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(SESSION 1906–7)

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(*Died 6th Nov.*, 1893, *and Sir. W. S. Church. Senior* [*Medical*] *Vice-President, acted as President until 1st March*, 1894.)
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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 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 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, W.

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

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, N.W.

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

1905 BOHR, CHRISTIAN, Copenhagen.

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OF LONDON

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P.—President.
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T.—Treasurer.
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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.
Cent. Com.—Member of Centenary 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.

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[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, AND FELLOWS WHO HAVE COMPOUNDED.

Elected

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

1877 **Abercrombie,** *JOHN*, M.D., Consulting Physician to, and late Lecturer on Forensic Medicine at, Charing Cross Hospital; Augill Brough, Westmorland. C. 1896-8. *Referee*, 1898-1906. *Trans.* 2.

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

1885 ACLAND, THEODORE DYKE, M.D., Physician to St. Thomas's Hospital, and Physician to the Hospital for Consumption and Diseases of the Chest, Brompton; 19, Bryanston square, W. C. 1906-7. *Ho. Com.* 1906-7. *Referee*, 1905-6.

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

1879 ALLCHIN, WILLIAM HENRY, M.D., F.R.S.Ed., Senior Physician to the Westminster Hospital; 5, Chandos street, Cavendish square, W. 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, W.

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

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

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, W.

Elected

1893 BAILEY, ROBERT COZENS, M.S., Spanish place mansion, Manchester square, W.

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

1900 BALDWIN, ASLETT, 6, Manchester square, W.

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

1885 BALLANCE, CHARLES ALFRED, M.V.O., Pruss. O.C.3, M.S., Surgeon to St. Thomas's Hospital; Surgeon to the National Hospital for the Paralysed and Epileptic, Queen square; 106, Harley street, Cavendish square, W. *Referee*, 1904-7. *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, W. C. 1895-7. *Referee*, 1897-1907. *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, W. C. 1892. S. 1899-1902. *Referee*, 1896-9. *Trans.* 2.

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

1893 BARRETT, HOWARD, 49, Gordon square, W.C.

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

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

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

1859 BARWELL, RICHARD, Consulting Surgeon to the Charing Cross Hospital; 55, Wimpole street, W. 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; Fairfield, Chesham Bois, Bucks. C.1885. V.P.1904-5. *Referee*, 1886-96. *Trans.* 4.

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

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

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

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, W. C. 1906-7.

1897 BEDDARD, A. P., M.D., Assistant Physician to Guy's Hospital; 44, Seymour street, W. *Trans.* 1.

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, W. C. 1900-2. *Referee*, 1896-1900, 1906-7. *Trans.* 1.

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

1877 BENNETT, SIR WILLIAM HENRY, K.C.V.O., Surgeon to St. George's Hospital; 1, Chesterfield street, Mayfair, W. C. 1893-4. *Referee*, 1892-93, 1899-1907. *Trans.* 4.

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

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, W.

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

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

1901 BLAIKIE, J. BRUNTON, M.D., C.M., 80, Brook street, Grosvenor square W.

1883 **Bland-Sutton,** JOHN, Surgeon to the Middlesex Hospital; Surgeon to the Chelsea Hospital for Women; 47, Brook street, Grosvenor square, W. C. 1906-7. *Trans.* 6.

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

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

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

1904 BOLTON, CHARLES, M.D., 16, Devonshire street, W.

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

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

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

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, W. C. 1904-6. *Referee*, 1895–1904. *Trans.* 1.

1890 BRADFORD, JOHN ROSE, M.D., D.Sc., F.R.S., Physician to University College Hospital; 8, Manchester square, W. *Referee*, 1899-1907. *Trans.* 1.

1907 BRAILEY, ARTHUR R., M.C.; 11, Old Burlington street, W.

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

1901 BREWERTON, ELMORE WRIGHT, 84, Wimpole street, W.

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

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

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, W. C. 1885. V.P. 1905-6. *Referee*, 1881-4, 1891-7. *Trans.* 5.

1905 BROWN, WILLIAM CARNEGIE, M.D., 32, Harley street, W.

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, W.

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, W. 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, W.

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

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, W. C. 1888-9. V.P. 1907. *Referee,* 1880-87. *Lib. Com.* 1882-7. *Trans.* 2.

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, W. 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, W. *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, W.

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, W.

1873 **Butlin,** HENRY TRENTHAM, D.C.L., Surgeon to St. Bartholomew's Hospital; 82, Harley street, Cavendish square, W. C. 1887-8. V.P. 1904-6. *Referee,* 1893-1904. *Trans.* 4. *Pro.* 1.

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

Elected

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, W. *C.* 1907.

1899 BUZZARD, EDWARD FARQUHAR, M.D., 78, Wimpole street, Cavendish square, W.

1868 **Buzzard,** THOMAS, M.D., Physician to the National Hospital for the Paralysed and Epileptic; 74, Grosvenor street, Grosvenor square, W. C. 1885-6. V.P. 1905-7. *Referee*, 1887-1905.

1885 CAHILL, JOHN, M.D., 12, Seville street, Lowndes square, S.W.

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, W.

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

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

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

1901 CARGILL, LIONEL VERNON, 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, W.

1888 CARLESS, ALBERT, M.S., Professor of Surgery in King's College, London; Surgeon to King's College Hospital; 6, Upper Wimpole street, W.

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

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

Elected

1888 CAUTLEY, EDMUND, M.D., B.C., 15, Upper Brook street, W. *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, S.W. 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, W. C. 1898-1900. *Referee*, 1891-8. *Lib. Com.* 1885-98. *Trans.* 8.

1906 CHARLES, SIR RICHARD HAVELOCK, K.C.V.O., M.D., M.Ch., 9, Manchester square, W.

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, W. *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, W. C. 1897-9. *Referee*, 1894-7, 1904-7. *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, W. C. 1885-6. V.P. 1892-4. T. 1894-1907. *Referee*, 1874-81. *Ho. Com.* 1898-1907.

1879 CLARK, ANDREW, D.Sc., Surgeon to, and Lecturer on Surgery at, the Middlesex Hospital; 71, Harley street, Cavendish square, W. C. 1906-7.

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, W.

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

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, W. C. 1899-1901. *Lib. Com.* 1905-7. *Trans.* 1.

1905 CLOGG, HERBERT SHERWELL, M.B., M.S., 143, Harley street, Cavendish square, W.

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

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

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

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

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, W.

