut off, and that this incompleteness of the operation is not infrequently due to the cæcal end of the appendix being partially invaginated. (3) Then, again, there is the very difficult question, which has been touched upon by more than one speaker, *viz.* when to operate; and this is a question that is also of great interest and importance to the physician, who otherwise is perhaps more concerned with the conditions that lead up to appendicitis, which were not within the scope of the debate. When the diagnosis of perityphlitic abscess has been arrived at, of course the operation practically immediately follows. Mr. Cripps would not wait for abscess, but would advise operation in the earlier stage of appendicitis. But great emphasis has rightly been laid by many speakers, especially by Mr. Gould, Mr. Turner, and Mr. Waterhouse, upon the importance of anticipating abscess by dealing with the appendix in what is called the quiescent period of appendicitis. This view must, of course, be guarded by great care in diagnosis; for whilst on the one hand if diseased appendices are allowed to remain there is the risk at any moment of abscess with general peritonitis, with the result of raising the mortality of operative interference to about 25 per cent., instead of 2 per cent. or less, for the simple operation; on the other hand, healthy appendices are sometimes removed, leaving behind the malady from which the patient has been promised relief. The danger must be recognised that the operation may be too lightly undertaken, and may become too much of a routine practice, and not sufficiently safeguarded by careful and accurate diagnosis. (4) Future complications in quiescent cases may, I ventured to hint, be sometimes avoided by a careful preparation of the patient for a

few days before the operation. (5) I think the Society will feel especially indebted to Mr. Lockwood for his most valuable contribution to the debate by illustrating so clearly on the screen those points in the pathology of diseased appendices which are so intimately concerned with the future of the cases operated on. (6) Another point of great importance to the future comfort of the patient was touched upon by several speakers, viz. especially in cases requiring large incisions, the avoidance as much as possible of cutting across muscular fibre, and the importance of separating rather than dividing the muscles with the view of obviating ventral hernia and the necessity of afterwards using a large and cumbersome truss. I have only ventured to refer to a few of what seemed to me, as I listened to the discussion, the most important questions which were touched upon and determined by it. I must not let this opportunity pass without offering the sincere thanks of the Society to those gentlemen who have taken part in the debate and especially to those—Mr. Hugh Lett, Mr. Battle, Mr. Gask, Mr. Baldwin, Mr. Spencer, Mr. Barling, Mr. Lockwood, Mr. Lawrence Jones, Dr. Hawkins, Mr. Clogg, and Mr. Fairbank—who have at great labour prepared the statistics of some thousands of cases, which have been brought before the Society.

APPENDICITIS

AT

MIDDLESEX HOSPITAL

STATISTICAL TABLES OF CASES OF OPERATION FOR APPENDICITIS

FURNISHED BY

ASLETT BALDWIN, F.R.C.S.Eng.

SURGICAL REGISTRAR TO THE HOSPITAL

1902.

Appendectomy for Relapsing Appendicitis.

26 cases recovered without any complication.

1 case died. Laparotomy for intestinal obstruction was performed the third day after the primary operation. Age 40. P.M.—Some peritonitis.

1 case aborted the ninth day after the operation; recovered.

had a superficial abscess in the abdominal wall; vered.

Abscess.

bscess was opened and drained, the moved; recovered without com-

pened and drained, the fluid appeared in right rther surgical treat-

1 case, the abscess was opened and drained, the appendix not removed; a sinus persisted for 8 months, which was opened; a suture removed; recovered.

1 case, the abscess was opened and drained, the appendix not removed; 24 days after primary operation symptoms of acute peritonitis. Laparotomy was done; miliary tubercles found on peritoneum; recovered.

13 cases, the abscess was opened and drained; the appendix was removed; recovered without complications.

1 case, the abscess was opened and drained; the appendix was removed; fluid appeared in right pleura; recovered without further surgical treatment.

1 case, the abscess was opened and drained; the appendix was removed; died. P.M.—Peritonitis. Age 38.

Appendicitis with General Peritonitis.

1 case laparotomy and appendectomy; recovered without complication. Age 14.

1 case appendectomy; abdomen washed out; 30 days after primary operation pus discovered in left pleura; rib resected [illegible] recovered. Age 13.

1 case died [illegible] tomy. Age 10.

1 case died [illegible]

1 case died [illegible] patient [illegible] appendicitis [illegible] No P.M.

1903.

Appendectomy for Relapsing

53 cases recovered without

1 case recovered; a sinus persisted, which healed after three sutures had been removed.

Appendicitis with Abscess.

19 cases, the abscess was opened and drained, the appendix not removed, recovered without complications. Two of these were the same case, readmitted after two months. On the second occasion the abscess was opened both through the abdomen and through the thigh. Readmitted in September, 1904, with sinus. Appendix found and removed; it was very large and contained pus. Many adhesions. Three small abscesses formed under the scars which were opened. On December 17th, 1904, the sinuses were freely opened up. Patient left hospital February 2nd, 1905, with a sinus 2¼ inches long, discharging a little non-offensive material.

1 case, the abscess was opened and drained, the appendix not removed; died. P.M.—peritonitis. Age 43.

9 cases, the abscess was opened and drained; the appendix was removed; recovered without complication.

1 case, the abscess was opened and drained; the appendix was removed; died. Age 21. P.M.—peritonitis.

1 case, the abscess was opened and drained; the appendix was removed; died. P.M.—Subphrenic abscess. Age 18.

1 case, the abscess was opened and drained; the appendix was removed; died. Cerebral softening. Age 51. Embolism of right middle cerebral artery (clot probably embolic).

[illegible] abscess was opened and drained; the [illegible] was removed; died. Pulmonary embolism of right common and internal [illegible] 30.

Appendicitis with General Peritonitis.

1 case, died; after appendectomy. Age 12. No P.M.

1 case, died; after laparotomy. Age 28. P.M.—peritonitis.

1 case died; after appendectomy. Age 8. P.M.—Peritonitis.

1904.

Appendectomy for Relapsing Appendicitis.

45 cases recovered without complication.

Appendicitis with Abscess.

16 cases, the abscess was opened and drained; the appendix not removed; recovered without complications. One readmitted five months later for sinus, which healed after scraping twice. One readmitted in three weeks for small sinus, which got well without operation.

4 cases, the abscess was opened and drained; the appendix not removed; died. (1) P.M., age 30, appendix abscess. (2) P.M., age 30, abscess and peritonitis. (3) Age 7, no P.M. (4) Age 16, a second abscess was opened eighteen days after the first. P.M.—Peritonitis.

11 cases, the abscess was opened and drained; the appendix was removed; recovered. In one case an abscess formed three times under the scar. Sinuses formed which healed after being freely opened.

2 cases of residual abscess opened and drained; recovered. One had abscess opened and drained six months previously, the appendix not removed. One had appendicitis in 1899, a second attack in February, 1904. In April, 1904, an operation was done for removal of the appendix. It was not found, but two concretions were removed. In September the fibrous remains of the appendix

were removed. Admitted December, 1904, with abscess in left side of abdomen, which was opened and drained.

Appendicitis with General Peritonitis.

1 case, the appendix was removed; recovered. Readmitted three months later with sinus, which healed after a suture had been removed. Readmitted February 18th, 1905, for pain and tenderness and some rigidity in appendix region. Temp. 102·2°. Been in bed for a week. Had pain in right side of abdomen about once a week, which lasted two to three hours since leaving hospital.

3 cases the appendix was removed; died. (1) Age 20; no P.M. (2) Age 9; no P.M. (3) Age 34. P.M.—Peritonitis.

I have made three tables of all the cases operated upon for appendicitis in the Middlesex Hospital during the years 1902, 1903, 1904, with the results up to the patient's discharge from hospital. I have been furnished with 100 printed letters asking patients for their after-histories. These I have sent out as follows:—A letter has been sent to all the cases operated upon in 1903, to those operated upon for abscess without removal of the appendix in 1904, and to several of the latter cases in 1902, with the following result:

1903.

(1) *Appendectomy for Relapsing Appendicitis.*

38 cases quite well.

1 case, a little aching when weather changes; otherwise well.

1 case has pain after exertion; otherwise well.

2 cases have some bulging of the scar; otherwise well.

1 case, some weakness about site of operation; otherwise well.
11 cases, no answer.

(2) *Abscess opened and drained, Appendix not removed.*
9 cases quite well. (1 died of pneumonia in January, 1904.)
3 cases have pain occasionally on right side.
3 cases well, but have hernia at site of scar.
1 case, no reply.

(3) *Abscess opened and drained, Appendix removed.*
6 cases well.
3 cases, no reply.

1904.

Abscess opened and drained, Appendix not removed.
11 cases well.
1 case well, but has hernia at site of scar.
1 case well, but had to have sinus scraped.
2 cases, no reply.
1 case had a sinus; died at home Oct., 1904. ? Cause.
1 case well, but has a sinus.

1902.

Abscess opened and drained, Appendix not removed.
2 cases quite well.
1 case well, but sometimes has sharp pain in right side of abdomen.

I have to thank the surgeons in charge, Mr. Henry Morris, Mr. Andrew Clark, and Mr. Pearce Gould, for kindly giving me permission to use their cases.

ASLETT BALDWIN.

SUMMARY OF RESULTS OF FORTY-FOUR CONSECUTIVE CASES OF APPENDICITIS TREATED BY OPERATION

BY

A. S. BARLING, M.R.C.S., L.R.C.P.

SURGEON TO THE ROYAL LANCASTER INFIRMARY

During the last five years I have operated on 44 cases of appendicitis during the acute stage. There were 9 deaths, all due to septic absorption and shock.

The complications met with were—

Empyema . . .	1
Acute obstruction . .	1
Cholecystitis . . .	1

The empyema was on the right side. The first symptoms pointing to the chest appeared six days after the drainage of an adherent abscess. The pleura was opened in the usual manner, and was found to contain very fœtid pus and gas. No opening existed through the diaphragm. A piece of the seventh rib was excised, and the child recovered.

The case of acute obstruction occurred in a boy aged 7 years, and was due to a coil of ileum being strangulated by a band in the *left* iliac region. The child had had an abscess, in connection with the appendix, drained six weeks previously. The wound had healed, and the patient had been running about for a fortnight when the obstruction took place. He was operated on three days afterwards, and eventually recovered.

Another interesting complication took place in a young man. Twelve days after an abscess had been drained

the temperature, which had been normal for seven days, began to rise, and a tender area developed over the site of the gall-bladder. This was soon followed by a distinct localised swelling, four inches by three inches, which moved with respiration, and had a very distinct margin. There was sickness, and constipation. The abdomen was flaccid, and the wound was draining efficiently. The temperature varied between 100° and 101° F. for six days, when it began to fall, and, at the same time, the tumour lessened in size, and in three days had disappeared.

As regards the after-results in cases where the appendix has not been removed, I have examined or heard from twenty-five patients during the last few weeks. In no case has there been any recurrence, and their general health remains satisfactory.

SOME CASES OF APPENDIX (LOCALISED) SUPPURATION.

BY

WILLIAM HENRY BATTLE.

A.—In five cases there was a return of symptoms after a localised collection of pus had been evacuated and the patient apparently recovered.

1.—E. M—, aged 45. History of attacks of appendicitis for five years, then an abscess which burst into the rectum, and during the same illness one which required incision in front. Later there was a return of symptoms on three occasions, so appendix removed March 30th, 1900.

Appendix enlarged, thickened, kinked, old perforation at the bend, closed by omentum. Interior dilated and strictured.

2.—F—, aged 43. A sudden attack of abdominal pain with vomiting, followed a week later by abrupt collapse. Temperature not taken for some days, collapse so extreme. Large abscess evacuated. Healed slowly, and for some time pus came on pressure from region of umbilicus. Here adhesions formed, producing obstruction of the bowels, relieved by operation. Later, symptoms of appendicitis developed, and the appendix was removed.

Appendix twisted at right angle. Strictured with dilatation beyond, and scar of old perforation where small gut was adherent.

3.—F—, aged 39. Was in St. Thomas's Hospital September, 1899, when appendix abscess opened. Taken to Royal Free October 23rd, 1900, having had three attacks of illness and abdominal pain. Appendix removed October 25th. It was adherent in the right iliac fossa. Its walls were so thick, as a result of chronic inflammation, that the lumen was almost obliterated.

She had afterwards some painful attacks in the lower abdomen, and cystic and adherent ovaries, etc., were removed in St. Thomas's. After this she quite recovered.

4.—M—, aged 26. Appendix abscess in November, 1902, which discharged through the scrotum. Symptoms of appendicitis on February 24th, 1903. Removal of appendix March 10th. Dense adhesions. Three omental bands ligatured and removed. A few pockets of pus were found amongst the adhesions.

Appendix dilated, contained pus. A perforation sealed by plastic lymph quarter of an inch from tip.

5.—F—, aged 24. On January 23rd, 1905, a large pelvic abscess incised and drained. She left on January 21st, and was sent back for symptoms of appendicitis on February 14th. Appendix removed. It hung down into the pelvis, and was kinked and adherent.

B.—Cases in which a second abscess formed, the appendix not having been removed at the first operation.

6.—M—, aged 40. Appendix abscess incised July 10th, 1899. He was re-admitted and a sinus scraped in September. This closed, but an abscess formed and was opened in November. Another abscess formed, he was re-admitted on February 20th, and the appendix excised on February 22nd, 1900.

It was adherent to the pelvic wall, and separated with difficulty. Its walls were much thickened, and in some parts cicatrised; it contained a concretion.

7.—B. M—, aged 14. Seen in consultation February 21st, 1901. Symptoms commenced five days before. Abscess opened and drained February 22nd. Appendix not seen. The parents refused to have appendix removed. In June, 1901, an acute abscess developed. The surgeon who operated removed the appendix. Fæcal fistula followed, but boy recovered.

Appendix large, dusky red. Scar of old perforation.

8.—M—, aged 11. Admitted to a fever hospital, and transferred to St. Thomas's on January 3rd, 1903, with a statement that there had been a discharge of pus from the rectum on December 21st. He had been there for seven days, having been admitted for supposed typhoid fever. On January 1st there had been a return of pain, and on February 7th, after admission, an abscess over the appendix was opened. The appendix was excised on March 18th.

It was bent on itself, adherent anteriorly to scar and to the omentum. The terminal inch was completely cut off by the fibrous constriction, apparently at the site of a previous perforation, and in it there was a small collection of pus. In the proximal part there was a "date-stone" fæcal concretion.

9.—F—, aged 24. Admitted to St. Thomas's Hospital May 2nd, 1902. Attack of pain six months before, but only in bed two days. Symptoms of present attack ten days. Abscess incised. Re-admitted with another abscess on June 25th, which was incised, the appendix being removed later.

It was dilated, and contained a large fæcal concretion. The end was adherent to the cicatrix in abdominal wall, and had been perforated.

10.—M—, aged 27. Admitted to St. Thomas's February 27th, 1903, and appendix abscess (nine days' duration) opened on the 28th. Re-admitted on April 30th, 1904, and another abscess of five

days' duration opened May 2nd. Appendix removed May 20th.

It was two inches long, walls very thick and hard, lumen difficult to define, and at one part completely obliterated.

C.—Cases of sinus formation after localised abscess had been opened and drained.

11.—F—, aged 23. Girl, admitted to St. Thomas's Hospital with appendix abscess. Wound closed to a mere track leading to iliac fossa, which would not heal. A concretion was struck with a bullet probe and removed with urethral forceps. The sinus closed.

Appendix not removed.

12.—M—, aged 19. Admitted St. Thomas's in 1895 with a sinus following an operation in Manchester for appendix abscess. This was slit up and packed. Recovery followed.

He was re-admitted April 13th, and died April 17th from peritonitis, after an operation for the relief of intestinal obstruction due to fibrous contraction of the ileum.

No appendix found.

13.—M—, aged 22. A soldier admitted for a sinus in the right side of the abdomen, St. Thomas's Hospital, July 31st, 1899. Appendicitis in May (Ireland). Second attack (Salisbury Plain) August, third attack on board troopship, and was landed at Port Said in November, 1898, when abscess was opened. Appendix removed September 7th, 1899. Left cured September 29th.

Appendix.—A fæcal concretion, the size of a cherry stone, situated three-quarters of an inch from its base, and at its proximal side a complete stricture. At the tip there is a perforation of the mucous membrane, but the peritoneal covering is intact. There were many adhesions.

14.—M—, aged 22. Admitted to St. Thomas's Hospital, March 1st, 1901, when an abscess of some fourteen days' duration was incised. Left on the 20th. A small sinus had not healed, and was scraped April 13, and packed. This was repeated in May, but without success. On May 24th appendix removed. Wound closed without trouble, and he left on June 20th.

Appendix.—Adherent to the abdominal wall at the end of the sinus, and curled up. Chronic inflammation with dense walls and omental adhesions.

15.—F—, aged 40. First attack of appendicitis March, 1903, second attack in May, and abscess opened by median incision on the 18th of that month. Sinus in mid-line persisted. Probe passed down in direction of cæcum, discharge fæcal. Operation October 21st. Removal of appendix through incision through rectus sheath. Very dense adhesions, which completely enveloped appendix.

Appendix large, subject of chronic inflammation, with much thickening. The sinus opened directly into it.

16.—F—, aged 35. Appendix abscess opened January 5th, 1905. Sinus persisted; operation for removal of appendix February 17th. History of attacks of abdominal pain of unknown nature.

Appendix opening into granulation-lined cavity, very much thickened and friable.

D.—In two cases operation was performed for commencing peritonitis when there had been previous localised suppuration.

17.—F—, aged 14. Underwent operation by median incision for peritonitis in a London hospital in May, 1900. She was then sent to a convalescent home, and there an abscess formed, which was opened through the right linea semilunaris in July. She was admitted to St. Thomas's on December 21st of the same year for commencing peritonitis with three days' history of pain and the presence

of free fluid in the abdomen. Incision with displacement of rectus. Lavage.

Appendix—some old adhesions—contained a concretion over which there was a recent perforation.

18.—F—, aged 21, nurse. She had undergone operation in December, 1901, for appendix abscess, Morrison's incision being used. Operation for peritonitis twenty hours after commencement of symptoms, April 30th, 1902, Horton Manor Asylum. Removal of appendix, lavage, drainage. Much free fluid.

Appendix pelvic in situation. Chronic catarrhal inflammation. A large perforation with sloughy edges.

E.—Cases in which the appendix was removed when the abscess had closed and imflammatory symptoms subsided.

19.—F—, aged 14. Admitted to St. Thomas's Hospital for ventral hernia January 15th, 1901, the result of operation for appendix abscess twelve months before. At the operation for cure of this hernia the appendix was removed.

Appendix. Two inches in length, adherent to cæcum. Evidences of former inflammation, lumen not obliterated but cæcal end very narrowed. Some adhesions of omentum to scar.

20.—M—, aged 12. Admitted to St. Thomas's Hospital, November 8th. Pelvic abscess opened through the mid-line. First attack, fæcal discharge from wound for about a fortnight; wound closed. Readmitted February 10th, 1902, and appendix removed February 14th.

Appendix to outer side of cæcum, apex being twisted forwards and outwards, strictured, and containing two concretions.

21.—F—, aged 22. Was in St. Thomas's Hospital November 8th to December 18th, 1903, and an appendix abscess opened; first attack. A con-

cretion was passed by the wound eight days later. On March 2nd, 1904, the appendix was removed.

Appendix contained a concretion, beyond which there was a complete stricture at the site of a former peforation; beyond this, again, there was a cavity lined by mucous membrane, which occupied the distal half inch.

22.—M—, aged 18. An appendix abscess was opened in St. Thomas's Hospital on the 13th March, 1904. On the 12th April the appendix was removed.

Appendix adherent to under and inner part of the cæcum; lumen patent; remains of former perforation visible.

23.—M—, aged 36. Was in St. Thomas's Hospital May 5th to May 30th, 1902. Appendix abscess opened. Appendix removed at private surgical home, June 19th, 1902. Operation was a difficult one, there being many and firm adhesions.

Appendix lying towards pelvis in indurated and thickened omentum. Chronic inflammation with much enlargement. It contained a concretion which was easily felt when the iliac fossa was first examined.

24.—M—, aged 57. In St. Thomas's Hospital, June 17th to July 18th, 1902, for a large appendix abscess which was opened and drained. Was readmitted August 15th, and the appendix removed August 19th. He left September 3rd.

Appendix surrounded by adhesions; chronic inflammation.

25.—M—, aged 7. Was admitted to St. Thomas's Hospital November 1st. Appendix abscess opened November 4th. Appendix removed November 28th, and he left December 12th.

Appendix very large, thick and hard, size of a little finger. Chronic inflammation. There was a scar of former perforation.

F.—Cases in which the appendix was removed after abscess had apparently given way into the bowel, or been opened *per rectum*.

26.—R—, aged 50. Stout man, seen in consultation March 27th, 1903. Gave the following history:—Eighteen months before an attack of severe abdominal pains with vomiting. Five weeks ago similar attack. Swelling three weeks. Hard, very tender swelling with rounded outline on right side, between umbilicus and ant. sup. sp. Movable on deeper parts, but attached to linea semilunaris. March 29th, abscess opened. April 11th, appendix removed.

Appendix very thick and stiff from chronic inflammation. Adherent to the abdominal wall.

27.—A. B. L—, aged 14. Fever with occasional vomiting and pain in lower abdomen for three weeks or more, beginning abruptly May 5th, 1903. Seen in consultation June 4th. Abscess opened *per rectum* June 5th. Appendix removed June 20th. Drainage. Sinus closed gradually after occasional rises of temperature, and a renewed discharge for a short time *per rectum*.

Appendix lying in a finger-like pocket between rectum and vagina, at the bottom of which were two concretions outside the appendix. This was short, very adherent, and had been perforated near the tip.

28.—Mr. C—, aged 30. Seen in consultation September 2nd, 1902. Symptoms of appendix inflammation began on August 29th, and were accompanied with marked fever. On September 8th three actions of the bowel took place, the motions being offensive, and contained mucus and blood. The temperature fell to normal, and there was no further rise. The swelling in the iliac fossa rapidly subsided. On October 27th the appendix was removed. It passed under the ileum, and presented above the juncture of cæcum and ileum. Many adhesions.

Appendix bent, strictured, and cystic; full of mucus

fluid. The scar of a recent perforation was present midway in its length.

29.—F—, aged 25. Seen in consultation at Finchley December 18th, 1904. Symptoms of appendicitis for two days. A sudden fall in the temperature took place, and she passed a very loose pale yellow stool; at the same time the local condition improved. On December 24th the temperature rose again, and there was some return of local discomfort and swelling. On December 25th another fall of temperature occurred, and she passed a motion similar to the one passed on the 24th. After this she seemed without further symptoms. Admitted to St. Thomas's Hospital on February 6th, 1905. The appendix was removed on the 8th. There were many adhesions.

Appendix.—The open stump of the appendix was found in an old abscess cavity. The distal portion was not found. Two fæcal concretions were in this cavity, the various pockets of which were scraped. Drainage.

30.—H—, aged 6. Seen in consultation November 19th, 1904. Ill four days with symptoms of acute appendicitis. On the 21st there was a rapid fall of temperature, and general improvement, with diminution of the swelling, which was in right hypogastric region. Appendix removed December 12th. Adherent in front of upper part of right kidney, surrounded by omentum. Sigmoid on right side.

Appendix strictured, with ball-like concretion beyond it, and beyond that appendix almost in two pieces being adherent to cæcum.

G.—Cases in which the appendix was removed when localised suppuration was present.

31.—F—, aged 4. Admitted to St. Thomas's Hospital June 27th, 1902. First attack. Abscess at brim of pelvis. Incision and removal of appendix and drainage.

Appendix adherent to intestines.

32.—M—, aged 13. Admitted Royal Free Hospital February 20th; left March 18th, 1900. Small abscess; drainage.

Appendix large, much thickened; strictured towards the distal extremity.

33.—F—, aged 23. Admitted to St. Thomas's Hospital September 18th, and left November 3rd, 1903. Small abscess which contained a concretion; drained.

Appendix perforated near its base.

34.—M—, aged 16. Admitted to St. Thomas's Hospital October 24th, and left November 9th, 1904. At the operation, October 26th, a small abscess was found about the caput cæci.

Appendix in a condition of recent catarrhal inflammation.

35.—F—, aged 54. Admitted to St. Thomas's Hospital January 25th, and transferred to surgical side February 1st, 1905. There was a history of several attacks of indigestion with vomiting. On January 11th a violent attack with vomiting and pain in iliac region. A hard, smooth, fixed swelling in iliac fossa.

Appendix lying to outer side of cæcum; not adherent; recent catarrhal inflammation. An abscess behind the caput cæci; drainage.

36.—M—, aged 57. Transferred to surgical ward, St. Thomas's Hospital, April 13th, 1903, from medical ward, where he had been under observation for twenty days. Second attack of appendicitis. All symptoms had subsided at time of operation, April 14th; but suppuration was found about the cæcum. Appendix removed. Died April 18th from cardiac seizure.

Appendix difficult to find and remove. There was a large hole in it, with a broken concretion outside. The small intestine was adherent.

37.—F—, aged 54. Admitted to medical ward, St. Thomas's Hospital, January 25th, with symptoms referred to iliac fossa. Transferred to surgical side and operation performed February 1st. Abscess beneath the cæcum. Appendix removed; drainage.

Appendix lying to outer side of cæcum, not adherent. Catarrhal inflammation.

H.—Cases in which a second abscess developed, but in which the appendix was not removed.

38.—F—, aged 5. Under treatment February, 1897, for appendix abscess in Royal Free Hospital. Re-admitted August, 1897, for another abscess, with bulging of the scar. August 17th abscess opened and scraped. Appendix not removed. Subsequent history unknown.

39.—C. M—, aged 16. Appendix abscess of three or four weeks' duration, and of large size, opened in St. Thomas's Home and drained. The wound closed satisfactorily, but in Switzerland, some months later, another abscess formed.

I.—Cases in which a concretion was found in the pus at the time of operation. Appendix not removed.

40.—F—, aged 14. St. Thomas's Hospital January 24th to February 23rd, 1905, first attack. Operation after seven days' duration of symptoms.

41.—F—, aged 51. St. Thomas's Hospital January 10th to February 6th, 1904. Some pain two months. Swelling three weeks. More acute symptoms three days. Opened in the loin. In both the concretion was of moderate size.

K.—Case in which a slough of the appendix was found in the abscess.

42.—M—, aged 28. St. Thomas's Hospital August 15th to 17th. Third attack. First attack nine months before. Abscess of moderate size. The main part of the appendix had sloughed off.

L.—The condition of the appendix in three fatal cases.

43.—M—, aged 25. Admitted March 19th, died March 24th, 1902. Three weeks' history of appendicitis at time of operation, and appendix apparently quiet. Abscess about and under surface of caput cæci drained. A concretion about half an inch long was removed.

Appendix.—Found *post-mortem* under cæcum, not forming part of the abscess cavity. The top had sloughed off. An opening was found in the cæcum half an inch from base of appendix. General peritonitis.

44.—M—, aged 45. St. Thomas's Hospital June 20th to 30th, 1902. Second attack. Died suddenly ten days after the abscess was opened. At the *post-mortem* local condition was satisfactory. There was no peritonitis. Cause of death not ascertained.

Appendix thickened, and adherent to outer side of cæcum.

45.—M—, aged 15. In St. Thomas's Hospital early in 1898 for large appendix abscess, which was treated by incision, and later by counter incision. Re-admitted May 2nd, and died July 31st, 1898, apparently from exhaustion.

Appendix.—Not found. Very offensive cavity, with very thick adhesions surrounding it. No trace of tubercle about it, but the lungs were riddled with tubercle.

Fatal case. No *post-mortem* examination.

46.—F—, aged 28. Seen in consultation June 30th, 1902, and abscess of twelve days' duration opened and drained.

Died June 3rd. Symptoms of acute yellow atrophy of liver.

M.—Condition of the appendix in three cases in which an appendix abscess ruptured spontaneously, discharging its contents into the general peritoneal cavity.

All were the first attack, and were treated in St Thomas's Hospital.

47.—M—, aged

history of six days' illness. On the morning of the 19th, 11.15, sudden severe attack of pain in the abdomen, with vomiting and rapid increase of pulse. About four hours afterwards abdominal section. Lavage, drainage. Died on the 20th, peritonitis.

Appendix long and curled on itself, proximal two inches healthy, remainder acutely inflamed. The tip had sloughed off. The abscess had given way to the outer side.

48.—F—, aged 18. Symptoms of appendicitis on October 27th, but after a few hours rest continued work. Sudden attack of pain during the night of November 2nd; admitted November 3rd. Incision over abscess and in left linea semilunaris, lavage, drainage. Went home December 11th, 1904. (Opening in abscess towards pelvis.) Re-admitted 1905. Appendix removed January 18th.

Appendix two inches long. Strictured half an inch from the tip, below which was a small perforation. Adherent to caput cæci and at first indistinguishable.

49.—M—, aged 33. Four days before admission symptoms of appendicitis, but he resumed work on the following day. Sudden increase of pain sixteen hours before admission, November 4th, 1904. Incision over abscess and in linea alba, lavage, gauze packing and drainage. Opening in abscess to outer side. On January 4th, 1905, appendix removed.

Appendix one and a quarter inches long. Two strictures. Evidence of former perforation below the distal one.

Death from rupture of abscess at time of operation.

50.—M—, aged 29. Admitted to the Royal Free Hospital January 21st, 1897. Died January 24th. Symptoms commenced three days before. Incision January 22nd, and drainage. Died thirty-six hours later from general peritonitis. The abscess cavity

had ruptured into general peritoneal cavity, the surrounding adhesions being very fragile. It was thought this resulted from necessary movements at operation.

Appendix was found, at the *post-mortem* examination, to have been recently perforated.

SUMMARY OF THE LIST OF CASES OF (LOCALISED) APPENDIX SUPPURATION

The number of cases in all 61, of which 11 still retain their appendices. They are not, therefore, given in this list.

The following summary of the fifty remaining has been prepared to show the rough pathological condition of the appendix in the various groups which have a bearing on the question as to the advisability of removal of the appendix during the quiet interval following abscess.

A and B.—When the symptoms of appendicitis recurred, or another abscess formed—

Kinked, strictured, with signs of old perforation, 3. Strictured, 2. Kinked, 1. Showing signs of recent inflammation, 1. Strictured, with a retained concretion, 1. Chronic inflammation, with signs of old perforation, 1. Concretion and old perforation, 1.

C.—Sinus formation after abscess—

Stricture, concretion, old perforation, 1. Strictured, 1. Recent perforation, 1. Chronic inflammation with old perforation, 1.

In 2, condition of appendix unknown.

D.—Removal during a later peritonitis when recovered from abscess treated elsewhere.

Concretion with recent perforation 1. Recent perforation 1.

E.—Removal after closure of abscess. No further symptoms.
Concretion, old perforation 1. Strictured 1. Kinked, stricture concretion 1. Old perforation 1. Stricture concretion, old perforation 1.* Marked chronic inflammation 1. Marked chronic, old perforation 1.

F.—Removal after rupture into bowel.
Marked chronic inflammation 1. Stricture 1. Kinked, stricture, recent perforation 1. Concretion, recent perforation 1. Stricture concretion 1.

G.—Removal during operation for evacuation of pus.
One not stated. Stricture 1. Recent inflammation 2. Recent perforation 1. Concretion, recent perforation 1. Chronic inflammation 1.

L.—Three fatal cases, in which appendix had not been removed.
Partial slough 1. Concretion removed at operation 1. Chronic inflammation 1.

M.—Rupture of abscess into peritoneum, spontaneous.
One fatal; long, curled on itself, acute inflammation with slough. Stricture 2.

In thirteen cases concretions were found when the appendix was excised. In No. 21 a concretion was found in the abscess when opened, and another in the appendix when removed. In another, No. 40, where a concretion was found in the pus, the patient has recently returned complaining of pain in iliac fossa, where there is a tender swelling.

In the total of 61, 54 recovered, 7 died.

Cause of death, No. 45, tubercle of lungs and exhaustion. No. 46, acute yellow atrophy of liver.

Three peritonitis. 43.—Damage to bowel in attempt to find appendix when the abscess was opened. 50.—Intra-peritoneal rupture during operation. 47.—Intra-peritoneal spontaneous rupture.

Two cardiac disease. 44.—Ten days after abscess opened. 36.—Four days after abscess opened.

The treatment employed in these fifty cases, which include all which have a bearing on the condition of the appendix, was as follows :

In 5 the appendix was removed after a calculated interval following rupture into bowel.

In 11, *simple incision with drainage.* 6 recovered. 5 died. In 1 the appendix was found to have sloughed. In 3 concretions were removed, one of them *died*—peritonitis. In 1 concretion removed from a sinus. In 2 a second abscess formed at a later date. In 1 the abscess ruptured posteriorly during operation—*died*—peritonitis. In 1 the patient *died* of acute yellow atrophy of liver. In 1 chronic abscess and exhaustion and tubercle of lungs. One died suddenly—cardiac seizure ten days later.

In 7 *the appendix was afterwards removed,* at a time selected.

In 16 *there was a return or persistence of symptoms,* and the appendix was removed. Appendicitis 5. Abscess 5. Peritonitis 2. Sinus 4.

In 2 the appendix was removed when the patients had recovered from an operation for the relief of symptoms due to rupture into the peritoneum.

In 1 case the patient did not recover after operation for intra-peritoneal rupture.

In 7 the abscess was opened, the appendix removed, and the abscess cavity drained. Six recovered, 1 died from cardiac seizure four days later.

In 1 a sinus persisted after appendix abscess which had been treated elsewhere.

APPENDICITIS

AT

CHARING CROSS HOSPITAL

STATISTICAL TABLES OF CASES OF OPERATION FOR APPENDICITIS

DURING THE YEARS 1902, 1903, AND 1904

FURNISHED BY

H. S. CLOGG, M.S., F.R.C.S.

ASSISTANT SURGEON TO THE HOSPITAL

AND

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SURGICAL REGISTRAR TO THE HOSPITAL

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1. *Number of cases.*—125.
   Of these 61 were operated upon in quiescent period.
   5 were operated upon in acute stage (without abscess).
   51 were operated upon for abscess formation.
   8 were operated upon for general peritonitis.
2. *Sex.*—Males 74; females 51.
3. *Number of attacks* of Appendicitis—
   50 had 1 attack.
   28 had 2 attacks.
   23 had 3 attacks.
   21 had four or more attacks.
   2 cases had chronic pain in right iliac region only.

4. *Suppuration occurred*—
   In 1st attack in 35 cases.
   In 2nd „ 10 „
   In 3rd „ 10 „
   In 4th „ 2 „
   In 5th „ 2 „
5. *Age at which 1st attack occurred*—
   Before 10 years in 11 cases.
   Between 10—20 years in 49 cases.
   Between 20—40 years in 45 cases.
   After 40 years in 10 cases.
6. *Situation of appendix* with relation to cæcum, in cases in which appendix was actually removed—
   Behind and external in 38 cases.
   Behind and internal in 24 cases.
   Directed towards or into pelvis in 11 cases.
   Behind in 8 cases.
   Downwards, below cæcum, in 5 cases.
   In cases with abscess, and in which the appendix was not found, the position of the abscess indicated that the appendix lay—
   To the outer side of cæcum in 14 cases.
   „ inner „ 9 „
   Below the cæcum in 1 case.
7. *Condition of appendix.*—Peritoneum showed signs of present or past inflammation in all the cases save 12. Of these 12 cases all but 1 showed definite macroscopical or microscopical signs of disease of the organ.

   Stenosis, partial or complete, occurred in 26 cases (of these, 10 showed distension on distal side of constriction) *gangrene* in 9, *perforation* in 9, *concretion* in 6, *kinking* in 7, *obliteration of lumen* of distal portion in 3, *tubercular* disease in 1.
8. *Incision* used.
   In *suppurating cases.*
   Anterior incision only in 43 cases.
   Anterior incision and incision in loin in 7 cases.
   Posterior incision in loin only in 1 case.

In *non-suppurating cases* (1903—1904 only).—All by anterior incision.
By McBurney's method in 35 cases.
Through sheath of rectus in 14 cases.
Oblique incision direct through muscle in 3 cases.

9. In the 51 *cases of localised suppuration* the appendix was removed in 22 at the time the abscess was opened.

10. *Special complications during the operation*—
(A) In one case operated upon in quiescent period the appendix could not be found on account of the mass of adhesions.
(B) In two quiescent cases the cæcum was in danger of being opened, owing to the close adhesion of the appendix to it. A portion of the wall of the appendix had to be left attached to the cæcum.
(C) A case of chronic intestinal obstruction. No definite attack of appendicitis; "bilious" for years. Constipation usual. Obstruction of varying degree for two months before operation. At operation an omental band was found passing from cæcum to a loop of small gut, which was kinked thereby. Another loop of gut was partially strangulated under this band. Appendix lay in a mass of adhesions, and was removed. Patient made a good recovery.

11. *Mortality.*—All the non-suppurative cases recovered.
Of the *abscess cases* (51), 6 died. 3 from general peritonitis.
1 from septicæmia ten hours after operation.
1 from pylephlebitis.
1 from general tuberculosis.
Of the *general peritonitis* cases (8), 7 died.

12. Results of cases of *localised suppuration.*
All abscesses were drained except 2, in which abscess

was small; one of these healed without further suppuration. The other developed a deep abscess on 7th day. This was drained and patient recovered.

All the other cases, except 8, healed up in 3 to 8 weeks. Of the remaining 8 cases, 2 healed in 3 months; 1 healed in 5 months; 3 went home with a sinus, and were lost sight of; 1 was known to have a sinus after 9 months; the remaining case had a sinus for 3 months, and then died of pylephlebitis.

13. *Early complications* after operation.

A. *Fæcal fistula* 3 cases.

1. Case operated upon in acute stage, without abscess formation. Fæcal fistula formed on 18th day, and persisted, in spite of two attempts to close it, till patient was lost sight of. Fistula closed and reopened twice after patient left hospital.
2. Case of general peritonitis. Fæcal fistula formed, and patient died on 10th day after operation. P.M. Perforation of appendix, duodenum, and sigmoid.
3. Case of abscess. Fæcal discharge on 2nd day. On 4th day appendix (sloughed) came away with a concretion. Fæcal discharge then ceased.

B. *Local abscess formation.* 2 cases.

1. Case operated upon in acute stage, without suppuration. Abscess formed in cæcal region on 4th day.
2. Case of small abscess. Not drained. Abscess re-formed on 7th day. All well till that day.

C. *Secondary abscess formation.* 5 cases.

1. *Pelvic abscess.* Abscess in iliac fossa opened and drained through usual in-

cision. Pus escaped by rectum, and through wound on 10th day, and also a few days later. Healed in 4 weeks.

2. *Pelvic abscess* (?) Abscess in iliac fossa opened. Temperature markedly raised for 3 weeks. Mass of exudation in pelvis felt *p.v.* This subsided with the temperature, without any escape of pus being noticed.
3. *Abscess in left iliac region and pelvis.*—Abscess in right iliac region opened. On eleventh day a tender swelling was noticed above left Poupart's ligament and felt *per rectum*. Enemata had no effect on swelling. By the sixteenth day temperature normal and mass had disappeared.
4. *Pelvic abscess.*—Right iliac abscess opened and drained. On seventh day temperature rose. On twelfth day a mass felt in Douglas's pouch. Mass smaller and temperature normal by seventeenth day. Pus passed *per rectum* on eighteenth and twentieth days, and mucus and blood for three days after this. Patient discharged with sinus in three and a half weeks. Uterus was fixed by exudate.
5. *Abscess on left side of abdomen* (? ?).—Operation during quiescent period, two and a half weeks after first attack. Appendix removed without difficulty. Temperature reached 102°—105° each day till seventh, when rigor occurred. Pain and tenderness in left flank. Temperature was normal by tenth day, and pain and tenderness disappeared without visible escape of pus.

D. *General suppurative peritonitis.*—3 cases.

1. Abscess opened on tenth day of attack. All well for twenty-four hours, then general peritonitis set in, and patient died on fourth day. No P.M.
2. Abscess opened on ninth day of attack. Died of general peritonitis on third day. P.M.—Large retro-peritoneal abscess, communicating with general abdominal cavity by a small hole close to appendix.
3. Abscess opened on twelfth day of attack. All well for fourteen days, then temperature rose and vomiting, etc., commenced. Abdomen washed out on nineteenth day. Death twelve hours later. P.M.—No cause found for the onset of general peritonitis.

E. *Septicæmia.*—1 case. Died ten hours after operation for abscess. P.M.—Signs of septicæmia.

F. *Phlebitis and thrombosis.*—1 case. Case operated upon during acute attack without abscess. Thrombosis of left femoral vein three weeks after, and of right femoral vein later.

G. *Pneumonia and broncho-pneumonia.*—3 cases, immediately following operation. In 2 the operation was performed in quiescent period. The other case had an abscess in right iliac region. Abscess opened and appendix removed. Temperature rose at once, and profuse foul expectoration occurred. Death on seventh day. No P.M. Appendix showed tubercles.

H. *Hæmatemesis.*—2 cases.

1. The case of septicæmia already mentioned.
2. Simple case of appendicectomy. Vomiting incessantly for three and a half days

after operation. On second day vomit contained altered blood.

I. *Parotitis.*—1 case. Bilateral. Swelling occurred on third and sixth days respectively, after opening of appendical abscess. Subsided without suppuration.

J. *Second incision* in loin was necessary in one case on fifteenth day after abscess had been opened and drained from front.

K. *Rigors.*—1 case. Case of acute abscess. Opened and drained. Temperature normal and case doing well. Rigor occurred on nineteenth and again on twenty-fourth day. Temperature fell rapidly on each occasion after the rise accompanying the rigor. Nothing found to account for rigor. Healed in five weeks. Patient discharged without further trouble.

L. *Subphrenic abscess.*—Case of general peritonitis lingered for four weeks after operation and then died. P.M.—Right subphrenic abscess, and abscesses in pelvis, and to outer side of ascending colon.

Late Complications.

A. *Recurrence of symptoms* in a case of abscess. Appendix not removed. Wound healed in four weeks. Patient returned to hospital a week after discharge with abdominal pain, vomiting, and temp. 100·2°. Rapid recovery.

B. *Recurrent abscess.*—One month after a large retro-peritoneal abscess had healed another abscess formed under scar. Healed in five weeks.

C. *Persistent sinus.*—2 cases returned to hospital with sinus.

(*a*) Abscess opened. Appendix not removed. Healed in seven weeks. Two months later wound reopened and

sinus remained. Scraped three times. Concretion removed. Sinus persists.

(*b*) Abscess opened and drained by anterior and posterior incisions. Appendix not removed. Sinus persisted. Nine months later sinus scraped; healed in three months. Two months later abscess under scar. Opened and appendix removed; healed in four weeks. Four months later reopened and healed again in a month.

D. *Œdema* of left leg four months after simple appendicectomy; œdema persisted; cause?

E. *Bulging of abdominal wall* around the scar, due to paralysis of muscular fibres; 2 cases.

1. Easy appendicectomy; McBurney's method; wound healed by first intention; three months later the scar was sound, but there was bulging of abdominal wall around the lower part of scar.

2. Abscess opened in front and in loin; healed in five weeks. One month later patient was readmitted with large area of bulging beneath scar, and also below and internal to scar over a considerable area. *Operation.*—Appendix removed, and muscles were overlapped, layer by layer, in closing the wound.

F. *Tubercular peritonitis.*—1 case. This case is referred to above (E. 1). Five months after appendicectomy patient came to hospital complaining of discomfort and distension of abdomen, constipation with one attack of diarrhœa, slight vomiting, and marked loss of weight and strength. No tubercles had been seen at the operation. Appendix was not microscoped.

APPENDICITIS

AT

ST. BARTHOLOMEW'S HOSPITAL

STATISTICAL TABLES

FURNISHED BY

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SURGICAL REGISTRAR TO THE HOSPITAL

General Statistics of Cases of Appendicitis and their Complications occurring at St. Bartholomew's Hospital during a period of ten years. Years 1894—1903 (*inclusive*).

Year.	Total No. of cases.	Total No. of operations.	Total No. of deaths after operation.	Total No. of deaths without operation.	Operative mortality.	General peritonitis.	Subphrenic abscess.	Empyema.	Pyæmia and multiple abscesses.	Fæcal fistula.	Thrombosis of veins.	Liver abscess.	Bronchitis and pneumonia.	Strangulation of intestine.
1894	41	10	7	0	70 %	5	—	—	—	—	—	—	—	—
1895	38	12	3	2	25 %	2	—	—	—	3	—	—	1	—
1896	81	45	14	2	31·1 %	12	—	—	—	2	1	—	1	1
1897	85	40	10	3	25 %	7	1	—	—	2	—	—	—	—
1898	97	60	9	1	15 %	8	1	—	2	3	—	—	—	—
1899	122	80	16	2	20 %	10	2	—	—	2	—	—	—	—
1900	120	89	12	0	13·4 %	13	—	—	—	4	—	—	—	1
1901	166	123	25	0	20·03 %	20	1	2	—	4	1	1	2	—
1902	205	171	19	1	11·1 %	19	2	—	3	4	—	—	1	—
1903	215	165	21	1	16·3%	19	1	1	1	6	2	—	—	1

Complications occurring in Cases of Appendicitis recovering after Operation (in the same period of ten years).

Year.	Total No. of operations.	Total No. of recoveries.	General peritonitis.	Subphrenic abscess.	Empyema.	Localised peritonitis or residual abscess.	Fæcal fistula.	Thrombosis of veins.	Sinus on discharge from hospital.	Ventral hernia.	Bronchitis and pneumonia.	Parotitis.
1894	10	3	—	—	—	—	—	—	—	—	—	—
1895	12	9	—	—	—	—	2	—	—	—	—	—
1896	45	31	1	—	—	—	1	1	1	—	1	—
1897	40	30	1	—	—	—	1	—	—	—	—	—
1898	60	51	1	—	—	1	2	—	4	1	—	—
1899	80	64	—	—	—	—	2	—	1	1	—	—
1900	89	77	1	—	1	1	2	—	1	—	1	—
1901	123	98	2	1	—	—	2	1	5	—	2	1
1902	171	152	2	—	—	4	4	1	6	5	1	—
1903	165	144	1	—	1	—	5	2	2	—	—	—

APPENDICITIS

AT

ST. THOMAS'S HOSPITAL

YEARS 1900-1-2-3-4

STATISTICAL TABLES FURNISHED BY
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Number of cases admitted . .	947
Number of deaths . . .	168
Percentage mortality . . .	17·7

On these 947 patients there were performed 863 operations, with 131 deaths after operation, the mortality being 15·2 per cent.

Of these 131 deaths after operation, 98 occurred in cases of general peritonitis; 29 occurred in cases of abscess; 2 occurred in cases of excision in acute stage: viz. (1) After 15 days of vague febrile symptoms, nature of case uncertain: death from pylephlebitis (cp. table). (2) On fourth day of third attack; infection of peritoneum, pleura, and pericardium (cp. table), and 2 occurred in cases of excision in quiescent stage: viz. (1) Death from general peritonitis (cp. table). (2) Death from peritonitis, possibly tuberculous; no post mortem (cp. table).

These two deaths after excision in quiescent stage occurred in 507 instances of that operation.

After these 863 operations, the following complications occurred:

		Instances.
in abdomen (lower)	ventral hernia	6
	residual or secondary abscess	17
	fæcal fistula	15
	abscess in abdominal wall	1
	obstruction by adhesions	5
	obstruction by band	1
	volvulus	1
	secondary infection of peritoneum	3
in abdomen (upper)	subphrenic abscess	5
	pylephlebitis	2
	subhepatic abscess	1
in thorax	broncho-pneumonia	3
	pneumonia	2
	gangrene of lung	1
	pleural effusion	1
	empyema	1
	pulmonary embolism or thrombosis	2
in distant parts	thrombosis in veins of leg	2

And the following complications were found to be present at the time of operation:

Perforation of bowel	1
Pyo-salpinx (right)	1

Details of Complications.

Ventral hernia.—All after incision and drainage of abscess.

Residual abscess.—In 9 cases second abscess in right iliac region *before* leaving hospital. In 1 case second abscess in left iliac region before leaving hospital. In 7 cases second abscess [illegible] leaving hospital, the interval [illegible]

8, 9, and 24; of these, the abscess lay under the scar of the earlier incision in 6 cases; in 1 case it presented above pubes in middle line. In all cases a primary abscess had been incised and drained.

Fæcal fistula.—In 13 cases after incision and drainage of abscess, of which two died. In one case operation on third day of a first attack, appendix adherent to inflamed small intestine; spontaneous opening of wound later, fæcal extravasation and general infection of peritoneum (fatal). In one case incision and drainage of abscess; fæcal fistula on fourteenth day; death two months later, with tuberculous peritonitis.

Abscess in abdominal wall.—Excision of gangrenous appendix on sixth day of a first attack; large abscess developed in abdominal wall (fatal).

Obstruction by adhesions.—In three cases incision and drainage of abscess; obstruction in one case four months later (operation, recovery), in one case fourteen months later (operation, recovery), in one case one month later (operation, death). In one case excision of appendix in quiescent interval and right uterine appendage five years previously (operation, recovery). In one case general peritonitis, immediate excision and lavage; obstruction three days later (operation, death).

Obstruction by band.—Incision and drainage of abscess, healed on 72nd day; sudden obstruction by band (adhesion) on 82nd day (operation, recovery); appendix subsequently excised on 105th day.

Volvulus.—Incision of abscess and removal of appendix. Death nine days later, with volvulus of small intestine (gangrenous), and general peritonitis.

Secondary infection of peritoneum.—In one case excision of appendix on fourth day of third attack; five days later sudden rise of temperature, followed by rigors and continuance of fever; death fourteen days later, from peritonitis, pleurisy, and pericarditis. In one case excision of appendix seven days after subsidence of pain, appendix adherent; death from

general peritonitis. In one case excision on ninth day of fourth attack; caseous tuberculous glands in mesentery; wound healed; death on twenty-fifth day, with signs of general peritonitis; no post mortem.

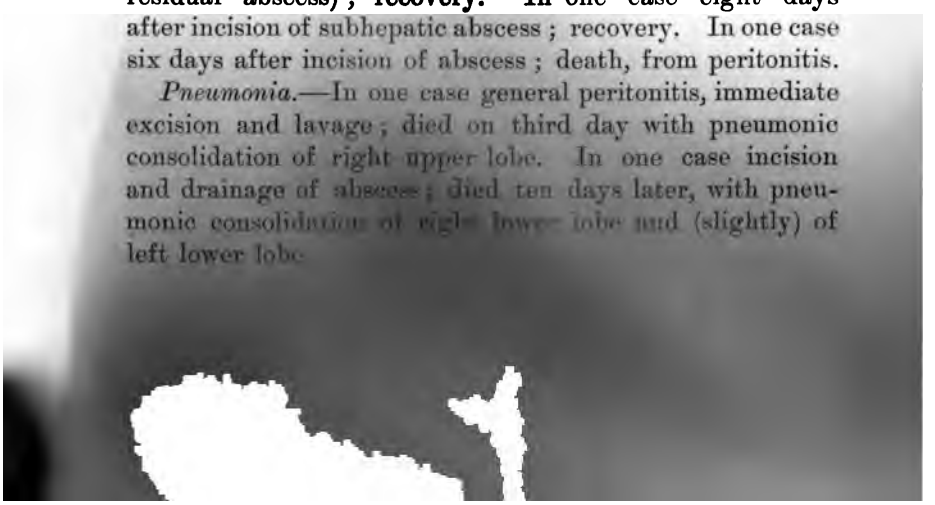
Subphrenic abscess.—In one case, after incision and drainage of abscess around appendix, patient returned to hospital, one month after leaving, with right subphrenic abscess; operation; recovery. In three cases of incision and drainage of abscess around appendix, right subphrenic abscess was incised and drained after an interval of 14, 16, and 26 days respectively; two recoveries, one death (gangrene of lung, pleurisy). In one case general peritonitis, operation, lavage; right subphrenic abscess incised and drained nine days later; death.

Pylephlebitis.—In one case excision of appendix after vague febrile symptoms for fifteen days, suggesting enteric fever (Widal's reaction positive, but leucocytes 30,000); wasting and pyrexia afterwards, with no enlargement of liver; death two and a half months later, purulent infiltration of liver. In one case, incision and drainage of abscess; death two days later, from portal thrombosis and early suppuration in liver. (In one case operation for *hepatic abscess only*; post-mortem, appendix found in dense mass of adhesions.)

Subhepatic abscess.—In one case incision of abscess in right iliac fossa on sixth day, two days later incision in right loin into subhepatic abscess; death.

Broncho-pneumonia.—In one case, five days after incision and drainage of abscess in left iliac fossa (cp. residual abscess); recovery. In one case eight days after incision of subhepatic abscess; recovery. In one case six days after incision of abscess; death, from peritonitis.

Pneumonia.—In one case general peritonitis, immediate excision and lavage; died on third day with pneumonic consolidation of right upper lobe. In one case incision and drainage of abscess; died ten days later, with pneumonic consolidation of right lower lobe and (slightly) of left lower lobe.



Gangrene of lung.—In one case incision and drainage of abscess; twelve days later signs of pneumonic consolidation. Post mortem, consolidation and commencing gangrene of both right and left lower lobes.

Pleural effusion.—In one case general peritonitis; immediate excision and lavage; death on ninth day, with right pleural effusion.

Empyema.—In one case general peritonitis; immediate excision and lavage; parotitis; empyema on right side incised and drained on eighteenth day; recovery.

Pulmonary embolism or thrombosis.—In one case incision and drainage of abscess; sudden pain in left side and hæmoptysis ten days later; recovery; no thrombosis detected in veins of extremities. In one case incision and drainage of abscess; sudden death (twelve hours), with rapid respiration, four days later; no post mortem; no thrombosis detected in veins of extremities.

Thrombosis in leg.—In one case excision of appendix in quiescent interval; thrombosis and œdema of left leg below knee on fourteenth day; condition lasted three weeks; recovery. In one case thrombosis of femoral vein of left leg eighth day after incision and drainage of abscess; recovery.

Complications found at Time of Operation.

(1) *Perforation of bowel.*—In one case general peritonitis, cæliotomy and lavage, ascaris lumbricoides found free in abdominal cavity; post mortem, perforation of small intestine in pelvis and of ascending colon.

(2) *Pyosalpinx.*—In one case general peritonitis, excision and lavage; death four days later; post mortem, outer two inches of right Fallopian tube distended with pus.

APPENDICITIS

AT

ST. GEORGE'S HOSPITAL

STATISTICAL TABLES SHOWING PRESENT CONDITION OF SEVENTY-THREE PATIENTS AFTER OPERATIONS FOR APPENDICITIS

PERFORMED DURING THE YEARS 1900—1902.

FURNISHED BY

LAWRENCE JONES, M.B., B.S., F.R.C.S., ENG.

REGISTRAR TO THE HOSPITAL

174 patients written to. 73 replies.

Acute cases with Abscess, 34 cases.

(1) *Appendix removed,* 19 cases.

"Quite well in every way," 12 cases.

"No pain, but very slight bulging of wound," 2 cases.

"Slight pain occasionally, with constipation," 1 case.

"Slight pain sometimes, with a little bulging of wound," 1 case.

"Very slight occasional pain," 2 cases.

"Quite well, but has developed a double inguinal hernia, which he attributes to wearing a belt," 1 case.

(2) *Appendix not removed,* 15 cases.

"Quite well in every way," 10 cases.

"Another acute attack, necessitating removal of the appendix, but now quite well," 2 cases.

"A little pain and bulging of wound," 1 case.
"Pain only on coughing or over-exertion," 1 case.
"Recovery complicated by thrombosis, which has since been repeated; locally quite well," 1 case.

Acute cases, without Abscess, 12 cases.
"Quite well in every way," 7 cases.
"Occasional pain," 2 cases.
"Occasional pain and slight bulging," 2 cases.
"Bulging of wound about the size of an egg," 1 case.

Subacute or Chronic, 27 cases.
"Quite well in every way," 16 cases.
"Very little tenderness," 2 cases.
"Very slight bulging," 2 cases.
"A little occasional pain," 1 case.
"Constipation," 1 case.
"Sudden spasms of pain at times, passing away quickly," 1 case.
"Tenderness and a little bulging," 1 case.
"Pain only on over-exertion," 2 cases.
"Large ventral hernia and gastric trouble," 1 case.

All the complaints were represented as being quite trivial in nature, and did not in any way inconvenience the patients, except in two, one with "large ventral hernia and gastric trouble," and the other with "pain in the region of the wound." In the latter case the patient attributed various other symptoms to the operation, and, I think, need not be counted.

The "bulging of the wound" was only in the former of the two cases in any way marked; all the other cases were slight.

I asked the patients whether they had worn belts; they all had done so with the exception of one, who had *no* hernia.

APPENDICITIS

AT

ST. GEORGE'S HOSPITAL

(OPERATION IN EVERY CASE)

STATISTICAL TABLES

FURNISHED BY

LAWRENCE JONES, M.S., F.R.C.S.Eng.,
SURGICAL REGISTRAR TO THE HOSPITAL.

				Deaths.	Complications.
Acute Appendicitis with General Peritonitis.					
1st attack . . . 1	*Males*	0			
2nd attack . . . 0					
3rd attack or more . 1	*Females*	2	... ...	1	
		— 2			
Acute Appendicitis with Abscess.					
1st attack . . . 10	*Males*	12	Appendix removed . . 7	2	Thrombosis 1.
2nd attack . . . 2					
3rd attack or more . 2	*Females*	9	Appendix *not* removed . 14	4	
? attack . . . 7		—21			
Acute Appendictis.					
1st attack . . . 4	*Males*	10			
2nd attack . . . 5					
3rd attack or more . 5	*Females*	6	... ...	2	One case developed pneumonia, empyema, liver abscess, cerebral abscess.
? attack . . . 2		—16			
Chronic, Subacute, or Quiescent Appendicitis.					
1st attack . . . 3	*Males*	13			
2nd attack . . . 5					
3rd attack or more . 9	*Females*	6			
? attack . . . 2		—19			
	Total	58		9 deaths	

				Deaths.	Complications.
te Appendicitis with General Peritonitis.					
1st attack . . . 0	*Males*	7			
2nd attack . . . 0					
3rd attack . . . 0	*Females*	0	... ...	7	
? attack . . . 7		— 7			
te Appendicitis with Abscess.					
1st attack . . . 15	*Males*	14	Appendix removed . . 13	1	Fæcal fistula 3.
2nd attack . . . 5					Fæcal fistula 3; thrombosis 2; pulmonary embolism 1; pleurisy 1.
3rd attack or more . 1	*Females*	9	Appendix *not* removed . 10	...	
? attack . . . 2		—23			
Acute Appendicitis.					
1st attack . . . 8	*Males*	6			
2nd attack . . . 2					Arthritis 1; pneumonia 1; fæcal fistula 1.
3rd attack or more . 1	*Females*	7	... ...	...	
? attack . . . 2		—13			
Chronic, Subacute, or Quiescent Appendicitis.					
1st attack . . . 6	*Males*	5			
2nd attack . . . 4					
3rd attack or more . 6	*Females*	11			
		—16			
	Total	59		8 deaths	

1902.

				Deaths.	Complications.
Acute Appendicitis with General Peritonitis.					
1st attack . 0	*Males* 3				
2nd attack . 0					
3rd attack . 0	*Females* 1	... ...		3	
4th attack . 0	—4				
	Males 26	Appendix removed . . 19		2	Fæcal fistula 1; empyema 1.
	Females 8	Appendix *not* removed . 15		2	Pulmonary embolism 1; fæcal fistula 1; pleurisy 3; thrombosis 2; empyema 1.
	—34				
	Males 13				
	Females 5	... ...		1	Fæcal fistula 2; bronchitis 1; thrombosis 1.
	—18				
	Males 19				
	Females 15	... ...		...	Pulmonary embolism 1.
	—34				
	Total 90			8 deaths	

1903.

				Deaths.	Complications.
...ith General					
. . 6	*Males* 5				
. . 1					
... or more . 0	*Females* 3	... ...		2	
... . . 1	— 8				
...endicitis with Abscess.					
... attack . . . 37	*Males* 38	Appendix removed . . 43		3	Empyema 1; pleurisy 1; fæcal fistula 1.
... attack . . . 11					
... attack or more . 5	*Females* 21	Appendix *not* removed . 16		1	Fæcal fistula 3; pneumonia 1; pleurisy 1.
? attack . . . 6	—59				
... Appendicitis.					
1st attack . . . 17	*Males* 12				
2nd attack . . . 7					
3rd attack or more . 5	*Females* 17	... ...		1	Fæcal fistula 1; pyosalpinx 1.
? attack . . . 0	—29				
Subacute, Chronic, or Quiescent Appendicitis.					
1st attack . . . 12	*Males* 12				
2nd attack . . . 7					
3rd attack or more . 5	*Females* 18	... ...		...	Thrombosis 1; bronchitis 1.
? attack . . . 6	—30				
	Total 126			7 deaths	

1904 (not complete)

					Deaths.	Complications.
Acute Appendicitis with General Peritonitis.						
1st attack	1	*Males*	5	... ...	7	
2nd attack	2					
3rd attack or more	1	*Females*	2			
? attack	3		— 7			
Acute Appendicitis with Abscess.						
1st attack	28	*Males*	22	Appendix removed . . 32	6	Thrombosis 1; fæcal fistula 1.
2nd attack	7					
3rd attack or more	3	*Females*	19	Appendix *not* removed . 9	1	Bronchitis 1; fæcal fistula 1.
? attack	3		—41			
Acute Appendicitis.						
1st attack	16	*Males*	17	... ...	1	Splenic abscess and pylephlebitis 1; mania and pleurisy 1; bronchitis 2.
2nd attack	7					
3rd attack or more	4	*Females*	13			
? attack	3		—30			
Subacute, Chronic, or Quiescent Appendicitis.						
1st attack	12	*Males*	19	... ...	...	Thrombosis 1; pleurisy 4; pneumonia 1.
2nd attack	7					
3rd attack or more	9	*Females*	12			
? attack	3		—31			
		Total	109		15 deaths	

1900, 1901, 1902, 1903, 1904.

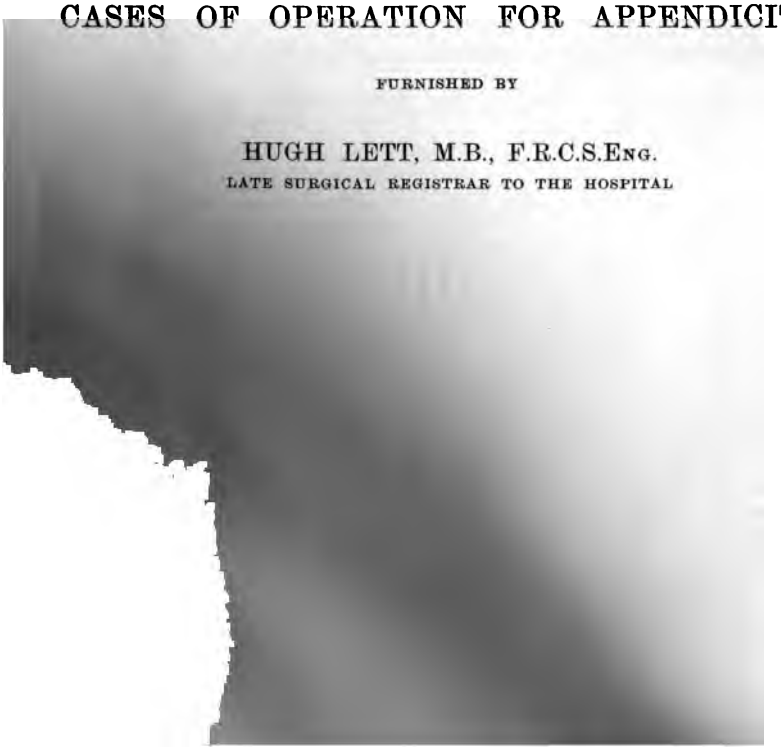
				Deaths.	Complications.
Appendicitis with neral Peritonitis.					
attack . 8	*Males* 20				
d attack . 3					
d attack or more 1	*Females* 8	... ...		20	
attack . . 16	—28				
Appendicitis with scess.					
t attack . 107	*Males* 112	Appendix removed . . 114		14	Fæcal fistula 6; empyema 2; thrombosis 2; pleurisy 1.
. 29					
k or more 13	*Females* 66	Appendix *not* removed . 64 (7 cases required second operation)		8	Fæcal fistula 8; bronchitis 1; thrombosis 4; pleurisy 5; pneumonia 1; empyema 1; pulmonary embolus 2.
ack . . 29	—178				
te Appendicitis.					
1st attack . 53	*Males* 58				
2nd attack . 24					Splenic abscess and pylephlebitis 1; pneumonia, empyema, cerebral abscess, and liver abscess 1; fæcal fistula 4; bronchitis 3; thrombosis 1; pyosalpinx 1; mania and pleurisy 1; pneumonia 1; arthritis 1.
3rd attack or more 19	*Females* 48	... ...		5	
? attack . . 10	—106				
Chronic, Subacute, or Quiescent Appendicitis.					
1st attack . 44	*Males* 68				
2nd attack . 32					Thrombosis 2; pleurisy 4; pneumonia 1; bronchitis 1; pulmonary embolism 1.
3rd attack or more 41	*Females* 62	... ...		...	
? attack . . 13	—130				
	Total 442			47 deaths	

APPENDICITIS

AT THE

LONDON HOSPITAL

STATISTICAL TABLES OF ONE THOUSAND CASES OF OPERATION FOR APPENDICITIS



FURNISHED BY

HUGH LETT, M.B., F.R.C.S.Eng.

LATE SURGICAL REGISTRAR TO THE HOSPITAL

TABLE D (*London Hospital Series*).

Cases of Abscess and of General Peritonitis in this Series.

—	Number of cases.	No. of previous Attacks of Appendicitis.				
		Not stated.	None.	One.	Two.	Three or more.
Abscess	499	200	187	67	19	26
General peritonitis without abscess	101	66	31	3	1	0
Total	600	266	218	70	20	26

TABLE E (*London Hospital Series*).

Table to show the Complications met with in the 1000 *cases.*

Complications.	Number of cases.
Fæcal fistula	49
Thrombosis of the femoral vein	12
Intestinal obstruction	10
Broncho-pneumonia	17
Pleurisy with effusion...	14
Pleurisy without effusion	2
Empyema	7
Acute bronchitis	4
[illegible]mbolism	1
[illegible]suppurative	4
[illegible]	4
[illegible]	11
[illegible]	11

[illegible]ppendicitis was associated with
[illegible]nancy.

TABLE F.—*Table to show the Frequency of "Further Attacks" after Various Operations for Appendicitis.* (*London Hospital Series.*)

Type	No.	Abscess or General Peritonitis.						Interim Operations.		
		Number of Cases.	Appendix Removed.		Not Removed.		Not Stated.		Number of Cases.	+	O
			+	O	+	O	+	O			
		11	0	4	0	6	0	1	12	1	11
		8	0	3	0	3	0	0	5	0	5
		33	0	4	1	14	0	4	30	0	30
		14	1	3	4	6	0	0	5	0	5
		30	0	13	6	29	0	1	30	1	29
		20	0	5	0	10	0	1	20	3	17
		47	1	23	5	18	0	2	45	3	42
		34	1	16	2	11	0	0	32	2	30
		47	2	21	1	13	0	4	43	0	43
		23	1	9	2	12	0	1	20	1	19
		[illegible]	6	101	21	122	0	14	242	11	231

[illegible] column indicates the number of patients who had further "attacks;" the sign O indicates the number of patients who had no further "attacks."

STATISTICAL TABLES

OF

TWO HUNDRED CASES OF OPERATION

FOR APPENDICITIS.

FURNISHED BY

C. B. LOCKWOOD, F.R.C.S.Eng.,

SURGEON TO ST. BARTHOLOMEW'S HOSPITAL.

Statistical Tables of Two Hundred Cases of Operation for Appendicitis; furnished by C. B. Lockwood, F.R.C.S.Eng.

(The word *First* means that the wound healed by first intention.)

No.	Sex and age.	Position of appendix.	Kind of appendicitis, microscopic.	Complications.	Results.		Remarks.
					Immediate.	Ultimate.	
1	F. 32	Pelvic brim	Mucous membrane inflamed; distended lumen	Muco-membranous colitis	First	Improved	Preceded by influenza and pneumonia.
2	M. 10	Iliac fossa	Chronic ulcerative with two big concretions, one above and one below kink	Thrombosis of mesenteric veins; intestinal obstruction	Died	—	—
3	M. 26	Retrocolic	Fœcal and bacterial contents; distended; ulceration of mucosa	Mucous colitis	First	Cured	Pain left side of abdomen.
4	F. 34	Brim of pelvis	Purulent and septic infiltration; stenosed and dilated	Hæmatosalpinx	First	Cured	—
5	M. 12	Iliac	Mucosa destroyed; many bacteria in peritoneal lymph; acute septic inflammation	Acute local abscess	Drained	Recovered	—
6	F. 22	Pelvic	Distension of lumen and glands; ulceration of mucous membrane; bacterial invasion; concretions; septic contents	—	First	Cured	Pain under liver at gall-bladder; diarrhœa.
7	M. 27	Inguinal hernia (congenital sac)	Concretion; ulceration of mucosa	—	First	Cured	Truss became painful.
8	M. 8	Pelvic brim	Acute inflammation; mucosa denuded; peritoneum infected	Acute iliac abscess	Drained	Cured	Early operation.
9	F. 19	Pelvic	Acute inflammation; septic	Pelvic abscess	Drained	Cured	No perforation.
10	M. 27	Ileocolic fossa	Chronic inflammation; septic contents	—	First	Cured	—
11	F. 35	Iliac	Constriction; dilatation; muco-purulent contents	—	First	Cured	—
12	M. 60	Iliac	Distended; fæcal contents; mucosa denuded and invaded by bacteria	—	First	Cured	—

		Iliac	Constriction; dilated and thinned; a cyst; ulceration of mucosa; tuberculous	—	First	Well 4 years after	Colitis.
		Iliac	Acutely inflamed; acute kink; distended; septic contents	—	First	Cured	On point of rupture.
		Pelvic	Inflamed adherent omentum; constricted; distended	—	First	Cured	—
		Pelvic	Ulceration of mucosa; kinked; distended with fæces	—	First	Cured	Son also operated upon for acute appendicular abscess with fæcal concretion. Floating cæcum. Spurious relapse.
		Iliac; adherent	Ulceration of mucosa; muco-purulent contents; lymphadenitis	—	First	Cured	Ten days after third severe attack.
		Iliac	Ulceration of mucosa; distended; septic contents	—	First	Cured	Healed perforation.
		ıac; adherent	Septic contents; superficial ulceration of mucosa	—	First. Drain for blood	?	Attack 10 years before recurrence; cæcum injured and sutured.
	32	External	Lymphadenitis; septic contents	—	First	Cured 5 years after	—
	M. 18	External	Concretion; gangrene; perforation; mucosa destroyed; bacteria throughout. Concretion	Acute abscess, localised	Drained	Cured	Meso-appendix gangrenous.
22	M. 23	Pelvic	Perforation; gangrene; concretion	Septic peritonitis; fæcal fistula ileum, due to ulceration	Drained	Cured	Pelvis drained; meso-appendix gangrenous.
23	F. 45	Iliac; adherent	Ulceration of mucosa; peritoneum infected	Small abscess	First	Cured	Well 6 years after.
24	M. 16	Iliac	Constricted, two places; distended; superficial ulceration	—	First	Cured	—
25	M. 32	Iliac	Commencing ulceration of mucosa, distal end obliterated by inflammatory tissue throughout	—	First	Cured	—
26	M. 24	Iliac; adherent	Healed perforation	—	First	Cured	Scar weak, outer end.
27	F. 32	Iliac	Kinked; distended septic contents; commencing superficial ulceration of mucosa	—	First	Cured	—

No.	Sex and age.	Position of appendix.	Kind of appendicitis, microscopic.	Complications.	Results.		Remarks.
					Immediate.	Ultimate.	
28	F. 26	Iliac	Superficial ulceration of mucosa; septic contents; culture, colon bacillus	—	First	Cured	Appendicular colic.
29	F. 14	Iliac	Perforated ulceration of mucosa; lymphadenitis	Abscess	Drained	Cured	—
30	M. 35	Iliac	Septic contents; lymphadenitis	—	Partial superficial suppuration]	Weak scar. Cured	—
31	M. 35	Pelvic; dense adhesions	Distended; ulceration of mucosa; septic contents; invasion	—	Drained	Cured	Operation took 2¼ hours.
32	M. ? 15	Iliac; adherent	Slight inflammation of peritoneal coat	—	First	—	After 2 slight attacks.
33	M. 14	Pelvic brim	Septic contents; mucosa destroyed; gangrenous	Pus in iliac fossa and pelvis	Drained	Recovered	Semilunar incision; fresh pus let out 7 days after first from pelvis and abdomen.
34	F. ? 17	Iliac	Lymphadenitis	—	First	—	—
35	M. 25	Iliac	Ulceration of mucosa; septic contents; concretion	—	First	Cured	Came under my care for varix of leg veins.
36	F. 22	Iliac	Hardly any changes; slight old blood extravasation	—	First	Not 3 mos. after.	—
37	M. ? 15	Iliac; adherent omentum	All coats inflamed; mucosa destroyed	—	First	Cured	Appendix removed, Nov., 1896. Dec., 1899, in for radical cure of hernia; well 9 yrs. after.
38	M. 14	Iliac	Mucosa ulcerated; septic contents; perforated and infected; concretions, 2	Acute suppurative peritonitis	Drain	Recovered	—
39	M. 10	Iliac; omental cap	Mucosa destroyed; septic contents; perforation; concretion	Acute suppurative peritonitis	Drains	Recovered	Suprapubic drain; well 6 years after.
40	F. 16	Iliac	Mucosa destroyed; septic contents; pus; perforated	Acute iliac abscess	Drain	Cured	Subsequently a ventral hernia was repaired.

41	M. 40	Iliac	Fibrous obliteration	—	Hæmatoma; suppuration	Cured	—
42	M. 15	Iliac	Dilated lumen; lymphadenitis	Lymphatic glands enlarged	First	Cured	—
43	M. 20	Iliac	Tuberculosis of peritoneal coat; tubercle bacilli not found; tuberculous peritonitis	Tuberculous peritonitis	First	Much improved	Not diagnosed. Went to Australia.
44	M. 29	Iliac	Septic contents; ulceration of mucosa	—	First	Cured	Dilated cæcum caused spurious relapse.
45	M. 32	Iliac; adherent to cæcum	Mucosa destroyed; fibrous obliteration	—	First	Cured	In Bartholomew's and London; 20 attacks.
46	M. 6	Iliac	Mucosa destroyed; remainder infected; perforated	Subcæcal acute abscess	Drain	Recovered	—
47	M. 14	Retrocolic	Empyema of appendix	—	Slight suppuration	Cured	Pus escaped during operation.
48	M. ? 30	Iliac	Septic contents; superficial ulceration of mucosa	—	Slight suppuration	Cured	Hæmatoma.
49	F. 10	Pelvic	Moderately inflamed	Septic sinus at time of operation after abscess had been opened	Drained	Died	Uncontrollable vomiting.
50	F. ? 25	Iliac	Concretion; bacterial mass; ulceration of mucosa; bacterial invasion	—	First	Cured	—
51	F. ? 35	Iliac; adherent	Slight chronic inflammation	—	First	Cured	20 attacks.
52	M. 25	Iliac	Lymphadenitis	—	First	Cured	—
53	M. 35	Iliac; adherent	Chronically inflamed and obliterated	Pain over gall-bladder; blood and oxalate of lime in urine; tenderness in iliac fossa	First	Cured; seen two years after	Exploratory incision in right semilunar line —oblique over appendix. Diagnosis doubtful; trouble in either gall-bladder, kidney, or appendix.
54	M. 38	Iliac	Mucocele	—	First	Cured	—

No.	Sex and age.	Position of appendix.	Kind of appendicitis, microscopic.	Complications.	Results.		Remarks.
					Immediate.	Ultimate.	
55	F. 37	Iliac	Columnar-celled carcinoma	—	First	—	Afterwards resection of cæcum; relapse 10 months; intestinal anastomosis and inguinal colotomy. Died six months later.
56	M. 68	Iliac	Bacterial contents; ulceration of mucosa	Albuminuria	First	Cured	—
57	F. 45	Pelvic	Mucous membrane destroyed; septic contents concretion	Uterine fibroids; acute abscess, pelvic	Drained	Died	—
58	M. 37	Inguinal hernia	Chronic inflammation; several shot	—	Suppurated	Cured	—
59	F. 6	Iliac	Perforated at operation; slight changes; peritoneum inflamed	Iliac abscess; pelvic abscess	Drained	Cured	Intestinal obstruction; sapræmia; obstruction by band 6 months after. Recovered after operation.
60	M. 11	Iliac	Tuberculous empyema of appendix	—	Suppurated	—	Died about a year after of nephritis and empyema.
61	F. 54	Iliac; adherent	Fibrous obliteration; lumen size of tubular gland, complete at distal end	—	First	Cured	Ten years' interval between attacks.
62	M. 37	Iliac	Constricted; ulceration of mucosa; septic phlebitis	Multiple liver abscesses	Drained	Died	Portal pyæmia, probably due to streptococci.
63	F. 13	Iliac; adherent	Lymphangitis and lymphadenitis	—	First	Cured	—
64	F. 34	Iliac	Constriction (mucosa intact); septic contents, streptococci	—	First	Cured	—
65	F. 18	Iliac; adherent	Peritoneum much inflamed	—	Drained first	Cured	Right broad ligament inflamed.
66	F. 24	Iliac	Kinked; Bacterial contents; Superficial ulceration } slight	—	First	Doubtful, 14 months	Operation considered exploratory.

67	M.? 10	Pelvic	Acute; ulceration of mucosa; lymphadenitis	—	First	—	—
68	F. 39	Iliac	Fibrous obliteration; also ? carcinoma	—	First	Well 14 mos. after	—
69	M. 33	Ileo-cæcal fossa	Kinked; bacterial contents; ulceration of mucosa	—	First	Cured	—
70	M. 21	Iliac	Ulceration of mucosa; muco-pus and bacilli in lumen	—	First	Cured	—
71	F. 19	Retrocæcal; adherent	Slightly inflamed peritoneum	Probably enteroptosis	First	Doubtful. Occasional attacks of pain; since married]	Hepatic flexure of colon prolapsed.
72	F. 40	Iliac	Pigmentation of cells in mucosa	—	First	No result	Exploratory median incision.
73	M. 15	Iliac	Kinked; thread-worms and bacterial contents; ulceration of mucosa	—	First	Cured	—
74	M. 28	Pelvic brim	Bacterial contents; ulceration of mucosa	Mucous colitis	First	Cured	Exploratory.
75	F. 52	Iliac	Fæcal and bacterial contents; slight inflammation	Chronic dyspeptic constipation	First	Improved	—
76	M. 36	Retrocolic	Bacterial and muco-purulent contents; ulceration of mucosa	Mucous colitis	First	Cured	Mucous colitis; pain for a time left side of abdomen.
77	M. 16	Retrocolic	Bacterial contents; ulceration of mucosa; concretions	Mucous colitis	First	Cured	Mucous colitis; pain left side.
78	F. 44	Iliac	Bacterial contents; concretion; ulceration of mucosa	Infection of left ovary and tube	First: median incision	Cured	Found by accident during removal of inflamed left ovary and tube. Right previously removed
79	F. 32	Iliac	Distended; fæcal contents; ulceration of mucosa; lymphadenitis	—	First	Cured	—
80	M. 21	Iliac	Purulent contents; ulceration of mucosa	—	First	Cured 4 years after	—
81	F. 35	Iliac	Inflammatory obliteration	—	First	Cured	—
82	M. 28	Iliac	Mucosa ulcerated	—	First	Cured	Attack of pleurisy during convalescence.
83	F. 28	Ileo-cæcal fossa	Purulent and bacterial contents; ulceration of mucosa; bacterial invasion]	—	First	Cured	—

No.	Sex and age.	Position of appendix.	Kind of appendicitis, microscopic.	Complications.	Results.		Remarks.
					Immediate.	Ultimate.	
84	M. 16	Pelvic	Kinked; distended; bit of glass in contents; fæcal and bacterial invasion of mucosa	Rigors (bacterial invasion)	First	Cured	—
85	M. 16	Iliac	Fæcal contents; concretions; ulceration of mucosa	Thrombosis, mesenteric veins	—	Died	Exploratory.
86	M. 15	Iliac	Constriction; fæcal contents; distension; all coats infected	—	First	Cured	Epithelial lining of lumen thinned, but intact.
	M. 30	Iliac; adherent	Ulceration of mucosa; enormous thickening of all coats	—	First	Cured 5 years after	—
	F. 29	Iliac	Septic contents; ulceration of mucosa; coats greatly thickened	—	First	Cured	? Slight enteroptosis.
	M. 36	Iliac	Kinked; distended; fæcal-bacterial contents; ulceration of mucosa	—	First	Cured	—
	M. 9	Iliac	Ulceration of mucosa; lymphadenitis	—	First	Cured	—
	F. 29	Iliac	Septic infection of all coats, especially peritoneal	Perforated, acute abscess	Drained	Cured	Thrombosis of left iliac vein during convalescence.
	F. 10	Iliac	Stenosis; dilated; thread-worms and fæcal contents; ulceration of mucosa	—	First	Cured Feb. 9th, 1905	—
	F. 23	Pelvic; adherent	Ulceration of mucosa; fibrous obliteration	—	First	Cured	—
	F. 27	Iliac	Ulceration of mucosa (all coats thickened)	—	First	Cured	—
	28	Iliac	Stenosis (by compression of greatly thickened œdematous coats)	Right inguinal hernia (came for hernia)	First	Cured	Radical cure at same time.

96	M. 24	Iliac; adherent	Ulceration of mucosa; lumen full of granulation tissue	—	First	Cured	Recovery slow.
97	M. 9	Pelvic	Gangrenous; perforated; many bacteria in remains of mucosa	Intestinal obstruction; general septic peritonitis	Drained	Died	See 119 for sister also operated upon for acute pelvic appendicular abscess; recovered.
98	F. 40	Iliac; adherent	Distended; muco-purulent and fæcal contents; mucosa denuded	Probably kinked in ileo-cæcal fossa	First	Cured	—
99	M. 26	Iliac	Ulceration and destruction of mucosa; acute septic inflammation; lumen closed; bacteria throughout	Acute iliac abscess	Drained	Cured	—
100	F. 22	Iliac	Stenosis; distended; fæcal contents and concretion; ulceration of mucosa	—	First	Cured	—
101	F. 36	Iliac	Stenosis; ulceration of mucosa; purulent septic contents	—	First	Cured	—
102	M. 25	Ileo-cæcal fossa, adherent	Inflammatory obliteration of lumen	—	First	Partial cure	? Colitis.
103	M. —	Pelvic brim	Mucosa inflamed; inflamed submucosa and peritoneum	—	First	—	—
104	F. 33	Pelvic	Mucosa destroyed; fibrous obliteration	Floating cæcum	First	Not cured	—
105	M. 42	Iliac	Inflammatory obliteration	—	First	Slight improvement	—
106	F. 39	Pelvic	Ulceration of mucosa; granulating	—	First	Cured	—
107	M. 17	Iliac	Kinked; distended; concretions; mucosa denuded; bacterial contents	—	First	Cured 4 years after	5-inch-long appendix, semilunar incision.
108	F. 27	Iliac	Kinked; concretion; mucosa denuded; lymphadenitis	—	First	Cured	—
109	M. 14	Iliac	Concretion; septic contents; ulceration of mucosa; perforation	Acute iliac abscess	Drained	Recovered; cured 4 years after	—
110	M. 35	Iliac	Fibrous obliteration; carbonate of lime crystals in remains of lumen	—	First	Cured	Had excessive pain during attacks; 2 years' history.