1860 **Couper,** JOHN, Consulting Surgeon to the Royal London Ophthalmic Hospital and to the London Hospital; 80, Grosvenor street, W. 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, W. C. 1893-4. *Referee*, 1892-3. *Ho. Com.* 1895-8.

Elected

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

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

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

1874 **Cripps,** WILLIAM HARRISON, Surgeon to St. Bartholomew's Hospital; 2, Stratford place, Oxford street, W. C. 1890-91. V.P. 1906-7. *Trans.* 1.

1882 CROCKER, HENRY RADCLIFFE, M.D., Physician to the Skin Department, University College Hospital; 121, Harley street, Cavendish square, W. C. 1903-4. *Trans.* 3.

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

1888 CULLINGWORTH, CHARLES JAMES, M.D., D.C.L., LL.D., Consulting Obstetric Physician to St. Thomas's Hospital; 14, Manchester square, W. *Referee,* 1896-1907.

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, W. *Trans.* 1.

1898 CURRIE, A. STARK, M.D., 20, Oxford terrace, Hyde park, W.

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, W. 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, W. C. 1896-7. V.P. 1901-2. *Trans.* 4.

Elected

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

1896 DAUBER, JOHN HENRY, M.A., M.B., B.Ch., Gynæcologist to the Hospital for Women, Soho square; 39, Hertford street, Mayfair, W.

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

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

1878 DENT, CLINTON THOMAS, Surgeon to, and Lecturer on Surgery at, St. George's Hospital; 61, Brook street, W. C. 1890, 1905-7. 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, W. *Trans.* 1.

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

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, W. P. 1896-8. C. 1874-5. V.P. 1887. *Referee*, 1869-73, 1882-6. *Sci. Com.* 1867, 1879, 1889-96. *Trans.* 16.

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, W.

1888 DONELAN, JAMES, M.B., M.C., Chevalier, Crown of Italy, Physician to the Italian Hospital, Queen square; 6, Manchester square, W.

Elected

1877 **Doran**, ALBAN HENRY GRIFFITHS, Surgeon to the Samaritan Free Hospital; 9, Granville place, Portman square, W. C. 1893-4. *Lib. Com.* 1891-3, 1899-1907. *Referee*, 1898-1907. *Trans.* 4.

1891 DOVE, PERCY W., M.B., 84, Crouch hill, N.

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

1893 DRYSDALE, JOHN H., M.D., 11, Devonshire place, W. *Trans.* 1.

1865 **Duckworth**, SIR DYCE, M.D., LL.D., Physician to, and Lecturer on Clinical Medicine at, St. Bartholomew's Hospital; 28, Grosvenor place, S.W. C. 1883-4. V.P. 1902-4. *Referee*, 1885-97. *Trans.* 2.

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

1880 DUNBAR, JAMES JOHN MACWHIRTER, M.D., Hedingham House, Clapham Common, S.W. *C.* 1907.

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

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

1874 **Durham**, FREDERIC, M.B., Consulting Surgeon to the North-West London Hospital; 52, Brook street, Grosvenor square, W.

1905 EASON, HERBERT LIGHTFOOT, M.D., M.S., 24, St. Thomas's street, S.E.

1868 **Eastes**, GEORGE, M.B.Lond., 35, Gloucester terrace, Hyde Park, W. 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, W.

1891 EDDOWES, ALFRED, M.D., 28, Wimpole street, W.

Elected

1883 EDMUNDS, WALTER, M.C., 2, Devonshire place, Portland place, W. *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, W.

1902 ENGLISH, THOMAS CRISP, B.S., Assistant Surgeon to St. George's Hospital and to the Grosvenor Hospital for Women and Children; 47, Upper Brook street, W. *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, W.

1904 EVANS, JOHN HOWELL, 63, Grosvenor street, W.

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, W.

1879 EVE, FREDERIC S., Surgeon to the London Hospital; Surgeon to the Evelina Hospital for Sick Children; 61, Harley street, Cavendish square, W. C. 1897-9. *Referee*, 1902-7. *Trans.* 4.

1877 **Ewart,** WILLIAM, M.D., Physician to St. George's Hospital and to the Belgrave Hospital for Children; 33, Curzon street, Mayfair, W. C. 1895-7. *Lib. Com.* 1897-1907. *Sci. Com.* 1889-1902. *Trans.* 3. *Pro.* 1.

1900 FAIRBAIRN, JOHN SHIELDS, M.B., Assistant Obstetric Physician to St. Thomas's Hospital; 42, Wimpole street, W.

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

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

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

Elected

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, W. *Referee*, 1891-6. C. 1896-8. *Dis. Com.* 1896-1907. *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, W.

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, W.

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

1896 FOULERTON, ALEXANDER GRANT RUSSELL, Middlesex Hospital, W., 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, W. 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, W. C. 1904-5. *Trans.* 1.

1871 FRANK, PHILIP, M.D., 3, Elvaston place, South Kensington, S.W. *C.* 1907.

1902 FRENCH, HERBERT, M.D., 26, St. Thomas's street, S.E. *Trans.* 3.

1896 FREYER, P. J., M.D., M.Ch., Surgeon to St. Peter's Hospital; 27, Harley street, Cavendish square, W. *Trans.* 1.

Elected

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, W.

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

1883 FULLER, HENRY ROXBURGH, M.D., 45, Curzon street, Mayfair, W. C. 1906-7.

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

1899 FÜRTH, KARL, M.D., Physician to the German Hospital, 39, Harley street, W.

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, W. C. 1892. *Referee*, 1882-91, 1896-1907. *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, W.

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, W. C. 1867. V.P. 1880-81. *Referee*, 1855-65. *Trans.* 9.

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, W. C. 1902-4. *Sci. Com.* 1889-1902. *Lib. Com.* 1896-1902, 1904-7. *Referee*, 1906-7. *Trans.* 8.

Elected

1905 GASK, GEORGE ERNEST, The Warden's House, St. Bartholomew's Hospital, E.C.

1887 GAY, JOHN, 119, Upper Richmond road, Putney, S.W.

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, W. C. 1883-4. L. (June) 1887-99. V.P. 1899-1900. *Sci. Com.* 1879. *Bldg. Com.* 1889-92. *Referee*, 1885-7, 1900-7. *Lib. Com.* 1871-6. *Ho. Com.* 1898-1900. *Trans.* 1.

1898 GIBBES, CUTHBERT CHAPMAN, M.D., 89, Harley street, W.

1880 GIBBONS, ROBERT ALEXANDER, M.D., Physician to the Grosvenor Hospital for Women and Children; 29, Cadogan place, S.W. C. 1896-7. *Trans.* 1.

1893 GILES, ARTHUR EDWARD, M.D., B.Sc., Assistant Surgeon, Chelsea Hospital for Women; 10, Upper Wimpole street, W.

1894 GILL, RICHARD, 72, Wimpole street, W.

1906 GOADBY, KENNETH W., 21, New Cavendish street, Portland place, W.

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, W. S. 1892-4. L. 1895-1907. *Referee*, 1886-91. *Ho. Com.* 1898-1907. *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, W.

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

Elected

1897 GOODBODY, F. W., M.D., 6, Chandos street, Cavendish square, W. *Trans.* 1.

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

1883 GOODHART, JAMES FREDERIC, M.D., Physician to Guy's Hospital; Consulting Physician to the Evelina Hospital for Sick Children; 25, Portland place, W. C. 1903–4. *Referee*, 1900-3. *Lib. Com.* 1893-6.

1895 GOSSAGE, ALFRED MILNE, M.B., 54, Upper Berkeley street, W. *Trans.* 1.

1877 GOULD, ALFRED PEARCE, M.S., *Hon. Treasurer*, Surgeon to, and Lecturer on Surgical Pathology at, the Middlesex Hospital; 10, Queen Anne street, Cavendish square, W. C. 1892-3. S. 1898–1901. V.P. 1904–6. T. 1906-7. *Referee*, 1895-8. *Ho. Com.* 1892-8, 1904–6. *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, W.

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, W. C. 1891. *Referee*, 1888-90. *Lib. Com.* 1884-6. *Trans.* 8.

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

1868 **Green**, T. HENRY, M.D., Physician to the Charing Cross Hospital, and to the Hospital for Consumption, Brompton; 74, Wimpole street, Cavendish square, W. V.P. 1907. 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, W. *Referee*, 1902-7.

Elected

1903 GRÜNBAUM, OTTO F. F., M.D., B.C., 34, Wimpole street, Cavendish square, W. *Trans.* 1.

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, W. 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, W.

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

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

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

1881 **Hall**, FRANCIS DE HAVILLAND, M.D., Physician to the Westminster Hospital; 47, Wimpole street, Cavendish square, W. C. 1901-3. *Referee*, 1893-7.

1891 HAMER, WILLIAM HEATON, M.D., 55, Dartmouth park hill, Highgate, N.

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, W.

1905 HANDLEY, WILLIAM SAMPSON, M.S., Assistant to the Middlesex Hospital, late Surgeon to Outpatients, Samaritan Free Hospital, 77, Wimpole street, Cavendish square, W.

Elected

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

1893 **Harley,** Vaughan, M.D., 25, Harley street, Cavendish square, W.

1901 Harmer, William Douglas, M.B., 45, Weymouth street, Portland place, W.

1892 Harold, John, M.B., 91, Harley street, Cavendish square, W.

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

1904 Harrison, Charles Joseph, M.D., B.S., 6, Swan Walk, Chelsea, S.W.

1901 Hartigan, T. J. P., 94, Harley street, W.

1896 Hartley, Percival Horton-Smith, M.V.O., M.D., 19, Devonshire street, Portland place, W. *Sci. Com.* 1897-1902. *Trans.* 1.

1870 **Haward,** J. Warrington, *President;* Consulting Surgeon to, and Lecturer on Clinical Surgery at, St. George's Hospital; 57, Green street, Grosvenor Square, W. C. 1885. S. 1888-91. V.P. 1894-5. T. (June) 1895-1906. P. 1906-7. *Lib. Com.* 1881-4. *Sci. Com.* 1889-91. *Bldg. Com.* (*Sec.*) 1889-92. *Ho. Com.* 1892-1907. *Trans.* 3.

1891 Hawkins, Herbert Pennell, M.D., B.Ch., Physician to St. Thomas's Hospital; 56, Portland place, W.

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, W.

1905 Head, Henry, M.D., F.R.S., 4, Montagu square, W.

Elected

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

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

1900 HERN, WILLIAM, Dental Surgeon to the Middlesex Hospital, late Dental Surgeon to, and Lecturer on Dental Surgery and Pathology at, the Royal Dental Hospital of London; 7, Stratford place, W.

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, W. C. 1904–6. *Ho. Com.* 1904–6.

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

1883 HERRINGHAM, WILMOT PARKER, M.D., Assistant Physician, St. Bartholomew's Hospital; 40, Wimpole street, Cavendish square, W. C. 1903-4. *Lib. Com.* 1902-3, 1904-7. *Ho. Com.* 1903-4. *Trans.* 3.

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

1906 HERTZ, ARTHUR FREDERICK, M.B., B.C., The College, Guy's Hospital, S.E. *Trans.* 1.

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, W. *Trans.* 3.

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

1906 HINE, THOMAS GUY MACAULAY, M.B., B.C., 19, Lower Seymour street, W.

1904 HOBSON, H. OVERTON, M.D., 32, Upper Berkeley street, W.; 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, W. 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, W. C. 1904-6.

1898 HORDER, THOMAS J., M.D., 141, Harley street, W. *Trans.* 1.

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, W. *Referee*, 1897-1907. *Trans.* 1.

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

1906 HUGHES, GERALD S., Bolingbroke Hospital, Wandsworth common, S.W.

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

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

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, W. C. 1870. V.P. 1882. P. 1894-6. *Referee*, 1876-81, 1883-94. *Lib. Com.* 1864-5. *Trans.* 15. *Pro.* 2.

1888 HUTCHINSON, JONATHAN, Jun., Surgeon to the London Hospital; 1, Park crescent, W. *Trans.* 4.

Elected

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

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, W. C. 1889.

1883 JESSOP, WALTER H. H., M.B., Ophthalmic Surgeon to St. Bartholomew's Hospital; 73, Harley street, W. C. 1906-7. *Referee*, 1901-6.

1881 JOHNSON, GEORGE LINDSAY, M.D., Cortina, Netherhall gardens, South Hampstead, N.W., and 55, Queen Anne street, W.

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, W. *Trans.* 1.