No.	Sex and age.	Position of appendix.	Kind of appendicitis, microscopic.	Complications.	Results.		Remarks.
					Immediate.	Ultimate.	
111	M. 23	Retrocolic	Septic contents; ulceration of mucosa	—	First	Cured 3 years after	—
112	M. 72	Iliac	Acute infection; many bacilli; perforated	Acute iliac abscess and peritonitis	Drained	Died	Acute septic appendicitis supervening on chronic ulceration.
113	F. 25	—	Fæcal and bacterial contents; distended; mucosa denuded and invaded	—	First	Cured	—
114	M. 25	Iliac	Fæcal contents; concretions; ulceration of mucosa; invasion	—	First	Cured	—
115	F. 14	Iliac	Fæcal contents; ulceration of mucosa; infiltration of mucosa; septic peritoneum	Iliac abscess, acute	Drained	Cured	Bacteria in lumen and in peritoneal lymph morphologically similar.]
116	M. 37	Iliac	Perforation (supervening on stenosis); mucosa nearly destroyed; cocci and bacilli in inflamed peritoneum	A purgative brought on crisis; acute iliac abscess	Drained	Cured a year after	—
117	M. 22	Pelvic	Distended; purulent contents; ulceration of mucosa	—	First	Cured	Long incision; abdominal wall weak.
118	F. 35	Iliac	Distended; purulent and fæcal contents; mucosa denuded	—	First	—	—
119	F. 16	Pelvic	Ulceration of mucosa; perforated	Acute pelvic abscess	Drained	Cured	For brother, see 97.
120	M. 35	Iliac	Ulceration and destruction of mucosa; muco-purulent and septic	— contents]	First	Cured 4 years after	—
121	M. 16	Retrocolic	Distended; muco-purulent contents; ulceration of mucosa; bacterial invasion	—	First	—	—
122	M. 25	Iliac	Stenosis; ulceration of mucosa	—	First	Cured 3 years after	—
123	M. —	—	Ulceration of mucosa	—	First	—	—
124	M. 10	Pelvic	Fæcal concretion; distension; ulceration of mucosa; bacterial	— invasion]	First	Cured 3 years after	For sister, see 196.

125	F. 16	Undescended cæcum; under liver; above iliac crest	Ulceration of mucosa; septic peritoneum	Several loculi of stinking pus	Drained	Cured 3 years after	For brother, see 197.
126	M. 27	Iliac	Constricted; distended; mucopurulent; denuded of epithelium	—	First	Cured 3 years after	Since—flatulence and constipation, both slight
127	M. 15	Retrocolic	Distended; pus and intestinal bacteria; ulceration of mucosa	—	First	Cured	—
128	M. 16	Retrocolic	Ulceration of mucosa	—	First	Cured	—
129	M. 27	Ileo-cæcal fossa	Tuberculous; tubercle bacilli in peritoneum; ulceration of mucosa	Tubercle of lung	First	Tubercle progressed *in situ*	Silk extruded.
130	F. 32	Iliac	Distended; fæcal contents; ulceration of mucosa; bacterial invasion	—	First	Cured	—
131	F. 51	Iliac	Kinked; distended; concretion; ulceration of mucosa; bacterial invasion	—	First	Cured	Afterwards jaundice.
132	M. 36	Iliac	Ulceration of mucosa; purulent contents	(Subdiaphragmatic abscess) septic sinus; previous operation	Drained	—	Sinus not healed two years after, but otherwise well.
133	F. 28	Iliac	Distended; concretions; ulceration of mucosa	Extensive adhesions due to gastric ulcer	First	—	Died two months later of acute intestinal obstruction.
134	M. 15	Iliac	Kinked; distended; superficial ulceration of mucosa	—	First	Cured	—
135	M. 30	Pelvic; adherent	Constricted; distended, purulent contents; ulceration of mucosa; superficial: purulent infiltration	—	First	Cured 2½ years after	—
136	F. 28	Iliac	Normal; slightly distended with fæces	Riedel's lobe	First	—	Riedel's lobe subsequently removed, *v.* 'Lancet,' July 25th, 1903, p. 223.
137	F. 39	Iliac	Kinked; distended; denuded	—	First	Cured	Naked-eye diagnosis of tubercle not confirmed by microscope.
138	M. 42	Iliac	Much thickening; mucosa denuded of epithelium and glands; lumen compressed	—	First	Cured 2 years after	—

No.	Sex and age.	Position of appendix.	Kind of appendicitis, microscopic.	Complications.	Results.		Remarks.
					Immediate.	Ultimate.	
139	F. 22	Iliac	Ulceration of mucosa; bacterial invasion; perforation; remains of mucosa and the peritoneum have many bacteria in them	Right Fallopian tube and broad ligament inflamed; acute pelvic abscess	Drained	Cured 2 years after	
140	F. 39	Iliac	Kinked; stenosed at kink; distended, mucous, epithelium, and pus-cells; mucosa thinned	—	First	Cured	—
141	F. 17	Iliac	Fæcal contents; ulceration of mucosa; bacterial invasion	—	First	Cured 2½ years after	—
142	F. 32	Iliac	Constricted; fæcal mass; ulceration of mucosa	—	First	Cured	—
143	M. 41	Iliac; adherent	Ulceration of mucosa; contained muco-pus	—	First	Cured 5 years after	—
144	M. 10	Iliac	Fibrous constriction and obliteration of lumen; distension; muco-purulent septic contents; ulceration of mucosa	—	First	Cured	Rigors.
145	M. 19	Iliac; adherent	Dilated; denuded; muco-purulent contents: invasion	—	First	—	Sept. 14th, 1901, acute appendicular abscess opened; Sept. 9th, 1902, weak scar; continued appendicitis; removal appendix
146	M. 26	Iliac	Fibrous obliteration and strictures; lumen distended; ulceration of mucosa; muco-purulent septic infiltration	—	First	Cured	Fibrous tissue obliterating lumen contains many pigment cells.
147	F. 25	Iliac	Fibrous obliteration; slightly distended; ulceration of mucosa	—	First	Cured	—
148	M. 29	Pelvic	Ulceration of mucosa; muco-purulent septic contents; intestinal bacteria	—	First	Cured 3 years after	—

		[illegible]	? Inflamed; probably normal	Part of cæcum much inflamed	First	Cured, 1½ years after	—
		Iliac	Kinked; ulceration of mucosa; fæcal contents; bacterial invasion	—	First	Cured 3 yrs. after	—
		Pelvic	Ulceration of mucosa; muco-purulent and bacterial contents	—	First	Cured 2 yrs. after	At operation lumen full of pus.
		Iliac	Kinked; superficial ulceration of mucosa; mucosa nearly intact, but peritoneum and mucosa and submucosa much inflamed	—	First	Cured	Entered Army; naked-eye evidence marked.
	45	Iliac	Ulceration of mucosa, deep	—	First	Cured 2 yrs. after	Could not work for 2 years before operation; now at work. Cæcum was adherent to front abdominal wall; occasional slight pain when standing.
154	M. 28	Iliac; adherent	Ulceration of mucosa, deep; lumen nearly obliterated	—	Drained first	Cured 18 months after	Previous attempt by another surgeon.
155	M. 19	Retrocolic; adherent	Distended; fæcal contents; ulceration of mucosa; bacterial invasion	—	Suppurated	—	Fæcal contents escaped during removal.
156	F. 7	Pelvic brim	Ulceration of mucosa; muco-purulent bacterial contents, bacilli and probably streptococci; gangrene	Pus in iliac fossa and pelvis; right Fallopian tube infected	Drained	Died	Three days of very acute sepsis. T. 102.° P. 130.
157	M. 5	Iliac	Ulceration of mucosa, deep; purulent and bacterial contents	—	First	Cured	—
158	F. 8	Outside cæcum	Epithelium and glands destroyed; lumen full of pus and bacteria; bacteria in mucosa and peritoneal lymph	Acute iliac abscess	Drained	Recovered	—
159	F. 7	Pelvic	Ulceration of mucosa; purulent and bacterial contents; perforation	Acute pelvic abscess	Drained	Died	—
160	F. 14	Iliac; adherent	Ulceration of mucosa; bacilli in peritoneal lymph	Periappendicular abscess	Drained	Cured	—

No.	Sex and age.	Position of appendix.	Kind of appendicitis, microscopic.	Complications.	Results.		Remarks.
					Immediate.	Ultimate.	
161	M. 21	Iliac	Ulceration of mucosa, deep	—	Suppurated	Cured 2 yrs. after	—
162	M. 34	Iliac	Constricted; lumen obliterated; distended; ulceration of mucosa; muco-purulent bacterial contents	—	First. Hæmatoma	Cured 2 yrs. after	Ultimately lost some buried sutures.
163	M. 45	Iliac	Ulceration of mucosa, slight; inflammatory obliteration of lumen	—	First	Cured. Well 2 years after	—
164	M.?23	Pelvic brim	Ulceration of mucosa; muco-purulent bacterial contents; invasion	—	First	—	Semilunar incision.
165	M. 9	External to cæcum and colon	Mucosa nearly destroyed; remains of mucosa full of bacteria, also peritoneum; bacilli; cocci; streptococci	Acute abscess	Drained	Recovered	(Weak scar ?).
166	M. 19	Iliac; omental adhesions	Ulceration of mucosa; fæcal contents; bacterial invasion; fistula; (concretion escaped Dec. 13th, 1901)	Intraperitoneal appendicular fistula	Cat-gut and fishing-gut	Suppurated; Recovered	Dec. 13th, 1901, abscess; Jan. 4th, 1902, empyema; April 7th, 1902, appendix excised; weak scar.
167	M. 43	Iliac	Chronic inflammation of coats; mucous glands dilated; crystals of carbonate of lime (?) mucocele	—	First	Cured	—
168	F. 43	Ileo-cæcal	Superficial ulceration of mucosa; fibrous obliteration of lumen	—	First	Cured	—
169	M. 16	Iliac	Distended; superficial ulceration of mucosa; muco-purulent bacterial contents; infiltration of mucosa	—	First	Cured	Fœtal type.
170	M. 29	Iliac	Superficial ulceration of mucosa	—	First	Cured	Fœtal type.
171	M. 17	Iliac	Stenosis of lumen; epithelium denuded; all coats infected	—	First	Cured	Appendix wrapped in omentum.

172	F. 21	Iliac	Ulceration of mucosa; distended; muco-purulent bacterial contents, infiltrating mucosa	—	First	Cured	—
173	M. 34	Iliac	Mucosa destroyed; bacilli in peritoneal lymph	Acute abscess and much peritonitis	Drained	Died	Operation Aug. 8th; died Aug. 10th; alcoholic
174	M. 39	Pelvic	Distended; fæcal contents; coats thinned	Cæcum in pelvis; neurasthenia	First	Doubtful	—
175	M. 45	Pelvic; adherent	Mucosa destroyed; lumen obliterated; (? commencing mucocele)	—	First	Cured 2 years after	—
176	F. 23	Pelvic	Kinked; fæcal contents; mucosa denuded; bacterial invasion	Floating cæcum	—	Died	Probably a mechanical obstruction.
177*	F. 43	Iliac	Old blood and pigment in mucosa, otherwise intact	Probably enteroptosis	First	Cured 2 years after	Semilunar incision.
178	M. 25	Iliac	Purulent and bacterial contents; ulceration of mucosa: bacterial invasion	—	First	Cured	Three slight attacks; temperature normal throughout.
179	M. 64	Iliac	Distended; muco-purulent and fæcal contents; denudation of mucosa; infiltration	—	First	Cured 2 years after	Violent pain during attacks—about eight in two years.
180	M. 24	Iliac; many adhesions	Inflamed connective tissue, with some smooth muscle; nothing to identify as appendix	Abscess opened, 1901; atrophied appendix removed, March 22nd, 1902	First	Cured 3 years after	
181	F. 17	Iliac	Ulceration of mucosa; purulent and bacterial contents	—	First	Cured	Mucous colitis; slow recovery.
182	F. —	Iliac	Distended; denuded; fæcal contents; concretions, six; thread-worms	—	First	—	—
183	F. 36	Iliac	Ulceration of mucosa; obliteration of lumen, muco-pus and bacteria	Pus escaped at operation	First	Cured	—
184	M. 28	Iliac	Mucosa denuded (very little); mucosa, sub-mucosa and meso-appendix much inflamed; muco-pus; no bacteria	—	First	Cured	—
185	F. 26	Iliac	Ulceration of mucosa; obliteration of lumen	—	First	Cured	—

* Died afterwards of carcinoma of sigmoid flexure.

No.	Sex and age.	Position of appendix.	Kind of appendicitis, microscopic.	Complications.	Results.		Remarks.
					Immediate.	Ultimate.	
161	M. 21	Iliac	Ulceration of mucosa, deep	—	Suppurated	Cured 2 yrs. after	—
162	M. 34	Iliac	Constricted; lumen obliterated; distended; ulceration of mucosa; muco-purulent bacterial contents	—	First. Hæmatoma	Cured 2 yrs. after	Ultimately lost some buried sutures.
163	M. 45	Iliac	Ulceration of mucosa, slight; inflammatory obliteration of lumen	—	First	Cured. Well 2 years after	—
164	M.? 23	Pelvic brim	Ulceration of mucosa; muco-purulent bacterial contents; invasion	—	First	—	Semilunar incision.
165	M. 9	External to cæcum and colon	Mucosa nearly destroyed; remains of mucosa full of bacteria, also peritoneum; bacilli; cocci; streptococci	Acute abscess	Drained	Recovered	(Weak scar ?).
166	M. 19	Iliac; omental adhesions	Ulceration of mucosa; fæcal contents; bacterial invasion; fistula; (concretion escaped Dec. 13th, 1901)	Intraperitoneal appendicular fistula	Cat-gut and fishing-gut	Suppurated; Recovered	Dec. 13th, 1901, abscess; Jan. 4th, 1902, empyema; April 7th, 1902, appendix excised; weak scar.
167	M. 43	Iliac	Chronic inflammation of coats; mucous glands dilated; crystals of carbonate of lime (?) mucocele	—	First	Cured	—
168	F. 43	Ileo-cæcal	Superficial ulceration of mucosa; fibrous obliteration of lumen	—	First	Cured	—
169	M. 16	Iliac	Distended; superficial ulceration of mucosa; muco-purulent bacterial contents; infiltration of mucosa	—	First	Cured	Fœtal type.
170	M. 29	Iliac	Superficial ulceration of mucosa	—	First	Cured	Fœtal type.
171	M. 17	Iliac	Stenosis of lumen; epithelium denuded; all coats infected	—	First	Cured	Appendix wrapped in omentum.

172	F. 21	Iliac	Ulceration of mucosa; distended; muco-purulent bacterial contents, infiltrating mucosa	—	First	Cured	—
173	M. 34	Iliac	Mucosa destroyed; bacilli in peritoneal lymph	Acute abscess and much peritonitis	Drained	Died	Operation Aug. 8th; died Aug. 10th; alcoholic
174	M. 39	Pelvic	Distended; fæcal contents; coats thinned	Cæcum in pelvis; neurasthenia	First	Doubtful	—
175	M. 45	Pelvic; adherent	Mucosa destroyed; lumen obliterated; (? commencing mucocele)	—	First	Cured 2 years after	—
176	F. 23	Pelvic	Kinked; fæcal contents; mucosa denuded; bacterial invasion	Floating cæcum	—	Died	Probably a mechanical obstruction.
177*	F. 43	Iliac	Old blood and pigment in mucosa, otherwise intact	Probably enteroptosis	First	Cured 2 years after	Semilunar incision.
178	M. 25	Iliac	Purulent and bacterial contents; ulceration of mucosa: bacterial invasion	—	First	Cured	Three slight attacks; temperature normal throughout.
179	M. 64	Iliac	Distended; muco-purulent and fæcal contents; denudation of mucosa; infiltration	—	First	Cured 2 years after	Violent pain during attacks—about eight in two years.
180	M. 24	Iliac; many adhesions	Inflamed connective tissue, with some smooth muscle; nothing to identify as appendix	Abscess opened, 1901; atrophied appendix removed, March 22nd, 1902	First	Cured 3 years after	
181	F. 17	Iliac	Ulceration of mucosa; purulent and bacterial contents	—	First	Cured	Mucous colitis; slow recovery.
182	F. —	Iliac	Distended; denuded; fæcal contents; concretions, six; threadworms	—	First	—	—
183	F. 36	Iliac	Ulceration of mucosa; obliteration of lumen, muco-pus and bacteria	Pus escaped at operation	First	Cured	—
184	M. 28	Iliac	Mucosa denuded (very little); mucosa, sub-mucosa and meso-appendix much inflamed; muco-pus; no bacteria	—	First	Cured	—
185	F. 26	Iliac	Ulceration of mucosa; obliteration of lumen	—	First	Cured	—

* Died afterwards of carcinoma of sigmoid flexure.

No.	Sex and age.	Position of appendix.	Kind of appendicitis, microscopic.	Complications.	Results.		Remarks.
					Immediate.	Ultimate.	
186	M. 26	Pelvic brim	Distended; fæcal contents; lymphadenitis; mucosa intact; distal lumen. Inflammatory obliteration	—	First	—	—
187	M. 26	Retrocolic; adherent	Ulceration of mucosa; muco-purulent bacterial contents. Invasion of mucosa	—	Died; septic peritonitis	—	Small aperture in back of cæcum.
188	F. 3	Iliac	Ulceration of mucosa; muco-purulent and bacterial contents; invasion of mucosa; peritoneal lymph full of bacteria	Perforated septic peritonitis; subphrenic and hepatic abscesses	—	Died	—
189	F. 23	Iliac	Kinked; ulceration of mucosa; fæcal contents; concretions; fæcal extravasation into lymphoid tissue and lymph paths	Septic endometritis 5 months before operation, following confinement	First	—	Semilunar incision.
190	F. 12	Pelvic brim	Gangrenous patch; ulceration of mucosa; purulent and fæcal contents; bacteria abound in mucosa, peritoneum, and meso-appendix	Pus in iliac fossa and pelvis	Drained	Cured	Operation 16 hours after attack began.
191	F. 27	Iliac	Distended slightly; fæcal contents; epithelial lining intact; mucosa and sub-mucosa slightly inflamed	—	Suppurated; silk extruded	Cured	At operation the fæcal contents felt hard.
192	F.17	Iliac	Ulceration of mucosa; purulent and bacterial contents infiltrating mucosa	—	First	Cured	Brother died after operation for appendicitis.
193	M. 11	Iliac; adherent omentum	Ulceration of mucosa; purulent contents	Peri-appendicular abscess	First	Cured	Catgut throughout.
194	M. 9	Pelvic brim	Ulceration of mucosa; fæcal contents; bacterial invasion; lymphadenitis	(Calcareous nodule at base of meso-appendix)	First	Cured	Lymphatic glands of neck and elsewhere enlarged.

		—o; adherent	Stenosis; distension; mucosa destroyed; mucous contents	—	First	Cured	Well for some months and died a year after of cancer of the liver (columnar-celled).
197	F. 16	Pelvic	Constricted; distended; muco-pus	—	First	Cured	For brother see No. 124.
198	M. 19	Ileo-cæcal fossa	Distended; muco-purulent bacterial contents; lymphadenitis	—	First	Cured	For sister see No. 125.
199	M. 42	Iliac	Distended; fæcal contents; mucosa ulcerated, with bacterial invasion	—	First	Cured	Six attacks in five years.
200	M. 23	Iliac	Kinked; distended; purulent bacterial contents; ulceration of mucosa; bacteria in mucosa and peritoneal lymph	—	First	Cured	Condition of peritoneum unsuspected. One moderate attack with rigors.
	F. 28	Pelvic; adherent	Acute bend on itself; ulceration of mucosa; muco-purulent contents; some bacteria	—	First	Well 4 months after	—

* Died a year after of columnar-celled carcinoma of the rib and liver.

XXXVIII.

SUMMARY OF 200 CASES.

Males, 118. Females, 82.

Microscopical condition of appendix after removal.

		No. of Case.
Unrecognisable	1	180
Normal	2	36, 136
Doubtful	3	71, 72, 177
Carcinoma	1	55
? Carcinoma.	1	68
Tuberculous	4	13, 43, 60, 129
Intra-abdominal fistula . . .	1	166
Mucocele	2	54, 167
Melanosis	1	72
With ulceration or denudation of mucosa	123	
(In 37 of these bacteria had penetrated into the mucosa.)		
Inflammatory obliteration of lumen .		
Kinked		
Constricted or stenosed . . .		

NOTE.—The last four are often present together in the same appendix.

Some of the contents.

		No. of Case.
Shot	1	58
Glass	1	84
Carbonate of lime crystals . . .	1	110
(Probably present in [illegible] 167 and 194.)		
Thread-worms	3	[illegible], 92, 182
F[illegible]		
[illegible]		

Complications.

		No. of Case.
Intra-abdominal suppuration*. (26 Recovered—13 males, 13 females. 7 died—3 males, 4 females.)	33	
Septic sinus (1 died.)	2	49, 132†
Thrombosis of mesenteric (2 died. Both had concretions and ulceration of mucosa.)	2	2, 85
Muco-membranous colitis	1	1
Mucous colitis	5	3, 13, 76, 77, 181
Hæmatosalpinx	1	4
Inflammation of right Fallopian tube and broad ligament of uterus	4	65, 78, 139, 156
Septic endometritis preceding	1	189
Inflammation of left Fallopian tube and ovary	1	78

Other complications, subsequent.

		No. of Case.
Pleurisy, recovered	1	82
Empyema, recovered	1	166
Multiple liver abscess, died	1	62
Subphrenic abscess, recovered	1	132
Subphrenic and hepatic abscesses, died	1	188
Thrombosis left iliac vein, recovered	1	91
Intestinal strangulation by band, recovered	1	59

* Appendix removed in all and microscopically examined.

† In both an abscess had been opened and the appendix left. In two others, 166 and 180, abscesses had been opened. The condition of these four appendices was as follows: 49, moderately inflamed; 132, active disease; 166, active disease and intra-peritoneal fistula; 180, unrecog-

		No. of Case.
Acute obstruction by old adhesions following gastric ulcer . .	1	133
(Another patient (176) with a floating cæcum died, probably of a mechanical obstruction.)		
Not cured	8	1, 36, 75, 102,
(Of these, 3 were exploratory. Cancer and tubercle not included.)		104, 105, 136, 174
Spurious relapse	2	16, 44

Positions occupied by appendix.

Pelvic brim . . .	10	7 males, 3 females.
Pelvis . . .	27	14 „ 13 „
External to cæcum and right colon	3	
Right flank (under liver) .	1	
In inguinal hernia . .	2	
Retrocolic	11	
Ileo-cæcal	6	
Iliac	140	

Ten times the pelvic appendix was associated with intra-peritoneal abscess: 9, 22, 33, 49, 59, 119, 139, 156, 159, 190.

STATISTICAL TABLES OF ONE HUNDRED CONSECUTIVE CASES OF OPERATION FOR APPENDICITIS

FURNISHED BY

W. G. SPENCER, M.S., F.R.C.S.Eng.

SURGEON TO THE WESTMINSTER HOSPITAL

A recent series of 100 consecutive cases ending January 31st, 1905. All the cases were operated upon without delay; the chronic ones without waiting for another attack, all the rest, including doubtful cases, as soon as possible. In most of the cases, or nearly all, the diagnosis has been made or, at any rate, suggested before the cases came under my notice.

	Total.	Recoveries.	Deaths.
1. Chronic and subacute simple cases .	35 ...	35 ...	0
2. Apparently limited abscess incised and drained without immediate removal of the appendix	8 ...	4 ...	4
3. Septic inflammation in and around the appendix; extensive abscesses; septic peritonitis; in all immediate removal of appendix	57 ...	48 ...	9
	100	87	13

TABLE 1.—Appendix removed and wound closed without drainage. No hernia or other complication noted.

TABLE 2.—All the eight cases occurred amongst the first twenty of the series, and appeared to be cases of circumscribed abscesses. In the four recoveries excision was done at a second operation, and in all there was extensive disease of the appendix and around it. Among the four who died, one died three days later without more being done; in the other three a second operation was performed, and further suppuration exposed whilst dissecting out the appendix. Two developed septic empyema and died, one after the empyema had been drained, and the fourth died after further suppuration.

TABLE 3.—In all the cases, after the first twenty of the series, the appendix was invariably removed. In 34 of the recoveries the wound was sutured except for the drainage opening. All healed within about a month except one, in which there persists a mucous fistula, from which periodical menstrual fluid is discharged. The right tube, ovary, and appendix formed a suppurating mass in a girl of 17, who is now in good health, and the fistula is closing (1). The only hernia noted is in a small boy, some muscle having been destroyed by suppuration, but this can be repaired as soon as the boy is strong enough (2).

In 14 cases of recovery there was extensive abscess, or pelvic or general peritonitis. The large wound was wholly filled with gauze, and the secondary sutures inserted between the seventh and twenty-fifth days, on an average about the tenth day. All were extensive cases, but without marked tympanites, and all are soundly healed without hernia or fistula.

The nine deaths occurred as follows:—(1) A man of 24 had, six days after the acute onset, a gangrenous appendix, sticky general peritonitis and tympanites. The latter increased, and he died after three days. (2) A man aged 38 had acute intestinal obstruction with fæcal vomiting, for which he was massaged until the fifth day. He then was found to have gangrenous appendicitis, with

pus filling the lower half of the abdomen and marked tympanites; and he died three days later. (3) A boy of 15 had gangrenous appendicitis and general peritonitis. After operation he seemed to do well, and his bowels were well opened, until the fifth day, when suddenly signs of intestinal obstruction set in. This proved to be due to a volvulus of the small intestines in the pelvis, and he died twenty-four hours after reduction of the volvulus. (4) A girl of 15 had gangrenous appendicitis and purulent peritonitis, acute symptoms having been present for ten days. Tympanites increased after operation, and she died on the sixth day. (5) A woman of 40 had an enlargement of the liver area, along with signs of acute appendicitis. Gangrene of the inner layers of the appendix was found, and the wound healed, but the liver area increased without the site of metastatic suppuration being discovered, and she died after thirteen days. No *post-mortem* examination. (6) A woman of 60 had acute gangrenous appendicitis, with gangrenous infiltration of the loin. She seemed doing well when, on the sixth day, the gauze plug was omitted. Soon profuse venous hæmorrhage occurred, and I re-plugged the wound. There was no further bleeding, but the patient then did badly, and died on the eighth day. (7) An alcoholic patient had a similar condition, along with bronchitis. Although both urine and fæces escaped from the wound, it tended to improve, but the bronchitis increased, and he died eight days after the operation. (8) A man of 48 had a mass above an inflamed appendix, which turned out to be irremovable cancer of the colon and omentum. On the nineteenth day he had convulsions, and died quickly. (9) A man of 65 had also a mass above an inflamed appendix, but this proved to be a distended gall-bladder containing a large calculus, which was removed, together with the appendix. He seemed to be progressing well, and was actually writing a letter stating this, when he fell back dead. No *post-mortem* examination.

In spite of a number of severe cases, an improvement

of results has followed the invariable removal of the appendix; indeed, in many cases this course is absolutely essential for success. No ill results are attributable to it; no deaths occurred from shock or other operative complication.

Addendum July, 1905.—(1) The fistula has closed; (2) the ventral protrusion seems to be disappearing under a belt.

CASES OF APPENDICITIS,

WITH OPERATION AND HISTORY AFTER DISCHARGE FROM HOSPITAL,

DURING THE YEARS 1901, 1902, AND 1903,

UNDER THE CARE OF THE MEDICAL AND SURGICAL STAFF OF

GUY'S HOSPITAL

COLLECTED BY

RALPH THOMPSON, CH.B., F.R.C.S.,

RESIDENT SURGICAL OFFICER; LATE SURGICAL REGISTRAR.

DURING the years 1901, 1902, and 1903, 315 cases of appendicitis, with or without complications, have been operated upon at Guy's Hospital.

The cases in 1904 have not been included in any way, as these cases are too recent for a fair consideration of the patients' condition in 1905.

Of these 315 cases 202, including 37 deaths following upon operation, were traced in February, March, or April, 1905, either by means of letters or by actual examination.

Of the cases seen by medical men after operation, 67 in number, 18 per cent. suffered from ventral hernia. Of the cases reported upon by the patients themselves, 98 in number, 5 per cent. suffered from ventral hernia, a discrepancy which is not difficult of explanation, but the facts show how necessary it is to follow up cases personally.

The number of cases unrelieved, or only partially relieved, indicate that it is not enough to trace the cases only so far as their discharge from hospital.

Messrs. O. V. Payne, H. M. Clarke, F. Hardy, and B. W.

II. Operation of Appendicectomy for Relapsing Appendicitis complicated by Presence of unsuspected, or undiagnosed, Abscess.

Total number of cases		14
Deaths		0
Recoveries		14
Complications during convalescence:		
Pelvic abscess and femoral thrombosis		1
After-history in February, March, or April, 1905:		
Complete recoveries	9	
Ventral hernia	1	
	—	10

III. Operation for Appendicectomy complicated by Presence of suspected or diagnosed Abscesses.

Total number of cases		28
Deaths:		
From general peritonitis	1	
„ hyperpyrexia	1	
	—	2
Recoveries		26
Complications during convalescence:		
Fæcal fistula		1
After-history in February, March, or April, 1905:		
Complete recoveries	12	
Ventral hernia	2	
Suffering from fits following operation	1	
	—	15

IV. Operation of Appendicectomy complicated by Presence of General Peritonitis.

Total number of cases		15
Deaths:		
From general peritonitis	3	
„ general peritonitis and broncho-pneumonia	1	
„ volvulus of jejunum	1	
	—	5

VI. Operation of Laparotomy for General Peritonitis without Appendicectomy.

Total number of cases . . .		18
Deaths :		
From general peritonitis . . .	9	
„ general peritonitis and bronchitis	1	
„ fæcal fistula and general peritonitis	1	
„ intestinal obstruction . .	1	
Not accounted for	2	
	—	14
Recoveries		4
Complications during convalescence :		
Fæcal fistula	1	
Pleurisy	1	
	—	2
After-history in February, March, or April, 1905 :		
Complete recoveries	2	

VII. Operation of Division of Appendix between Ligatures.

Total number of cases . . .	1
Deaths	0
Recoveries	1

CASES OF APPENDICITIS,

WITH OPERATION AND HISTORY AFTER DISCHARGE FROM HOSPITAL,

DURING THE YEARS 1901, 1902, AND 1903,

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Messrs. O. V. Payne, H. M. Clarke, F. Hardy, and B. W.

Lacey have rendered assistance in collecting and examining cases.

ANALYSIS OF CASES.

I. Operation of Simple Appendicectomy for Relapsing Appendicitis.

Total number of cases		171
Deaths:		
From general peritonitis	2	
„ septicæmia	1	
„ pulmonary embolus	1	
„ gangrene of the lung	2	
„ ulceration of colon	1	
„ insanity and suicide	1	
	—	8
Recoveries		163
Complications during convalescence:		
Hæmoglobinuria	1	
Hæmaturia	1	
Fæcal fistula	1	
Femoral thrombosis	4	
Pleurisy	1	
Tonsillitis	1	
Pneumonia	2	
	—	11
After-history in February, March, or April, 1905:		
Complete recoveries	72	
No relief	6	
Partial relief	5	
Movable kidney	2	
Ventral hernia	5	
Pyuria in 1903	1	
Dead, of phthisis, 1904	1	
Chronic tubal nephritis, 1905	1	
Painful adhesions divided six months after operation	1	
Disease of great trochanter	1	

II. Operation of Appendicectomy for Relapsing Appendicitis complicated by Presence of unsuspected, or undiagnosed, Abscess.

Total number of cases		14
Deaths		0
Recoveries		14
Complications during convalescence:		
Pelvic abscess and femoral thrombosis		1
After-history in February, March, or April, 1905:		
Complete recoveries	9	
Ventral hernia	1	
	—	10

III. Operation for Appendicectomy complicated by Presence of suspected or diagnosed Abscesses.

Total number of cases		28
Deaths:		
From general peritonitis	1	
„ hyperpyrexia	1	
	—	2
Recoveries		26
Complications during convalescence:		
Fæcal fistula		1
After-history in February, March, or April, 1905:		
Complete recoveries	12	
Ventral hernia	2	
Suffering from fits following operation	1	
	—	15

IV. Operation of Appendicectomy complicated by Presence of General Peritonitis.

Total number of cases		15
Deaths:		
From general peritonitis	3	
„ general peritonitis and broncho-pneumonia	1	
„ volvulus of jejunum	1	
	—	5

Recoveries		10
Complications during convalescence :		
Subdiaphragmatic abscess and pleurisy	1	
After-history in February, March, or April, 1905 :		
Complete recoveries	6	
Ventral hernia	2	
	—	8

V. OPERATION OF INCISION OF ABSCESS WITHOUT APPENDICECTOMY.

Total number of cases		68
Deaths :		
From general peritonitis	4	
„ gangrenous mesentery	1	
„ diarrhœa	1	
„ general peritonitis and broncho-pneumonia	1	
„ secondary abscesses near appendix, and pleurisy, with pneumonia	1	
	—	8
Recoveries		60
Complications during convalescence :		
Tonsillitis and otorrhœa	1	
Urticaria.	1	
Fæcal fistula	2	
Empyema	1	
Femoral thrombosis	1	
	—	6
After-history in February, March, or April, 1905 :		
Complete recoveries	24	
Unrelieved	2	
Partial relief	2	
Recurrent abscess	1	
Persistent sinus	1	
Ventral hernia.	5	
	—	35

VI. Operation of Laparotomy for General Peritonitis without Appendicectomy.

Total number of cases . . .		18
Deaths :		
From general peritonitis . . .	9	
„ general peritonitis and bronchitis	1	
„ fæcal fistula and general peritonitis	1	
„ intestinal obstruction . .	1	
Not accounted for	2	
	—	14
Recoveries		4
Complications during convalescence :		
Fæcal fistula	1	
Pleurisy	1	
	—	2
After-history in February, March, or April, 1905 :		
Complete recoveries	2	

VII. Operation of Division of Appendix between Ligatures.

Total number of cases . . .	1
Deaths	0
Recoveries	1

List of Cases of Appendicitis with Operation, 1901–3.

No.	Reference.	Sex and Age.	Operation.	Result.	Complications.	After-history.
1	H 74, 1901	M. 16	Appendix divided between ligatures	Recovery	None.	Not traced.
2	H 143, „	M. 18	Appendicectomy	Recovery	None	In St. Helena as soldier in 1905.
3	H 202, „	M. 8	Appendicular abscess incised	Recovery	None	Not traced.
4	H 291, „	M. 19	Appendicectomy	Recovery	None	Well in April, 1905.
5	H 514, „	M. 42	Appendicectomy	Recovery	None	Not traced.
6	L 612, „	M. 28	Appendicectomy	Recovery	None	Well in April, 1905.
7	L 246, „	M.	Appendicular abscess incised	Recovery	None	Not traced.
8	L 253, „	M. 41	Appendical abscess incised in India; recurrent abscess burst five years later	Recovery	None	Not traced.
9	L 288, „	M. 27	Appendicectomy	Recovery	None	Not traced.
10	L 417*a*, „	F. 24	Appendicectomy	Recovery	None	Well in April, 1905.
11	L 484, „	F. 37	Appendicular abscess incised	Recovery	None	Large ventral hernia in 1905.
12	L 544, „	F. 16	Appendicectomy	Recovery	None	Not traced.
13	L 566, „	M. 24	Appendicectomy	Recovery	Hæmoglobinuria one month after operation	Not traced.
14	L 607, „	M. 29	Appendicectomy	Recovery	None	Not traced.
15	B 12, „	M. 29	Appendicectomy	Recovery	None	Not traced.
16	B 80, „	F. 25	Appendicectomy	Recovery	None	Pain on left side as bad as before operation; ? ovarian, April, 1905.
17	B 136, „	M. 26	Appendicectomy	Recovery	None	Well in April, 1905; slight bulging at scar.
18	B 171, „	F. 20	Unsuspected abscess incised; appendicectomy	Recovery	None	Not traced.
19	B 177, „	F. 32	Appendicectomy	Recovery	None	Eventually developed disease of great trochanter.
20	B 433, „	M. 8	Appendicectomy	Recovery	None	Not traced.
21	B 601, „	M. 35	Appendicectomy	Recovery	None	Well in April, 1905.

22	B 607, 1901	M. 47	Appendicectomy	Recovery	None	Had pain in right side for 18 months.
23	B 629, „	M., adult	Appendicectomy	Recovery	None	Well in April, 1905.
24	J 33, „	F. 17	Appendicectomy	Recovery	None	Not traced.
25	J 128, „	M. 26	Appendicectomy	Recovery	Fæcal fistula	Deficiency in abdominal wall, but no rupture in 1905; otherwise well.
26	J 192, „	M. 14	Appendicectomy	Recovery	None	Well in April, 1905; in Navy.
27	J 351, „	M. 48	Appendicectomy	Recovery	None	Well in April, 1905.
28	S 56, „	M. 41	Appendicectomy	Recovery	None	Well in April, 1905.
29	S 114, „	M. 32	Appendicectomy	Recovery	Complicated 12 days after operation by left femoral thrombosis	Not traced.
30	S 136, „	F. 14	Appendicectomy	Recovery	None	Well in April, 1905.
31	S 169, „	M. 26	Appendicectomy	Recovery	None	Not traced.
32	S 178, „	F. 21	Appendicectomy	Recovery	None	Not traced.
33	S 199, „	M. 36	Appendicectomy	Recovery	None	Not traced.
34	S 226, „	F. 25	Appendicectomy	Recovery	None	Not traced.
35	La 19, „	F. 24	Appendicectomy	Recovery	None	Not traced.
36	S 28, „	M.	Appendicectomy	Recovery	None	Not traced.
37	La 54, „	F.	Appendicectomy	Recovery	None	Not traced.
38	La 696, „	M. 24	Unsuspected abscesses incised; appendicectomy	Recovery	None	Bulging of wound. No actual rupture. Well, April, 1905.
39	La 82, „	F.	Appendicectomy	Recovery	None	Not traced.
40	La 113, „	M. 35	Unsuspected abscess found; appendicectomy	Recovery	None	Not traced.
41	La 137, „	M. 21	Appendicectomy	Recovery	None	Not traced.
42	La 169, „	F.	Appendicectomy	Recovery	None	Several operations since. No relief.
43	D 8 „	M. 21	Appendicectomy	Recovery	None	Not traced.
44	La, private case, 1903	M. 25	Appendicectomy; unsuspected abscess found and incised	Recovery	None	Well in April, 1905.
45	D 39*a*, 1901	M. 22	Appendicectomy	Recovery	None	Not traced.
46	D 52, „	F. 21	Appendicectomy	Recovery	None	Not traced.
47	D 55, „	F. 33	Appendicectomy	Recovery	None	Not traced.
48	D 89, „	M. 5	Appendicectomy; with radical cure of hernia	Recovery	None	Not traced.
49	D 124, „	M. 36	Appendicectomy	Recovery	None	Not traced.

No.	Reference.	Sex and age.	Operation.	Result.	Complications.	After-history.
50	D 168, 1901	F. 25	Appendicectomy	Recovery	None	Not traced.
51	D 173, „	M. 34	Appendicectomy	Recovery	None	Not traced.
52	D 10A, „	M. 17	Laparotomy for general peritonitis	Recovery	None	Not traced.
53	D 28, „	M. 45	Appendical abscess incised	Recovery	None	Not traced.
54	D 76, „	M. 18	Appendical abscess incised March, 1900; recurrence of pain, appendicectomy, 1901	Recovery	None	Not traced.
55	F 35, „	F. 13	Appendicectomy	Recovery	Complicated by hæmaturia and albuminuria	Not traced.
56	T 14, „	M. 9	Abscess incised; appendicectomy	Recovery	None	Well in March, 1905.
57	T 16, „	M. 15	Abscess incised; appendix left	Recovery	Complicated by tonsillitis and otorrhœa	Two attacks of pain two months after operation. Not traced further.
58	T 33, „	M. 3½	Abscess incised; appendix left	Recovery	None	Not traced.
59	T 172, „	M. 11	Suppurative peritonitis; laparotomy; appendicectomy	Recovery	None	Well in March, 1905.
60	T 233, „	M. 28	Appendicectomy	Recovery	None	Not traced.
61	T 326, „	F. 57	Appendicular abscess and general peritonitis; laparotomy	Death	General peritonitis	—
62	T 343, „	M. 28	Abscess incised; appendix left	Recovery	None	Ventral hernia in April, 1905, otherwise well.
63	W 128, „	F. 24	Appendicectomy	Recovery	None	Well in April, 1905.
64	W 163, „	M. 15	Abscess incised	Recovery	None	Not traced.
65	W 306, „	M. 18	Abscess incised	Death	Subpleural hæmorrhages; gangrenous mesentery; acute enteritis	—.
66	W 333, „	M. 23	Abscess incised	Recovery	None	Not traced.