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

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

1887 JONES, HENRY LEWIS, M.D., Medical Officer in charge of Electrical Department at St. Bartholomew's Hospital; 143, Harley street, Cavendish square, W. *Referee*, 1906-7.

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

1907 JONES, ROBERT, 11, Nelson street, Liverpool.

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, W. 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, W.

Elected

1882 KEETLEY, CHARLES R. B., Senior Surgeon to the West London Hospital; 56, Grosvenor street, Grosvenor square, W. C. 1901-3. *Lib. Com.* 1903-7. *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, W.

1901 KELYNACK, T. N., M.D., 120, Harley street, W.

1857 **Kiallmark**, HENRY WALTER, 5, Pembridge gardens, W. 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, W. C. 1900-2. *Referee*, 1905-7. *Trans.* 4.

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

1900 LAKE, RICHARD, Surgeon Laryngologist, Mount Vernon Hospital for Consumption; Surgeon, Royal Ear Hospital; 60, Harley street, W. *Trans.* 1.

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

1884 LANE, WILLIAM ARBUTHNOT, M.S., Surgeon to Guy's Hospital and to the Hospital for Sick Children, 21, Cavendish square, W. 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, W. C. 1904–5.

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

1865 **Langton**, JOHN, Surgeon to, and Lecturer on Clinical Surgery at, St. Bartholomew's Hospital; Surgeon to the City of London Truss Society; 20, Bentinck street, Manchester square, W. C. 1881–2. V.P. 1895-7, *Referee*, 1885-95. *Lib. Com.* 1879-80, 1888-95, *Trans.* 2.

Elected

1898 Latham, Arthur Carlyle, M.D., Physician to St. George's Hospital; 38, Portland place, W.

1890 Law, Edward, M.D., C.M., 8, Wimpole street, Cavendish square, W.

1898 Lawford, J. B., Ophthalmic Surgeon and Lecturer on Ophthalmology, St. Thomas's Hospital; Surgeon to Royal London Ophthalmic Hospital; 99, Harley street, W. *Referee*, 1906-7.

1890 Lawrie, Edward, M.B., Surgeon Lieutenant-Colonel, Indian Medical Department; late Residency Surgeon, Hyderabad, Deccan; Harley Lodge, 115a, Harley street, W.

1893 Lawson, Arnold, Ophthalmic Surgeon to the Children's Hospital, Paddington Green; 12, Harley street, Cavendish square, W.

1900 Leaf, Cecil Huntington, M.A., M.B.; 75, Wimpole street, Cavendish square, W.

1896 Lee, William Edward, M.D., 36, Finsbury pavement, E.C.

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, W. *Trans.* 2.

1899 Legge, Thomas Morison, M.D., 18, Cheyne row, Chelsea, S.W.

1895 Leslie, Robert Murray, M.D., Assistant Physician to Royal Hospital for Diseases of the Chest; 152, Harley street, Cavendish square, W.

1886 Lewers, Arthur Hamilton Nicholson, M.D., Obstetric Physician to the London Hospital; 43, Upper Brook street, W. *Trans.* 1.

1891 Little, Ernest Muirhead, Surgeon to the National Orthopædic Hospital; 5, Duchess street, Portland place, W.

1889 Little, John Fletcher, M.B., 125, Harley street, Cavendish square, W.

Elected

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, W. C. 1901-3. *Trans.* 5.

1897 LOW, HAROLD, 10, Evelyn gardens, S.W.

1906 LOW, VINCENT WARREN, M.D., B.S.; 146, Harley street, W.

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, W. 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, W. *Referee*, 1903-7. *Trans.* 1.

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

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

1905 MCCANN, FREDERICK JOHN, M.D., 5, Curzon street, Mayfair, W.

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, W. C. 1886-7. *Lib. Com.* 1882-5. *Referee*, 1890–1905.

1889 **MacAlister**, *DONALD*, M.A., B.Sc., M.D., LL.D., D.C.L., Principal of Glasgow University, President of the General Medical Council, Consulting Physician to Addenbrooke's Hospital; Linacre Lecturer and Tutor, St. John's College; University Lecturer in Medicine, St. John's College, Cambridge.

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

Elected

1898 McFADYEAN, SIR JOHN, The Royal Veterinary College, Camden Town, N.W.

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, W.

1902 MACKENZIE, HECTOR WILLIAM GAVIN, M.A., M.D., 34, Upper Brook street, W.

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

1880 MADDICK, EDMUND DISTIN, 31, Putney hill, S.W.

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, W. *Sci. Com.* 1889-1902.

1880 MAKINS, GEORGE HENRY, C.B., Surgeon to St. Thomas's Hospital; Consulting Surgeon to the Evelina Hospital for Children; 47, Charles street, Berkeley square, W. C. 1899-1900. *Referee*, 1898-9, 1902-7. *Trans.* 2.

1885 MALCOLM, JOHN DAVID, M.B., C.M., Surgeon to the Samaritan Free Hospital; 13, Portman street, Portman square, W. *Trans.* 5.

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, W. *Referee*, 1904-7.

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, W. C. 1882-3, 1889. S. 1885-7. V.P. 1891-3. *Lib. Com.* 1880-1. *Trans.* 4.

Elected

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

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, W. C. 1906-7. *Referee*, 1904-6.

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

1891 MAY, WILLIAM PAGE, M.D., B.Sc., 14, Great Cumberland place, W.

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

1894 MICHELS, ERNST, M.D., Surgeon to the German Hospital; 48, Finsbury square, E.C. *Trans.* 3.

1907 MILLER, REGINALD HENRY, M.B., B.S.Lond., 22A, Cavendish square, W.

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

1907 MOLLISON, WILLIAM MAYHEW, M.C.Cantab.; Warden's House, Guy's Hospital, S.E.

1873 **Moore**, NORMAN, M.D., *Hon. Librarian*, Physician and Lecturer on Medicine to St. Bartholomew's Hospital; 94, Gloucester place, Portman square, W. C. 1891-2. L. 1899-1907. S. 1896-9. *Referee*, 1886-90. *Ho. Com.* 1898-1907. *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, W. C. 1895–7. *Dis. Com.* 1896–7. *Referee*, 1901-7. *Trans.* 2.

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

Elected

1874 **Morris**, HENRY, M.A., Surgeon to the Middlesex Hospital; 8, Cavendish square, W. 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, W. *Sci. Com.* 1889-1902. *Trans.* 1.

1898 MORRISON, JAMES, M.D., 23, Weymouth street, W.

1885 MOTT, FREDERICK WALKER, M.D., F.R.S., Assistant Physician, Charing Cross Hospital; Pathologist to the London County Council; 25, Nottingham place, W. *Referee*, 1900-7. *Sci. Com.* 1899-1904. *Trans.* 1.

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

1902 MUMMERY, JOHN PERCY LOCKHART, B.A., 10, Cavendish place, W. *Trans.* 1.

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, W.

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

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, W. *Sci. Com.* 1889-1902. *Trans.* 1.

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

Elected

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

1906 NITCH, CYRIL ALFRED RANKIN, M.S., 69, Harley street, W.

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

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, W. C. 1906-7. *Trans.* 1.

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

1860 **Ogle,** WILLIAM, M.D., late Superintendent of Statistics in the Registrar-General's Department, Somerset House; 10, Gordon street, Gordon square, W.C. *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, W.

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, W. C. 1897. *Lib. Com.* 1896-7. *Trans.* 1.

1905 ORMOND, A. W., 37, Queen Anne street, Cavendish square, W. *Trans.* 1.

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

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, W. C. 1890-91. V.P. 1906-7. *Referee*, 1884-9. *Lib. Com.* 1886-8. *Ho. Com.* 1906-7. *Trans.* 4.

Elected

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, W. S. 1904-7. *Lib. Com.* 1902-4.

1905 PARDOE, JOHN GEORGE, M.B., 77, Wimpole street, W. *Trans.* 1.

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

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

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

1883 PASTEUR, WILLIAM, M.D., Physician to the Middlesex Hospital; Consulting Physician to the North-Eastern Hospital for Children; 4, Chandos street, Cavendish square, W. C. 1906-7.

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

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

1891 PATON, EDWARD PERCY, M.D., M.S., 53, Queen Anne street, Cavendish square, W.

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

1894 PEGLER, L. HEMINGTON, M.D., 58, Harley street, W.

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

1890 PERRY, SIR EDWIN COOPER, M.D., Physician to Guy's Hospital; The Superintendent's House, Guy's Hospital, S.E.

1895 PHEAR, ARTHUR G., M.D., Assistant Physician and Pathologist to the Metropolitan Hospital; 47, Weymouth street, Portland place, W. *Trans.* 2.

Elected

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

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, W. *Trans.* 1.

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, 1889-1904. *Lib. Com.* 1879-81.

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

1884 PITT, GEORGE NEWTON, M.D., Physician to, and Pathologist at, Guy's Hospital; 15, Portland place, W. S. 1902-6. *Lib. Com.* 1906-7. *Referee*, 1897-1902. *Trans.* 3.

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, W. *Referee*, 1897-1907.

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

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

1884 POLLARD, BILTON, B.S., Surgeon to University College Hospital; Consulting Surgeon to the North-Eastern Hospital for Children; 24, Harley street, Cavendish square, W. C. 1907. *Trans.* 1.

Elected

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

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

1867 **Powell,** SIR RICHARD DOUGLAS, Bart., K.C.V.O., M.D. 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, W. P. 1904-6. S. (Oct.) 1883-5. C. 1887-8. V.P. 1902-4. *Referee*, 1879-83, 1886. *Trans.* 3.

1887 POWER, D'ARCY, M.A., M.B., Surgeon to, and Lecturer on Surgery at St. Bartholomew's Hospital; Consulting Surgeon to the Victoria Hospital for Children, Chelsea; 10A, Chandos street, Cavendish square, W. *Lib. Com.* 1896-1907. *Trans.* 4.

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, 1889-1904. *Lib. Com.* 1872-8.

1905 PRICE, FREDERICK WILLIAM, M.B., 133, Harley street, W.

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, S.E., and Beachcote, The Bungalows, Walmer Beach.

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

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

Elected

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, W. C. 1893-4. *Lib. Com.* 1887-93, 1899-1907. *Referee*, 1897-1907. *Trans.* 1.

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

1893 RANKIN, GUTHRIE, M.D., 4, Chesham street, Belgrave square, S.W. *Trans.* 2.

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

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

1869 **Read**, THOMAS LAURENCE, 11, Petersham terrace, Queen's gate, S.W. 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, W. C. 1904-6. *Cent. Com.* 1904–5.

1903 RICH, EVELYN ARTHUR, 12, Wilton street, Grosvenor place, S.W.

1887 RICHARDSON, GILBERT, M.A., M.D., 19, Putney hill, S.W.

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, W. C. 1881-2. V.P. 1900-2. *Referee*, 1873-80, 1889-97. *Trans.* 6.

1900 RIVIERE, CLIVE, M.D., 19, Devonshire street, Portland place, W. *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, W.

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

Elected

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, W. C. 1894-5. *Referee*, 1899-1907. *Sci. Com.* 1889-1902.

1898 ROBERTSON, F. W., M.D., "Ravenstone," 37, Lingfield road, Wimbledon, Surrey.

1901 ROBINSON, GEORGE HENKELL DRUMMOND, M.D., 17, Seymour street, Portman square, W.

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, W. *Trans.* 1.

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, W. *Trans.* 11. *Pro.* 1.

1890 ROLLESTON, HUMPHRY DAVY, M.D., Physician to St. George's Hospital, and to the Victoria Hospital for Children; 55, Upper Brook street, Grosvenor square, W. *Referee*, 1906-7. *Trans.* 1.

1906 ROLLESTON, J. D., M.D, Grove Hospital, Tooting, S.W.

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

1868 ROUGHTON, EDMUND WILKINSON, B.S., M.D., Surgeon and Surgical Tutor to the Royal Free Hospital; 38, Queen Anne street, W. *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, W. C. 1902-4. *Lib. Com.* 1900-2. *Referee*, 1900-2.

Elected

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

1906 ROWLANDS, ROBERT PUGH, M.S., 6, St. Thomas's street, S.E.

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, W. *Trans.* 1.

1900 RYALL, CHARLES, 62, Harley street, Cavendish square, W.

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

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, W. *Trans.* 1.

1902 SAMBON, LOUIS W., M.D., 1, Palace garden mansions, W.

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, W.

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, W.