69	P 377,	,,	M. 38	Appendicectomy	Recovery	None	Well in March, 1905.	
70	Pe 50,	,,	M. 29	Appendicectomy	Recovery	Pleurisy with sterile effusion; hæmorrhage from bowel twice	Not traced.	
71	Pe 138,	,,	M. 13	Appendicectomy	Recovery	Tonsillitis	Not traced.	
72	Pe 193,	,,	F. 18	Appendical abscess incised; recurrence; opened again	Death	General peritonitis; sloughing appendix	—	
73	Pe 388,	,,	M. 19	Appendical abscess incised	Recovery	None	Well in March, 1905.	
74	C 7,	,,	M. 16	Appendicular abscess incised; appendix left	Recovery	Fæcal fistula	Well in March, 1905.	
75	C 32,	,,	M. 8½	Appendicular abscess incised	Recovery	Urticaria	Well in March, 1905.	
76	C 79,	,,	M. 10	General peritonitis; laparotomy	Death	Gangrenous appendix	—	
77	C 71,	,,	M. 7	Appendical abscess incised	Death	Diarrhœa	—	
78	C 142,	,,	M. 7	Appendicectomy with general peritonitis	Recovery	Recurrent abscess opened	Not traced.	
79	C 200,	,,	M. 14	Abscess incised; appendicectomy	Recovery	None	Not traced.	
80	C 202,	,,	M. 14	General peritonitis; appendicectomy	Recovery	None	Well in March, 1905.	
81	C 211,	,,	F. 20	Appendicular abscess incised; appendicectomy	Recovery	None	Not traced.	
82	C 265,	,,	M.	General peritonitis; laparotomy	Death	—	—	
83	C 428,	,,	F. 20	Abscess incised	Recovery	None	Well in March, 1905.	
84	C 515,	,,	M. 18	Appendical abscess incised	Recovery	None	Not traced.	
85	C 534,	,,	F. 19	Appendical abscess incised	Death	Second abscess opened *per vaginam*; pleurisy and hypostatic pneumonia	—	
86	C 528,	,,	M. 10	Abscess incised; appendicectomy	Recovery	None	Well in March, 1905; slight ventral hernia.	
87	C 542,	,,	M. 35	General peritonitis; laparotomy	Death	Distended cæcum punctured; localised abscess; œdema of lung; fatty liver	—	

No.	Reference.	Sex and age.	Operation.	Result.	Complications.	After-history.
88	C 553, 1901	F. 18	General peritonitis; appendicectomy	Death	General peritonitis	—
89	C 573, „	M. 8	Appendicular abscess incised	Recovery	Recurrent abscess incised; recurrent pain later	Not traced.
90	C 595, „	F. 8	Abscess incised; appendicectomy	Recovery	None	Not traced.
91	C 611, „	M. 12	General peritonitis; appendicectomy	Death	General peritonitis	—
92	C 682, „	M. 13	General peritonitis; laparotomy	Death	General peritonitis and subdiaphragmatic abscess	—
93	C 705, „	M. 28	General peritonitis; laparotomy	Death after acute intestinal obstruction; general peritonitis and congestion of lungs		—
94	H 29, 1902	F. 20	Appendicectomy	Recovery	None	Not traced.
95	L 58, „	F. 27	Appendicectomy	Recovery	None	Not traced.
96	L 186, „	M. 50	Appendicectomy	Recovery	None	Not traced.
97	L 190, „	M. 13	Appendical abscess incised; appendicectomy	Recovery	None	Not traced.
98	L 219, „	M. 22	Appendicectomy	Recovery	None	Not traced.
99	L 610, „	M. 46	Appendicectomy	Recovery	Thrombosis left leg	Well in February, 1905.
100	L 613, „	M. 14	Appendicectomy	? Recovery	None	Not traced.
101	L 615, „	M. 21	Appendicectomy	Recovery	None	Well in February, 1905.
102	L 695, „	M. 27	Appendicectomy	Death	P.M. showed ulceration of colon	—
103	B 21, „	M. 28	Appendicectomy	Recovery	None	Not traced.
104	B 129, „	F. 28	Appendicectomy	Recovery	None	Well in February, 1905; perhaps a ventral hernia.
105	B 156, „	M. 33	Appendicectomy	Recovery	None	Well in February, 1905.
106	B 260, „	M. 17	Abscess incised; appendix left	Recovery	None	Well in February, 1905.
107	B 295, „	M. 12	Appendicectomy	Death	P.M. gangrenous bowel and general peritonitis	—

110	B 346,	„	M. 25	Appendicectomy	Recovery	None	Not traced.
111	B 411,	„	M. 17	Appendicectomy	Recovery	None	Well in February, 1905.
112	B 412,	„	M. 18	Appendicectomy	Recovery	None	Not traced.
113	B 573,	„	M. 27	Appendicectomy	Recovery	None	Well in February, 1905.
114	S 18,	„	F. 20	Appendicectomy	Recovery	None	General health poor, but local condition good in February, 1905.
115	S 27,	„	F. 24	Appendicectomy	Recovery	None	Not traced.
116	S 63,	„	F. 35	Abscess incised (unsuspected); appendicectomy	Recovery	None	Well in February, 1905.
117	S 73,	„	M. 19	Abscess incised; appendicectomy	Recovery	None	Not traced.
118	S 83,	„	M. 21	Appendicectomy	Recovery	None	Well in February, 1905.
119	S 195,	„	F. 15	Appendicectomy	Recovery	None	Well in February, 1905.
120	S 203,	„	F. 23	Appendicectomy	Recovery	None	Well in February, 1905.
121	S 238,	„	F. 25	Unsuspected abscess found, appendicectomy	Recovery	Pelvic abscess during convalescence	Well in February, 1905, patient says, left leg swelled after pelvic abscess opened (*per rectum*).
122	S 240,	„	F. 30	Appendicectomy	Recovery	None	Well in February, 1905.
123	S 288,	„	F. 22	Appendicectomy	Recovery	None	Well in February, 1905.
124	S 291,	„	F. 26	Appendicectomy	Recovery	None	Well in February, 1905.
125	S 411,	„	F. 14	Appendicectomy	Recovery	None	Well in February, 1905.
126	S 428,	„	M. 32	Appendicectomy	Recovery	None	Not traced.
127	S 467,	„	F. 51	Appendical abscess incised	Recovery	None	Not traced.
128	S 473,	„	M. 11	Unsuspected abscess found, appendicectomy	Recovery	None	Well in February, 1905.
129	S 479,	„	M. 13	Appendicectomy	Recovery	None	Well in February, 1905.
130	S 490,	„	F. 14	Unsuspected abscess found, appendicectomy	Recovery	None	Well in February, 1905.
131	S 494,	„	M. 12	Appendical abscess incised	Recovery	None	Well in February, 1905.
132	S 580,	„	M. 26	Appendicectomy	Recovery	None	Well in February, 1905.
133	S 581,	„	M. 33	Appendicectomy	Recovery	None	Well in February, 1905.
134	J 12,	„	M. 22	Appendicectomy	Recovery?	None	Not traced.
135	J 36,	„	F. 20	Appendicectomy	Recovery	None	Well in February, 1905.
136	D 62,	„	M	Appendicectomy	Recovery	None	Well in February, 1905.
137	J 108,	„	F. 35	Appendicectomy	Recovery	None	Well in February, 1905.

No.	Reference.	Sex and age.	Operation.	Result.	Complications.	After-history.
138	J 127, 1902	F. 25	Appendicectomy	Recovery	Swelling of leg 14 days after operation	No relief of symptoms, seen in February, 1905.
139	J 304, „	M. 68	Appendical abscess incised	Recovery	None	Not traced.
140	J 350, „	F. 28	Appendicectomy	Death	P.-M., gangrenous cæcum, general peritonitis	—
141	J 526, „	M	Appendical abscess incised	Recovery	None	Not traced.
142	D 45, „	F. 10	Appendical abscess incised	Recovery	None	Well in February, 1905.
143	D 94, „	M. 24	Appendicectomy	Recovery	None	Well in February, 1905.
144	D 103, „	M. 32	Appendical abscess incised	Recovery	None	Well in February, 1905.
145	D 104, „	F. 20	Appendicectomy	Recovery	None	Pain in right iliac fossa whenever patient catches cold (February, 1905).
146	D 161, „	F. 52	Appendicectomy	Recovery	None	Well in February, 1905.
147	D 168, „	M. 39	Appendicectomy	Recovery	None	Only partial relief (traced February, 1905).
148	D 203, „	M. 14	Appendicectomy	Recovery	None	Well in February, 1905.
149	D 229, „	F. 22	Appendicectomy	Death	Septicæmia	—
150	F 41, „	M. 26	Appendicectomy	Recovery	None	Well in February. 1905.
151	F 65, „	F. 19	Appendicectomy	Recovery	None	Not traced.
152	F 111, „	F. 16	Appendical abscess incised	Recovery	None	Pain in appendical region came on two years after operation, especially during periods; some thickening can be felt February, 1905.
153	F 169, „	M. 20	Appendicectomy	Recovery	None	Not traced.
154	F 181, „	F. 50	Appendicular abscess incised	Recovery	Fæcal fistula	Not traced.
155	St 33, „	F. 29	Appendicectomy	Recovery	None	8½ months pregnant, Feb., 1905; wound not stretched at all; quite well.
156	St 74, „	M. 39	Unsuspected abscess found; appendicectomy	Recovery	None	Not traced.
157	St 81, „	M. 32	Appendicectomy	Recovery	None	Well in February, 1905.
158	St 165, „	M. 20	Appendicectomy	Recovery	None	Not traced.

	La 56,	„	F. 31	Appendicectomy	Recovery	Pneumonia	Well in February, 1905.
	La 80,	„	F. 22	Appendicectomy	Recovery	None	Well in February, 1905; has some pain due to moveable kidney.
163	La 134,	„	F. 23	Appendicectomy	Recovery	None	Not traced.
164	W 28,	„	M. 16	Appendicectomy	Recovery	None	Not traced.
165	Pe 236,	„	M. 22	Appendicectomy	Recovery	None	Well in February, 1905.
166	Pe 239,	„	M. 13	Unsuspected abscess found; appendicectomy	Recovery	None	Well in February, 1905.
167	Pe 271,	„	M. 35	Appendicectomy	Recovery	None	Not traced.
168	Pe 304,	„	M. 28	Appendicectomy	Recovery	None	Well in February, 1905.
169	C 3,	„	M. 51	General peritonitis; appendicectomy	Death	Fæcal fistula; general peritonitis	—
170	C 24,	„	F. 31	Pelvic abscess incised; appendicectomy	Recovery	None	Well in February, 1905.
171	C 266,	„	F. 17	Appendical abscess incised; appendicectomy	Recovery	None	Well in February, 1905.
172	C 267,	„	M. 10	Appendical abscess incised; appendicectomy	Recovery	None	Well in February, 1905.
173	C 342,	„	F. 10	Abscess incised; appendicectomy	Recovery	None	Not traced.
174	C 382,	„	M. 8	Appendicular abscess incised; appendicectomy	Recovery	None	Not traced.
175	C 595,	„	M. 22	Appendicular abscess incised; appendicectomy	Recovery	None	Not traced.
176	C 55,	„	M. 18	Appendicular abscess incised	Recovery	None	Not traced.
177	C 133,	„	M. 54	General peritonitis; laparotomy	Death	General peritonitis	—
178	C 233,	„	M. 18	Appendicular abscess incised	Recovery	None	Not traced.
179	C 359,	„	F. 19	Appendicular abscess incised	Recovery	None	Ventral hernia developed; operated upon March, 1905.
180	C 369,	„	M. 14	Appendicular abscess incised	Recovery	None	Not traced.
181	C 514,	„	M. 12	General peritonitis; laparotomy	Recovery	Fæcal fistula	Well in February, 1905.

No.	Reference.	Sex and age.	Operation.	Result.	Complications.	After-history.
182	W 270, 1902	F. 14	Appendical abscess incised	Recovery	None	Still has pain when she catches cold, sufficient to prevent her working.
183	P 210, „	M. 41	Appendicular abscess incised	Recovery	None	Well in February, 1905.
184	P 200, „	M. 39	Appendicular abscess incised	Recovery	None	Probably ventral hernia.
185	P 136, „	M. 21	Appendicular abscess opened *per rectum*	Recovery	None	Well in February, 1905.
186	P 240, „	M. 19	Appendicular abscess opened	Recovery	None	Well in February, 1905.
187	P 232, „	M. 6	Appendicular abscess opened	Death	2nd abscess had formed, which was opened; general peritonitis	—
188	P 333, „	M. 34	Appendicular abscess opened	Death	General peritonitis	—
189	P 11, „	F. 19	Appendicectomy; appendix as band producing obstruction	Recovery	None	Well in February, 1905.
190	J 34, 1903	M. 35	Appendicular abscess incised	Recovery	None	Well in February, 1905.
191	J 65, „	F. 29	Appendicectomy	Recovery	None	Not traced.
192	J 67, „	F. 35	Appendicular abscess incised; recurrence of abscess; incised; appendicectomy later	Recovery	None	Not traced.
193	J 185, „	F. 21	Appendicectomy	Recovery	None	Not traced.
194	J 214, „	M. 22	Unsuspected abscess found; appendicectomy	Recovery	None	Not traced.
195	J 215, „	M. 17	Appendicectomy	Recovery	None	Well in February, 1905.
196	J 266, „	M. 52	Appendicectomy	Recovery	None	Feb., 1905, says that exertion causes internal dragging pain; eventually closed.
197	J 272, „	F. 15	Appendicectomy	Recovery	Fæcal fistula produced by operation for obstruction	Well in Feb., 1905; "slight rupture at site of wound."

STATISTICAL TABLES

OF

WO HUNDRED CASES OF OPERATION

FOR APPENDICITIS.

FURNISHED BY

C. B. LOCKWOOD, F.R.C.S.Eng.,

SURGEON TO ST. BARTHOLOMEW'S HOSPITAL.

No.	Reference.	Sex and Age.	Operation.	Result.	Complications.	After-history.
220	S 242, 1903	F. 7	Appendical abscess incised, appendicectomy through second incision	Recovery	None	Well in February, 1905.
221	S 318, „	F. 27	Appendicectomy	Recovery	None	Well in March, 1905.
222	S 320, „	F. 19	Appendicectomy	Recovery	None	No relief. Nephrorrhaphy, 1905.
223	S 364, „	M. 19	Appendical abscess incised	Recovery	None	Recurrent pain, ventral hernia, ? relief.
224	S 428, „	M. 13	General peritonitis laparotomy	Death	Gangrenous appendix	—
225	S 439, „	M. 29	Appendicectomy	Recovery	None	Well in February, 1905.
226	S 450, „	M. 45	Appendicectomy	Recovery	None	Well in February, 1905.
227	S 459, „	M. 34	Appendicectomy	Recovery	None	Well in February, 1905.
228	S 559, „	M. 13	Appendicectomy	Recovery	None	Well in February, 1905.
229	S 592, „	M. 17	Appendicectomy	Recovery	None	Well in February, 1905, very slight ventral hernia.
230	S 598, „	M. 29	Appendicectomy	Recovery	None	Well in February, 1905.
231	S 615, „	M. 36	Appendicectomy	Recovery	None	Well in February, 1905.
232	S 689, „	M. 58	Appendicectomy	Recovery	None	Well in February, 1905.
233	S 702, „	M. 10	Appendicectomy	Recovery	None	Well in February, 1905.
234	B 30, „	M. 11	Appendical abscess incised	Recovery	None	Well in February, 1905.
235	B 37, „	M. 32	Appendical abscess incised	Recovery	None	Well in February, 1905.
236	B 38, „	M. 13	Appendical abscess incised	Recovery	None	Well in February, 1905.
237	B 90, „	F. 27	Appendicectomy	Recovery	None	Well in February, 1905.
238	B 145, „	M. 32	Appendicectomy	Recovery	None	Well in February, 1905.
239	B 235, „	F. 31	Appendicectomy	Recovery	None	Re-admitted five months later with pyuria. Relieved.
240	B 296, „	F. 25	Appendicectomy	Recovery	None	Well in February, 1905.
241	B 316, „	F. 10	Appendicectomy	Recovery	None	Ventral hernia in 1905.
242	B 422, „	M. 48	Appendicectomy	Recovery	None	Well in February, 1905.
243	B 437, „	M. 26	Appendicectomy	Recovery	None	Died of phthisis, October, 1904.
244	B 488, „	F. 17	Appendicectomy	Recovery	None	Suffering in February, 1905, from chronic tubal nephritis.
245	B 490, „	F. 17	Appendicectomy	Recovery	None	Not traced.
246	B 501, „	M. 30	Appendicectomy	Recovery	None	Well in February, 1905.

247	F	74,	„	M.	Appendicectomy	Recovery	None	Not traced.
248	F	76,	„	M. 29	Appendicectomy	Recovery	None	Well in February, 1905.
249	D	2,	„	M. 18	Appendicectomy	Recovery	None	Well in February, 1905.
250	D	43,	„	F. 15	Appendicectomy	Recovery	None	Well in February, 1905.
251	D	59,	„	M. 45	Appendicectomy	Recovery	None	Not traced.
252	D	69,	„	F. 35	Appendical abscess incised	Death	General peritonitis	—
253	D	78,	„	F. 34	Appendicectomy	Recovery	None	Now complains (March, 1905) of pain every month like old pain.
254	D	113,	„	M. 26	Appendicectomy	Recovery	None	Not traced.
255	D	115,	„	M. 23	Appendicectomy	Recovery	None	Well in March, 1905.
256	D	144,	„	M. 20	Appendicectomy	Recovery	None	Well in March, 1905.
257	D	150,	„	F. 33	Appendicectomy	Recovery	None	Some pain in appendicial region in March, 1905.
258	D	173,	„	F. 25	Appendicectomy	Recovery	None	Not traced.
259	St	90,	„	M. 24	Appendicectomy	Recovery	None	Not traced.
260	St	56,	„	M. 16	Appendicectomy	Recovery	None	Well April, 1905.
261	La	3,	„	F. 18	Appendicectomy	Recovery	None	Admitted 6 months later, abdominal adhesions divided.
262	La	11,	„	F. 29	Appendicectomy	Death	Pulmonary embolus 4th day after operation	—
263	La	20,	„	M. 34	Appendicectomy	Death	Gangrene of lungs and tuberculous bronchopneumonia on 25th day	—
264	La	53,	„	M. 22	Appendicectomy	Recovery	None	Well in April, 1905.
265	La	60,	„	M. 29	Appendicectomy	Recovery	None	Not traced.
266	La	110,	„	F. 38	Appendicectomy	Recovery	None	No relief April, 1905.
267	La	116,	„	M. adult	Appendical abscess incised	Recovery	None	Large ventral hernia April, 1905.
268	La	175,	„	M. 25	Unsuspected abscess found, appendicectomy	Recovery	None	Well in April, 1905.
269	T		„	F	Abscess incised, appendicectomy	Recovery	None	Well in March, 1905.
270	T		„	F	Abscess incised, appendicectomy	Recovery	None	Well in March, 1905, although pregnant, child nearly full term, no ventral hernia, no stretching of scar.

No.	Reference.	Sex and Age.	Operation.	Result.	Complications.	After-history.
908	C 657, 1903	M. 19	General peritonitis; laparotomy	Death	—	—
909	C 672, „	M. 11	General peritonitis; appendicectomy	Recovery	None	Well in March, 1905.
910	C 692, „	F. 35	Abscess incised; appendicectomy	Recovery	None	Not traced.
911	C 719, „	F. 18	General peritonitis; appendicectomy	Death	Peritonitis; bronchopneumonia	—
912	C 734, „	M. 11	General peritonitis; appendicectomy	Recovery	None	Well in March, 1905.
913	C 754, „	F. 21	Abscess incised; appendicectomy	Recovery	None	Well in March, 1905.
914	C 809, „	F. 12	General peritonitis; appendicectomy	Death	Volvulus of jejunum relieved by operation but death followed general peritonitis	—
—	Private case, 1903	F. adult	Appendicectomy	Recovery	Femoral thrombosis both legs	Well in March, 1905.

STATISTICAL TABLES OF ONE HUNDRED AND FORTY CASES OF OPERATION FOR APPENDICITIS

PERFORMED BY

G. R. TURNER, F.R.C.S.Eng.

AT

ST. GEORGE'S HOSPITAL, FROM 1895 to 1904.

Following the hospital method of classification, they are divided into—(1) Acute, (2) acute with abscess, (3) subacute, and (4) chronic cases.

Subacute cases may be taken to be those not operated upon in the first twenty-four hours after admission, but not chronic in their nature. *Chronic* cases are those operated on during a period of quiescence—between attacks. The *acute* cases were operated on as soon as possible after admission.

Of the 140 *cases,* 53 had had well-marked previous attacks; 125 operations were done "during the attack"; 15 operations were done "between the attacks"; 101 were acute or acute with abscess (10 of whom died, 1 from chloroform); 24 were subacute (all recovered); 15 were chronic (all recovered). The appendix was removed in 117 out of 140 cases operated on.

Of the 101 acute cases, 35 had had well-marked previous attacks. In 38 out of the 117 cases in which it was removed the appendix was gangrenous. Stercoliths are noted in 40 cases.

(*a*) *The acute cases with abscess* were 57 in number, 6 of whom died; 15 had had previous attacks. The appen-

dix was removed in 34 of these cases, was not seen in 23. Of these 23 all recovered but 1, who was admitted moribund. Two have had subsequent trouble demanding operation.

(*b*) *The acute cases without abscess* were 44 in number, 4 of whom died. In 10 cases acute general septic peritonitis was present at time of operation. In 13 cases there was recent perforation; 10 had had well-marked previous attacks. In all the cases the appendix was removed.

Of the 24 subacute cases, 10 had had well-marked previous attacks; 6 were complicated with peritoneal tubercle. All recovered.

Of the 15 chronic cases, all had had several previous attacks, one no less than thirteen, others six and four. In one case there was evidence of an old healed perforation. In 1 during convalescence there was thrombosis of the left femoral vein. All recovered.

Age and Sex.

Males, 83; Females, 57 { 29 children (under 16). 111 adults.

Only 6 over 40. Greatest age, two cases of 56.

Adults, 111 { 26 over 30. 85 between the age of 16 and 30.

Mortality.

Acute cases. . . .	9·9 per cent.
Acute and subacute cases .	8 „ „
Subacute cases . . .	*Nil.*
Chronic cases . .	*Nil.*
140 cases, mortality . .	7·1 per cent.

Of the 10 fatal cases, in 6 abscess was present; in all the appendix was gangrenous. All were acute cases. One died from chloroform, 2 were admitted moribund,

1 was complicated by abortion at the sixth month, 4 had general septic peritonitis present at the time of operation, 1 died on the nineteenth day from sloughing of cæcum (no peritonitis), 1 developed peritonitis on the fourth day from premature removal of gauze-packing.

In 7 of the cases the fatal was the first attack, in 1 the second, in 1 the third.

Complications.

Six cases, tubercular peritonitis; 1 case, perforated duodenal ulcer; 1 case, perforated gastric ulcer; 1 case, acute rheumatism; 1 case, pneumonia after operation; 1 case, thrombosis of femoral vein after operation; 1 case, pulmonary embolism after operation; 1 case, acute mania after operation; 1 case, intestinal obstruction from another cause. All recovered.

Deaths.

1. *Acute.*—Seventh day of disease. General septic peritonitis present at operation. Died fifth day after operation.

2. *Acute.*—Fourteenth day of disease. Acute general peritonitis. Died ninth day after operation.

3. *Acute.*—Twelfth day of disease. General septic peritonitis present at operation. Abortion six months' fœtus. Died sixth day after operation.

4. *Acute, with abscess.*—Fifteenth day of disease. Appendix not seen. General peritonitis. Died fifth day after operation.

5. *Acute, with abscess.*—Thirtieth day of disease. Sloughing extending into cæcum. Died of exhaustion. No peritonitis. Died nineteenth day after operation

6. *Acute, with abscess.*—Seventh day of disease. Perforation; died of general peritonitis. Died fourth day after operation.

7. *Acute, with abscess.*—Ninth day of disease. Gauze-

packing removed on the fourth day; peritonitis then rapidly followed. Died seventh day after operation.

8. *Acute, with abscess.*—Seventh day of disease. The abscess apparently had burst on his admission. He died with all the symptoms of chloroform-poisoning at time of operation. Died on the operating-table.

9. *Acute, with abscess.*—Seventh day of disease. General septic peritonitis, practically moribund on admission. Died twenty-four hours after operation.

10. *Acute.*—Eighth day of disease. General septic peritonitis; pus everywhere; practically moribund on admission. Died four hours after operation.

In all these cases the appendix was partly or entirely gangrenous. They were not seen at the hospital until the second, tenth, eleventh, fifth, third, eighth, sixth, fifth, seventh, sixth days after onset of first symptoms. In other words, only 2 were seen before the fifth day, 2 on that day, 2 on the sixth day, and 1 on the seventh, eighth, tenth, and eleventh days.

Details of Cases.

No. of Case.	Hospital No.	Sex.	Age.	No. of attack.	Nature of attack.	Remarks.	Result.
	1895						
1	1284 Tr.	M.	29	2	Acute	A. gangrenous *c.* stercolith, removed	R.
2	Med. Tr.	M.	—	Several	Acute	Gen. peritonitis present at time of operation	R.
3	Med. Tr.	M.	—	Several	Acute *c.* abscess	A. not removed	R.
4	Med. Tr.	M.	8	Several	Acute *c.* abscess	Stinking pus in peritoneal cavity; irrigation; pneumonia	R.
	1896						
5	374	M.	8	1	Acute *c.* abscess	Gangrenous A.; much lymph on intestines; A. removed	R.
	1897						
6	May Tr.	M.	25	3	*c.* Gen. peritonitis	A. removed; extensive general peritonitis found; death on fifth day (seventh day of disease)	D.
7	July Tr.	F.	56	1	Acute *c.* abscess	Perforation at base of A., which was removed; did well	R.
	1898						
8	649 Tr.	F.	23	1	Acute *c.* abscess	Chronic abscess, containing stercolith; A. could not be found	R.
9	917	M.	25	2	Recurrent	Adhesions; stricture of A.	R.
10	666 Tr.	M.	8	1	Catarrhal	Adhesions; catarrhal A.; tuberculous glands	R.
11	1101	M.	23	3	Acute *c.* abscess	A. not seen	R.
12	1618	F.	24	1	Acute *c.* abscess	Small abscess; inflamed A. removed; stercolith size of pea	R.
13	1953	M.	22	2	Acute *c.* abscess	Small quantity of foul pus, thickened, adherent; A. removed; stercolith	R.
	1899						
14	24	M.	7	1	Acute gangrenous	No pus; A. gangrenous *c.* stercolith	R.
15	574	M.	14	1	Acute *c.* abscess	A. gangrenous *c.* stercolith; removed	R.

No. of case.	Hospital No.	Sex.	Age.	No. of attack.	Nature of attack.	Remarks.	Result.
	1899						
16	904	M.	16	5	Recurrent	A very adherent; removed	R.
17	915	M.	18	1	Acute *c.* abscess	A. gangrenous *c.* concretion; removed	R.
18	1615	F.	28	Chronic	Catarrhal	A. removed	—
19	1789	F.	32	1	Acute *c.* abscess	Large abscess on inner side of cæcum; A. not seen; one of Gilbert Barling's "non-adherent abscesses" at back of pelvis (tenth day of disease)	D.
20	1928	M.	14	2	Subacute	I.P. two months previously for appendicitis; A. adherent to bladder; removed; enlarged glands; tubercles all over peritoneum	R.
21	1940	F.	15	1	Acute *c.* abscess	A. gangrenous; perforated; removed	R.
22	1951	F.	20	2	Acute *c.* abscess	A. not removed	R.
	1900						
23	208	F.	13	1	Acute *c.* abscess	Large abscess containing gas; A. not seen; patient returned May, 1901, with acute attack; A. then removed. See S.R. 604, 1901	R.
24	371	F.	17	2	Acute *c.* Gen. peritonitis	I.P. under Dr. Penrose three months previously with appendicitis; at operation, general septic peritonitis, perforated appendix; irrigation; did well	R.
25	517	M.	10	?	Acute *c.* abscess	Large abscess; A. not removed	R.
26	540	M.	40	Several previous	Acute *c.* abscess	Large abscess; A. not seen	R.
27	547	M.	18	2	Chronic	A. adherent *c.* stercolith	R.
28	664	F.	14	1	Acute	A. inflamed and few adhesions removed	R.
29	674	M.	29	1	Acute	A. thickened; adherent to external iliac vein; removed	R.
30	1011	M.	18	2	Subacute	I.P. under Mr. Dent one year previously; A. many adhesions and kinked	R.
31	1171	M.	23	1	Subacute	A. removed	—
32	1431	M.	25	Several	Recurrent	A. very adherent; thickened; removed	R.
33	1608	M.	38	1	Acute *c.* abscess	Large abscess, containing gas and pus; A. not seen; but on sixth day after operation sloughed A. and stercolith came away; death on nineteenth day, after two days' acute symptoms (thirtieth day of disease)	D.

34	1791	M.	22	3	Subacute	A. much thickened and adherent; complete fibrous stricture one inch from end	R.
35	1890	M.	23	6	Subacute	A. adherent and thickened; removed	R.
36	353 M.R.	F.	35	1	Acute *c.* abscess	A. not seen	R.
37	1797	M.	33	2	Subacute	A. removed; few adhesions	R.
	1901						
38	133	F.	19	1	Abscess	A. gangrenous at tip; stercolith; small pelvic abscess	R.
39	344	M.	21	1	Acute *c.* abscess	A. acutely inflamed; almost gangrenous, adherent; post-cæcal abscess	R.
40	517	M.	17	1	Acute rheumatic	A. swollen and œdematous; much clear serous fluid free in abdomen; apparently a rheumatic case; also acute rheumatism of joints at same time	R.
41	602	F.	14	2	Acute	A. removed. (Also an abscess had been opened in February, 1900; see S.R. 208)	R.
42	805	M.	26	1	Acute	Admitted on April 29 for subacute symptoms; on evening of May 1 symptoms of perforation occurred. *Operation.*—A. perforated, fæcal matter escaping	R.
43	808	M.	1	11	Acute *c.* abscess	Pus; A. extensively ulcerated and adherent; stricture at centre	R.
44	1014	M.	24	1	Subacute	Adherent A. removed	—
45	1026	F.	25	2	Acute	A. catarrhal; formal amputation; stercolith pushed into cæcum	R.
46	1034	F.	32	1	Acute *c.* abscess	Large post-cæcal abscess; gangrenous A. stercolith; A. not removed; sinus open some months; eventually healed	—
47	1116	M.	28	2	Acute *c.* abscess	A. removed	R.
48	1220	F.	43	1	Acute	A. enlarged, adherent, removed	R.
49	1326	M.	22	1	Acute *c.* abscess	Foul abscess; A. not removed, but stercolith found and taken away	R.
50	1898	M.	16	1	Acute	A. acutely inflamed; stercolith removed; no pus	—
51	2037	M.	56	1	Acute *c.* abscess	A. not seen	R.
52	2087	F.	22	13	Recurrent	A. removed; adherent, kinked; catarrhal inflammation	R.
	1902						
53	174	M	5	1	Acute gangrenous	Death on ninth day; no continuation notes; ? cause	D.
54	277	F.	19	1	Acute	A. removed; swollen, kinked, inflamed; part of mm. nearly gangrenous; no pus	R.
55	278	F.	15	1	Acute *c.* abscess	Small abscess; A. not seen	R.

No. of case.	Hospital No.	Sex.	Age.	No. of attack.	Nature of attack.	Remarks.	Result.
	1902						
56	443	F.	17	1	Subacute	A. adherent; removed; intestines, etc., studded with *tubercles*; worm-like structure (? nature) found free in peritoneal cavity	R.
57	454	M.	20	3	Subacute	A. removed; many adhesions; small pulmonary embolism after operation	R.
58	458	M.	48	1	Subacute	A. very adherent; removed	R.
59	710	F.	36	1	Subacute	A. kinked and enlarged; removed	R.
60	764	M.	17	2	Acute *c.* abscess	Foul abscess; A. not seen; patient discharged with sinus; stercolith then came away, and sinus healed	R.
61	829	M.	11	1	Acute *c.* abscess	Foul pus; A. not seen	R.
62	896 M.R.	M.	49	14	Acute *c.* abscess	Large retro-cæcal abscess; A. not seen	R.
63	1022	M.	19	1	Subacute	A. thickened; stricture; removed	R.
64	1102	M.	28	1	Subacute	A. thickened; adherent; removed	R.
65	1439	M.	11	1	Acute *c.* abscess	Small abscess; A. swollen, covered with lymph; stercolith; removed	R.
66	1685	M.	13	1	Acute *c.* abscess	Foul abscess; A. not seen	R.
67	1687	M.	29	6	Recurrent	A. very adherent, thickened and kinked; removed	R.
68	1690	F.	30	4	Recurrent	A. thickened and adherent; removed	R.
69	1856	M.	14	1	Acute *c.* abscess	Foul post-cæcal abscess; A. not seen; patient returned May, 1903, with another acute abscess	R.
70	1932	F.	39	1	Subacute	A. kinked and adherent; removed	R.
71	1932	F.	39	1	Subacute	A. very adherent; large concretion, and a little pus inside; scattered tubercles over peritoneum	R.
72	855 M.R.	M.	28	1	Acute	Notes lost	R.
	1903						
73	59	F.	30	1	Acute *c.* abscess	Perforated A.; some pus; death on the fourth day (seventh day of disease)	D.
74	72	F.	35	1	Acute	A. inflamed, kinked, small stercolith; *ovary* inflamed and enlarged	R.
75	235	M.	14	1	Acute *c.* abscess	Large foul abscess; A not seen	R.
76	323	M.	2	1	Subacute	A. found in hernial sac; slightly inflamed; removed	R.

77	332	F.	22	1	Acute	Plastic appendicitis; pus at apex of A.; no stercolith; A. removed	R.
78	685	F.	22	1	Subacute	A. catarrhal; stercolith; removed	R.
79	338	M.	18	1	Acute	A. kinked, thickened; stricture at end; removed	R.
80	601	F.	14	1	Acute *c.* gen. perit.	A. sloughed, removed; intense general peritonitis; pus everywhere; counter-incisions; general irrigation; died four hours after operation	D.
81	867	F.	24	2	Acute	A. kinked, inflamed, adherent	R.
82	983	F.	18	1	Acute *c.* gen. perit.	Sixth day of attack; simulated perforated gastric ulcer: intense general peritonitis; A. perforated, gangrenous; removed; *abortion* followed (six months' fœtus), and death on sixth day	D.
83	999	F.	18	1	Acute	A. gangrenous; stercolith; removed; no pus	R.
84	1063	M.	20	1	Plastic; quiescent	Many adhesions; A. removed in two pieces	R.
85	1149	M.	44	1	Acute *c.* abscess	Large abscess; A. not seen; stercolith; removed	R.
86	1156	F.	22	2	Subacute	Onset coincident *c.* menstruation; A. removed in quiescent stage; 5½ inches long; stercolith.	R.
87	1163	M.	16	1	Acute *c.* abscess	A. gangrenous; stercolith; removed; did well until removal of gauze plugs by H.S. on fourth day, then developed general peritonitis, and died in spite of second laparotomy (ninth day of disease)	D.
88	1219	F.	18	Chronic	Quiescent	A. removed; stercolith	R.
89	1228	M.	29	7	Acute *c.* abscess	Large retro-cæcal abscess; gangrenous A.	R.
90	1256	F.	24	4	Acute	A. densely adherent; cæcal end dealt with first	R.
91	1281	F.	17	1	Acute *c.* abscess	A. gangrenous and perforated: removed	R.
92	1486	M.	45	1	Acute	A. swollen and inflamed; no perforation or stercolith; formal amputation	R.
93	1758	M.	16	1	Acute	A. inflamed; adherent; removed	R.
94	1827	M.	13	1	Acute *c.* abscess	A. not seen; large retro-cæcal abscess	R.
95	1836	F.	30	3	Chronic; quiescent	A. removed; many adhesions	R.
96	1839	M.	29	2	Acute *c.* abscess	A. not seen; retro-cæcal abscess; pneumonia after operation	R.
97	1913	M.	32	1	Acute *c.* abscess	A. perforated and gangrenous; removed	R.
98	1921	F.	39	1	Acute *c.* abscess	A. gangrenous; stercolith; removed	R.
99	2020	F.	22	2	Acute	A. catarrhal; removed; two stercoliths; Dr. Dakin operated six weeks later, and found inflamed mass of omentum on left side	R.
100	2023	F.	21	1	Subacute	A. catarrhal; removed	R.
101	2032	F.	38	1	Subacute	A. catarrhal; removed	R.

No. of case.	Hospital No.	Sex.	Age.	No. of attack.	Nature of attack.	Remarks.	Result.
	1903						
102	1528 M.R.	M.	22	?	Acute	—	R.
	1904						
103	131	F.	24	1	Acute *c.* abscess	Foul abscess; A. not seen	R.
104	132	M.	23	1	Acute *c.* abscess	Large abscess; A. perforated; removed	R.
105	220	F.	22	3	Recurrent quiescent	A. thickened; adherent; removed; *thrombosis* of left femoral vein occurred	R.
106	224	F.	24	4	Acute *c.* abscess	Small abscess outside cæcum; A. not seen	R.
107	401	M.	28	2	Acute *c.* abscess	A. removed; distended with pus; minute perforation	R.
108	473	M.	21	1	Acute	A. gangrenous and perforated; removed stercolith	R.
109	492	M.	26	2	Recurrent quiescent	A. removed; distended with sero-pus; old perforation at base	R.
110	567	M.	22	1	Subacute	A. thickened; removed	R.
111	577	M.	25	1	Acute *c.* abscess	A. gangrenous, almost perforated; removed four stercoliths	R.
112	596	M.	22	Several	Subacute	A. adherent and thickened; removed; caseous glands in mesentery	R.
113	640	F.	32	1	Acute	A. acutely inflamed; removed	R.
114	653	M.	26	2	Acute *c.* ruptured abscess	Abscess apparently ruptured in surgery; walked into hospital; A. removed; died on table from *chloroform* (seventh day of disease)	D.
115	733	M.	21	1	Acute *c.* abscess	Large abscess, containing foul gas and thick pus under tension	R.
116	825	M.	8	7	Acute *c.* gen. perit.	Gangrenous perforated A. *c.* stercolith removed; foul purulent fluid free in all parts of peritoneal cavity	R.
117	979	F.	39	1	Acute	Case resembled perforated gastric ulcer; epigastrium opened first; acute inflamed; A. removed; temporary acute *mania* followed	R.
118	992	M.	25	1	Acute	A. distended with pus; removed	R.
119	1009	M.	22	4	Recurrent	A. removed	R.
120	1092	M.	28	Several	Acute hæmorrhagic	A. removed; hæmorrhage into walls and lumen of A.	R.
121	726	M.	23	?	Perforated gastric ulcer	A. 4 inches long; full of fæcal material; removed	R.

122	1071	M.	31	?	Perforated duodenal ulcer	A. 3½ inches long; slight adhesions; two stercoliths; removed	R.
123	1177	M.	19	1	Acute *c.* gen. perit.	Sent up on sixth day, hopeless; general peritonitis; almost moribund; huge collections of pus everywhere; large stercolith; A. gangrenous and perforated, not seen	D.
124	1248	F.	24	1	Acute *c.* abscess	A. perforated; adherent to iliac vessels; removed	R.
125	1612	M.	40	1	Acute *c.* abscess	Large abscess; gangrenous retro-cæcal A. removed; stercolith	R.
126	1790	F.	23	1	Acute *c.* abscess	A. not removed	R.
127	1860	F.	25	2	Acute *c.* abscess	Gangrenous A.; stercolith	R.
128	1866	M.	14	2	Acute	Large A.; tip adherent to internal abdominal ring	R.
129	1945	M.	13	1	Acute	Tubercular peritonitis; misplaced cæcum	R.
130	1946	M.	18	2	Acute	Intestines covered with lymph; general peritonitis	R.
131	2032	F.	20	3	Subacute	Stercolith	R.
132	2036	F.	24	1	Acute *c.* abscess	Deep pelvic and sub-colic abscess; stercolith; perforation	—
133	2041	M.	14	1	Acute *c.* abscess	Deep pelvic burrowing abscess; perforation stercolith; intestinal obstruction; internal strangulation of small intestine in hole of mesentery; median laparotomy	R.
134	2123	F.	9	1	Acute *c.* abscess	Perforation; stercolith bronchitis	R.
135	2121	F.	25	1	Acute	Subsequent pleurisy	R.
136	2129	F.	31	Several	Acute *c.* abscess	Perforation near cæcum	R.
137	2200	M.	20	2	Acute	General peritonitis; no adhesions; infected intestines; perforation	R.
138	2202	M.	33	Several	Chronic	A. very adherent to iliac vessels	R.
139	1875	F.	21	3	Subacute	Two stercoliths; A. distended; suppurating ovaries on both sides	R.
140	1905 20	M.	23	1	Acute	Gangrenous A.; three stercoliths; general peritonitis	R.

SUMMARY AND CONCLUSIONS

BY

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AND

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In accordance with a resolution of the Council, we have tried to arrange some of the statistics which are contained in these very valuable papers. But, of course, no attempt is made to deduce arguments from them, or to do more than put them in a convenient form for reference. It is evident that papers prepared with so great care and diligence demand minute study, and must be perused as they stand, and not in abstract.

(1) *Operations during a Period of Quiescence.*

These cases are described under diverse titles, as chronic, relapsing, or quiescent. Perhaps we may take it as a fair measure of all of them, that the patient, at the time of the operation, was in perfect or nearly perfect health, free from pain and fever, and there was no abscess. In every case, the surgeon could choose his time for the operation; there was no urgent need for immediate interference. But this view seems to need some slight correction. It is probable that, in a few of the cases, the patient was not wholly free from some slight discomfort, hardly to be called pain, in the region of the appendix. For the statistics of St. George's Hospital make no distinction between *chronic* and *sub-acute* cases; and the statistics of St. Thomas's Hospital include, among "operations in the quiescent stage," one case of operation

only seven days after the subsidence of pain, tenderness, and fever, and one case of operation on the ninth day after the fourth attack of the disease. It is to be noted that these two cases, which might perhaps be called *sub-acute* rather than *chronic*, were the only two cases which ended fatally, out of no less than 507 operations done during the quiescent stage.