1879 SAVAGE, GEORGE HENRY, M.D., Lecturer on Mental Diseases at Guy's Hospital; 26, Devonshire place, W. C. 1898-9.

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

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

Elected

1905 SCOTT, SYDNEY RICHARD, 46, Queen Anne street, W.

1892 SEGUNDO, CHARLES SEMPILL DE, M.B., B.S., 97, Gloucester place, W.

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

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, W. 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, W.

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

1882 SHARKEY, SEYMOUR JOHN, M.D., Physician to, and Joint Lecturer on Medicine at, St. Thomas's Hospital; 22, Harley street, Cavendish square, W. C. 1899-1900. *Referee*, 1897-9, 1902-7. *Trans.* 2.

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

1886 SHAW, LAURISTON ELGIE, M.D., Physician to Guy's Hospital; 64, Harley street, Cavendish square, W. *Referee*, 1903-7.

1903 SHERREN, JAMES, 40, Devonshire street, W.

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

1899 SHUTTLEWORTH, GEORGE EDWARD, M.D., Parkholme East Sheen, S.W., and 27, New Cavendish street, W.

1899 SIMPSON, WILLIAM JOHN RITCHIE, M.D., 31, York terrace, York gate, Regent's park, N.W.

Elected

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

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

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, W. C. 1899-1900.

1866 SMITH, HEYWOOD, M.A., M.D., 25, Welbeck street, Cavendish square, W.

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

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, W. *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.

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

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, W.C. *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, W. *Referee*, 1894-7.

1887 SPENCER, WALTER GEORGE, M.B., M.S., Surgeon to, and Lecturer on Physiology at, the Westminster Hospital; 35, Brook street, Grosvenor square, W. *Trans.* 2. *Referee*, 1905-7. *Lib. Com.* 1904-7.

Elected

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, W.

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, W.

1875 Spitta, EDMUND JOHNSON, 41, Ventnor Villas, Hove. C. 1903-5.

1903 SPRIGGS, EDMUND IVENS, M.D., Assistant Physician to, and Lecturer on Pathological Chemistry at, St. George's Hospital, and Assistant Physician to the Victoria Hospital for Children, 48, Bryanston street, Portman square, W.

1885 SQUIRE, JOHN EDWARD, C.B., M.D., Physician to the Mount Vernon Hospital for Consumption; 5, Harley street, Cavendish square, W. *Trans.* 2.

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

1907 STEVEN, JOHN LINDSAY, M.D.Glas., 16, Woodside place, Glasgow.

1899 STEWART, PURVES, M.D., 94, Harley street, W. *Trans.* 1.

1856 Stocker, ALONZO HENRY, M.D., Peckham House, Peckham, S.E.

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, W. C. 1904-6.

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, W.

1896 SWAN, CHARLES ROBERT JOHN ATKIN, M.B., B.Ch., 3, Chester place, Hyde Park square, W.

Elected

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, W. *Referee*, 1904-7.

1875 TAY, WAREN, 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, E.C.

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, W. *Trustee*, 1893-1903. S. 1889-93. C. 1894-6. *Sci. Com.* 1889-1902. *Referee*, 1887-8, 1899-1907. *Trans.* 5.

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, W. *Trans.* 1.

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

1905 THOMPSON, ARTHUR RALPH, 4, St. Thomas's street, S.E.

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

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

1862 **Thompson**, REGINALD EDWARD, M.D., 13, Cheyne gardens, Chelsea, S.W. C. 1879. S. 1880-82. V.P. 1883-4. *Referee*, 1873-8. *Sci. Com.* 1867. *Trans.* 2.

Elected

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, W. *Trans.* 3.

1892 THOMSON, STCLAIR, M.D., Physician for Diseases of the Throat in King's College Hospital; 28, Queen Anne street, Cavendish square, W. *Trans.* 2.

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

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

1872 TOMES, CHARLES SISSMORE, M.A., F.R.S., 9, Park crescent, Portland place, W. C. 1887. V.P. 1897-99. *Lib. Com.* 1879.

1882 TOOTH, HOWARD HENRY, C.M.G., M.D., *Hon. Secretary*, Physician to the National Hospital for the Paralysed and Epileptic, Queen square; Assistant Physician to St. Bartholomew's Hospital; 34, Harley street, Cavendish square, W. S. 1906-7. C. 1905-6. *Referee*, 1902-5. *Sci. Com.* 1896-1902.

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, W. 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, W.

Elected

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

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

1882 TURNER, GEORGE ROBERTSON, Surgeon to, and Joint Lecturer on Surgery at, St. George's Hospital; 41, Half Moon street, Piccadilly, W. C. 1903-4. *Trans.* 1.

1898 TURNER, WILLIAM, M.B., M.S., Assistant Surgeon, Westminster Hospital; 17, Harley street, Cavendish square, W.

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, W. *Trans.* 3.

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

1892 TWEEDY, SIR 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, W.

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

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

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, W.

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, W.

Elected

1884 WAKLEY, THOMAS, 16, Hyde Park Gate, S.W. C. 1905-6.

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

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

1900 WALKER, JOHN WILLIAM THOMSON, M.B., Assistant Surgeon to the North-West London Hospital, and to St. Peter's Hospital; 30, Queen Anne street, W. *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, N.W. *Referee* 1895-1907. *Trans.* 1.

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

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, W.

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

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

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, W.

1877 **Warner**, FRANCIS, M.D., Senior Physician to, and Lecturer on Materia Medica and Therapeutics at, the London Hospital; 5, Prince of Wales terrace, Kensington Palace, W. *Referee*, 1905. C. 1899-1901. *Trans.* 3.

Elected

1894 WATERHOUSE, HERBERT FURNIVALL, C.M., Senior Assistant Surgeon and Lecturer on Anatomy, Charing Cross Hospital; Surgeon, Victoria Hospital for Children; 81, Wimpole street, W. *Referee*, 1905-7.

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

1891 Weber, FREDERIC PARKES, M.D., Physician to the German Hospital, Dalston; 19, Harley street, W. *Trans.* 4. *Pro.* 1.

1857 WEBER, SIR HERMANN, M.D., Consulting Physician to the German Hospital; 10, Grosvenor street, Grosvenor square, W. 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., 16, Lower Seymour street, Portman square, W.

1903 WEST, CHARLES ERNEST, 132, Harley street, W.

1877 WEST, SAMUEL, M.D., Physician and Lecturer on Medicine to St. Bartholomew's Hospital; Consulting Physician to the Royal Free Hospital; 15, Wimpole street, Cavendish square, W. C. 1894-5. *Lib. Com.* 1892-4. *Trans.* 8.

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

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

1881 WHITE, WILLIAM HALE, M.D., Physician to, and Lecturer on Materia Medica at, Guy's Hospital; 65, Harley street, Cavendish square, W. C. 1900-2. *Referee*, 1888-97, 1899-1900. *Trans.* 5.

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

Elected

1897 WHITFIELD, ARTHUR, M.D., 21, Bentinck street, Manchester square, W. *Trans.* 1.

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

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

1865 **Willett**, ALFRED, Consulting Surgeon to St. Bartholomew's Hospital; Surgeon to St. Luke's Hospital; 6, Oxford square, Hyde park, W. *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.

1902 **Willett**, JOHN ABERNETHY, M.B., 26, Upper Wimpole street, Cavendish square, W.

1888 WILLIAMS, CAMPBELL, 18, Queen Anne street, W.

1866 **Williams**, CHARLES THEODORE, M.V.O., 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, W. 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, W.C. C. 1905-6. *Trans.* 1.

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.

1901 WILLIAMS, LEONARD, M.D., Physician to the French Hospital, 8, York street, Portman square, W.

Elected

1905 WILLIAMSON, HERBERT, M.B., B.C., 84, Wimpole street, Portland place, W.

1903 WILLIAMSON, OLIVER K., M.A., M.D., 55, Upper Berkeley street, W.

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

1892 WRIGHT, Sir ALMROTH EDWARD, M.D., Ch.B., 7, Lower Seymour street, W. *Referee.* 1906-7. *Trans.* 2.

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

1904 YOUNG, ROBERT ARTHUR, M.D., B.Sc., Assistant Physician to the Middlesex Hospital, and to the Brompton Hospital, 8, Mansfield street, Portland place, W.

LIST OF RESIDENT FELLOWS

ARRANGED ACCORDING TO

DATE OF ELECTION

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.
1862 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.
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.
John Langton.
Alfred Willett.
Sir Alfred Cooper.
1866 Samuel Jones Gee, M.D.
1866 Charles Theodore Williams, M.D., M.V.O.
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.
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.
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.
1876 Sir Thomas Barlow, Bart., K.C.V.O., 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.
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 Malcolm A. Morris.
A. E. Cumberbatch, M.B.
Arthur E. J. Barker.
Sir Fredk. Treves, Bart., G.C.V.O., C.B.
Andrew Clark, D.Sc.
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.
1879 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.
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.
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.
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.
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.

1883 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.
Sidney Harris Cox Martin, M.D., F.R.S.
Thomas Wakley.
F. Swinford Edwards.
James Johnston, 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.
Henry Lewis Jones, M.D.
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.
1888 John Anderson, M.D., C.I.E.
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.
1889 Montagu Handfield-Jones, M.D.
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.
William T. Holmes Spicer, M.B.
Seymour Taylor, M.D.
W. G. O. White-Cooper, M.B.
James Jackson Clarke, M.B.
Leonard G. Guthrie, M.D., B.Ch.
G. William Hill, M.D., B.Sc.
Edward Law, M.D., C.M.
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.D.
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.

1891 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.
Charles Sempill de Segundo, M.B.
Sir John Tweedy.
J. S. Selwyn-Harvey, M.D.
StClair Thomson, M.D.
Henry Rayner, M.D.
H. Marmaduke Page.
Sir 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.
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.
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.V.O., 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.
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.
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.

1898 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.

1898 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.
Thomas Herbert Kellock, M.D.

1899 James Hugh Thursfield, M.D.
Lindley Marcroft Scott, M.D.
Louis Bathe Rawling, 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.
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.
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.
Arthur Evans, M.S.
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.
Harold L. Barnard, M.S.
Fallon Percy Wightwick, M.D.

1903 Edmund Ivens Spriggs, M.D.
Otto F. F. Grünbaum, M.D., B.C.
Samuel William Carruthers, M.D.
John Russell Ryan, M.D., B.Ch.
Oliver K. Williamson, M.D.
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.
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.
HerbertLightfoot Eason, M.D., M.S.
John Fawcett, M.D.
Herbert J. Scharlieb, C.M.G., M.D., B.S.
William Henry Battle.
1905 Herbert Sherwell Clogg, M.B., M.S.
William Foster Cross.
Thomas Jefferson Faulder.
George Ernest Gask.
William Sampson Handley, M.S.
Henry Head, M.D., F.R.S.
Frederick John McCann, M.D.
Harry Edwin Bruce Bruce-Porter, M.D.
Sydney Richard Scott.
Arthur Ralph Thompson.
Herbert Williamson, M.B., B.C.
William Cecil Bosanquet, M.D.
1905 Francis Frederick Muecke, M.B., B.S.
A. W. Ormond.
John George Pardoe, M.B., C.M.
Frederick William Price, M.B.
1906 Sir Richard Havelock Charles, K.C.V.O., M.D., M.Ch.
Arthur Frederick Hertz, M.B., B.C.
Thomas Guy Macaulay Hine, M.B., B.C.
Gerald S. Hughes, M.B., B.S.
Kenneth W. Goadby.
Vincent Warren Low, M.D., B.S.
Cyril Alfred Rankin Nitch, M.S.
J. D. Rolleston, M.D.
Robert Pugh Rowlands, M.S.
1907 Arthur R. Brailey, M.C.
Reginald Henry Miller. M.B., B.S.
William Mayhew Mollison, M.C.

NON-RESIDENT FELLOWS

Elected

1866 *Allbutt, Sir 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*, The Gables, Wokingham, Berks.

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

1905 *Anderson-Berry, David*, West Brow, Sedlescombe road South, St. Leonard's-on-Sea.

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

1903 *Ascherson, William Lawrence*, M.B., B.C., The Sanatorium, Mundesley, Norfolk.

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

1902 *Bailey, William Henry*, M.D., 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.* 3.

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.

1396 *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.

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

Elected

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*, Napier House, Cheltenham.

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, East gate, 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.

1903 *Burrows, Harold*, M.B., B.S., 1, The Cams, Grove road, Southsea.

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

Elected

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

1900 *Carlyon, T. B.*, Brinklow, near Coventry.

1888 *Carter, William Jeffreys Brcher*, 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

1888 *Clarke, Robert Henry*, M.B., Oakfield, Warlingham, Surrey.