What is the mortality of the operation done in the quiescent stage? In answer to that question we have the statistics of six hospitals as follows:—Charing Cross, 61 cases, 0 deaths; St. George's, 130 cases, 0 deaths; Guy's, 171 cases, 8 deaths; London, 364 cases, 4 deaths; Middlesex, 128 cases, 1 death; and St. Thomas's, 507 cases, 2 deaths. Altogether, 1361 cases, with 15 deaths—1·1 per cent.

What were the causes of death in these cases? Setting aside the London Hospital statistics, which do not help us here, we have 11 deaths in 997 cases. Of these 11 deaths, 1 was from insanity and suicide, 1 from ulceration of the colon, 1 from pulmonary embolism, and 2 from gangrene of lung, 1 from septicæmia, 1 from peritonitis, probably tubercular, and 4 from general peritonitis.

What were the nature and the incidence of complications not ending fatally? Setting aside those cases where complications occurred, not during convalescence but at some later period, and setting aside the London Hospital statistics, which do not help us here, we have 986 cases of recovery after operation during quiescence. The complications during convalescence, in these 986 cases, were as follows: Superficial abscess 1, sinus 1, fæcal fistula 1, obstruction by adhesions 1, swelling in left side of abdominal cavity 1, hæmoglobinuria 1, hæmaturia 1, hæmatemesis 1, abortion 1, tonsillitis 1, pulmonary embolism 1, pleurisy 5, pneumonia and broncho-pneumonia 5, and thrombosis 8. These 30 instances of complications may not represent so many as 30 separate cases, but we cannot be far wrong if we assume that the incidence of complications is at the rate of 3 per cent. of all operations done

during the quiescent period. The occurrence of pneumonia in 5 cases may have been due in part to the anæsthetic.

(2) *Operations for Abscess without General Peritonitis.*

Under this head are included all cases where pus was found in the neighbourhood of the appendix at the time of operation; and we make no distinction between those cases where the appendix was removed and those where it was left. The cases are all alike thus far, that there was no general peritonitis. Setting aside the statistics from St. Thomas's Hospital, which do not help us here, we have, from five hospitals, 863 cases. Of these, 83 died = 9·62 per cent. We may put the mortality, roughly, at 10 per cent. The figures are as follows: Charing Cross, 51 cases with 6 deaths; St. George's, 178 cases with 22 deaths; Guy's, 110 cases with 10 deaths; London, 431 cases with 35 deaths; and Middlesex, 93 cases with 10 deaths. We think that this death-rate of 10 per cent. is higher than that which is generally anticipated in such cases.

What were the causes of death in these cases? Setting aside the statistics of St. George's and the London Hospital, which do not help us here, and setting aside two of the Middlesex Hospital cases, where the causes of death are not stated, we are left with 24 cases in which we know the causes of death. Of these 24 cases, 1 died of diarrhœa, and 1 of general tuberculosis, 1 cerebral embolism, 1 pulmonary embolism, 1 (three months after operation) pylephlebitis, and 1 gangrene of mesentery, 2 pneumonia or broncho-pneumonia, *plus* extension of abdominal inflammation, and 1 subphrenic abscess, 1 hyperpyrexia, 1 septicæmia (death 10 hours after operation), and 10 general peritonitis. We may put it, roughly, that half the deaths after operation for localised abscess, and probably more than half, are due to further infection of the general peritoneal cavity.

What were the nature and the incidence of complications in these cases? Here the statistics of St. George's Hospital make no distinction between the complications which occurred in the fatal cases and those which occurred in the successful cases. The total number of the cases was 178, and of these 22 died, and the complications in all the 178 cases taken together were as follows: Out of 114 cases where the appendix was removed 14 died; and the complications were: fæcal fistula 6, empyema 2, thrombosis 2, and pleurisy 1. Out of 64 cases where the appendix was not removed 8 died; and the complications were: fæcal fistula 8, bronchitis 1, thrombosis 4, pleurisy 5, pneumonia 1, empyema 1, pulmonary embolus 2. It is to be noted that the mortality, 1 in 8, is exactly the same in the two sets of cases; but the complications were much more frequent in those cases where the appendix was not removed. Moreover, of these non-removal cases, 7 "required second operation." These figures, taken by themselves, would seem to show that the removal of the appendix, in cases of localised abscess, adds nothing to the danger of the operation, and tends to prevent complications during recovery. And the statistics from Guy's Hospital seem to point toward the same conclusion. Out of 42 cases of abscess, where the appendix was removed, only 2 died = 5 per cent. Out of 68 cases, where the appendix was not removed, 8 died = nearly 12 per cent. The latter set of cases also shows a much more frequent incidence of complications. On the other hand, the statistics from the Middlesex Hospital give 40 cases where the appendix was removed, with 5 deaths = 12·5 per cent., and 53 cases where it was not removed, with 5 deaths = 9·4 per cent. And Mr. Turner's statistics give a mortality of 15 per cent. in removal cases, against 4 per cent. in non-removal cases; but in more than one of the fatal cases the abscess had already burst into the general peritoneal cavity, and they must therefore be excluded.

Mr. Battle's statistics seem to point in the same direction

as those from St. George's and Guy's. Perhaps it is impossible, by statistics, either to prove or to disprove that opinion, which certainly was held by the majority of those who took part in the discussion of this subject—that the simple free incision of an appendix-abscess is not of itself a dangerous operation, and that there is danger if any prolonged search be made for the appendix at the time of operation. But the figures make it impossible to doubt that the appendix, left behind, does materially increase the risk of complications during or after convalescence.

(3) *Operations for Appendicitis with General Peritonitis.*

We cannot here either exclude or include all those cases of acute appendicitis where some peritonitis, but not "general peritonitis," was found at the time of operation. In the statistics from the London Hospital, the cases headed *Operation during attack: local peritonitis: no pus*— are kept separate from the cases headed *Operation in Cases with General Peritonitis with or without Abscess.* The mortality of the former cases was 15 per cent.; of the latter, 76 per cent. In the statistics from Guy's Hospital, the cases of *Operation of Appendicectomy complicated by presence of General Peritonitis* are kept separate from the cases of *Operation of Laparotomy for General Peritonitis without Appendicectomy.* The mortality of the former cases was 33 per cent.; of the latter, 78 per cent. But we can hardly be far wrong if we assume that some cases stood, as it were, on the borderland. Perhaps something might be gained if we exclude all those cases where it is expressly stated that the peritonitis was "local," and put together all those cases where it is expressly stated that there was "general peritonitis" at the time of operation. In some of them the appendix was removed at the operation; in others it was left. The figures are as follows: Charing Cross, 7 deaths in 8 cases; St. George's, 20 in 28; Guy's, 14 in 18; London, 127 in 176; Middlesex, 9

in 12. Total, 242 cases, with 177 deaths = 73 per cent. Of these 177 deaths, the vast majority were but the last stage of the condition in which the patients were admitted to hospital; and where the immediate mortality is so heavy it would be useless to make a list of the complications which occurred in the minority of the cases.

(4) *Statistics of Personal Experiences.*

We do not think it our duty to arrange, or to review, the records of individual practice which have been added by Mr. Lockwood, Mr. Battle, Mr. Turner, and Mr. Barling to the statistics of the hospitals. But we feel that it is our privilege here to express, as members of Council, our admiration of the generous and laborious work which these gentlemen, and all who have prepared this long and elaborate series of papers, have given to their subject. These papers are, we believe, destined to be among the classics of medical literature.

TORSION OF THE GREAT OMENTUM

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PART I.

Introduction.

In the first volume of the 'Annals of Surgery,' pp. 405–430, published in 1900, Mr. Lawford Knaggs, of Leeds, drew attention to the occurrence of volvulus or torsion of the intestine, in association with a hernia. His interest had been drawn to the subject by two cases which occurred in his private practice. The evolution of our knowledge of any disease is almost always the same. The severe and fatal forms first attract attention; later, other and milder clinical manifestations are recognised; and finally, given the necessary circumstances (such as the presence of a hernia, as in this example), the condition is recognised as occurring frequently. Mr. Lawford Knaggs' paper is really an introduction of the subject from the period of

rarity to the middle stage. As yet it has not advanced to the third or "frequent" stage. Though only writing on a kindred subject, torsion of the omentum and not of the bowel, we must express our opinion that volvulus is a very common consequence of the strangulation of a hernia. When the strangulation is at the hernial orifice, this becomes the fixed point about which the protruded loop of bowel twists. The twisting agents are, firstly, the meteorism in the strangulated coil, and secondly, the movements of flexion of the thigh. If the strangulation is not tight, the point on which the bowel has twisted may be carried within the abdomen by the force of the twist, when the intestinal obstruction becomes intra-abdominal. Should the hernia alone be operated on and the volvulus not be reduced spontaneously, as will occur very easily when the twisted coil is set free from the hernia, the patient will die of unrelieved obstruction. These cases are rare, and naturally will continue so, because in strangulation of the bowel the volvulus is secondary to the obstruction, making what was merely a slight obstruction absolute. As an example of this, reference is made to a case recorded in the 'Erasmus Wilson Lectures,' 1904, pp. 58–60 (Constable & Co.), in which a volvulus was noted. It is a matter of common surgical knowledge that in the strangulation of a hernia the situation of the obstruction is at the hernial orifice. If there is torsion of the bowel, it will probably be intra-hernial, and very rarely intra-abdominal. The volvulus is undone, after the reduction of the bowel, by the pressure of the gas within it. But if surgeons will take the trouble to look carefully, volvulus of the gut within the hernia will be found a common, if not a very common, occurrence, but it is of little or no practical import; just as the torsion which can be seen, if looked for, accompanying almost every internal form of intestinal obstruction requires no special treatment. Torsion of the bowel in strangulated, obstructed, or painful and irreducible herniæ is far from an uncommon event. What, then, with regard to the omentum,

which is often alone or sometimes accompanies the bowel? Before entering upon the subject-matter of this paper, reference must be made to another structure, besides bowel and omentum, which descends through the inguinal canal into the scrotum, namely, the testis. The twists found in structures which have passed through this region are formed slowly by the addition of frequent small increments. The acute volvulus of the bowel in strangulated herniæ is an exception. Normally, the testis is in the scrotum by the eighth or ninth month of intra-uterine life. Imperfectly descended testes accompany the majority of the recognised cases of torsion of the cord, and are in the situation to become twisted by the movements of the trunk and legs. A careful examination of imperfectly descended testes showed that small degrees of torsion, half a complete turn, were commonly found in cases which had suffered from sudden and sharp attacks of pain in the gland. Attacks of pain in an imperfectly descended testis were given a marked significance by this fact.*

Both bowel and imperfectly descended testes in the inguinal region are often twisted. We should expect, therefore, the omentum, which is exposed to similar conditions when in a hernia, to suffer likewise. With the object of introducing this and of collecting all that is known on the subject within reasonable compass and in an accessible form, the authors have written this paper. No cases beyond the end of 1904 have been included.

History.

The first example of torsion of the omentum, described by Oberst in the year 1882, was found in the sac of a painful irreducible inguinal hernia of old standing of a man, aged 35. From this date to the year 1893, when Demons described an example, no other case was recorded. In 1898, 3 new examples were added, one being added

* 'Brit. Med. Journ.,' June 4th, 1904. Paper on "The Value of the Imperfectly Descended Testis."

by each of the following—Bayer, Eiselberg, and Monod; 10 more were published in 1900, 6 more in 1901, and 5 more in 1902. In 1903 a large number of new instances were described, 16 in number. Up to the end of 1904 only 7 more have been added. Four new ones are recorded in this paper, thus bringing the total number up to 53. The fact that the first instance was described in 1882 cannot be urged in favour of a new pathological condition occurring then for the first time. Neither can the fact that no cases are described in 1890 and 16 in 1903 be attributed to such a condition as that represented by the phrase "good and bad harvests." The most intelligible explanation of the history of the disease is that, although it had occurred for centuries and perhaps thousands of years, the condition was first *recognised* by Oberst in 1882. For the next eleven years it continued to be overlooked, until in 1893 Demons re-discovered it. The seeds sown by these authors did not bear fruit until 1898. And though 1899 was barren in this regard, the next year bore full fruit—10 cases. Since then surgeons have begun to notice the condition, and by the end of 1904, 53 cases in all are known.

A further point is of interest, viz. that Lucas-Championnière (Observations 15 and 17)[1] was the first to report more than one case. Yet it cannot be held that he is the only man who up to 1901 had seen 2 cases. To him must be given the credit of being the first to *recognise* 2 of his own. Nordmann (Observations 28 and 29) in 1903 reported 2 cases; Rudolph in the same year reported 4 others, and to him belongs the credit of observing the largest number of cases and publishing the longest contribution to the subject (Observations 30–33). Mauclaire in 1904 published 2 cases (46 and 47), and the authors of this paper now add 4 more (50–53).

History teaches us also that at first only the most obvious cases were recognised. And although we know that intra-abdominal torsion of the omentum must be infrequent,

[1] *The numbers in brackets refer to the cases recorded at the end of the paper.*

torsion of it within the sac of a strangulated or painful and irreducible epiplocele is a fairly common event. Both the history of the reported cases and our own observations bear out this suggestion. To exemplify how these cases may have been mistaken or overlooked, we have included a section on the inferential diagnosis.

Clinical Classification.

The cases of torsion of the omentum naturally fall into three main clinical groups :

(I) *Abdominal.*—In this type there is no hernia present, and the torsion is purely abdominal. There are 6 examples: Baldwin (22), Syme (23), Stewart (43), Noble (45), Scudder (48), Corner and Pinches (51).

(II) *Hernial.*—The torsion occurs solely within the sac. There are 6 examples : Oberst (1), Heinlein (26), Rudolph II (31), Capette (38), Corner and Pinches II and III (52 and 53).

(III) *Hernial and Abdominal.*—The torsion is not limited to the hernial sac, but extends into the abdomen, or there may be a series of twists in each. The remaining 41 cases belong to this variety. Two subdivisions may be recognised.:

(1) When the hernial tumour is present, 30 cases.

(2) When the hernial sac is empty, the usual contents being reduced into the abdomen, 11 cases.

Cases of the second class—the hernial—are the ones most easily overlooked, and will be found, we believe, to be the most frequent.

Clinical Diagnoses made in the reported Cases.

(I) *Abdominal.*—Of these 6 cases, 5 were diagnosed as appendicitis probably accompanied by a local abscess, the other as a suppurating omental hydatid.

(II) *Hernial.*—Strangulated hernia was the diagnosis made in 4 cases, irreducible hernia in one, and incarcerated hernia in the other.

(III) *Hernial and abdominal.*—In this large group the following diagnoses were made:

Strangulated inguinal hernia	14 cases.
Incarcerated inguinal hernia	1 case.
Irreducible inguinal hernia	2 cases.
"Inguinal hernia" ? condition	2 „
Strangulated umbilical hernia	1 case.
Reduction of hernia *en masse*	2 cases.
Sarcoma of testicle	1 case.
Appendicitis	9 cases.
Intestinal obstruction	1 case.
Abdominal tumour	1 „
Intra-peritoneal abscess	1 „
Chronic peritonitis	1 „
Torsion of omentum	1 „

In 4 cases no diagnosis was made. The diagnosis of torsion of the omentum was made once, by Rudolph III, 32.

Clinical Etiology.

Sex.—Torsion of the omentum occurs more frequently in males than in females. Of the 53 recorded cases, 36 were males, 17 females. Males, 67·92 per cent.; females, 32·07 per cent.

During the years 1898-1902 inclusively the number of cases of inguinal hernia admitted to St. Thomas's Hospital was 1080; of these 951 were males, 129 females —about 8 to 1. In this same period there were 191 strangulated inguinal herniæ, of which 184 were males, 7 females—about 26 to 1. The presence of a strangulated inguinal hernia in a woman should raise the suspicion of the presence of a uterine appendage or twisted omentum in the sac.

Years.	No. of cases.
0—10	0
11—20	1
21—30	5

Years.	No. of cases.
31—40	14
41—50	20
51—60	9
61—70	0
71—80	1
Not recorded	3

The most frequent period for the torsion to occur is middle life, from 35 to 55 years of age; 39 cases, 78 per cent., occurred during that period. The youngest example recorded was 17 (Chavannaz, 10). The oldest was 79 (Weiner, 9).

Hernia.—With the exception of the 6 purely abdominal cases, a hernia was present in every instance—90 per cent. The various forms of herniæ present were as follows:

Right inguinal	33 cases	70·2 per cent.
Left inguinal	9 „	19·1 „
? side inguinal	3 „	6·38 „
Ventral	1 case	2·1 „
Umbilical	1 „	2·1 „

In no case was there a femoral hernia present.

These ruptures had been previously reducible with the exception of four—L. Championnière and Mauban (17), Weisinger (16), Heitz and Bender (11), Makins (50).

Table of the Time which the Herniæ had been present.

Hernia had been present for	1— 5 years	4 cases
„ „ „ „	6—10 „	5 „
„ „ „ „	11—15 „	8 „
„ „ „ „	16—20 „	4 „
„ „ „ „	21—25 „	1 „
„ „ „ „	26—30 „	5 „
Duration of hernia not stated		16 „
Congenital sac		3 „
Since childhood		1 „

The above table shows the number of years during

which the herniæ have been present up to the time of the acute attack. In 24 out of 31 cases, 77·7 per cent., where the age of the hernia is definitely stated, it had been present over 10 years. The shortest time for which the hernia had been noticed is 2 years, Nordmann I (28). In 17 cases it is stated that a truss had been worn.

The size of the hernia seems variable and quite an indifferent matter. In one instance it is said to have reached to the knee—Mauclaire I (46). It was large in the majority of cases.

Torsion should be looked for carefully in all epiploceles of old standing, especially when painful, irreducible, and subject to attacks of pain or strangulated.

It is very rare for bowel to be present, because torsion of the omentum is usually a very slow development; and if bowel is present, some acute crisis will occur from its torsion before the omental twists have progressed sufficiently to cause trouble.

Clinical Symptoms.

Onset.—In only a few of these cases is any reference made to the onset of symptoms. In 21 cases (37·7 per cent.) the onset was sudden, and in 7 (13·2 per cent.) gradual. In the purely abdominal cases sudden onset was noted four times, gradual once. Below are given some examples of the method of onset.

Monod (5): Immediately after an effort the patient felt a sharp pain in the right iliac fossa, the pain being likened to a cut with a knife. *Noble* (45): Sudden onset with excruciating pain in the abdomen, attended by nausea and vomiting. *Malherbe* (41): Four hours after a meal sudden violent pain in the right iliac fossa, most intense at MacBurney's point. *Corner and Pinches I* (51): The patient had been ill for ten days with pain in the right side. *Broca and Chavannaz* (10): Suddenly one morning an irreducible tumour appeared in the right inguinal region without any signs of stra[illegible]

Scudder (48): Sudden onset, with abdominal pain and vomiting.

Pain.—Pain is the most constant of all the symptoms, and in nearly all the cases where any details are given it is said to have been present, and was one of the first symptoms to appear. In investigating this, it will be best to divide the cases into the three types.

(1) *Abdominal.*—Pain in the abdomen was present in all these cases; in four it was situated in the right iliac fossa, and in one it is said to have been diffuse, but most intense at MacBurney's point.

(2) *Hernial.*—In four cases pain is noted as being present in the hernia.

(3) *Abdominal and hernial.*—The following table shows the distribution of the pain felt:

Table showing the Site to which the Pain was referred in these Cases.

Pain referred to hernia alone . . .	14 cases.
„ „ abdomen alone . . .	17 „
„ „ both	7 „
Pain not mentioned in any particular region .	3 „

In 11 cases the pain is especially referred to the right iliac fossa, and in 1 is said to be most intense at MacBurney's point. As a rule, it is very acute and persistent, but in 1 case (Morrison, 39) there were exacerbations of pain from time to time. In most cases it was sufficiently severe to cause the patient to take to his bed.

Vomiting does not appear to be a very constant symptom. In 19 cases (35·8 per cent.) it is said to have been present; in 14 (26·4 per cent.) the absence of vomiting is noted; and in 20 (37·7 per cent.) cases no mention of it is made. Where it has been noted it is usually frequent and violent. In one case (Lejars) it is stated that the vomited matter was greenish. Nausea without vomiting was present in 9·4 per cent. of cases.

Condition of the bowels.—Diarrhœa in 2 cases; bowels open in 16 cases; bowels inactive in 11 cases; flatus alone passed in 5 cases.

From the above table, it will be seen that the most common condition is that the bowels were open, but this term is a vague one. In the majority of cases it is not stated on what day of the disease they were opened or how often. Absolute "obstruction" is noted 5 times and the passage of flatus alone in 5 instances. On the whole, the condition of the bowels seems very variable, but the most common condition is one of inactivity.

Pulse.—As a rule the pulse is accelerated, varying from 80 up to 120 per minute.

Temperature.—The temperature is usually slightly raised; in 21 cases (39·6 per cent.) it is noted that the temperature was above normal; in only 3 is it said to have been unchanged. The highest recorded temperature is 101·8° F.; the usual is between 100·4° F. and 101·4° F. In no case is it said to have been subnormal.

General appearance.—In only a few cases is any reference made to the facial expression; it appears to vary with the duration and acuteness of the symptoms. In 3 cases it is noted as being quite normal, in 1 to have resembled the facies of intestinal obstruction, in another the facies of peritonitis.

Clinical Signs.

Tumour.—In all but 3 of these patients a tumour could be felt on examination, but its situation varied.

(1) *Abdominal.*—In 4 of these a mass could be felt in the right iliac fossa; in the other 2 there was tenderness and rigidity in this region, but no definite tumour could be detected on palpation.

(2) *Hernial.*—All of these patients had a hard, painful, irreducible swelling in the inguinal region.

(3) *Hernial and abdominal.*—The following table shows the tumour present in this series:

Situation of Tumour.

A hernial tumour alone present	.	12 cases.
An abdominal tumour alone present		7 „
Tumours present in both situations	.	19 „
Neither tumour detected	. .	3 „

From this table it will be seen that it is most common for tumours to be present both in the hernia and in the abdomen; and it is very frequently noted that the hernial and abdominal tumours appear to be continuous.

The tumour is as a rule hard, tender on palpation, and dull to light rather than to heavy percussion, and is often said to have had an irregular surface. Dulness was noted nineteen times in this series. General distension of the abdomen is not a common sign; it was only noted 7 times—13·2 per cent. The following extracts from cases are illustrative of descriptions of the tumour:

Wiener (9).—"About midway between the anterior superior spine of the ilium on the right side and the free border of the ribs, extending from the axillary to the mammary line, a rounded tumour the size of an orange was readily felt."

Rudolph III (32).—"There was a small swelling in the right inguinal region, also an intra-abdominal swelling on the right side extending two fingers' breadth above the umbilicus, and stretching from the right anterior superior spine of the ilium to the middle line. The two swellings appeared to be continuous with each other."

Wiart and Renon (21).—"The abdomen was enlarged and a little painful over a small area in the right iliac fossa. From a level with the anterior superior spine of the ilium, and extending downwards and inwards towards the hernia, is a tumour the size of a turkey's egg. It is hard and lobulated."

Championnière and Mauban (17).—"The hernia was hard and painful, and the hernial tumour seemed to be continued into the abdomen in the direction of the inguinal canal."

Treatment Adopted.

In all these patients the severity of the attack was recognised, and in all but one operation was performed. In this case (Tremolière, 34) the general condition was too bad to warrant operation. The various incisions made were as follows:

(1) *Abdominal.*—In 5 instances .an incision was made over the right iliac fossa, in the other a middle line incision.

(2) *Hernial.*—In all 6 patients the incision was made over the hernia.

(3) *Hernial and abdominal.*—This table will show the different incisions made:

Hernial	14 cases.
Abdominal	15 „
Hernial and later prolonged upwards .	5 „
Doubtful	5 „

Two operations were performed on Demon's (2) patient, the first on the hernia, then later a laparotomy.

Although its true nature was not always recognised, yet in nearly all cases the tumour was removed.

Prognosis.

All of the cases recovered except 8. The fatal ones were recorded by Eiselberg (4), Monod (5), Heitz and Bender (11), Lejars (14), Moresco (25), Tremolière (34), Zeller (37), and Audier (44). Of these, Tremolière's was too bad for anything to be done; Audier's was operated on as a desperate measure; Monod's and Heitz and Bender's died of pneumonia; and Lejars' of delirium tremens. Zeller's record is so short that nothing can be said. Eiselberg's case died 38 hours after operation, presumably of peritonitis. These results show that when submitted early to operation the prognosis is good.

The mortality for these 53 cases, in which the majority of observers recognised the pathological condition of tor-

sion of the omentum for the first time, is 13·2 per cent. But if the cases in which the fatal result was not concerned with the surgeon are excluded, the percentage mortality is 4·1. There are very few conditions which can show so small a mortality in the first chapter of their history.

Inferential Diagnosis.

All diagnosis is inferential; the conclusion is arrived at by means of inferences drawn from observations. These inferences can only be deduced correctly when born of sound knowledge. The previous part of this paper[1] contains practically all that is known of torsion of the omentum. It was decided, therefore, to include a section on the inferential diagnosis, in which we will draw inferences from the cases of others and from our own. As a new disease was not discovered when the first case of omental torsion was reported, but merely an old one recognised, this section will be interesting and instructive in affording examples in which the twists were, or may have been, overlooked. Further, some authors have included under the head of omental torsion cases which, after mature consideration and study of the records of all available examples, we do not feel justified in accepting on the evidence adduced. The first three and the fifth instances to be quoted are examples of this.

To begin with, we will briefly summarise the conditions which should lead to a suspicion of the existence of torsion of the omentum. The subject is most commonly a man of middle age, with an inguinal hernia of some years' standing, which has become troublesome and given rise to certain symptoms which can be summed up as those of subacute intestinal obstruction. Vomiting may be absent, and both fæces and flatus may be passed. On examination, the hernia will most probably appear to be painful and irreducible. Usually a tumour can be felt both in

[1] See also 'American Journal of Medical Science,' 1905, and 'St. Thomas's Hospital Reports,' 1904.

the scrotum and on the right side of the abdomen. Should such a condition be found in the inguinal hernia of an adult woman, it will either contain an ovary and Fallopian tube or twisted omentum.[1] Occasionally only the abdominal tumour is present, the sac appearing to be empty. Such is the clinical picture in brief which should raise suspicions of omental torsion.

It has been stated that to Lucas-Championnière belongs the honour of being the first man to recognise two cases of torsion of the omentum. These were published separately in 1900 and 1901. In 1898, previous to having recognised the condition, he published a case, which is briefly reproduced.

Lucas-Championnière, 'Bull. Soc. Chir.,' 1898, pp. 195–197:

Male, aged 53. Right inguinal hernia since infancy; had worn a truss. For some time past the hernia had become more painful. On examination there was a large irreducible inguinal hernia, tender on pressure; the abdomen was hard and tender. The bowels were open.

Operation.—The inguinal canal was occupied by an enormous cord continuous with a tumour in the scrotum, which was very adherent to the sac. In the centre was a little black liquid, apparently altered blood; the mass was prolonged upwards into the abdomen, where it joined an enormous tumour which occupied all the anterior part of the abdominal cavity and descended into the pelvis. At the upper part it was in juxtaposition with the transverse colon and was adherent to the anterior abdominal wall. This tumour was thick, hard, lardaceous, and consisted of the entire omentum.

This case agrees with everything which has been said of omental torsion, with the exception that the torsion must have been very slow and subacute to account for the great size of the omentum and the many adhesions which it had contracted.

[1] "A Case of Cellulitis of the Round Ligament," Clin. S[illegible] 1903.

Potherat in 1900, previous to his recognition of the pathological *rôle* of torsion of the omentum, published a case which he says was like that recorded by Championnière in 1898, which we have just reproduced. The following is a brief summary:

Potherat, 'Bull. Soc. Chir.,' 1900, pp. 525–531:

Male, aged 16. Right inguinal hernia. When first seen there was a hard rounded tumour obscuring the testicle; above it was a hard, irregular, thickened cord entering the inguinal canal. The patient had had slight symptoms for some months, was unable to walk, and had suffered from loss of appetite and flesh.

Diagnosis.—Neoplasm of testicle and epididymis.

Operation.—Castration was performed, and the cord resected high up in the canal. On examining the specimen it was found to consist of a mass of thickened, heaped-up omentum which surrounded and buried the testicle; it was dark in colour and resembled that described by Championnière (1898). The condition was thought to be due to inflammatory lesions affecting an old omental hernia.

Like Championnière's, this seems to have been a case of chronic torsion of the omentum, but, unlike his, the trouble appeared to be confined to the scrotal region.

Verdelet and Rocher recorded a case in 1900 in which a hernia became irreducible, and at the operation there was found a condition in which torsion could very easily have occurred.

Verdelet and Rocher, 'Journ. de Med. de Bordeaux,' September, 1900, pp. 559–60; 'Gaz. Hebdom. des Scien. Med.,' June, 1900, pp. 291–293:

Male, aged 56. Reducible right inguinal hernia for forty years. The patient wore a truss. The hernia suddenly became irreducible without any symptoms being present, it remained irreducible for six months, and was [illegible] replaced. Three weeks later, after a fit of coughing, [illegible] became irreducible. After two days, during which [illegible]atedly tried, the hernia was partially reduced.

In the right side of the scrotum there was a soft lobulated mass, with a pedicle running up above Poupart's ligament. The tumour was not painful; it was dull on percussion, except in the upper part, where it was a little resonant. All had been reduced, except a rounded mass the size of an orange, which was irregular, hard, and lobulated, but not painful on pressure. From its upper part there was a pedicle going in the direction of the inguinal canal. It was movable in the scrotum, and situated behind the hernial mass.

Operation.—In the sac a large, fat, vascular mass was found, behind which was an irregular, lobulated, fatty tumour, the size of a fœtal head. There was a pedicle of the same nature with large vessels in it, running towards the inguinal canal. There was also a right hydrocele present. The tumour was formed by the "rolling up" of the omentum on itself.

They describe the tumour as consisting of "rolled up" omentum. That "rolling up" is not the same as "twisting" has not been recognised by all observers, some of whom have taken this case to be one of proved torsion. Had other symptoms than mere painless irreducibility of the hernia been present, it would have been probable that torsion of the stalk above this "rolled up" omentum had taken place. As it is, there is no evidence that torsion did occur, though under such conditions it is hard to realise that it did not.

In 1902 Spellissey, in a paper on the diagnosis of appendicitis, reported a most suggestive case. Though a hernial sac was present, all the signs and symptoms were intra-abdominal.

Spellissey, 'Annals of Surgery,' 1902, i, p. 769:

Female, aged 40. Right inguinal hernia for eight years. History of one day's illness, accompanied by absolute inactivity of bowels and vomiting.

Diagnosis.—Appendicitis.

Operation.—In the abdomen was found a large mass of omentum, weighing about two pounds, which was in a

gangrenous condition. No abscess was observed, and no torsion is mentioned. The patient died on the fourth day.

This case leads up to another American one, in which the symptoms were abdominal and no hernial sac was present. Although the account is not clear, some authors have included Eitel's case as one of omental torsion, *e. g.* Scudder (48), Noble (45).

Eitel, 'New York Med. Rec.,' May, 1899 (reference not confirmed):

Male, aged 44. Patient noticed the abdomen was of large size. It was tapped, and three gallons of fluid drawn off. Twenty-four days later it had again become distended, and 2½ gallons were drawn off. After tapping a tumour was felt in the hypogastrium, 7 inches long, 4 broad, and 4 thick, soft, movable, and neither tender nor painful on examination.

Operation.—Three days later. An omental tumour was found, rolled inwards and upwards, so producing constriction and partial obstruction to the return circulation in it. Its veins were enormously distended. It was unravelled without difficulty, and the omentum spread out, whereupon the dilation of the veins disappeared, and it became normal in appearance. The patient had been in the habit of carrying a heavy box, which pressed upwards and inwards just below the umbilicus. No torsion above the tumour formed by the rolling up of the omentum was present.

Again there is the word "rolled" which renders the diagnosis of torsion difficult. This, coupled with the fact that other clinical data, such as the condition of the liver, are wanting, plus the knowledge that only one case of known omental torsion has been accompanied by such ascites, makes it unwarrantable for us to include this case. Compare this case with that of Hochenegg (8) which showed twisting of the pedicle of a "rolled-up" omentum and ascites.

ion to four more cases, one of Mr.
f our own. By these examples

we wish to indicate the kind of case in which it seems probable that some twisting of the irreducible omentum is the cause of the pain and other symptoms. Any surgeon can realise how very easy it is to overlook a turn buried deeply at the root of a large omental hernia; and, owing to the absence of landmarks on the omentum itself, it is impossible to recognise even the straight position, much less the slightly twisted one. The dilated condition of the veins makes it obvious that there is some pressure obstructing the blood-flow in them. If this is not caused by the inguinal rings it must be brought about by some change in the omentum itself. Under these circumstances torsion seems to be the most plausible explanation. Moreover, we have observed it in two cases, Observations 52 and 53, which were very like the ones to be quoted.

Stephen Paget :

In the College of Surgeons' Museum there is a specimen (2621A) of omentum removed from a case of supposed strangulated hernia. The following account is taken from the notes furnished with the specimen.

Male, aged 63. Right inguinal hernia for 38 years. A truss had been worn. Recently the hernia came down and he was unable to replace it. Half of the tumour was returned by taxis, but a portion remained, and gave a "crumbling" sensation to the hand, suggestive of blood-clot. The hernia increased in size. The bowels were opened: temperature normal; no vomiting, no pain. Operation. There was a thick sac containing omentum. The constriction shown in the specimen had no connection with the external ring. There was a band of omentum arising below the constriction, adherent to the bottom of the sac. The lower half of the tumour was deep red in colour.

Corner and Pinches, 1 :

W. A., male, aged 25, was admitted to St. Thomas's Hospital for a painful irreducible inguinal hernia on the left side. At the operation it was noticed that the sac contained only omentum with some fluid; also the omentum, though swollen,

was not strangulated, and could be reduced by steady pressure without needing a "herniotomy." There was no evidence that the fluid came from the abdomen; indeed, the swollen omentum would seem to have acted as a cork, and prevented it. If so, the fluid formation must have been local—that is to say, in the hernial sac. Considering the extreme ease with which such a condition is overlooked during the manipulations of the operation, the case may very well have been one of torsion of the omentum.

Corner and Pinches, 2:

W. P—, male, aged 46, was admitted to St. Thomas's Hospital with a painful irreducible right inguinal hernia. There was no vomiting. The rupture was very large. At the operation it was found to consist of an enormous mass of omentum, with distended veins. More omentum was withdrawn from the abdomen, the stalk ligatured, and the mass removed. No torsion was noticed as it was not looked for, but the stalk was certainly not strangulated.

Corner and Pinches, 3:

F. G—, male, aged 40, was operated on for a huge painful and irreducible left inguinal hernia. It was found to consist of an enormous quantity of congested omentum, not strangulated, but adherent to the bottom of the sac. No torsion was noticed. But what, then, was the explanation of his having had painful attacks previously?

By means of these examples we hope to have shown that, from the knowledge previously gained, it has been possible to draw such inferences as are in favour or not in favour of torsion of the omentum being the key to the understanding of some clinical and pathological problems. Moreover, we hope that in such illustrations it has been possible to indicate the main directions in which our knowledge of the subject will be increased in the future.

Differential Diagnosis.

When the subject under consideration happens to be one the true clinical and pathological importance of which

there is reason to believe is not yet understood, more stress must be laid upon its diagnosis or recognition than upon the differential diagnosis. The concrete value of what we prefer to call the inferential diagnosis is to be placed far above the more abstract refinements of the differential diagnosis. Still, in view of the mistakes which have been made in the 53 cases collected in this paper, we have thought it desirable that something should be said.

(1) The instances of abdominal torsion, unaccompanied by the presence of any hernia, form about 10 to 12 per cent. of all cases of omental torsion. If the infrequence of the lesion is taken into account, as well as the fact that within the abdomen torsion is most unlikely to be overlooked, the subject of the differential diagnosis need not be considered in any detail. Five out of the six cases were diagnosed as appendicitis, with or without abscess. The sixth, occurring in Australasia, where obscure lesions are always guessed to be hydatids, was diagnosed accordingly. A tumour, or dulness with resistance, will always be present, and if found before the attack has commenced, will raise other suspicions than those of appendicitis. The condition is one which is to be recognised and treated properly during the course of operation rather than one to be diagnosed beforehand.

(2) When the torsion is in a hernial sac, 10 to 12 per cent., it will be found amongst the painful and irreducible, the incarcerated and strangulated herniæ. Here, as in the abdominal class, the surgeon should pay more attention to the recognition of the condition at operation than to its differentiation beforehand. A few words may be said upon the latter. In only two instances was bowel present, Mauclaire II (47) and Makins (50). The first point to be proved will be that omentum alone is in the sac. In no case was the omentum strangulated; therefore it should have an impulse on coughing and, perhaps, bowel might be coughed down. The impulse given on coughing to an omental hernia is far less definite than

that communicated to one containing bowel. It is especially difficult to make satisfactory observations on large epiploceles which have an ample heaving impulse communicated to them by the muscles of the abdominal wall. In consequence, it is not easy to make sure of a large non-strangulated omental hernia. But if these two points are ascertained, then the accompanying symptoms are probably produced by torsion or epiploitis, of which two the latter is caused by the former. Beyond this the diagnosis is only a conjecture, based on inference and such elementary differentiation as the above.

(3) In the third division, which includes 75 to 80 per cent. of the cases, both the abdominal conditions and a hernial sac are present. The conjunction of these two enables us to take the differentiation further than in the other classes—indeed, sufficiently far for a diagnosis rather than a conjecture to be made. Referring to what has been said above in considering the examples of abdominal and hernial torsion, the signs of both will be found in the condition now under discussion. Typically, there will be present a tumour in the abdomen and an inguinal hernia, on the same side and of considerable duration (ten years), which will present signs of strangulation or irreducibility with pain. More than this, the tumour in the abdomen and the tumour in the hernia will be continuous. General symptoms of subacute peritonitis or intestinal obstruction will accompany these local signs. Such things as a local tubercular peritonitis, an intra-peritoneal abscess, pelvic cellulitis, or a sub-peritoneal lipoma might give rise to a somewhat similar state of affairs. But it is of interest to note that the majority of the diagnoses made concerned the hernial condition—*e. g.* strangulated epiplocele—and when the abdominal signs predominated appendicitis was suspected. If the above signs and symptoms are found and special attention given to the diagnoses which learned and competent observers have already made in examples of omental torsion, it should be possible to arrive with reasonable certainty at a diagnosis. There is some prac-

tical importance in this because it may be necessary to open the abdomen as well as the hernial sac. Demons' case (2) died, most probably because this was not done at first. Rectal examinations have never revealed anything in these cases, and therefore will give very useful help in differentiation, *e. g.* appendix abscess or pelvic cellulitis.

Strangulated inguinal herniæ are very uncommon in women, yet a relatively high percentage of cases of omental torsion, connected with a hernia, occurred in them. In middle-aged women one must always remember that the Fallopian tubes and ovaries, usually in an abnormal condition, are found in these herniæ. A rectal or vaginal examination enables one to make the distinction at once.

As omental torsion has never been found in a femoral hernia, the differentiation of it from a partial enterocele (Richter's hernia) need not be discussed.

In a certain number of cases the hernia has been reduced without relief of the symptoms, although the sac is emptied. In these cases the obvious diagnosis of reduction without relief of the strangulation, or, as it is called, reduction *en masse*, is made. And nothing more can be inferred. Further, in a few more instances the attack or crisis came on whilst the hernial sac was empty, as in Peck's (7) and Noble's (45) cases. In these there is no guide to the fact that the case is not one of appendicitis. But as there had been a hernia of considerable duration present the suspicion of omental torsion might arise.

Pathogeny.

There are two ways of discussing factors which cause a pathological lesion. All the various agents can be classified and considered in their minutiæ, so as to make the account as full and perfect as possible; or details can be sacrificed and only the main factors taken into account, the object being to produce as brief an account

as is consistent with truth and accuracy. The latter method is the better for our purpose. No action in the world has a simple causation. There is always an innumerable host of subsidiary and perhaps insignificant considerations to be taken into every account which pretends to be full and complete.

The pathogeny of torsion of the great omentum will be considered when it occurs (1) in connection with a hernia, (2) when not.

(1) *When the Torsion is formed in connection with a Hernial Sac.*

Almost all the cases of omental torsion have occurred when the hernia has been of old standing. During the long interval between the formation of the hernia and the twisting of the omentum certain changes take place in the latter which are of great importance, because not until those changes have been brought about can the torsion take place. These changes may be regarded as having a threefold disposition: firstly, in the omentum at the neck of the sac (*a*); secondly, the omentum in the sac (*b*); and thirdly, the formation of adhesions (*c*). The first two will be considered together.

(*a* and *b*) When a quantity of omentum is expressed from the abdomen into a hernial sac, the part which is in the scrotum is exposed to comparatively little pressure, whilst that which lies in the inguinal canal is compressed into a roll or cord. The result is that the scrotal tumour becomes rounded and the inguinal stalk-like, connecting the former with the abdomen. Owing to the pressure in the inguinal canal, the part within it gets thinner and smaller; whilst the part without, owing to this pressure acting mostly on the veins and lymphatics, becomes œdematous and thicker, later becoming fibrous and often having fat deposited in it. The final condition may be likened to a cherry on its stalk. The cherry or rounded mass may hang symmetrically or asymmetrically upon its stalk; in

the latter case the cherry will tend to rotate upon its stalk with every change of position. Baldwin[1] has said: "All that seems primarily essential [for torsion] is the existence of a pedunculated tumour with no mechanical obstacles to its rotation. If the pedicle is situated so as to furnish a perpendicular axis for rotation, torsion (unipolar) is more apt to occur. If a perpendicular axis is afforded by the pedicle proper, and also by an adhesion at the bottom, torsion (bipolar) will be still more apt to take place."

(*c*) Common operative experience tells us that though a hernia is thought to be reducible, at the operation it is not uncommon to find the omentum adherent to the sac and irreducible. These adhesions arise from a subacute peritonitis, and are found chiefly at the lower part of the sac. It is not quite clear why this should be the situation so frequently selected. The lower part of the sac is subjected to a smaller amount of injury than is the part on which a truss presses. On the other hand, the lowest piece of the omentum is the portion replaced last when reducing the hernia, and knowing how frequently trusses are quite unconsciously worn on pieces of omentum, it would appear that some such traumatism to the piece of the omentum which hangs lowest is responsible for the adhesion at the bottom of the sac.

The following case has been recorded by Baldwin as illustrating the physical conditions necessary for the omentum to twist. No such torsion was found at the operation.

Baldwin's case:

Male, aged 29. Right inguinal hernia of one year's duration. An abscess formed in the right inguinal region, which was incised, pus escaping. Later a second abscess formed, which was treated in the same way, but an irreducible mass persisted in the inguinal canal, extending into the scrotum.

Diagnosis.—Omental hernia.

[1] Baldwin, 'Annals of Surgery,' 1902. vol. ii

Operation.—A small mass of omentum was found adherent at the bottom of the sac. On pulling down the omentum a mass was seen, which was 5 to 6 inches long and 1 inch in diameter. It was connected to the rest of the omentum by quite a narrow pedicle. When the tumour was examined it was found that after separating a few adhesions the mass could be spread out into quite a normal-looking piece of omentum. Here conditions most favourable for torsion were present, but it had not taken place (that is to say, it was not present at the operation).

The origin of the abscesses is of considerable clinical interest. Baldwin offers no suggestion. Can they have been due to torsion of the omentum, which had untwisted itself by the time of the last operation ? One would say no, for two reasons at least. Firstly, suppuration, though an occasional concomitant of omental torsion, is very uncommon ; secondly, although there had been two separate abscesses the omentum when spread out was a "normal-looking piece." As the omentum seems not to have been the offender, and the hernia was on the right side, it seems probable that the appendix was responsible. It is said in a paper[1] on the relations of the appendix and Meckel's diverticulum to hernial sacs, when the appendix is present along with other structures : "The most common of these is naturally the cæcum, next comes the omentum, and then the small intestine. These tend rather to protect the appendix and to minimise the chance of its making its presence felt." In an unrecorded case, whilst cutting away some omentum which was in a hernial sac, the appendix was found to have been divided. It was wrapped up in the omentum and quite indistinguishable until cut across.

No case of omental torsion in connection with a femoral hernia has been described. The difficulty is that the femoral ring and sac are very much smaller, admitting of less stretching than the inguinal ; consequently a very much smaller mass of omentum enters the hernia, and

[1] Corner and Spurrier, 'St. Thomas's Hospital Reports,' 1902, p. 376.

perhaps descends and reascends less often. In an umbilical hernia this objection is not present, and yet no case is recorded previous to this communication; the omentum soon gets too adherent for any rotation to take place. In recording Mr. Makins' case, we publish the first example of omental torsion in the sac of an umbilical hernia.

There is one example of a torsion in connection with a ventral hernia (18), but in this instance the hernia was in the lower portion of the abdomen, and gravity and the dragging movement would cause the formation of a pedicle and so predispose to torsion. This process of the formation of a pedunculated tumour appears to be as a rule a long one. In 29 cases the time which the hernia had been present is definitely stated, and in 22 of these it had been present over 10 years. The shortest time is given in Baldwin's case, where the hernia had only been present 1 year.

The changes in the omentum which result from its frequently descending into a hernia and being reduced appear in the formation of a rounded hard mass, which is symmetrically or asymmetrically placed upon the axis of its pedicle; or if there is an adhesion to the bottom of the sac, its pedicles. Its rotation may be unipolar or bipolar.

These are but predisposing passive factors, which cannot do anything unless assisted by some active force, such as is exemplified in the following classes.

(*a*) In the asymmetrical position, aided by gravity, the tumour will turn through a certain arc. But it seems inconceivable that more than half a turn could be accounted for, whilst there have been as many as eight turns!

(*b*) The tumour is no longer a soft mass of omentum, but is hard and irregular. In consequence, it does not accommodate itself to the various diameters of the inguinal canal like a fluid, but by rotation, just as the fœtal head does in descending through the pelvis at birth. Thus it is conceivable that by frequent descents of the hernia and its reduction by manipulation a number of turns can be

given to the tumour. In twelve out of twenty-three cases in which the number of twists is stated there were three or more present.

(*c*) These twists have been produced by a gradual process. Asymmetry of the tumour with regard to the insertion of its pedicle will aid. At first this rotation will not be sufficient to affect the circulation greatly ; the vessels will adjust themselves to the new conditions, and when next the tumour descends and is replaced, a little more rotation will be added.

Payr ('Verhandlungen der Deutschen Gesellschaft für Chirurgie,' 1902, p. 585) performed some experiments on animals to determine the cause of the torsion of abdominal viscera. In his opinion the circulation both in the pedicle and in the tumour play an important part. The veins are longer than the arteries ; when there is a light torsion of the pedicle the blood stagnates in their interior, these vessels curve themselves inward in a spiral manner around the resistant part of the pedicle, and the pressure within them begins the movements of torsion.

(*d*) When the omental tumour is down in the hernial sac, and especially if the heavier portion is in front, the movements of the legs, as in walking, will cause its rotation through half a circle. Moreover, the expulsion of an already twisted tumour from the abdomen into the scrotum can easily lead to a further half-turn being added there and perhaps more during the reduction.

Both Baracz (6) and Hochenegg (8) explain the production of the pedicle and tumour in a similar way. The initial torsion they both consider is due to attempts at the reduction of the hernia. The succeeding twists they suggest are due to the changing pressures of the surrounding abdominal organs, to the progressive swelling and œdema occasioned by the partially twisted pedicle, and to the jolting in walking and pressing.

If the inguinal canal tightly grips the omental pedicle, a fixed point is formed and any torsion present will be added to or undone by movements of the legs. In this case

the twist is entirely within the sac. If, on the other hand, the inguinal canal is lax, as it has been in every recorded case, there will be no very fixed point for rotation to take place upon until the insertion of the omentum at the transverse colon is reached. Twists have been found in every portion of the omentum, from close to its free edge up to the colon. In these cases the twist begins at the inguinal region and slowly travels up the omentum, just as one can twist any other apron which is fixed at one end by twisting the other. By these means intra-abdominal torsion arises in connection with omental herniæ.

(2) *Abdominal Torsion.*

Six cases have been recorded in which omental torsion took place without any hernia being present. These are recorded by Baldwin (22), Syme (23), Stewart (43), Noble (45), Scudder (48), and Corner and Pinches (51). Peck (7) has recorded a case, very similar to Noble's (45), in both of which the distal end was adherent to the right Fallopian tube. But, as there was an old-standing hernia present, it was placed in the hernial-abdominal class. Wiener (9) recorded a case, in which the appendix was responsible for the distal adhesion, which was disqualified as a pure abdominal case for the same reason as Peck's (7). In both of these the original twists may have been formed in connection with the hernia.

In all the 6 cases of pure abdominal torsion the tumour was situated on the right side of the abdomen, and in 4 cases the diagnosis of appendicitis was made. In 4 the appendix was removed. In another the distal extremity of the omentum was adherent to the right Fallopian tube.

The causes would seem to be:

(*a*) Asymmetry of the omental tumour between the two pedicles, its weight in consequence leading to "passive" torsion.

(*b*) Muscular efforts, *e.g.* straining, increase th

twist. An ever present and hardly appreciated active force, which may add to the twists already taken, is the pumping action of the diaphragm.

(*c*) The vascular mechanism described by Payr.

Pathological Notes.

Strangulation.—In no instance is it noted that the omentum was strangulated by the neck of the sac. It is stated several times that the ring would admit the index finger, or that it was wide. In one observation (Demons, 2), the ring was said to be a little tight. If careful observations are made of "strangulated" epiploceles, it will be found that torsion of the omentum is often confounded with this strangulation. If Payr's vascular mechanism in the production of torsion is as great as it is stated to be, then every strangulated epiplocele must be accompanied by twisting, the veins being obstructed and the arteries not.

Twists.—The following table shows the number of turns present in the pedicle in all cases where it has been definitely stated :

One twist	4 cases
One and a half twists	2 ,,
Two twists	4 ,,
Two and a half twists	2 ,,
Three to four twists	5 ,,
Four to five twists	2 ,,
Five to six twists	2 ,,
Six twists	3 ,,
Seven twists	No case
Eight twists	1 ,,
Described as many	6 cases

From this table it will be seen that the number of turns in the pedicle is extremely variable. The largest number present is eight ; the smallest, one. From two to five twists appears to be the most common number. In nearly all cases the turns are tightly drawn and are

adherent, but in one instance (Capette, 38) it is stated expressly that as soon as the adhesions at the top and bottom were broken down the whole fragment rapidly untwisted itself. Mr. Makins (50) had a similar experience.

Direction of the twist.—The direction of the twist, whether from right to left or from left to right, appears to be entirely accidental, and so is of no consequence. In these cases it was noted that the twist was from right to left five times; in the reverse direction once. Owing to the lack of a standard rule for surgeons, the observations upon this topic are worthy of little credence. The terms "clockwise" and "counter-clockwise" have been used only once.

Pedicle.—The pedicle is, as a rule, thin and rope-like; in four instances it is said to be of the size of the finger; in one, of the thumb. In one observation (Blake, 35) it is said to be a rope-like structure 1¼ inches thick, and 4 inches long. In Baracz' (6) case it was 12 cm. long, in Heitz and Bender's (11) 2 cm. long. It was noted several times that the vessels on the pedicle were tortuous and enlarged.

(*For further information reference must be made to the 'American Journal of Medical Science,'* 1905, *and the 'St. Thomas's Hospital Reports,'* 1904.)

PART II.

Observation 1.

Max Oberst, 'Centralblatt für Chirurgie,' 1882, pp. 441–447:

Male, aged 35. Right reducible inguinal hernia for twelve years. The hernia suddenly became irreducible. Symptoms for two days. Diagnosis: Irreducible inguinal hernia. Operation: A mass of incarcerated omentum was found with the twist entirely within the sac. The twist

was so tight that rupture of the pedicle seemed about to occur. Recovery.

Observation 2.

Demons, 'Revue de Chir.,' 1893, p. 159:

Male, aged 36. Inguinal hernia for twenty-nine years. Three days after an effort the patient had an acute attack with symptoms pointing to strangulation of the hernia. Diagnosis: Strangulated hernia. Operation: In the sac a large mass of gangrenous omentum was found. It was impossible to withdraw a healthy piece from the inguinal canal, so the omentum was removed at a point which did not seem as gangrenous as the rest. The next day the patient improved, but during the following night symptoms of peritonitis appeared. Laparotomy was performed. In the abdomen was found a large gangrenous mass, representing all the lower part of the omentum, which had been twisted upon itself. Three turns were situated just below the transverse colon. This torsion was not suspected at the former operation, when it prevented the extraction of the omentum from the inguinal canal. Recovery.

Observation 3.

Bayer, 'Centralblatt für Chirurgie,' 1898, p. 462:

Female, aged 54. Reducible left inguinal hernia for fifteen years. History of an attack of coughing followed by severe pain, the patient having sensation "as if a round body was revolving inside the hernia." The examination revealed no tumour, but pain was felt on examining the inguinal canal. On the next day a distinct tumour could be both seen and felt in this region. Diagnosis: Strangulated inguinal hernia. Operation: A mass of omentum was found within the abdomen with a pedicle about the thickness of a finger twisted four or five times. It was adherent to the neck of the sac by a band forming one point of support; the other point of support was the intra-

abdominal attachment of the omentum to the colon. The torsion was caused by the omentum revolving between these two points. Recovery.

Observation 4.

Eiselberg, 'Deutsche Medicinische Wochenschrift,' 1898, p. 260:

Male, aged 42. Right reducible inguinal hernia for three years. Sudden onset. Diagnosis: Strangulated omental inguinal hernia. Operation: Intra-abdominal torsion of omentum was found close to the transverse colon. The abdominal tumour was continuous with that in the hernial sac. Death thirty-eight hours after operation. At the post-mortem examination several recent ulcers in the stomach and duodenum were found. Death.

Observation 5.

Monod, 'Thèse de Reynier,' 1898:

Male, aged 40. Right reducible inguinal hernia for six years. Onset after a sudden and violent effort, accompanied by pain in the right iliac fossa. There was a large hard, tender swelling in the right side of the scrotum; the temperature was raised slightly, no vomiting. In the right iliac fossa a cord could be felt which appeared to be continuous with the scrotal tumour. Diagnosis: Strangulated omental inguinal hernia. Operation: A mass of omentum was found adherent to the bottom and twisted six times upon itself just above the neck of the sac. The patient died of bronchopneumonia.

Observation 6.

Baracz, 'Deutsche Zeitschrift für Chirurgie,' 1900, pp. 584–589:

Male, aged 42. Reducible left inguinal hernia for several years. Sudden attack of severe pain in the right inguinal region after lifting a heavy trunk; the hernia

became larger and irreducible. Two days later the inguinal pain had diminished, but severe paroxysmal abdominal pain had begun; the bowels were open. The left

FIG. 1.

Paracz' Case.

inguinal region was slightly prominent, and extending upwards from the left testicle was a hard tender strand running through the inguinal canal. The mass was irreducible. The abdomen was distended and tympanitic, with

shifting dulness in the flanks. Diagnosis: Strangulated inguinal hernia. Operation: A hard strand of omentum was present in the sac, being 12 cm. long and about the thickness of a finger. It was twisted five or six times on its own axis, and was adherent to the base of the sac by a broad band. The strand was connected with a tumour in the abdomen weighing 1 kilogramme and composed of the lower part of the omentum twisted several times around its own axis. Recovery.

Observation 7.

Peck, Reference not confirmed. Quoted by Wiener (9):

Female, aged 37. Reducible right inguinal hernia for twelve years. Sudden attack of abdominal pain accompanied by vomiting; temperature 100·5° F. Symptoms lasted for four to five days. An indistinct mass could be made out in the abdomen. Diagnosis: Abdominal tumour. Operation: The tumour was composed of the entire omentum rolled up in a vertical direction, filling the right side of the abdomen from the umbilicus to the pelvis. About two inches below the transverse colon a tightly twisted pedicle was present, consisting of the entire upper portion of the omentum twisted around its vertical axis. The omental tumour was not connected with the hernia, but was in contact with the right tube and ovary, which were adherent to it. Recovery.

Observation 8.

Hochenegg, 'Wiener Klinische Wochenschrift,' 1900, pp. 291–293:

Male, aged 41. Reducible right inguinal hernia for thirty years. Two years ago the patient had gastric disturbances, with sensations as if the stomach was being pulled aside by a heavy weight. While in a bath the hernia suddenly became larger and could only be reduced with difficulty. Twenty-four hours later there was a sudden attack of severe abdominal pain, accompanied by vomiting. The right thigh was kept flexed; the abdomen

was distended and tense. In the right inguinal region about a hand's breadth above Poupart's ligament and extending outwards into the flanks, a distinct mass could be felt, which was tender and dull on percussion. Diagnosis: Appendicitis. On the following day the abdomen was more distended and the mass had increased in size. The hernial sac was distended, fluctuating, and compressible. Operation: About 2 litres of bloody serum were present in the abdominal cavity. A non-adherent omental tumour

FIG. 2.

Wiener's Case.

was present; this mass was doubled on itself, the two halves being joined by adhesions. At the upper portion of it there was a slender pedicle which was twisted upon itself three or four times; above this the omentum was normal. Recovery.

Observation 9.

Wiener, 'Annals of Surgery,' 1900, vol. ii, pp. 648–662: Male, aged 79. Reducible right inguinal hernia for thirty

years. Four weeks before the acute attack there was a sudden and severe pain in the right iliac region, which lasted twenty-four hours and then subsided. After the attack the patient was quite well. Four days before operation there was another sudden attack of severe pain in the right iliac region; no vomiting or fever; the pain continued at intervals for four days; the bowels were opened regularly. Midway between the anterior superior spine on the right side and the free border of the ribs, and extending from the axillary to the mammary line, could be felt a rounded tumour, the size of an orange, which was tender on palpation and dull on percussion. The hernial sac was empty. Diagnosis: Intra-abdominal abscess. Operation: An omental tumour was found; the distal portion was adherent to an appendix epiploica on the ascending colon, the proximal portion was continuous with the rest of the omentum by means of a narrow pedicle which was twisted on itself five to six times. Recovery.

Observation 10.

Broca, 'Bull. Soc. Chir.,' 1900, p. 710; *Chavannaz,* 'Gaz. Hebd. Med. et Chir.,' 1900, p. 601:

Male, aged 17. A right hydrocele for 15 years. The patient wore a truss. Suddenly one morning an irreducible tumour appeared in the right inguinal region, without any signs of strangulation being present. Taxis was attempted for several days. A right scrotal tumour was present about the size of a chicken's egg; below this was a vaginal hydrocele. Above the hydrocele was attached a hard round cord, as thick as the middle-finger, irreducible, painless, and dull on percussion; this cord was prolonged into the inguinal region. Diagnosis: Omental hernia. Operation: A vaginal hydrocele was present; above this was a cyst of the spermatic cord invaginating the hernial sac, which contained omentum. The omentum was adherent to the small part of the sac projected into by the cyst. It was found that the lower part of the omentum

had formed two rings, which were round the part above it. The omentum was adherent to the peritoneum above this ring. Recovery.

Observation 11.

Heitz and Bender, 'Bull. Soc. Anat.,' 1900, pp. 957–960: Male, aged 50. Left irreducible inguinal hernia for

FIG. 3.

Broca's Case.

many years. Though the bowels had not been opened for two days, the patient had passed flatus. The hernia was painful, the pain being most marked at the neck of the sac. Abdomen slightly distended. Diagnosis: Obstructed

hernia. Operation: A large mass of omentum was found in the sac; in withdrawing the omentum two large turgescent masses appeared. There was a large quantity of sanguineous fluid in the abdomen. The omentum was removed and a "radical cure" done. The omentum was healthy in its upper part and in the lowest part, which was occupying the hernial sac. The middle portion was

FIG. 4.

Heitz and Bender's Case.

gathered together so as to form two or three fibrous cords at the end of which were fatty masses. Two of these had crossed and twisted round each other, forming a kind of strand 2 cm. long and plaited for 2½ turns. When untwisted it was seen to be made up of two distinct cords, the pedicles of the two fatty masses. The patient died of broncho-pneumonia.

Observation 12.

Potherat, 'Bull. Soc. Chir.,' 1900, pp. 525–531:

Male, aged ?. Onset with symptoms of strangulated inguinal hernia; taxis was applied and the tumour became smaller, but later the symptoms increased in severity. Diagnosis: Reduction *en masse.* Operation: In the sac was a small mass of black omentum, continuous with a larger and apparently gangrenous mass in the abdomen. This was twisted upon its own pedicle close to the transverse colon. The tumour was removed. Recovery.

Observation 13.

Walther, 'Bull. Soc. Chir.,' 1900, pp. 525–531:

Male, aged 22. Right inguinal hernia. Immediately after bicycle exercise the hernia suddenly became larger, was dull on percussion, and very painful. Diagnosis: Strangulated hernia. Operation: Two hours after onset of symptoms. In the sac was a mass of omentum twisted six times upon itself. The torsion had taken place between two adhesions, one at the bottom of the sac and the other immediately above the inguinal canal. There were two separate torsions present, one within the hernia, the other at the level of the neck of the sac. Recovery.

Observation 14.

Lejars, 'Bull. Soc. Chir.,' 1900, pp. 525–531:

Male, aged 44. Right inguinal hernia of old standing. The patient had all the signs and symptoms of appendicitis. A large mass was present in the right iliac fossa. There was a reducible inguinal hernia on the right side. Diagnosis: Appendicitis. Operation: There was a large [redacted] of omentum in the abdomen, the pedicle of which [redacted] or four times upon itself immediately [redacted] colon; the lower part descended

into the sac. The tumour was removed. The patient died of delirium tremens.

Observation 15.

Lucas-Championnière, 'Bull. Soc. Chir.,' 1900, pp. 525–531 :

Male, aged 45. Large left reducible inguinal hernia for 27 years. For several weeks it had been irreducible and painful, accompanied by violent griping pains and vomiting, but the bowels were not confined. On examination the hernia was irreducible and very painful; it was continuous with a mass in the abdomen which extended from the left groin nearly to the umbilicus. Diagnosis: Strangulated hernia. Operation: The scrotal mass was very adherent, it was continued up the inguinal canal into the abdomen, and was attached to the anterior abdominal wall; this portion was thin. The abdominal mass extended almost to the transverse colon, and joined the normal omentum by a narrow pedicle which was twisted upon itself. The tumour was removed and weighed 645 grms. Recovery.

Observation 16.

Wiesinger, 'Münch. med. Woch.,' 1901, p. 480 :

Male, aged 41. Right irreducible inguinal hernia for many years. Onset of attack with symptoms pointing to appendicitis; pain and tenderness in the right iliac fossa, with slight rise of temperature. Diagnosis: Appendicitis. Operation: The omentum in the sac was continuous with a mass in the abdomen, which was twisted upon itself many times, and had become necrotic. The tumour was removed. Recovery.

Observation 17.

Lucas-Championnière and *Mauclaire,* 'Bull. Soc. Anat.,' 1901, p. 241 :

Female, aged 55. Large [illegible] inguinal

hernia for 20 years. For 7 to 8 days the patient had had pain in the hernia and in the abdomen, accompanied by vomiting; the bowels were open. The hernia was hard and painful, and the tumour in it seemed to be continued into the abdomen. Diagnosis: Strangulated hernia. Operation on fourth day: The sac was thickened and contained blackish adherent omentum, which could be traced up the canal into the abdomen. Just below the transverse colon it was twisted upon itself in the form of a spiral and drawn tight. Recovery.

Observation 18.

Souligoux and *Deschamps*, 'Bull. Soc. Anat.,' 1901, p. 229:

Female, aged 35. Operation when 27 for salpingitis. Four months afterwards, following an effort, a reducible tumour about the size of the fist appeared at the site of the scar. The patient stated that she could feel three separate tumours. Suddenly, and without any apparent cause, the hernia became larger, painful, and irreducible. For two days neither fæces nor flatus were passed; no vomiting. In the middle line of the abdomen a hard painful tumour, the size of a fist, could be felt; it was dull on percussion. Operation four days after the onset of acute symptoms. In the sac was a small blackish piece of omentum, and a little blood-stained fluid; the mass was continuous with a large intra-abdominal tumour. The sac showed three loculi. The hernial tumour was continuous with the intra-abdominal mass by a thin pedicle; the intra-abdominal portion, rectangular in shape, 10 cm. long and 8 cm. round, was joined to the normal omentum above by two pedicles at its upper angles. One was very thin, and the other the size of the little finger. The latter was twisted 2½ times upon itself. Recovery.

Observation 19.

Delivein 'Wien. klin. Runds.,' 1901, p. 184:

'nguinal hernia for thirty years.

Onset with pain, slight rise of temperature, vomiting, and inaction of bowels. The hernia was irreducible but not strangulated; a finger could be passed through the ring. A tumour could be felt in the right iliac fossa, which was painful on palpation and dull on percussion. Diagnosis: Appendicitis. Operation: In the abdomen was a mass of omentum, twisted many times upon itself close to the transverse colon; the lower part was prolonged into the hernial sac. Recovery.

Observation 20.

Tuffier, 'Bull. Soc. Chir.,' 1901, p. 547:

Male, aged 48. Left reducible inguinal hernia. History of twenty-four hours' duration with all the signs of intestinal obstruction. There was an elongated tumour in the left iliac fossa, dull on superficial percussion, resonant on deep. Diagnosis: Intestinal obstruction. Operation: There was a little fluid in the abdomen; a large, black, omental tumour adherent to coils of distended gut was twisted twice on itself just below its junction with the colon. It was adherent below to the internal ring. Recovery.

Observation 21.

Wiart and *Renon*, 'Bull. Soc. Anat.,' 1901, p. 111:

Female, aged 53. Right reducible inguinal hernia for thirteen years. Onset with pain in the right iliac fossa. The abdomen was a little distended; the hernia could not be completely reduced. After two days' obstinate inaction of the bowels the abdomen became enlarged and a little painful over a small area in the right iliac fossa. From the level of the anterior superior spine of the ilium downwards and inwards towards the hernia a hard lobulated tumour the size of a turkey's egg could be felt which was not painful on palpation. It was dull on percussion. It extended to the umbilicus. The patient had known of this tumour for some time past. Diagnosis: Strangulated hernia. Operation: In the sac was a red congested mass of omentum the size of a chicken's egg. In the abdomen

was another omental mass about the size of a fœtal head; the neck of this mass was strangulated by the appendix, which was coiled twice round it. The appendix was removed and the omentum resected. The tip of the former was adherent to the omental pedicle, the rest of

FIG. 5.

Wiart and Renon's Case.

it was quite free and could be easily unrolled. The mechanism was in all probability as follows: Torsion of the omentum caused the appendix, which was adherent to it by its tip, to become twisted round the pedicle. Recovery.

Observation 22.

Baldwin, 'Annals of Surgery,' 1902, vol. ii, pp. 940–944: Male, aged 47. Onset with nausea and pain. On the next

day the pain had increased in severity; tenderness and muscular rigidity were present on the right side of the abdomen. Symptoms increased till the end of the fourth day. The most tender spot was situated just above Mac Burney's point. Diagnosis: Appendicitis. Operation: A mass of omentum was rolled up so as to form a distinct tumour, about the size of a large fig, with a very small pedicle about the size of a knitting-needle which was twisted upon itself eight times. The distal portion of the appendix was found to be obliterated. Both the appendix and the tumour were removed. Recovery.

Observation 23.

Syme, 'Intercol. Med. Journ. of Australasia,' 1902, pp. 444, 445:

Female, aged 51. Onset of attack with severe pain and the appearance of a swelling in the abdomen; no vomiting. The patient gave a history of having slipped and fallen seven days previously. A rounded tumour could be felt on the right side of the abdomen, extending from a point 1½ inches below the costal margin, almost to the iliac crest, and extending inwards to the umbilicus. The tumour was movable and dull on percussion. The tongue was a little coated, the bowels rather constipated, the temperature 98·4° F. No signs of a hernia present. Diagnosis: Suppurating omental hydatid. Operation: The small gut was found to be adherent to an omental tumour in the abdomen. This tumour was twisted several times upon itself. Recovery.

Observation 24.

Riedel, 'Verhandl. der Deutsch. [illegible] Chir.,' 1902, vol. i, p. 92:

Female, aged 31. Right ingu[illegible] had never worn a [illegible] pain, sickness, [illegible]

in the right iliac fossa; the hernia was irreducible, the bowels were open. Diagnosis: Appendicitis. Operation: The sac contained a mass of omentum continuous with another mass within the abdomen, which was twisted upon itself close to the transverse colon. Recovery.

Observation 25.

Moresco, 'Gaz. degli Osped. delle Clin.,' 1902, pp. 693–695:

Male, aged 36. Right reducible inguinal hernia for many years. Onset with symptoms of strangulated hernia, A painful tumour was present in the inguinal region. It was dull on percussion. The bowels were opened by means of purgatives. The temperature was slightly raised. There was no sickness. Diagnosis: strangulated omental hernia. Operation: the omentum was reduced from the hernial sac into the abdomen. Death a few hours later. Post-mortem: A large mass of omentum was found in the abdomen, twisted upon itself close to the transverse colon; it was continuous with that which had been present in the hernial sac.

Observation 26.

Heinlein, 'Münch. med. Woch.,' 1902, p. 1485:

Female, aged 30. Right inguinal hernia. An operation for radical cure was done two years previously. Three weeks before admission to the hospital the hernia had suddenly reappeared, accompanied by vomiting. Diagnosis: Strangulated hernia. Operation: A mass of omentum was found twisted upon itself within the sac. Recovery.

Observation 27.

Nordmann (1), 'Wien. klin. Woch.,' 1903, p. 459:

[illegible] reducible inguinal hernia for [illegible] dominal pain and headache.

Neither fæces nor flatus were passed. The hernia was hard and irreducible. A mass the size of a fist, tender on pressure, could be felt in the right iliac fossa. It appeared to be continuous with the hernia. Operation four days after the onset of symptoms. In the abdomen was a mass of congested omentum, twisted many times upon itself. The tumour was continuous with the portion of omentum present in the hernial sac. After removal, it was found to be 20 cm. long, 12 cm. broad, and weighed 250 grammes. Recovery.

Observation 28.

Nordmann (2), 'Wien. klin. Woch.,' 1903, p. 459:

Male, aged 37. Right reducible inguinal hernia since a child. For several weeks the patient had suffered from gastric disturbances, accompanied by abdominal pain. Sudden acute onset, with vomiting. The hernia was hard, tender, and irreducible. There was tenderness in the right iliac fossa over an area the size of a hand, with resistance to palpation. Diagnosis: Strangulated inguinal hernia. Operation: Five days after onset. In the abdomen there was a large matted mass of omentum, twisted upon itself and connected with a second mass in the hernial sac; at the lower part of this second mass was a small portion of omentum, also twisted upon itself. After removal the tumour was found to weigh 300 grammes, and to be 32 cm. long, 20 cm. broad. Recovery.

Observation 29.

Quènu, 'Bull. Soc. Chir.,' 1903, pp. 520–522:

Female, aged 51. Left reducible inguinal hernia for four years; the patient frequently had attacks of slight pain in the hernia. Onset with sudden attack of violent abdominal pain, especially acute in the [illegible]. The hernia was irreducible. Five days [illegible] painful tumour [illegible] descending [illegible]

opened; slight vomiting. The abdomen was soft except on the left side. Diagnosis: Irreducible inguinal hernia. Operation: Filling the sac there was an enormous mass of omentum, black in places, and slightly adherent; it could not be withdrawn. The whole omentum had been twisted upon itself, close to the transverse colon; two turns were present. Recovery.

Observation 30.

Rudolph (1), 'Wien. klin. Rundschau,' 1903, pp. 794–796:

Female, aged 53. Right inguinal hernia for 4 years. Onset with sudden pain in the hernia and in the right iliac region; no sickness; the bowels were opened three times. In the right iliac fossa there was a tender mass, the size of a fist, which extended down into the hernia, occupying the enlarged right labium majus. The tumour was hard and inelastic. *Per vaginam* and *per rectum* nothing abnormal was felt. No leucocytosis. Operation after 2 days. Intra-hernial torsion of omentum; many twists were present; the length of the twisted portion was 21 cm., the breadth 5 cm. Recovery.

Observation 31.

Rudolph (2), 'Wien. klin. Rund.,' 1903, pp. 811–813:

Female, aged 42. Left reducible inguinal hernia for 12 years. For 12 days it had been painful and irreducible; no sickness. The bowels were not opened, but flatus was passed freely. Diagnosis: Incarcerated inguinal hernia. Operation: The sac contained a mass of omentum which had been twisted upon itself in the inguinal canal. Recovery.

Observation 32.

'Wien. klin. Rund.,' 1903, pp. 834–836:

Right inguinal hernia for 20 years; for 1 year. Both sides had been

operated on, but the herniæ had recurred. The patient walked into hospital, complaining of abdominal pain; no sickness; the bowels had been opened. There was a small swelling in the right inguinal region. Unknown to her, there was also an intra-abdominal swelling on the right side which extended two fingers' breadth above the umbilicus and from the right anterior superior spine of the ilium to the middle line. The abdominal tumour appeared to be continuous with the inguinal hernia. *Per vaginam* and *per rectum*, nothing abnormal was found. Diagnosis: Intra-abdominal torsion of omentum with incarcerated epiplocele. Operation: intra-abdominal torsion of omentum was found. The tumour was removed and found to weigh 180 grammes. Recovery.

Observation 33.

Rudolph (4), 'Wien. klin. Rund.,' 1903, pp. 848–851:

Male, aged 35. Right reducible inguinal hernia for a long time. In the right inguinal region, concealing the testicle, was a smooth, hard, spindle-shaped swelling 25 cm. long and 10 cm. broad. Diagnosis: Sarcoma of the right testicle. Operation: In the inguinal region there was a congenital hernial sac containing a mass of omentum the size of a fist. This mass was twisted upon itself, the actual portion twisted being very thin. Recovery.

Observation 34.

Tremolières, 'Bull. [illegible] 1903, p. 693:

Male, aged 50. [illegible] When first seen the patient had [illegible] peritonitis [illegible] resistant [illegible] was pre[illegible] constipation [illegible] and the patient [illegible] men, a cord like [illegible]

peritoneal cavity like a sling. Below, it entered and was adherent to the sac of a right inguinal hernia; above, it went as far as the transverse colon, and could be followed to the splenic flexure. This portion of the colon was fixed. The cord when untwisted was seen to be the omentum, rolled upon itself and atrophied.

Observation 35.

Blake, 'Annals of Surgery,' 1903, pp. 99–102:

Male, aged 45. Right reducible inguinal hernia for 20 years. Onset with pain in the right side of the abdomen and in the hernia. Attempts at reduction failed; vomiting. Three days later there was a large tender scrotal hernia; a small portion only could be reduced. Temperature, 101°F. The lower right quadrant of the abdomen was filled with a mass which reached to the middle line. Diagnosis: Appendicitis. Operation: A large gangrenous mass of omentum was found in the abdomen, the lower end of which entered the sac and could not be withdrawn. There was a little fluid present. The omentum was not adherent, but the tip of it (in the sac) was enlarged. At its attachment to the transverse colon the omentum was twisted on itself 4½ times, forming a rope-like mass 1¼ inches thick and 4 inches long. The tumour was removed and found to weigh 2 lb. 12 oz. Recovery.

Observation 36.

Sonnenburg, 'Arch. Internat. de Chir.,' 1903:

Male, aged 32. Imperfectly descended right testicle. Right inguinal hernia for 25 years. Sudden onset of pain in the hernia, which became irreducible; no sickness; no rise of temperature. In the lower portion of the right side of the abdomen, just above Poupart's ligament, a swelling the size of a hen's egg could be felt. The right testicle could be distinguished in the inguinal canal at the bottom of the swelling. Diagnosis: Strangulated omentum

with inflammation. Operation: Two days after onset. In the sac was a little sanguineous fluid, the atrophic testicle, and a non-adherent mass of blue-black omentum. The latter was removed. It was found that it had been twisted upon itself just within the abdominal cavity. Recovery.

Observation 37.

Zeller, 'Freie Vereinig der Chir.,' Berl., Dec., 1902:

Male, aged ? Right inguinal hernia. The patient had all the symptoms of perforative appendicitis. There was a large tumour on the right side of the abdomen, which was painful on palpation; marked fever and complete obstruction were present. Diagnosis : Appendicitis. Operation on the seventh day. A mass of gangrenous omentum was found twisted through more than 360°. It formed a tumour as large as a child's head, the pedicle of which was thin. At the lower end of the tumour was another pedicle which was adherent to a right-sided hernial sac. The tumour was removed. The patient died.

Observation 38.

Capette, 'Bull. Soc. Anat.,' 1903, p. 539:

Male, aged 50. Left inguinal hernia. The patient when first seen had a rather large left inguinal hernia, which was not very tight. The symptoms of strangulation were not very well marked. Diagnosis : Strangulated hernia. Operation: Within the sac was a piece of omentum adherent by its two extremities, one to the bottom of the sac and the other at the level of the internal ring. The intermediate portion of omentum had been rolled up, so that its calibre was about that of a medium-sized cord, and was so arranged that, when the adhesions were broken down, the whole fragment rapidly untwisted itself. Recovery.

Observation 39.

Rutherford Morison, 'Northumb. and Durham Med. Jour.,' 1903, p. 211:

Male, aged 41. Left inguinal hernia for ten years. Recently the patient had had some pain and trouble in reducing it. While lifting heavy boxes he had sudden pain in the abdomen and the hernia came down. It was partially reduced on the next day, but the pain continued; the bowels were not opened, nor was flatus passed. At the end of three days the pain was worse and the patient vomited. The abdomen was distended and rigid all over, but especially so at the lower part. There was a firm, tender, indefinite swelling extending up from the left inguinal region to the umbilicus. Every two or three minutes there were exacerbations of pain. The lower quarter of the abdomen was dull on percussion. No hernia was actually present in scrotum. Diagnosis: Reduction of hernia *en masse,* with peritonitis. Operation: Four days after onset. In the inguinal canal there was a dark-coloured mass of omentum, which was attached by a long twisted cord to the bottom of the sac. Nearly all the omentum was a dark plum-colour, and close to its attachment to the transverse colon was twisted into a tight-rope-like structure. There was a little blood-stained fluid in the abdomen. The tumour was removed and was found to weigh 1 lb. Recovery.

Observation 40.

Vignard and *Giraudeau,* 'Arch. Prov. de Chir.,' April, 1903, pp. 206–230:

Male, aged 31. Right inguinal hernia since adolescence; imperfectly descended testicle on the same side. During the last nine years the patient had had attacks of pain in the right side of the abdomen unaccompanied by vomiting. The bowels were open. The temperature was 100·4° F. On the sixth day there was a painful swelling in the right

side of the scrotum surrounding the testicle; the scrotal pain got better, and during the third week the patient got up. The abdominal pain remained; there was pain on stooping and the patient was unable to do his work. In the right side of the abdomen there was a hard, painful tumour extending from the costal margin to the right side of the scrotum. Towards its middle it was about the size of the palm of the hand; below it thinned out and entered the hernia. It was dull on percussion. Diagnosis: Appendix abscess, the inflammation spreading to the sac of the inguinal hernia. Operation: One month after onset. The omentum, adherent to the abdominal wall, formed a blackish tumour extending into the inguinal region, where it was also adherent. The pedicle was twisted very tightly three times close to the transverse colon. The appendix contained three calculi and was removed. There was a second torsion just above the inguinal canal, not drawn very tightly. Recovery.

Observation 41.

Malherbe, 'Arch. Prov. de Chir.,' April, 1903, pp. 206–230:

Male, aged 28. Right inguinal hernia for six years. The patient gave a history of having had attacks of pain accompanied by vomiting, in the right inguinal region during the last two years. In this period he had had five attacks, each terminating when the hernia was reduced. Onset with a sudden attack of violent pain in the right iliac fossa; the hernia was reduced but the pain continued, being most intense at MacBurney's point. On the next day it was much better; later, it increased and became constant; there was slight fever, diarrhœa, and vomiting. There was some resistance to palpation in the right iliac fossa. No definite tumour was felt, but a little dulness on percussion was found over the resistant area. Temperature 101·8° F. Diagnosis: Appendicitis. Operation: Four days after onset. There was a large mass of omentum,

blackish in colour, adherent to the abdominal wall. At the junction of the upper and middle third the pedicle was twisted three times upon itself. The tumour was removed; the appendix, being slightly congested, was removed also. The tumour was 28 cm. long, 10 cm. broad, 15 cm. in circumference, and weighed 295 gr. Recovery.

Observation 42.

Barker, 'Brit. Med. Journ.,' 1903, vol. 1, p. 477:

Male, aged 24. Right reducible inguinal hernia for a long time. Onset of attack with sudden pain in the right groin accompanied by vomiting; the abdomen was distended, and there was a hard tender mass above Poupart's ligament. The temperature was raised. Diagnosis: Appendicitis. A mass of semi-gangrenous omentum was found as large as a fist, with torsion at its proximal portion which formed a pedicle. This had been twisted round and round until it formed a hard cord. Twelve half-turns were necessary before it was released. Recovery.

Observation 43.

Stewart, 'Journ. of the Amer. Med. Assoc.,' 1904, p. 767:

Male, aged 35. Sudden onset with severe abdominal pain, starting in the epigastrium and settling in the right iliac region; vomiting; temperature 101° F., pulse 120. There was no hernia present. The right side of the abdomen was held rigid, the most tender spot being at the outer border of the right rectus, on a level with the umbilicus. The bowels were open. Diagnosis: Appendicitis. Operation four days after the onset of acute symptoms. The abdomen was opened and the appendix removed; this was normal except for three or four minute hæmorrhagic spots beneath the mucous membrane. A mass was found, which proved to be the right lower portion of the omentum, gangrenous and twisted upon itself through

360°. The tumour was removed and found to be six inches long and three inches wide. Recovery.

Observation 44.

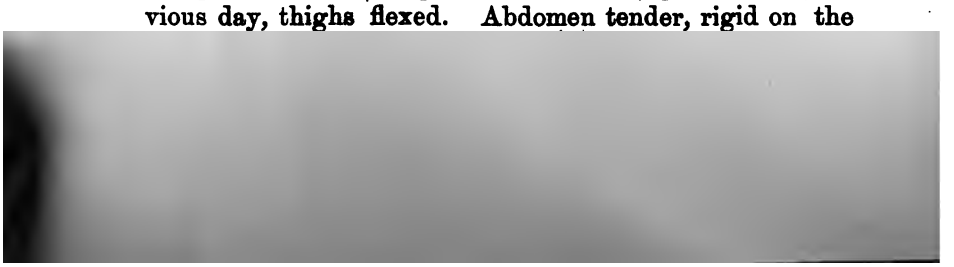
Audier, 'Thèse,' Lyon, 1904, 62 pages:

Female, aged 50. Right inguinal hernia. Onset with signs of partial intestinal obstruction; fæces and flatus were passed. There was a history of several similar attacks. The general condition was very bad. In the right inguinal region there was an elongated mass parallel to Poupart's ligament and extending above it. This tumour appeared to end close to the external ring. On palpation it was irregular, 8–9 cms. long. A tight pedicle at the upper part appeared to fix the tumour to the abdominal organs. It was dull on percussion. Diagnosis: Inguinal omental hernia. Operation four days after onset. In the sac was a large omental mass the size of a fœtal head, gangrenous in several places, adherent to the sac at its lowest part. Just within the abdomen was a second omental tumour connected with the first by a narrow pedicle, which was twisted several times upon itself. The second mass was connected by a pedicle with the intestine, probably the transverse colon. No definite torsion was observed in the upper pedicle. The patient died.

Observation 45.

Noble, 'The Med. and Surg. Monitor,' Indianapolis, June, 1904, pp. 261–263. 'Amer. Journ. of Obstet.,' March, 1904, pp. 364–366:

Female, aged 24. Gonorrhœa 2½ years previously. Sudden onset with excruciating pain in the abdomen, attended with nausea and vomiting. History of similar attacks at irregular intervals during the last two years. Temperature 100° F., pulse 90: bowels opened the previous day, thighs flexed. Abdomen tender, rigid on the



right side, and pressure over MacBurney's point elicited great pain. Dull note on percussion to the right of and below the umbilicus. Diagnosis: Appendicitis. Operation refused. Six days later sudden chill, followed by a temperature of 101° F.; pain increased. A mass could be felt midway between the anterior superior spine and the umbilicus. Operation: black mass in the abdomen two inches wide, five inches long, surrounded by recent adhesions to the fimbria of the right tube. With the colic origin as the proximal and the adhesion to the fimbria as the distal, an axis was formed around which the omentum had twisted upon itself. The appendix, although it took no part in the production of symptoms, was removed. Recovery.

Observation 46.

Mauclaire (1), 'Revue de Gyne. et de Chir. Abdom.,' 1904, pp. 425–430:

Male, aged 40. Very large inguinal hernia, which descended to the knee. Onset with symptoms of strangulation. Diagnosis: Strangulated hernia. Operation: In the sac there were 5–6 litres of serous fluid, the cæcum, ascending colon, and 50 cms. of small intestine. There were also present 7-8 hard omental cords, distinct from one another; some were vascular; the larger number were about the size of the little finger. Some of these were twisted round each other either at the middle or upper part; in length they were 20-25 cms. Recovery.

Observation 47.

Mauclaire (2), 'Revue de Gyne. et de Chir. Abdom.,' 1904, pp. 425–430:

Male, aged 45. Right reducible inguinal hernia for fourteen years. Suddenly it became painful and increased in size. In the scrotum there was an oval tumour the size of an apple; in the direction of the external ring a pedicle could be felt. The tumour was dull on percussion. No

enlargement of abdomen; diarrhœa; no vomiting; temperature 100·9° F. On the next day the tumour was a little smaller, less tender; diarrhœa persistent; vomiting commenced. Operation: On opening the sac, a thin pedicle was seen, twisted one and a half times upon itself; this was attached to an omental mass below, which was slightly adherent to the sac. The tumour was red and congested. There was a large quantity of sanguineous fluid in the sac. Recovery.

Observation 48.

Scudder, 'Annals of Surgery,' Dec., 1904, pp. 916–920:

Male, aged 25. Onset of attack with abdominal pain and vomiting, the pain becoming localised to the right side of the abdomen. Bowels not opened for three days. On the right side of the abdomen, a mass was felt occupying the whole of the iliac fossa, and extending to the hypochondrium and to the middle line; it was dull on percussion and not very tender on palpation. Diagnosis: Appendicitis. Operation: Eight days after onset. In the right iliac fossa there was a mass of friable dark omentum, with thrombosed vessels; this was continuous with a firm twisted mass of omentum, the size of an orange, lying close to the transverse colon. It was twisted one and a third times upon itself. The appendix was removed, as it was thickened and œdematous. Recovery.

Observation 49.

Trinkler, 'Deut. Zeitschrift für Chir.,' Band 75, 2–4, 1904, pp. 269–281:

Male, aged ?. Right inguinal hernia for ten years. Cryptorchid on same side. There was a history of previous attacks of abdominal pain. Onset with pain, constipation, no vomiting. A tumour could be felt in the right iliac fossa, resistant to palpation, dull on percussion. In the scrotum there was a swelling the size of a child's head, which obscured the testis. Diagnosis: Strangulated omen-

tal hernia. Operation: Herniotomy followed by laparotomy. In the sac there was a mass of omentum which was not strangulated; this was continued up into the abdomen, where it was twisted three times upon itself. Recovery.

Observation 50.

G. H. Makins, St. Thomas' Hospital; not reported previously:

Female, aged 44. Irreducible umbilical hernia for fourteen years. She had suffered from several attacks of pain in the hernia, sometimes accompanied by sickness. Acute onset with pain, bowels not opened, flatus passed, no vomiting. Diagnosis: Strangulated umbilical hernia. Operation: In the hernia there was some small intestine which was not strangulated. In a loculus of the sac there was a portion of omentum tightly twisted upon itself; this when freed spun round and untwisted. Recovery.

Observation 51.

Corner and Pinches (1) this paper:

Female, aged 37. The patient gave a history of having been ill for ten days, with pain in the right side. There had been no sickness, and the bowels had acted. The temperature was slightly raised. No hernia was present. There was a history of previous attacks of pain and discomfort in this region, but none were sufficiently severe to cause her to go to bed. A tender mass was present in the right iliac fossa, extending to just above the umbilicus. It was dull on percussion, and its outlines could not be clearly defined. *Per vaginam* and *per rectum* nothing abnormal was found. Diagnosis: Appendix abscess. Operation: On opening the abdomen there was a rush of dark green odourless fluid, which had evidently been imprisoned under tension. On inserting a finger, a cavity was entered on the inner and anterior aspect of which was felt a mass harder than the other walls. As pus was not

present, adhesions were broken down, and a mass of omentum delivered. A twist was seen at the proximal end; the mass was ligatured and removed. Recovery.

Observation 52.

Corner and Pinches (2), this paper:

Male, aged 41. Right reducible inguinal hernia for

FIG. 6.

Corner and Pinches' Case (2).

18 years. There had been previous attacks of pain in the hernia. For some days it had been irreducible, painful, and slowly enlarging. There was no sickness and the bowels were open. Previously there had been pain, when the hernia came down and remained

irreducible. Diagnosis: Strangulated inguinal hernia. Operation: The sac contained an enormous mass of dark-coloured omentum; traction on the mass easily withdrew it from the abdomen. The pedicle was twisted. After removal it was found to consist of a large mass, bifid at its free end, and by chronic inflammation adherent and divided into numerous lumps. Recovery.

Observation 53.

Corner and Pinches (3), this paper:

Male, aged 45. Large right inguinal hernia of old standing. For some time he had suffered from increasingly severe attacks of pain in the hernia. At the lower part of the sac was a small loculus, separated from the rest by an obvious constriction. Diagnosis: Strangulated hernia. Operation: There was no strangulation in the upper part or in the lower. When the latter loculus was laid open it was seen to contain a small rounded mass of omentum, which was not adherent, but was twisted on its pedicle. Recovery.

Observation 54.

Michaux, 'Sèance de la Société de Chir.,' Nov. 21st, 1900:

A piece of omentum strangulated by torsion was shown. No details were given, so that this case has not been included.

CHLOROFORM ANÆSTHESIA

A Special Discussion introduced by four short Papers describing Apparatus

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AN APPARATUS FOR ADMINISTERING CHLOROFORM VAPOUR MIXED WITH AIR IN ASCERTAINABLE PROPORTIONS. BY A. G. LEVY, M.D.

A NEW APPARATUS FOR THE DELIVERY OF CHLOROFORM AND AIR OF SUITABLE AND EASILY GOVERNED STRENGTH. BY A. D. WALLER, M.D., F.R.S.

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AN APPARATUS FOR THE DELIVERY OF CHLOROFORM VAPOUR OF KNOWN DILUTION. BY B. J. COLLINGWOOD M.B., B.C.CANTAB.

LIST OF SPEAKERS IN DISCUSSION

DR. F. W. HEWITT
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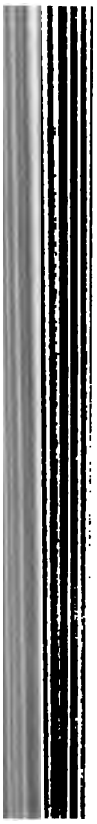

AN APPARATUS

FOR

ADMINISTERING CHLOROFORM VAPOUR MIXED WITH AIR IN ASCERTAINABLE PROPORTIONS

BY

A. G. LEVY, M.D.,

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(From the Laboratories of Pathological Chemistry and of Physiology at University College, London.)

Received April 7th—Read November 22nd, 1904.

THE instrument I have constructed is designed for regulating the strength of the chloroform vapour administered in the atmosphere inhaled, and at the same time for restricting its maximum strength within such limits of safety as are compatible with all practical exigencies. The instrument indicates with an approximate degree of accuracy the percentage of chloroform vapour inhaled. Absolute accuracy is only attainable under absolutely constant physical conditions such as may be provided for in a laboratory, but which cannot be insured for practical purposes.

The type of apparatus conforms to that originated by

John Snow,[1] of which the main principle is the suction of air over a surface of chloroform by the respiration, and the dilution of the mixture of air and chloroform vapour thus obtained by such a further proportion of air as may be desired previous to admission to the respiratory passages.

No further advance could be made upon the somewhat rough apparatus of Snow until the researches of Vernon Harcourt and Augustus Waller afforded convenient methods of estimating the proportion of chloroform contained in an atmosphere. Mr. Vernon Harcourt, with this facility at command, has recently produced an instrument[2] of a similar nature, of which the chief feature is an ingenious means of regulating the temperature of the chloroform. It restricts the vapour to narrower limits, but fails to eradicate the considerable errors due to variations in the respiration and to liability to agitation of the surface of the chloroform.[3]

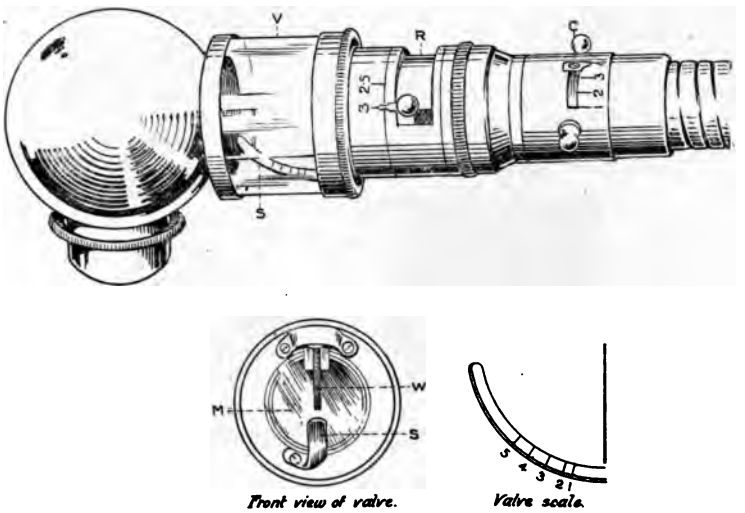
A fairly precise knowledge of the strength of the vapour is desirable during the critical period of the induction of narcosis, but thenceforth the aim of the administrator should be to restrict the strength of the vapour to the lowest degree that the exigencies of the operation and the individual peculiarities of the patient will allow; this particular strength can only be determined by clinical evidences, and being found, the only essential is that no fluctuations sufficiently gross shall occur to affect the maintenance of that narcosis.

These requirements I have fully provided for. Such inaccuracies as are liable to occur are comparatively slight, and being mainly inaccuracies of deficit cannot therefore exert any [illegible] effect. Furthermore, by giving [illegible] details, the [illegible] purposes

and the percentages of vapour be determined with considerable accuracy throughout a long operation.

The most disturbing conditions are those which are due to the variable force of the inspirations, and the consequent variation in rate at which the air current traverses the surface of the chloroform; but although these vital conditions are uncontrollable, they may to a certain extent be compensated for. With this end in

FIG. 1.

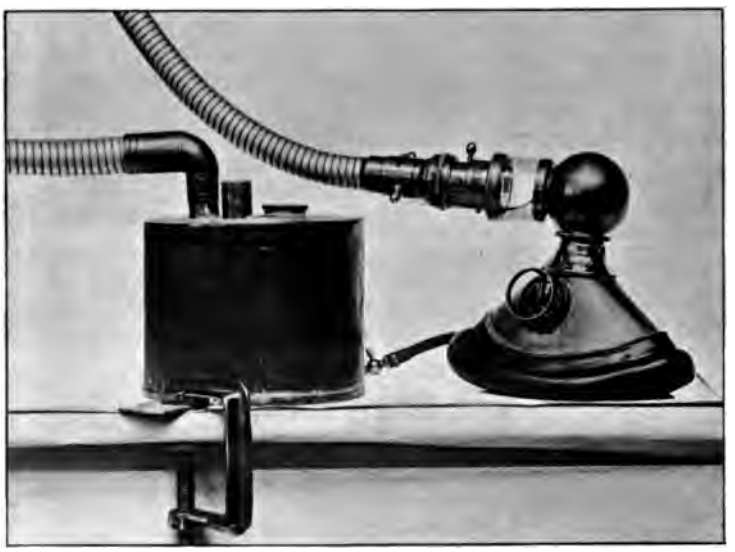


M. Mica valve. W. Spiral spring. S. Valve scale. V. Glass valve chamber. R. Regulator. C. Compensator.

view I register the strength of the inspirations by measuring the swing of a hinged inlet valve constructed of mica (Fig. 1). The hinge is made of oiled silk, and the necessary elastic resistance supplied by a length of thin, spirally-coiled manganin wire. The swing of the valve is measured off on a curved index engraved with marks denoting various degrees of force of inspiration. This contrivance is enclosed in a glass cylinder for convenience of observation.

For the sake of reference I have determined the value of these index marks in terms of the velocity of the corresponding air currents. This was carried out by employing a large glass water aspirator; by timing the outflow between two measured levels the rate of suction of the air occurring at any intermediate level could be calculated. By noting the relations of the swing of the valve to the contemporary levels of the water in the aspirator the value of the index

FIG. 2.



marks in terms of the velocity of the air-currents was determined.

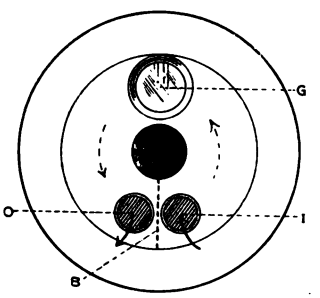
I will now proceed to describe the rest of the apparatus in detail (Fig. 2).

The chamber in which the chloroform is evaporated is annular in form (Fig. 3), of which the outer diameter is 8·2 cm., and the inner diameter is 2·5 cm. At one point it is intersected by a vertical partition, and in the roof, to one side of this, there is a short inlet tube, and a similar outlet tube on the other side.

The chamber is made of thin copper, and it forms part of the cover of a larger vessel of about a litre capacity. This latter is filled with water at 20° C., and serves as a water jacket to regulate the temperature of the chloroform. It is fitted with a clamp for fixing it firmly to the edge of the table upon which it rests.

The chamber is filled with chloroform to within 15 mm. of its roof, and this level is indicated by a metal peg attached to the side wall and viewed through a small glass window. A second peg lower down indicates the evaporation of a definite quantity of chloroform.

FIG. 2.



B. Bulkhead. I. Inlet tube. O. Outlet tube. G. Glass window. The arrows show the direction of the air-current.

The surface area of the chloroform is about 48 sq. cm. The mensuration of each inlet hole is 176 sq. mm., *i. e.* the area of a circle of 15 mm. diameter.

The chloroform chamber is connected with the regulating valves and face-piece by means of a length (equal 90 cm.) of aluminium pressure tubing, which has an internal diameter of 15·5 mm. The viscosity effect set up by the passage of the current of air through this length of tubing produces a measure of resistance, but this is quite insufficient to cause embarrassment of breathing.

The tube is connected at its further end directly to a cylindrical brass mounting which contains a circumferential air-slot of 18 × 2 mm., which may be covered up to any

desired extent by a revolving collar. This device I have termed the Compensator, and its use will be explained later. This mounting is screwed on to the tubular valves which constitute the Regulator. This consists of two cylindrical tubes fitted one within the other, the inner of which can be made to revolve within the outer by moving a lever. The distal ends (away from the face-piece) are blocked by metal discs, each having an equal sector (7—16 of the whole) cut out. In the circumference of the cylinders are cut slits equal also to 7-16ths of a circumference in length, of which the inner is 5·5 mm. broad; the internal diameter of the inner tube is 22·5 mm.

The sectors cut out of the discs communicate with the chloroform chamber through the medium of the flexible tube, and the slits give access to the air. These sets of apertures are so disposed as to act reciprocally, so that when one set is partly open the other is shut in the same proportion; thus the chloroform atmosphere aspired from the evaporating chamber may be diluted to any desired degree.

The lever before mentioned carries an index hand which travels over a scale indicating percentages of vapour. This scale was plotted out by means of a series of experimental estimations.

The air passes from this regulating apparatus through the glass valve chamber previously fully described. The valve also serves to bar the reflux of air during expiration.

A hollow metal globe connects this chamber to the face-piece, into one side of which is inserted a spring valve, which opens during expiration.

The percentage of chloroform vapour inhaled through this apparatus was estimated according to the method of Waller and Geets,[1] which consists of weighing a sample of the mixed gases in a counter-balanced glass bulb. By

[1] 'Brit. Med. Journ.,' June 30th, 1903; and 'Proc. Physiol. Soc.,' December, 1903.

paying due attention to detail, accurate results are by this means rapidly obtained.

For purposes of estimation I resorted to natural respiration through the face-piece, voluntarily controlling the inspirations to the various grades of strength marked on the valve scale, and thus the experiments were performed under conditions such as occur in actual practice. I availed myself in this connection of a device of Mr. Vernon Harcourt's of interposing a capacious air chamber between the face-piece and the sample bulb, thus avoiding the actual inhalation of a strong vapour.

It is necessary here to point out that the water bath does not maintain the chloroform at the same temperature under all conditions of breathing. Owing to its low specific heat, at the commencement of respiration the chloroform cools by reason of its evaporation very rapidly, and its temperature only becomes constant at from 2° to 7° C. below that of the water bath. The point of equilibrium of exchange varies according to the volume of air passing over the chloroform. When working out estimations, therefore, the chloroform had first to be brought to a steady temperature by taking a sufficient number of respirations, viz. from 30 to 50.

The effect of varying the rate of frequency of the respirations appears to be complicated, but the general result of increasing the rate is a slight diminution of percentage and *vice versâ*. However, the difference is not of grave importance, and I have adopted a uniform rate of 30 respirations per minute, which is the average rate under conditions of chloroform anæsthesia.

The marks on the valve scale correspond to the following rates of inspiration and their clinical expressions:

5	equals	870	c.c.	per	second	(forcible breathing).
4	„	690	„	„	(strong „	).
3	„	470	„	„	(moderate „	).
2	„	370	„	„	(weak „	).
1	„	260	„	„	(very weak „	).

There exists a great difference between the percentages of chloroform vapour taken up by the air when travelling over the surface of chloroform at the extremes of rate of flow. Thus, when the air slot of the regulator was completely closed the following results were obtained.

Velocity of air.		Percentage of vapour.
870	...	2·4
690	...	3·0
470	...	3·4
370	...	4·0
260	...	4·9

The important differences in the percentage of vapour corresponding to the various rates of the air-current have in the present instance been combated and brought to a common reading by means of the adjustable slit which I have styled the compensator. But it is only necessary to employ this in the series of higher percentages, 2·5 per cent. and over. At lower percentages than this the compensation is carried out automatically by reason of the viscosity effect upon the air-currents in the long connecting tube affecting more powerfully the low-pressure currents. As is well known, the flow through an aperture is approximately proportional to the square root of the driving pressure, whereas viscous flow, as flow through a long tube, is directly proportional to the pressure. Thus in weak breathing the current through the chloroform tube is relatively retarded and a proportionately larger amount of pure air is consequently admitted through the air slit of the regulator. A slight amount of over-compensation is unavoidable in the case of very faint aspirations. Such inspirations indicate either an obstructed breathing or an excessive degree of narcosis, so that it is an actual advantage to have the chloroform automatically reduced in strength.

TABLE I,

Showing the percentages obtained under varied conditions of inspiration up to 2 per cent.

	Breathing.	Index of regulator. 0·5	1·0	1·5	2·0 per cent.
IV.	Strong . .	0·5	1·0	1·4	1·9
III.	Moderate .	0·46	1·0	1·6	2·0
II.	Weak .	0·46	0·9	1·5	2·1
I.	Very weak .	0·3	0·8	1·3	1·9

At the 2·5 mark and over the automatic compensation progressively fails and the compensator has to be used. The rule for the use of this is simple: as the breathing sinks in force, the pointer of the compensating air-slit is adjusted to the number corresponding to that indicated on the valve-scale by the swing of the valve. The estimations then work out as follows:

TABLE II,

Showing percentages obtained with the index indicating from 2·5 to 3·5 per cent.

	Breathing.	Compensator.	Index of regulator. 2·5	3·0	3·5
IV.	Strong.	IV (shut) .	2·5	2·8	3·0
III.	Moderate.	III (4 mm.) .	2·6	2·9	3·4
II.	Weak.	II (12 mm.)	2·6	3·1	3·4
I.	Very weak.	I (18 mm.)	2·3	2·9	3·5

Here I have as far as possible purposely over-compensated the lowest value of breathing.

In the case of forcible breathing the percentage will be lower than that indicated by the index. Such forcible respirations only occur under somewhat exceptional circumstances, and need not be seriously taken into account. The following values are given for the sake of reference:

Index of Regulator.	0·5	1·0	1·5	2·0	2·5	3·0	3·5
Breathing forcible (V)	0·5	1·0	1·3	1·65	2·0	2·3	2·4

The use of the compensator is largely restricted to the period of induction, but if required thereafter its employment causes little inconvenience; it generally requires little adjustment, for the breathing as a rule settles down to a pretty constant strength.

There are certain inaccuracies to which this instrument is liable, and which require further comment.

Any agitation of the chloroform-container is apt to raise the percentage of vapour considerably. The vessel is therefore invariably clamped to the edge of the table upon which it rests.

The sinking of the level of the chloroform in the course of time causes a progressive decrease in the vapour evolved. Table III shows a series of readings to illustrate this point. The experimental conditions with the exception of the difference of the chloroform level were the same in each case.

TABLE III.

Amount of chloroform.	Percentage obtained.
2 ounces, normal level, 15 mm. from top	2·02
1·5 ounces, 18 mm. from top . .	1·97
1 ounce, 21 mm. from top . .	1·82
0·5 ounces, 24 mm. from top . .	1·7

Therefore if it be desired to maintain the accuracy of the readings after the evaporation of about half an ounce, the anæsthetic must be replenished.

The cooling of the water bath has also some effect upon the percentage of vapour. The water bath loses its heat but slowly, the external temperature when low being principally responsible for the loss. Some operating rooms have a temperature of 20° C., and then the loss is entirely negligable, for the conversion of one ounce of chloroform into vapour abstracts sufficient heat from

one litre of water to cause a fall of temperature of 2·5° C., but as the evaporation of this amount would take about an hour its influence is entirely obscured by the more important interchange with the external air.

If the room temperature be below 20° C. the water bath in time falls to an equality. A fall in temperature from 20° C. to 17·5° C. (63·5° F.), the lowest temperature likely to be experienced in an operating room, results in a decrease of the percentage from 2·0 to 1·85. Under such circumstances it would be necessary, for purposes of scientific accuracy to restore the temperature of the water bath by the addition of a little hot water.

For the first half hour the errors are very slight indeed. After a considerable lapse of time, and in the absence of the above-mentioned precautions, such an unfavourable combination of conditions may arise as would result in a decrease to 1·66 per cent. from an indicated 2 per cent. Such a deviation from the indicated percentage is, I believe, a maximum, but it develops slowly, and at a late stage of an operation even this is not, I think for practical purposes, of great importance; for then, as I have already pointed out, the main requirements of the anæsthetist are the capacity to dilute the chloroform to a minimum point of efficiency, an assurance of the absence of fluctuations, and a rough approximation of the actual percentage administered.

In use anæsthesia may be completely induced by this instrument in the average time of seven and a half minutes, proceeding gradually from a 0·5 per cent. mixture to a 3 per cent. or 3·5 per cent. This period is frequently uneventful, but is sometimes accompanied by excitement. When narcosis is complete, the conjunctival reflex being just, or not quite, abolished, the regulator is at once put back to the 2 per cent. or 2·5 per cent. mark, and is subsequently manipulated according to clinical indications. One, the most important of these evidences, is the respi-

tion, and an accurate measure of its force is rendered

he valve scale.

Attention must be paid to the avoidance of mechanical respiratory obstruction, and herein lies the only common difficulty which is likely to be experienced.

The strength of vapour is progressively reduced as time passes, and in the later stages of a long operation it may be very much attenuated.

It must, however, be remembered that very weak vapours are potent by reason of their continuous administration, and any intermittence may result in unexpected recovery.

A final word may be said in regard to the 3·5 per cent. limit; this is barely sufficient for the complete *induction* of anæsthesia, but it is certainly amply sufficient to produce muscular relaxation in the course of any class of operation, and I may, in this connection, add that a 2·5 to 3 per cent. vapour is frequently required in the earlier stages of most operations. A 3·5 per cent. vapour may be employed with confidence. Wrongfully, that is to say persistently, used, it will, in time, produce profound narcosis in many subjects, never sudden, but possibly occasionally dangerous. But this constitutes a wilful misuse of the instrument, for which there can be no excuse. The only danger of a moderately strong vapour lies in its unwitting use, and against such an accident I have, I believe, introduced every safeguard.

I have to express my thanks to Professor Vaughan Harley and Professor Starling and Dr. Waller for the laboratory facilities they have afforded me, and to Mr. W. Legge Symes for the practical interest he has displayed by revising the percentage scale of the instrument.

A NEW APPARATUS FOR THE DELIVERY OF CHLOROFORM AND AIR OF SUITABLE AND EASILY GOVERNED STRENGTH.

BY

A. D. WALLER, M.D., F.R.S.

Read November 22nd, 1904.

THE present apparatus for the delivery of chloroform and air will be readily understood from the accompanying photograph, representing the manner in which the administrator can test any given mixture on his own person before proceeding to its clinical application.

The apparatus consists of a three-wick lamp containing liquid chloroform, to be sucked by the wicks, and vapourised in the mixing chamber, into which air is pumped by an ordinary foot bellows as used by glass-blowers.

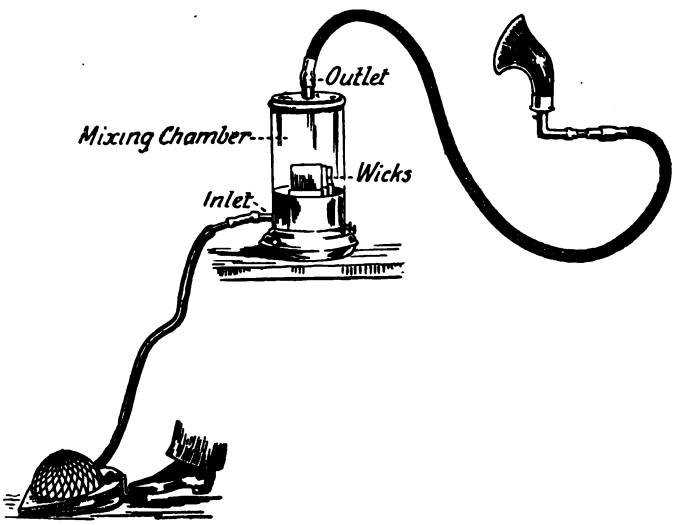
The volume of air supplied by the bellows is determined by an appropriate stop, of which the orifice has been adjusted to give with the bellows reservoir at ordinary tension the desired volume of air per minute—12 to 15 litres in the case of the human subject.

The volume of chloroform vapour taken up by the air is determined by the area of wick exposed and the volume of air supplied to the mixing chamber—in the case of the human subject a wick-area of 100 cm.^{2} obtained by turning up two of the three wicks was found to give a mixture of

about 2 per 100, with an air supply of 10 to 15 litres per minute.

The face-piece, in the case of the human subject, need not be closely applied, and is provided with orifices of overflow, the principle aimed at being to supply to that face-piece a constant flow of chloroform and air in liberal excess of the normal volume of inspired air.

For use in the laboratory, the mixture is pumped into



Apparatus for delivery of mixtures of air and chloroform vapour at percentages between 1 and 3 and quantities between 10 and 15 litres per minute.

the space in which the animal is confined for the purpose of inducing anæsthesia, and if it be required to maintain anæsthesia for a prolonged period, it is easy to work the pump by a motor and to reduce the strength of anæsthesia by lowering the wicks.

During operation upon [illegible] anæsthetic is administered in the [illegible] cannula, in which case the [illegible] pump is removed and the [illegible]

The method of estimation of the percentage depends upon the relative density of chloroform and of air.

Taking 1000 c.c. of $CHCl_3$ as weighing 5·325 grms., and 1000 c.c. of air as weighing 1·293 grms., the difference is 4·032 grms.; or, as a round number, 4 milligrammes per 1 c.c. So that with a 250 c.c. bulb 2·5 c.c. of chloroform vapour—*i.e.* 1 per 100—replacing an equal volume of air, give an increment of weight of 10 milligrammes; or briefly, each unit of chloroform vapour per cent. of chloroform + air is represented by a centigramme increase of weight; *e.g.* a 250 c.c. bulb filled with air and counterpoised is found to be 18 milligrammes heavier when filled with a mixture of chloroform and air, indicating a chloroform percentage of 1·8 per cent.

The first weighing is controlled by a second weighing, with the bulb washed out by air, and if thought desirable twin weighings are taken of two bulbs in series. For the present purpose it is sufficient to weigh the nearest milligramme, and it is not necessary to correct for temperature and pressure. A temperature of 18° is allowed for once for all by taking a capacity of rather more than 250 c.c.[1]

The wick apparatus examined by this method gave the following results, which ought to be sufficient to indicate that the apparatus in question affords a very convenient and easily-regulated means of anæsthesia—(1) for laboratory requirements; and (2) for clinical use. With a little experience of the apparatus it is easy to adjust the chloroform delivery to the requirements of an animal or patient by raising or lowering wicks. The administrator soon learns by his sense of smell the average numerical values of the percentages of the mixtures he considers to be suitable.

[1] The direction to the glass-blower is for bulbs of between 260 and 270 c.c. The percentage taken from cgrams. is checked if desired by the logarithmic formula: log. % = 1·8391 − log. V. + log. M. − log. B + ... serves to show that the direct reading ... sufficient.

The chief desideratum as regards administration to the human subject is uniformity; fluctuations of chloroform in-take from "much" to "none" and *vice versâ* are very objectionable. There is, however, no undue irregularity of in-take if, instead of reducing a quantity judged to be more than sufficient by lowering of wicks, a diminution of in-take be effected by regular and frequent removal of the face-piece for one or two chloroform-free respirations.

A first test of the delivery of the apparatus at ordinary room-temperature, with varying air-current and varying wick area, gave the following results:

Air-current.	1 wick (50 cm.²).	2 wicks (100 cm.²).	3 wicks (150 cm.²).
6 litres per min.	1·3%	2·6%	2·9%
12 „ „	1·1%	2·0%	2·2%
24 „ „	0·8%	1·5%	1·7%

With the above amounts the absolute volumes of chloroform vapour delivered to the mask were therefore

78 c.c.	156 c.c.	174 c.c.
132 c.c.	240 c.c.	264 c.c.
192 c.c.	360 c.c.	408 c.c.

And if we reckon a duration of inspiration equal to one half that of inspiration plus expiration, and a total volume of inspiration of six litres per minute, we obtain, as an approximate estimate of the corresponding volumes of chloroform vapour actually inspired:

39 c.c.	78 c.c.	87 c.c.
66 c.c.	120 c.c.	132 c.c.
48 c.c.	90 c.c.	102 c.c.

This last series of figures is obviously one in which there must be a considerable marginal allowance. The assumption that with inspiration amounting to a total volume of 6 litres and a total duration of ½ minute per minute, the volumes inspired are one half, one half, and one quarter the volumes delivered to the mask, cannot be strictly justified. Nevertheless, the calculation is useful

as showing that the fluctuations of chloroform inspired are not proportional with fluctuations of air-supply, and that accidental fluctuations of the latter may practically be disregarded. I estimate that the fluctuations of chloroform inspired with an air-supply raised from 12 to 24 litres per minute or lowered from 12 to 6 litres per minute do not exceed 25 to 30 per cent., and that an accidental fluctuation of 2 litres per minute is without sensible prejudice to uniform anæsthesia.

Obviously, however, care and watchfulness are necessary in this as in any other method; the anæsthesia is to be pressed or relaxed according to symptoms; more or less wick area is to be exposed as more or less anæsthetic is required.

A second test made to determine whether the delivery would sensibly diminish by reason of cooling by evaporation gave the following values:

Two wicks. Air-current = 24 litres per min.
At the 5th minute the percentage was = 1·4.
„ 25th „ „ „ 1·5.
„ 40th „ „ „ 1·4.
„ 55th „ „ „ 1·3.
„ 70th „ „ „ 1·2.

A third test made to examine the influence of the temperature of the chloroform gave the following:

Two wicks. Air-current = 12 litres per min.
At 8 to 10° the percentage was 1·4.
„ 20 to 22° „ „ 1·9.
„ 24 to 26° „ „ 2·0.

The percentage and absolute amount of chloroform inspired, although of the utmost importance in what may be termed the "institutes of anæsthesia," are of secondary importance in the practice of anæsthesia. The numerical scaffolding is essential to the study, discussion, and teaching of the principles of anæsthesia. In the absence of numerical measurements we have no right to assert that chloroform is not an uncertain drug; that for the ordinary should be continuously exhibited

in a dilution of between 1 and 2 per 100 in air. But for practical purposes the measurement of chloroform percentage during an administration would be a cumbrous and dangerous impediment. All that an instructed practitioner will care to do towards measurement will be to take a whiff of the mixture he is exhibiting and recognise the familiar pungency of 1, 2, and 3 per 100, and not run the risk of suddenly flooding the pulmonary sheet of blood of an unconscious or gasping patient by something between 5 and 10 per 100.

As regards the particular form of apparatus now presented, there are in my experience two simple precautions to be observed :

1. The wicks should be dry.

2. The mixing chamber should be flushed out by a few strokes of the bellows before the mask is applied to the patient's face. This preliminary precaution will more certainly have been secured if the adminstrator observes the practice of himself testing the mixture by the momentary application of the mask to his own face when he is about to exhibit.

A NEW APPARATUS FOR THE DELIVERY OF CHLOROFORM AND AIR OF SUITABLE AND EASILY GOVERNED STRENGTH.

BY

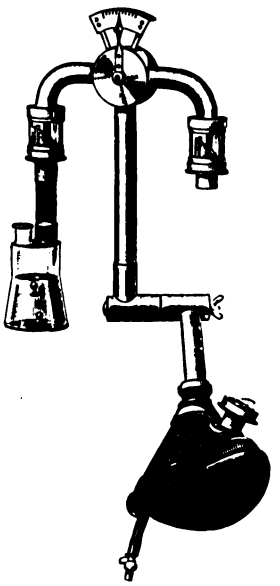
E. WILLETT, M.D.

Read November 22nd, 1904.

I HAVE been asked to describe the chloroform regulator invented by Prof. Vernon Harcourt, F.R.S. The apparatus is very ingenious and fairly simple, the idea being to give a mixture of air and chloroform in known quantities up to a maximum of 2 per cent. of chloroform.

It consists of a face-piece fitted with an air-cushion, attached to a hollow metal frame, terminating by means of a T-shaped junction in two apertures or inlets, each of which is fitted with a carefully-balanced valve to *admit* air only on inspiration: at the junction is an indicator or arm so arranged that when the indicator is completely pushed over towards one inlet the air is admitted at that inlet only; when it is placed midway, air is drawn in equally from the two inlets; below one inlet is suspended a small conical-shaped bottle with two necks, one neck being joined to the valvular inlet, the

other being open to the air; about half-way up the bottle is a zero-mark, below which the diameter of the bottle increases in a regular and known proportion (see below). Chloroform is poured into the bottle up to the zero-mark, and into the chloroform are dropped two small coloured specific gravity bulbs, one red, the other blue, which have been carefully prepared so that the red bulb *floats* as soon as the temperature of the chloroform falls as low as 15° C. (58° F.), while the blue one remains at the bottom until



the temperature of the chloroform sinks as low as, or below, 13° C. (55° F.), when it begins to float also. It has been calculated out carefully that when the red bulb floats and the blue one still remains at the bottom of the bottle, the atmosphere in a bottle of this size and shape does contain *exactly* 2 per cent. of chloroform. When the administration by means of the apparatus first begins, the chloroform being at the temperature of the air (unless previously cooled down), the air in the bottle would contain a per-

centage slightly above 2, but the evaporation of the chloroform causing its temperature to fall, the red bulb, previously at the bottom, quickly rises; when, during the continued administration and consequent evaporation, the temperature of the chloroform falls below 13° C. (as shown by the floating upward of the blue bulb) the required temperature can quickly be re-obtained by grasping the bottle with the hand, when the blue bulb at once resumes its proper position at the bottom.

Between the face-piece, which is fitted with an expiratory valve, and the T-shaped tap and indicator, is a universal joint, which can be fixed in any desired position by means of a screw.

The reason of the conical shape given to the bottle is as follows: as the evaporation of the chloroform proceeds, its surface gets proportionately further off from the orifice of the inlet; if the bottle were cylindrical, this would result in a diminution of the strength of the chloroform vapour, but the slightly increased area, met with as the surface of the chloroform descends, has been calculated out carefully in such a way as to preserve the same density of the vapour, viz. 2 per cent., whatever the height of the chloroform may be.

In certain cases it has been found practically that a 2 per cent. vapour is not sufficient to maintain deep narcosis: to remedy this, a small movable tube or throttle, known as the "*increase tube*" is supplied: by inserting this tube into the open orifice of the chloroform bottle, the percentage can be raised up to 2·5 or 3 per cent. at pleasure.

An objection having been raised by Professor Waller that oscillations or shaking of the apparatus during the course of an operation would raise the percentage of the vapour above the desired amount, I am showing a stand, recently devised by Professor Harcourt, to which the regulator is firmly clamped, connection with face-piece being obtained by means of an india-rubber tube, about 3 feet in length. By this means it appears that

any ordinary movement at the face-piece is preven from reaching the bottle, the surface of the chlorof remaining quite tranquil. I have had no personal perience of this modification.

When using the apparatus it is essential that complete coaptation of the face-piece to the patie face should be maintained, in order to secure a pro working of the valves.

AN APPARATUS FOR THE DELIVERY OF CHLOROFORM VAPOUR OF KNOWN DILUTION.

BY

B. J. COLLINGWOOD, M.B., B.C.CANTAB.

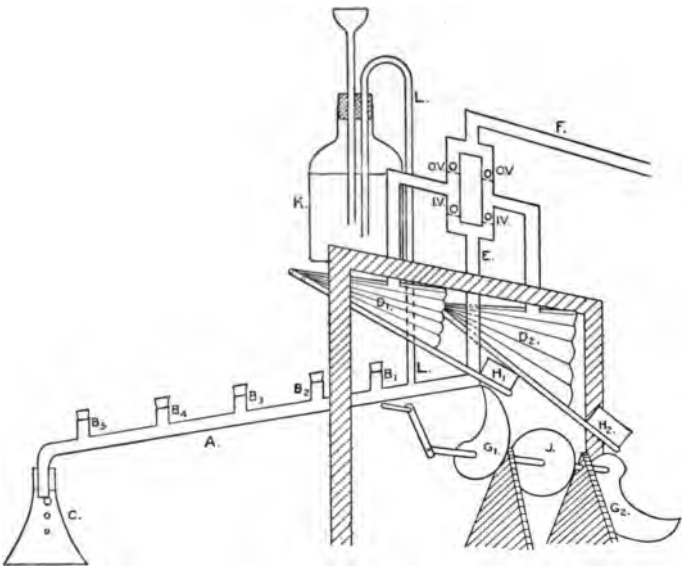
Read November 22nd, 1904.

IT has been clearly shown by recent observations that the chief danger in the ordinary methods of administering chloroform is due to the indefinite, irregular, and frequently excessive percentage of chloroform vapour that is being given to the patient. The paramount importance of the exact dosage of the drug has been of late strongly urged. ("Report of the Special Chloroform Commission of the British Medical Association," 'British Medical Journal,' 1902; "On the Dosage of Chloroform," by A. D. Waller, F.R.S., 'British Medical Journal,' April 23rd, 1898.)

The apparatus presented for criticism is intended to put into the hands of the anæsthetist the power of regulating the percentage of chloroform vapour to a greater point than, I believe, has hitherto been achieved.

The method consists of drawing air up an inclined tube down which a fine stream of chloroform is con-

stantly running. There are a series of inlets (B_1, B_2, B_3, B_4, B_5) along this tube which can be opened or closed at will. The amount of chloroform taken up by the air is regulated by opening one or other of these inlets. In this way the air is made to pass over a longer or shorter stream of chloroform. The inclined tube ends in a vessel (C) which receives all the chloroform which has not been vapourised.



The air is sucked in by two pairs of bellows (D_1, D_2) which act alternately. Each pair of bellows is connected with inlet and outlet ball-valves (I V, O V). The inlets of these valves are in communication by a T-piece with the inclined tube, the outlets in a similar manner leading to the delivery-tube (F).

The bellows are worked by a horizontal shaft on which two cams (G_1, G_2) are fixed, which close the bellows alternately. These cams are of such a size that one or other of the bellows is always being closed.

The opening of the bellows is brought about by weights (H_1, H_2) attached to the handles. Thus the bellows open independently of the turn of the shaft, and always at the same rate. That the rate of opening should be constant is a very important point, for any variation in the rate of intake current would, of necessity, produce a variation in the percentage of chloroform vapour.

In addition to the handle the operating shaft is provided with a grooved wheel (J), so that the apparatus can be worked either by hand or by motor.

The stream of chloroform enters the inclined tube (A) by a syphon tube (L) whose upper end dips in a Mariotte flask (K) containing chloroform. It was found that if a Mariotte flask was not used a rise of chloroform level in the flask of 3·5 centimetres caused a rise of percentage of chloroform vapour from 1·55 per cent. to 1·75 per cent.

The following table will show the constancy of percentage that can be obtained by the use of this apparatus:

Table showing Percentages of Chloroform Vapour obtained in Four Observations with various Inlets Open, and with Forty-eight Turns of Handle a Minute.

Observations.	Inlet No. 1. Per cent.	Inlet No. 2. Per cent.	Inlet No. 3. Per cent.	Inlet No. 4. Per cent.	Inlet No. 5. Per cent.
No. 1	0·85	1·00	1·40	1·60	2·00
No. 2	0·85	1·00	1·40	1·60	2·00
No. 3	0·85	0·95	1·40	1·60	2·00
No. 4	0·85	1·05	1·40	1·60	1·95

N.B.—The figures are correct to 0·05 per cent., corresponding on the scales to half a milligramme.

The method employed for estimating the percentages of chloroform was the densimetric method introduced by Waller and Geets ('British Medical Journal,' June 20th, 1903).

It will be noticed that in these observations the rate of turn of the handle was constant, namely forty-eight turns per minute. The next table shows how small a

difference in percentage is produced by great variations in the rate of revolutions.

Table showing Variations in Percentages of Chloroform Vapour, with Varying Rates of Turn of Handle in Case of Inlets No. 1 *and No.* 5.

Observations.	Inlet No. 1.		Inlet No. 5.	
	36 turns a minute. Per cent.	60 turns a minute. Per cent.	36 turns a minute. Per cent.	60 turns a minute. Per cent.
No. 1 . . .	0·90	0·80	2·15	1·90
No. 2 . . .	0·90	0·80	2·15	1·85
No. 3 . . .	0·90	0·80	2·15	1·90

These variations can be explained by the fact that the longer pause there is between each intake of the bellows, the longer time is given for chloroform to accumulate in the tube. It would thus appear that these small variations might be reduced to still smaller dimensions by using an inclined tube of smaller bore.

In both the above tables the observations were taken about half a minute after starting the apparatus. But further trials show that after five minutes' working there is only a very slight alteration in the percentage, the tendency being towards a slight fall. The inclined tube, therefore, must rapidly reach a constant temperature.

Experimental application.—It has long been known clinically that the longer anæsthesia has been established, the less anæsthetic is required. In this connection some interesting results have occurred when using this apparatus on cats and rats. I am enabled to give approximately the percentages of chloroform vapour required to abolish the corneal reflex after prolonged periods of full anæsthesia. I found that after half an hour 1·0 per cent. was sufficient, that after one hour 0·8 per cent. was required. The percentage gradually sank from hour to hour until after four hours 0·5 per cent. was enough, and after five hours 0·4 per cent. kept the reflex abolished. These figures are only applicable to an animal which has been fully anæsthetised,

for it appears that the amount required at any time is inversely proportional to the amount already given. Thus in one experiment a cat was anæsthetised for five hours, and during nearly the whole of that time it only received 0·6 per cent. of chloroform vapour, the result being that the corneal reflex did not disappear until the fifth hour was completed.

In conclusion, the points which can be urged in favour of this apparatus are as follows:

1. Constancy of percentage of chloroform vapour entirely independent of the rate or depth of the respirations of the recipient.

2. An accurate method of graduating percentage delivered.

3. Economy in chloroform; all that is not vapourised drops into the surplus bottle and can be used again.

4. A knowledge of the amount of air that is being delivered, forty-eight turns of the handle yielding about 8 litres of air.

DISCUSSION ON CHLOROFORM ANÆSTHESIA.

Dr. F. W. HEWITT, in opening the discussion, said that the great question before the Society that evening was, How should chloroform anæsthesia be induced and maintained? The question was an exceedingly important one, for there could be no doubt that by the observance of certain cardinal principles chloroform, the most manageable, but at the same time the most potent, of all anæsthetics, might be robbed of those terrors with which its name had so often been associated. Before proceeding to the consideration of these principles, it was necessary that there should be a clear definition of chloroform anæsthesia. There were certain interesting differences between (*a*) simple and (*b*) complex chloroform anæsthesia. By simple chloroform anæsthesia he meant that characteristic state of deep insensibility which was produced by the administration of chloroform to patients *not* undergoing any surgical operation. By complex chloroform anæsthesia he meant that condition which was characterised not only by the phenomena referable to the anæsthetic itself, but also by the phenomena referable to some surgical procedure which was taking place. The experience of some years had convinced him that it was necessary to realise the differences, often the great differences, between these two states before it was possible to thoroughly understand the nature of the various difficulties and accidents that might arise under this anæsthetic. He asserted that in a large proportion of the cases of chloroform anæsthesia in the operating theatre many of the phenomena customarily attributed to the effects of the anæsthetic were in reality referable to the operation. For example, reflex spasmodic retraction of the tongue, reflex laryngeal spasm, reflex general respiratory spasm, might one and all complicate a chloroform anæsthesia, which, in the absence of the particular procedures liable to induce such reflex effects, would have been uneventful. Thus, slight but long-continued laryngeal spasm, the outcome of prolonged peritoneal manipulation, might insidiously bring about an asphyxial state altogether foreign to simple chloroform anæsthesia. Or, to give an example from the circulatory side, the withdrawal from the abdominal cavity of a portion of intestine during well-established chloroform anæsthesia might be attended by pallor, feeble or arrested pulse, dilatation of the pupil, and in the worst cases separation of the lids and arrested breathing as the result of cerebral anæmia. He then discussed the question,

What were the cardinal principles upon which chloroform should be administered, and to what extent did the various instruments which had been devised carry these principles into effect? Most of the present-day authorities on chloroform anæsthesia placed at the head of this list the avoidance of vapour concentration beyond this or that percentage, some maintaining that 2 per cent. and others 4 per cent. should be the maximum. He held somewhat different views. He would emphasise the necessity of maintaining a free and unembarrassed airway during the introduction of chloroform vapour into the lungs. He had, himself, either tried or seen in use many most admirable appliances for adjusting chloroform percentages, and had observed on many occasions when such inhalers had been in use evidences of intercurrent asphyxia dependent either upon some unrecognised condition in the patient himself, preventing or retarding the free entry or exit of atmospheric air, or upon the narrow channels of the inhaler through which the patient had been breathing. The fact was, that in addition to the fully recognised paralytic effects of anæsthetics upon the respiratory centres there was an important tendency under these agents for the respiration to become intercurrently embarrassed, and it was the full recognition of the conditions which might lead to this intercurrent asphyxia which in his opinion formed the key to the correct understanding of the vast majority of difficulties and accidents under chloroform. The intercurrent asphyxia to which he specially referred might be brought about in a variety of ways and in many cases the evidences of it were so slight, save to the experienced ear, that the immediate cause of the condition was not recognised. The attention of the anæsthetist must be primarily directed to the maintenance of a free airway. The second principle in chloroform administration was the presentation to the patient of an equable and dilute chloroform atmosphere. He had never seen anything in practice to convince him that there was any special danger in a concentrated vapour except as regarded the amount of chloroform absorbed by the circulation from such a vapour. Snow, Clover, and Paul Bert demonstrated the important fact that over-dosage with chloroform might be avoided by the use of dilute percentage mixtures of chloroform vapour and air, but they all failed to explain satisfactorily deaths occurring in light anæsthesia, deaths which even dilute atmospheres of chloroform could not prevent, and which in his judgment were generally referable to intercurrent asphyxia. Whilst there were undoubtedly certain advantages in employing a percentage inhaler, there were also certain practical disadvantages which could not be lost sight of. Such inhalers were applicable in only a certain number of cases, so that even if their advantages were greater than they were one would still have to teach students simpler methods. They were comparatively cumbrous, complicated, and liable to

get out of order. They occupied the attention of the anæsthetist, which should be wholly directed to studying the special effects produced by chloroform in the patient before him. They were liable to become foul with blood or other fluids during operations, and were incapable, as a rule, of being sterilised. Certain of them were open to the grave objection of imposing a suction action upon the patient, involving a greater or less asphyxial element in the administration. In certain of them, again, the composition of the chloroform atmosphere was liable to considerable fluctuations if the inhaler were suddenly moved, this being in certain circumstances a grave objection. In all of them the induction was slow, many patients complaining of this in practice. In most of them the anæsthesia was not such as to satisfy all surgeons, being distinctly light in type, so that inconvenience, possibly of a dangerous character from a surgical standpoint, might be experienced. In hospital practice in which patients were wheeled from an anæsthetising room to a theatre, it was difficult to prevent partial recovery when using these inhalers. To the man of experience a percentage inhaler was probably useless, for after a few hundreds of administrations he was able to use any simple appliance such as a Skinner's mask in such a way as to keep up an equable vapour of the requisite and proper percentage. To the inexperienced man a percentage inhaler was complex and required a scientific attitude of mind which was not always to be found. Physiologically and academically, it was instructive to analyse the phenomena of chloroform anæsthesia, so to speak, by inhalers such as those of Dubois and Waller, which were free from the objection of suction action to which he had referred, and with them to be able to demonstrate to students the different stages of chloroformisation. But Lister, Waller, and Symes had shown—and his experience enabled him to endorse their views—that with a small flannel mask and a drop-bottle a dilute and equable chloroform atmosphere might be presented to the patient, and that with such appliances it was difficult to administer dangerously strong chloroform atmospheres. Indeed, with a small Skinner's frame and with a drop-bottle so adjusted that it was difficult to drench the absorbent surface, it was questionable whether there was any greater risk in administering chloroform than in using and attending to the working of a regulating inhaler. The third and last great principle in chloroform administration was to adjust the level or depth of the anæsthesia in each patient so as to obtain the proper conditions suitable for the particular operation without imperilling the safety of the patient. There was in each patient a particular degree of anæsthesia which should be maintained, although it was often difficult to discover the neutral line, so to speak, separating the too light from the too deep anæsthesia. In his endeavour to strike this neutral line, the anæsthetist must

not only be guided by the personal equation of his patient—some subjects requiring drops, whilst others required drachms—but by the nature and duration of the particular procedure in hand. There were undoubtedly many advantages from the point of view of the anæsthetist in what was now termed moderate anæsthesia. Provided the anæsthetist was alive to the absolute necessity of maintaining a free air-way and that the cornea was kept distinctly sensitive to touch, he questioned whether primary cardiac syncope, such as was feared by many, could ever occur. At the same time, this form of anæsthesia was open to two objections—(1) that it was often exceedingly inconvenient to the surgeon; and (2) that it favoured the occurrence of such reflex respiratory difficulties as those to which he had alluded. As a general rule an endeavour should be made to keep the corneal reflex just in abeyance, and in those cases in which reflex vaso-motor disturbances were prone to occur to lessen rather than to increase the depth of anæsthesia. When once a profound chloroform narcosis had been produced, reflex vaso-motor effects were more liable to occur than in lighter anæsthesia—a conclusion completely opposed to earlier teaching. It was highly probable that the so-called "surgical shock" which was formerly observed when operations were commonly begun during the light anæsthesia was in reality respiratory—*i.e.* asphyxial—in its nature. Up to the point at which the corneal reflex disappeared pulse observations were useless as regards the effects which chloroform was producing. But after this reflex had been lost the pulse should always be observed, for it would not only give valuable indications as to the depth of the anæsthesia, but it also afforded information as to any vaso-motor effects which the operation might be producing. He did not consider chloroform the best agent with which to induce chloroform anæsthesia, and whenever circumstances permitted some other anæsthetic or mixture of anæsthetics should be employed to tide the patient over the induction period. There were various ways in which this might be done, the best of all being to use the nitrous oxide-ether sequence till a point was reached at which soft snoring co-existed with the not yet abolished corneal reflex, and at this point to substitute chloroform for ether. From this point onwards the risk of chloroform was merely the toxic risk, which, with ordinary care, could be altogether avoided, the chief risk—that special to the excitement stage—having been entirely eliminated. In the ordinary run of surgical cases there was no better sequence than the nitrous oxide-ether chloroform sequence. Nitrous oxide was a boon to the patient because by its means consciousness was rapidly and pleasantly abolished; the nitrous oxide-ether sequence completely prevented all struggling and excitement and in two or three minutes rendered the patient fit for the change to chloroform; whilst chloroform itself was

eminently adapted for maintaining that form of anæsthesia which was most suitable from a surgeon's point of view and least likely to be followed by dangerous sequelæ. If too much ether was given it was not an easy matter—indeed, it was sometimes hazardous—to change to chloroform; but if the change was effected at the particular juncture described a smooth and satisfactory result followed.

Sir LAUDER BRUNTON observed that a careful distinction must be made between deaths from chloroform and deaths during chloroform anæsthesia. He had seen two deaths on the operating table; one was from shock in a case where chloroform could not be given, and the other was in a patient just beginning to recover from its effects. Chloroform was one of the most universal poisons known, and in sufficient concentration would destroy every living thing, from the lowest bacteria at work, and every living tissue. When introduced into the lungs of a rabbit by artificial respiration it would stop the heart very quickly, while ether left the heart unaffected. Notwithstanding this marked action on the heart, which he was accustomed to demonstrate to his class in St. Bartholomew's Hospital more than thirty years ago, he thought that the chloroform given by inhalation and not by insufflation paralysed the respiration before the heart. He considered that an apparatus for inhalation might be very useful as maintaining a more steady anæsthesia and preventing excess in inhalation; but he thought that such apparatus must always remain of limited application and that the successful administration of chloroform would always remain a question of knowledge and care on the part of the anæsthetist, and that his success would largely depend upon his following rules, in maintaining anæsthesia steadily and thoroughly until the operation was over, so as to avoid risk from shock by the operation, shock by the application of too concentrated a vapour to the patient's respiratory passages, and shock, spasm, and suffocation from inhalation of regurgitated food. But care was also required in regard to the position of the patient, in order that syncope might not be produced by the head being unduly raised, or suffocation induced by interference with the air passages or movements of the chest and abdomen.

Dr. DUDLEY W. BUXTON said that the main interest of the discussion centred in the question of giving known percentages of chloroform. He thought the reflex interference with respiration during induction to which Dr. Hewitt alluded was due in most cases to an excessive strength of chloroform. When the Vernon Harcourt inhaler was used these interferences seldom, if ever, occurred, as a continuous weak but increasing strength of vapour could be employed. It was, of course, most essential that any mechanical inhaler should be regarded merely as a help to the person giving the anæsthetic; he must know the condition

of his patient, the requirements of his patient, and could then, by graduating the dose to a suitable level, often save narcosis. A further important use of such inhalers was that the administrator could achieve accurately the requisite depth of narcosis in different operations—some needed a light and some a profound narcosis. In prolonged operations with severe shock the use of low percentage vapours often saved life by lessening the fall of blood pressure, since this fall went *pari passu* with the strength of the chloroform inhaled. In what Dr. Hewitt had called complex anæsthesia an adequate but light anæsthesia was safer than a profound narcosis, as Embley and Martin's work had shown that vagal inhibition, although dangerous or even fatal in profound narcosis, was of comparatively slight importance in slight narcosis. A prolonged use of the Vernon-Harcourt inhaler had convinced him of its value, and that its use made the administration of chloroform comparatively safe, certainly safer than when a method which supplied indefinite percentages of chloroform was employed.

The discussion was adjourned.

Resumed Discussion, December 13th, 1904.

Sir Victor Horsley, in resuming the discussion, said he was interested in the administration of chloroform, especially as he had been responsible for the appointment of the Committee of the British Medical Association. He thought the question of the administration of chloroform was really the question of the administration of a drug in known doses, and the suggestion that the personal attention of the anæsthetist without this knowledge was sufficient was a reactionary suggestion. He believed that this neglect of dose had been the cause of death in some cases, an excessive dose having been inadvertently given. The liminal point was probably the same for all adults; it was not minims for one person and drachms for another. For the induction period it was not necessary to go beyond a 2 per cent. vapour, and this might be inhaled for a long time without risk to life. This opinion was derived from his personal use of the Harcourt inhaler. The liminal point for the maintenance of anæsthesia was, however, different: as soon as the skin and superficial tissues were divided $\frac{1}{2}$ per cent. was enough, and this was quite safe. To this end the use of a regulator (which had been a revelation to him) was essential. A Skinner's mask and drop bottle even in skilful hands gave an inconstant administration which, he believed, might be the cause of death. An atmosphere might be suddenly presented of 4 per cent., which was certainly dangerous. The first danger in point of time, he believed, was from the heart and not from the respiration. The respiratory failure, which is the commonest

condition observed, occurred, as a rule, in the course of prolonged anæsthesia. For regulating the dose any method was better than the open method. The intercurrent asphyxia alluded to by Dr. Hewitt did not occur in chloroform anæsthesia unless, as he believed, the patient had had too much chloroform. Reflex spasms produced by operative procedure he had never seen, excluding operations involving the vagus nerve, although he had often seen asphyxia from too much chloroform. The tongue he had often seen fall back from too much chloroform. Reflex vasomotor phenomenon had never been demonstrated; the fall of blood-pressure was probably due to a primary effect on the heart. The influence of the rate of respiration was of little importance clinically, because when the patient began to breathe slower he also breathed less deeply, and, therefore, inhaled less chloroform. Stimulation of a sensory nerve, as the sciatic, caused a rise in blood-pressure which was of benefit to the patient. Clinically chloroform produced a fall of blood-pressure and of respiratory efficiency proportionate to the dose. The statement that deaths occurred during light anæsthesia and were unpreventable was not proved, because there was no record of the dose which was really being administered; he believed that if a constant percentage were given they would cease to occur. It was a dangerous statement to make that death might occur in light anæsthesia as tending to lead the inexperienced to give more chloroform than was necessary. Deaths occurred from a too high percentage being given by the open method. He had little doubt that some form of chloroform regulator would before long be almost universally adopted by the profession.

Dr. LAWRIE said: The numerous and one-sided discussions on the dangers of anæsthesia, which have been so frequent of late years, have given the profession and the public a morbid dread of chloroform, which has seriously restricted its advantages. One result of this astonishing policy is that anæsthesia specialists are about the worst paid body in the profession, which would assuredly not be the case if his principles had been adopted. No substitute for chloroform has yet been discovered, and for every case of anæsthesia at present there would be hundreds if his views were put into universal practice. The point which had impressed him most in the present discussion was the entire absence of any reference, even by Sir Lauder Brunton, to the work of the Hyderabad Commission on Chloroform, or to the experiments of Drs. Gaskell and Shore. His Highness the Nizam of Hyderabad spent a huge sum of money upon the chloroform commission, as well as upon the experiments of Gaskell and Shore, solely in the interests of the profession. The principal value of the Hyderabad Commission's experiments consisted in the work of Dr. Bomford, now Surgeon-General with the Government of India. In all the manometer experiments

every incident that occurred throughout each experiment was noted by Bomford on the tracing itself as the drum revolved, with the result that the Commission obtained a unique record of facts. The tracings of all the experiments, together with Dr. Bomford's invaluable notes, were photographed, and copies of all the photographs were presented by the Nizam's government to the leading universities and medical societies in Europe and America. He would like to know how many of those present had studied them, and whether the younger members of the profession had ever heard of them. Dr. Lawrie went on to say that the general results established by the Hyderabad Commission were illustrated by his diagram (exhibited) of an uncomplicated chloroform administration, the original tracing of which he handed round. The Commission proved that diluted chloroform, provided it is not too dilute, and the patient's or animal's breathing is regular, gradually causes an accumulation of the anæsthetic in the blood sufficient to produce first unconsciousness, which, if he only knew it, is the golden region for the anæsthetist, then narcosis, then cessation of the respiration, and finally death from stoppage of the heart. A higher concentration can only bring about the same sequence of events in a shorter time, and if the concentration is so excessive as to cause struggling and holding of the breath, the animal becomes more or less asphyxiated, and gasps in irregular overdoses which kill him with still greater rapidity. Incidentally he might mention an extraordinary clinical fact. The excessive concentration, which in some cases leads to rapidly fatal overdosing, in others conduces, in a way which nothing else can, to the regular breathing, by which alone safety is insured under chloroform, and then the highest concentration is every whit as safe in the induction stages of anæsthesia as the lowest concentration. To give chloroform safely, what is required? In the first place, it is absolutely indispensable to realise that chloroform has no direct action on the heart. On this essential principle the administration is commenced at the point marked (1) on the diagram, and continued as far as the point (*a*), if unconsciousness alone is desired, or the point (*b*) if complete anæsthesia is necessary. During the production of unconsciousness or anæsthesia the duties of the anæsthetist are twofold, and they demand the whole of his skill and attention. In the first place, he has to maintain regular respiration throughout, and in the second place, he has to remove the chloroform when the patient has had enough. If the Hyderabad rules are adhered to "accidents" under chloroform are impossible, since the region of danger, shown in the diagram in red, is never, under any circumstances, entered, and the chloroformist has the patient and the anæsthetic at all times entirely under his control. No form of inhaler can relieve the anæsthetist of his responsibilities, and, as Dr. Hewitt has pointed

out, an apparatus introduces an element of danger into the administration which is exactly proportioned to the amount of skill and care required for its management. In conclusion, Dr. Lawrie said he believed many present would agree with him that the work of the Hyderabad Commission on Chloroform had not yet received the attention to which it is entitled, and he expressed the hope that the Society would be pleased in the near future to appoint a committee of impartial men to examine and report upon it, with special reference to the part played on the Commission by Surgeon-General Bomford.

Dr. PAUL CHAPMAN said that neither on the first evening of this discussion before the Society, nor at Oxford, when he not only explained its mechanism in Balliol College Hall, but gave a practical demonstration of its use before the surgical section of the British Medical Association, had Dubois's apparatus met with the fair criticism which it deserves from those engaged in experiments on the production of percentage vapours. Seeing that Dubois's apparatus supplied the requisite and definite percentage vapours in any quantity, and that the percentage of chloroform vapour was not variable through any carelessness on the part of the administrator, he was at a loss to explain the reason why criticism of the apparatus was avoided. Dubois's apparatus really did furnish constant percentage vapours, and independently of any action on the part of the patient. To introduce the patient into the mechanism, and make him furnish the motive force by which the percentage of the vapour he is inhaling is produced, seemed to be a wrong principle at the very outset. It is not unfair to say, for the fact is patent, that experiments have been made, and are being made, by those who use apparatus constructed like those of Professor Vernon Harcourt and Dr. Levy on the suction principle, in which the patient himself is actually part of the mechanical apparatus which it is desired to test. Any use of a machine of which the patient furnished the motive power by his own inhalation, and so became an integral part of the machine, was an unnecessary mechanical experiment. They were experiments upon the patient, and he thought all such methods were very dangerous and wholly unjustifiable. Had no deaths occurred during the use of Professor Vernon Harcourt's apparatus? He had seen none reported, but he should like to have a distinct reply to this question. If they had occurred, no explanation or discussion of side issues could evade the unpleasant fact that experiments were being carried on, with a patient as part of the machine, to examine into the value of the other parts of the machine. He thought such experiments should not be carried on. The Dubois apparatus did not depend on such a plan. The vapour was passed by propulsion through a chamber with free exit. No obstruction whatever was afforded to the patient's breathing.

Absolutely definite percentages could be supplied by this method. It was difficult to get more than a 2 per cent. vapour, and impossible without active and rhythmical interference on the part of the operator. It was not possible by any overzeal to increase the percentage of chloroform vapour. The apparatus could easily be used without assistance; the turning of the handle became a purely automatic act, in the same way that walking was an automatic act. He had used the apparatus in every possible case, using no selection, and had never had any anxiety of any kind arising therefrom. It really did what it professed to do, and was absolutely satisfactory in every way.

Dr. DUDLEY W. BUXTON, speaking as Secretary of the British Medical Association Chloroform Committee, said he had never met with or heard of any deaths due to the administration of chloroform with the Vernon-Harcourt apparatus, neither had he seen any symptoms of danger from chloroform while it was being used.

Mr. F. EVE considered that an apparatus preventing the administration of a toxic dose was a great desideratum. The Vernon-Harcourt apparatus, which was the only one of which he had had experience, seemed to him cumbersome and not suitable for some important operations as those about the mouth; it involved fatigue of the inspiratory muscles in long operations. He preferred a skilful anæsthetist using a mask and drop bottle to a mediocre one using the Vernon-Harcourt or any other apparatus. Cyanosis during anæsthesia he believed to be an important factor in producing so-called "surgical shock," and this with other ill effects made him object to the routine use of nitrous oxide and ether as a preliminary to chloroform anæsthesia.

Mr. G. ROWELL said he had had an opportunity of using Dr. Levy's apparatus, which had confirmed his opinion that to obtain the best results in the induction of chloroform narcosis it was commonly necessary to increase the strength of vapour to as much as 3 per cent. Its use also showed that so mild a vapour as 1 per cent. was sufficient to maintain the required degree of narcosis in the majority of cases after a considerable time. Owing to the range of percentages possible with this apparatus, anæsthesia might be considerably more rapidly induced in resistant patients than is possible with other forms of apparatus which do not provide more than 2 per cent. Five minutes was the shortest, and seven and a half minutes the longest period of induction with the apparatus in his experience. The propriety of administering chloroform by means of an apparatus which provides a definite strength of vapour depends upon the assumptions that, as Dr. Waller stated, the effects of chloroform are proportionate to the amount inhaled, and that too strong a vapour is a common source of danger. These

propositions are by no means proven, and the onus of proof lies upon those who advocate the use of these apparatuses. The aim of science is to so master facts that they can be expressed in figures, but in attempting this with the dose of chloroform the physiologist is confronted by the psychological aspects of the question, which at present are, unfortunately, unable to be investigated experimentally. In the human subject the condition of the higher centres of the brain largely affects the question of the dosage of chloroform. One peculiarity of drugs that principally (or early) affect the human brain is the markedly different result that the same dose will produce in different individuals. The notorious variability in the capacity of different individuals for alcohol illustrates this point. The main question in the dosage of chloroform is that of the resistance of the brain centres to the drug. This varies with the following, amongst other factors, far more than with the body weight and strength: conditions of mental excitement, hysteria, terror of the anæsthetic, fatigue, want of sleep, the particular condition of nutrition of the centres at the time of taking the anæsthetic, toxæmia in all its forms, and, perhaps, most important of all, since most variable, the habits of the patient. The administration of anæsthetics is both a science and an art. The art derives little from physiology, and the science is knowledge mainly based upon a long series of clinical observations, and reasonings upon the multitude of symptoms displayed by patients. The only scientific way of administering chloroform is to carefully observe all the patient's symptoms, and to act accordingly. His experience leads him to agree with Dr. Hewitt that in the hands of a well-trained administrator an improperly high percentage of chloroform is not a common source of danger. Interference with respiration, from the large number of causes, reflex and otherwise, through which it may arise, is the commonest cause of danger. This interference with respiration may be the reflex effect of too strong a vapour, but it is not always so. It is often apparently of central origin in the induction period, and often due to the operation. To remedy such inefficient breathing at its earliest manifestation is one of the chief duties of an administrator. This can be less easily done when an apparatus is used. While any addition to our knowledge of the subject is welcome, while it is impossible to predict what the march of science may achieve, even in the direction of administering chloroform by machinery, one is at present amply justified in protesting against a premature, futile, and possibly dangerous, attempt to reduce this, the most complicated question in therapeutics, to a mere matter of weights and measures.

Dr. J. F. W. Silk associated himself with all the opinions expressed by Dr. Hewitt. The death rate from chloroform was something under 1 in 3000; the majority of deaths seemed to

occur at the hands of an inexperienced administrator. He was of opinion that comfort and safety in the administration of chloroform could best be improved by taking greater care in the education of the student, who should have more opportunities of obtaining experience in the administration of anæsthetics before he left the hospital and that the routine use of pure chloroform was to be deprecated. As a substitute he suggested the more frequent employment of some mixture of chloroform and ether, which was certainly safer than pure chloroform, produced a type of anæsthesia hardly to be distinguished from it, and could be given in a very simple form of apparatus. Dr. Silk thought, too, that greater discrimination should be exercised in selecting competent administrators, and that in the interests of the patient it was not wise to accept without inquiry the first person who might offer himself for the task. He was not convinced that it would be of any great advantage to introduce any of the machines for giving chloroform that had been shown. He was of opinion that they were of necessity a great deal too complicated to be likely to supplant simpler methods; that if they did not encourage they certainly did but little to discourage the view that successful administration depended upon the machine rather than upon the administrator. The importance of a knowledge of the exact proportion of chloroform in the vapour was, he believed, overestimated; the susceptibility of individual patients to its action varied so much that nothing could supply the place of individual attention to the particular patient.

Dr. J. BLUMFELD had had personal experience of the Vernon-Harcourt apparatus, the Dubois and Dr. Levy's apparatus. Too much had been claimed for the Vernon-Harcourt. Its disadvantages were the long time taken in the induction of anæsthesia with it, its cumbrousness, and the difficulty of getting a satisfactory condition of relaxation of the patient even with its maximum power. An advantage was the less quantity used by it, leading to a diminution of the after-effects. The true course of improving the safety of chloroform anæsthesia was to improve the education of the ordinary administrator. He did not think that any regulating inhaler would give greater safety in the administration of chloroform.

Mr. C. CARTER BRAINE had tried the Vernon-Harcourt inhaler in fifteen cases. The reason why he had not more to report was due to the fact that he had not been satisfied with the results obtained. A great drawback to its general employment is the inability to obtain anæsthesia within a reasonable time, but given anæsthesia induced, then he found great ease in maintaining it. One case was quoted in which during the administration all the symptoms of an overdose were present. In a later case during transit from the anæsthetic room to the theatre the apparatus collapsed, and about two ounces of chloroform were

upset over the patient's face; in another case the valve regulating the air supply did not work, and thus the whole inspiration passed over the chloroform, with the result that 2 per cent. was being administered, even though the indicator stood at 10 per cent. In six cases out of fifteen he had failed to induce anæsthesia with the apparatus, and had to change to other methods. The need for the so careful application of the face-piece was a disadvantage, and also the fact that the inhaler was frequently in the way of the surgeon or his assistant, even during abdominal operations. Of seven cases in which Dr. Levy's apparatus was used all had been successful, and he much preferred it to the Harcourt inhaler.

Dr. Herbert Scharlieb showed a few of the tracings obtained by Professor Schäfer and himself in a series of experiments investigating the effects of chloroform on animals, chiefly dogs, in the Physiological Laboratory of the University of Edinburgh. Whilst admitting it was impossible to reproduce in a laboratory experiment *all* the conditions that *might* arise in the human subject, he insisted that the facts so obtained only showed that the superadded dangers met with in man demanded the abandonment of guesswork methods of administering a potentially lethal drug, and the adoption of some means of scientifically measuring the percentage of chloroform vapour actually given. The following tracings were then demonstrated: (1) Showed the ordinary result of administering chloroform in as strong a percentage of vapour as is obtainable at the ordinary temperature of a room, viz. a rapid fall of blood-pressure with a marked effect on the respirations. The animal was subsequently recovered by chest compression, which had the advantage of acting on the heart as well as of aerating the lungs. (2) Showed the results obtained under exactly similar conditions, but with alcohol added to the chloroform in the proportion of one part absolute alcohol to nine parts of chloroform, viz. the blood-pressure and the respirations were well maintained. (3) Showed cardiac inhibition as a result of a too strong chloroform vapour, the animal being recovered by chest-compression. (4) Showed the same. The heart stopping before the respiration, and the subsequent spontaneous but ineffectual heart recovery. (5) Showed cardiac inhibition as a result of stimulation of the vagus. There was spontaneous, but ineffectual, recovery of the heart; respiration not recovered spontaneously, but the animal was recovered by chest compression five minutes after the cessation of respiration. (6) Showed the effect of a dose of atropine administered hypodermically prior to the commencement of the chloroform administration. The atropine abolished the arrest of the heart, as was obtained in tracings 3 and 4, and it permitted a diminu[illegible] beat. (7) Showed [illegible]

the inhibition obtained—as in tracing 5—by stimulation of the vagus. These tracings showed the following facts: (1) The danger in chloroform administration is due to the fact that the drug, if administered in too high a percentage, excites the cardial centre in the medulla oblongata and stops the heart, and secondarily the respiration. (2) Exactly the same result can be got during chloroform anæsthesia by artificially stimulating the vagus. (3) The prior administration of atropine (gr. $\frac{1}{100}$—$\frac{1}{50}$) entirely prevents heart-stoppage in both cases (1 and 2), although in both cases the heart-beats are rendered weaker and blood-pressure falls. (Dose of ·00002 gramme per kilo, body-weight administered hypodermically, effect begins to show itself in about 15 to 20 minutes and lasts about three hours.) (4) The addition of alcohol to the chloroform (in the proportion of one part alcohol to nine parts chloroform) greatly diminishes the tendency to produce inhibition and causes both blood-pressure and respiration to be well maintained, even with high percentage of chloroform vapour in the air respired. (5) Chloroform does not produce vaso-dilation but vaso-constriction. It acts directly on the muscular tissue of the arteries. The indications of these results are: (1) The prior administration of gr. $\frac{1}{50}$ atropine (if necessary in two doses of gr. $\frac{1}{100}$ each, the first dose one hour before the operation, the second immediately before the operation). (2) The use of a mixture of chloroform and alcohol containing 10 per cent. of absolute alcohol. (3) The necessity of scientifically controlling the percentage of chloroform. In conclusion he urged that it was not a question of *this or that* inhaler, but what were the *best* means to accurately measure the dose? Whilst a drop-bottle and a piece of lint might be quite safe in the hands of expert anæsthetists, they were not safe, but absolutely and unjustifiably dangerous, in the hands of the inexperienced. The time surely had come when fossilised methods should be abandoned in favour of *any scientifically accurate* apparatus giving definite percentage vapours.

Mr. H. Bellamy Gardner said that their teaching should be simplified as far as was possible, for the administration of anæsthetics was anxious work and plain dogmatic rules were of extreme value in cases of doubt and in emergencies. Much difference of opinion with regard to chloroform was due to a common error in the conception of the corneal reflex and to errors in the technique of eliciting it. During the administration of anæsthetics the anæsthetist's attention should be primarily devoted to maintaining efficient respiration and a free airway, because in the human subject the difficulties and dangers of anæsthesia were far more largely due to asphyxial factors [illegible] to the action of the drugs used, the [illegible] readily learned in two or three [illegible] fact was not yet sufficiently

recognised nor appreciated by workers in the laboratory who were not in the constant habit of handling the anæsthetised human being. In order to keep the airway clear all forms of stertor should be relieved directly they arose, whether buccal, nasal, glossal, tonsillar, palatal, epiglottic, mucous, laryngeal, or tracheal, whether they were purely mechanical or reflex, because if allowed to persist they tended to deepen and to introduce an unnecessary asphyxial factor into the narcosis. With regard to the actual dosage of chloroform the clinical evidence yielded by the degree of reflex response in the upper eyelid when the centre of the cornea was touched by the finger was the most valuable measure of the depth of narcosis, and if elicited in a proper manner it was neither necessary nor safe to abolish the same completely in both eyelids during any surgical operation under chloroform. The wrong method is to hold the upper eyelid open with one finger and touch the cornea with another finger. The eyelid cannot close if it be held open. The proper method is to raise the margin of one of the upper eyelids by gently inserting the pulp of one finger only between the lids, then drawing the upper lid upwards till the cornea is well exposed to brush definitely against the centre of the cornea and edge of the upper lid with that finger and let go suddenly. By feeling and watching the brisk, moderately rapid and slow responsive closures of the upper lid, the presence of the first, second, and third stages of narcosis respectively are demonstrated. During the persistence of a "weakly active" corneal reflex in, at any rate, one of the eyes impairment of respiratory activity from the action of chloroform upon the medullary centre was never observed, but central respiratory depression and commencing failure of function often supervened directly the corneal reflex was entirely abolished. Expressed in another way—when the corneal reflex was entirely absent in the human subject a condition in which central respiratory failure was common had been established. With regard to the dosimetric apparatus which was shown at the last meeting, applying this doctrine, he would ask only this question, Could the corneal reflexes be abolished in the human subject during their use? If they could, he maintained that a condition in which central respiratory failure was common could be produced, and they were, therefore, no safer than any other method.

Dr. A. D. WALLER thought that idiosyncrasy should not be appealed to until after we had obtained some knowledge of the amount of drug exhibited. He referred to the importance of interference with respiration attributable to the retention of chloroform in the blood. The [illegible] dangerous in the hands of unskilful [illegible] dosage was the chief point [illegible] desirable [illegible]

offered to inspiration should be between 1 and 2 per 100. To effect this, what kind of apparatus was preferable, the plenum or vacuum apparatus? The plenum, in Dr. Waller's opinion; the apparatus in which chloroformisation is made to depend upon the patient's respiration being, in his opinion, unsafe.

Dr. WILLETT had used the Vernon-Harcourt apparatus 300 times, but did not consider that it came up to what was claimed for it; it was only of use in selected cases.

Dr. LEVY said the percentage should be regulated as accurately as possible, and that it should not be subject to rapid fluctuations from alterations in the rate of draught through a suction apparatus, or from other fortuitous conditions. The percentage tension of the vapour was all-important in regulating the tension in the blood, and the mass of vapour passing through the lung was a matter of comparatively small importance. The Vernon-Harcourt apparatus he believed to be too inaccurate to allow conclusions to be drawn as to the strength of vapour delivered.

Dr. HEWITT, in concluding discussion, said it was a very difficult task to attempt to focus the discussion which had taken place. There seemed to be a good deal of misconception on the part of the physiologist as to what the clinical anæsthetist meant by anæsthesia, just as there was misconception on the part of the anæsthetist as to what the physiologist intended to describe by this term. Experience had convinced him (Dr. Hewitt) that there were essential points of difference between the two conditions; and until these were recognised the physiologist and the anæsthetist could hardly meet upon the same platform. It was not a difficult matter to define and demonstrate these differences. Sir Victor Horsley had said that he was content with a light chloroform anæsthesia. But he (Dr. Hewitt) felt that he was echoing the opinions of his colleagues when he said that in a large number of cases in practice, and especially in abdominal cases, it was necessary to obtain complete muscular relaxation. It was to be regretted that certain surgeons who had promised to attend and to state that they regarded this profound degree of narcosis as necessary for the successful performance of certain operations were absent this evening. He would point out that in practice the actual patency of the air-way was a very important factor. Many physiologists imagined that the introduction of chloroform vapour into the human organism could be precisely imitated by anæsthetising a rabbit through a tracheal tube. This was not so. There were numerous gradations between a very free air-way on the one hand and a completely obstructed air-way on the other. A patient with a very free air-way was more likely than a patient with a naturally obstructed air-way to pass through the stages of chloroform anæsthesia without other evidences of obstruction. If the air-way was absent that all tendency

towards intercurrent asphyxia was absent. Mr. Eve had laid great stress upon cyanosis, but this was only a symptom of oxygen deprivation. It was one of the later symptoms. Partial occlusion of the air-tract first increased the rate and depth of breathing (and up to this point it was often a positive advantage), it then led to duskiness of the features and a deepening of the anæsthesia from the intercurrent deprivation of oxygen. Finally cyanosis, dilatation of the pupil, separation of the lids, pallor, and pulselessness supervened. He more particularly referred, in this connection, to inspiratory as opposed to expiratory obstruction. Stertor, the audible expression of one variety of obstruction, generally contributed to anæsthesia. The respiratory pump might, in fact, be thrown out of gear just as much by alterations in its internal channels as by alterations in its motor mechanism. Sir Victor Horsley had said that he had never seen respiratory arrest from reflex tongue retraction or reflex laryngeal spasm dependent upon surgical procedures. All he (Dr. Hewitt) could say was that such conditions were not uncommon, especially in comparatively light anæsthesia. In former days the true nature of many of the phenomena brought about by operative procedures was not understood; the respiratory element was not recognised, and patients were thought to be liable to sudden cardiac arrest. Unfortunately, mistakes of a similar kind were still made. In the next place, he must again insist that cases occurred in which extraordinarily large or extraordinarily small quantities of chloroform were required, and such cases needed widely different management. He submitted that no regulating apparatus could in all cases produce the physiological effects of chloroform in a given time, and with the expenditure of a given quantity of the drug; and that, whether a regulating inhaler were used or not, the anæsthetist had to discover the degree of susceptibility of his patient. The sudden effects of concentrated chloroform vapour were in his (Dr. Hewitt's) opinion respiratory. He had used the Dubois, the Vernon Harcourt and the Waller instruments, and had seen Dr. Levy use his appliance. All had been tried in the theatre of St. George's Hospital. He considered that the Dubois and the Waller apparatus, both of which supplied a chloroform atmosphere to the patient without any suction action on the part of the latter, were to be preferred to the other instruments. In connection with chloroform mixtures, he might remind the Society that the so-called "A.C.E." mixture was originally suggested by the Society, and that if used properly it produced a chloroform anæsthesia on much the same principles as those now being advocated by the supporters of regulating inhalers. The dilution of the chloroform prevented, to a considerable extent, the introduction of dangerous quantities of the drug into the circulation. A mixture of two parts of chloroform to three

parts of ether was, in fact, an excellent anæsthetic for use, especially in country practice, it having been found that the alcohol of the "A.C.E." mixture was of little or no value. In summing up, he might say with Dr. Waller that the whole discussion turned upon one point: Should we or should we not use a regulating inhaler? It was a most difficult matter to say what the feeling of the meeting was, but it certainly seemed that at the present juncture the evidence brought forward in favour of such inhalers was not sufficiently convincing to entitle one to abandon simpler means of chloroformisation. It was, however, necessary to maintain an open mind and for those holding the one view to meet and to observe the procedures of those holding the other. He agreed with Dr. Buxton that a smoother induction period was generally obtainable with a regulating inhaler than with a Skinner's mask and drop bottle; but he contended that it was possible with the latter simple appliances to imitate, so to speak, the gradual and equable administration which could be effected by the more complex instruments. He also agreed with Dr. Buxton as to reflex vaso-motor effects being more common in the deepest chloroform anæsthesia. He did not deny that a properly working regulating inhaler such as that of Dubois or Waller had certain advantages; but it seemed to be the general opinion of the Society, at all events at present, that such advantages were rather more than counterbalanced by the disadvantages which he (Dr. Hewitt) and other speakers had enumerated.

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