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.

Elected

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., Teignmouth, South Devon.

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

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., Bellevue, Commissioner road, Rangoon, Burma.

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

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

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.

Elected

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

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

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

1898 Edkins, J. S., M.B., Brambles, Watford road, Northwood, Middlesex.

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., Scothorne house, Chevallier road, Felixstowe.

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 Roiston*, M.D., Westcott, Dorking.

1902 *Fennell, Charles Henry*, M.A., M.D., Hawkhurst, Upper Station road, Bexhill.

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

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

1903 *Forbes, Norman Hay*, Drumminor, Church Stretton, Salop.

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.* 3.

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

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

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.

Elected

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.

1905 *Grünbaum, Albert Sidney Frankau*, M.D., 38, Clarendon road, 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.

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*, 1899-1906.

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.

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

Elected

1899 *Hillier, A. P.*, M.D., Markyate 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.*, Ardmore, Torquay.

1905 *Howard, Alan Campbell Palmer*, M.D., C.M., c/o Bank of Montreal, 22, Abchurch lane, Lombard street, E.C.

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

1903 *Hulbert, Henry Louis Powell*, M.A., M.B.. B.C., Assistant Medical Officer for Schools, Manchester; The Education Offices, Deansgate, Manchester.

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., Consulting Surgeon to Guy's Hospital; Lordine Court, Ewhurst, Hawkhurst, Kent. 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.

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.]

1907 *Jones, Robert*, 11, Nelson street, Liverpool.

Elected

1865 *Jordan, Furneaux*, Consulting Surgeon to the Queen's Hospital, Birmingham; 10, Ferndale, Teignmouth.

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, etc.; 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.* 2.

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

1892 *Lazarus-Barlow, Walter Sydney*, M.D., Fernholme, Woodside Park, Finchley, N.W. *Sci. Com.* 1892—1902.

1886 *Lediard, Henry Ambrose*, M.D., Surgeon to the Cumberland Infirmary; 26, 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, Middlesex.

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

Elected

1897 *Levy, Alfred G.*, M.D., (*temporary address*) 41, Devonshire street, Portland place, W. *Trans.* 1.

1905 *Lewis, Frank Benjamin*, Springfield House, 144, London road, St. Leonard's-on-Sea.

1898 *Lindsay, James*, M.A., M.D., 3, Queen's Elms, Belfast.

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

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

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.

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.* 3.

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.

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.

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., F.R.S., Regius Professor of Medicine in the University of Oxford; 13, 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; 27, Rothsay road, Bedford. 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

1869 *Payne, Joseph Frank*, M.D., Consulting Physician to, and late Lecturer on Medicine at, St. Thomas's Hospital; Lyonsdown house, New Barnet, Herts. C. 1887. V.P. 1906-7. *Referee*, 1890-1906. *Sci. Com.* 1879. *Lib. Com.* 1878-85, 1889-1906.

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.

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

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

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

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.

1905 *Rashleigh, John Cosmo Stuart*, M.D., Throwleigh, Okehampton, Devon.

Elected

1902 *Raw, Nathan*, M.D., B.S., 66, Rodney street, Liverpool. *Trans.* 1.

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., 8, North terrace, Adelaide, South Australia.

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

1906 *Rickett, Gerald Russell*, M.B., B.C., North road, Sherborne, Dorset.

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*, M.D., I.M.S., 47, Park street, Calcutta. *Trans.* 5.

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

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

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.

Elected

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.* 1889-1904. *Trans.* 1.

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

1906 *Sedgwick, Richard Ernest*, M.D., B.C., 3, Brunswick street, Carlisle.

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.

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

Elected

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

1896 *Stephens, John William Watson*, M.B., B.C., The Johnston Laboratory, The University, Liverpool.

1907 *Steven, John Lindsay*, M.D., 16, Woodside place, Glasgow.

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.

1906 *Stewart, Ian Struthers*, M.D., 15, Merchiston place, Edinburgh. *Trans.* 1.

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; North grange, Headingley, 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.

Elected

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

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.

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.

1876 *Venn, Albert John*, M.D., Huntersdale, Virginia Water, Surrey.

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.

1897 *White, Charles Powell*, Pathological Department, Victoria University, Manchester.

1881 *Whitehead, Walter*, F.R.S. Ed., Consulting Surgeon to the Manchester Royal Infirmary, Manchester and Salford Lock Hospital, and Manchester and Salford Skin Hospital; late Professor of Clinical Surgery, Owens College, Victoria University; Birchfield, 235, Wilmslow road, Manchester. *Trans.* 1.

1885 *Whitla, Sir William*, M.A., M.D., LL.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.

1887 *Willett, Edgar*, M.D., Farmleigh, Worth Park, Crawley, Sussex. *Trans.* 1.

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

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

1890 *Wills, William Alfred*, M.D., late Assistant Physician to the Westminster Hospital; late Senior Physician to the North-Eastern Hospital for Children; Rotherhill, Stedham, Midhurst.

Elected

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

1906 *Wilson, H. Douglas*, M.D., 3, Ripon road, Harrogate.

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., Flax Bourton, near Bristol.

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

1899 *Wynter, Andrew Ellis*, M.D., 17, Eastfield road, Westbury-on-Trym, Bristol.

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

1906 *Young, Samuel Leggatt Orford*, M.B., B.C., Petersfield Lodge, Cambridge.

SERVICE FELLOWS.

1907 BATTYE, WALTER ROTHNEY, M.B., B.S., H.B.M. Consulate, Meshed, North Persia, c/o H. S. King & Co., 9, Pall Mall, S.W.

1903 BENTHAM, ROBERT, Royal Naval Hospital, Malta.

1905 HOLT, MAURICE PERCY, 67B, Shooter's Hill, Blackheath, S.E., and c/o Messrs. Holt, 3, Whitehall place, S.W. *Trans.* 1.

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

1905 KILKELLY, COLONEL CHARLES RANDOLPH, C.M.G., 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, S.W.].

1903 RANKING, ROBERT MAURICE, M.B., B.C., Hong Kong.

1907 SCOTT, GEORGE, M.B., C.M.Aberd., Lieut.-Col. R.A.M.C., 48, Fountainhall Road, Aberdeen (and c/o Messrs. Holt & Co., 3, Whitehall Place, S.W.).

1903 SMITHSON, MAJOR ARTHUR ERNEST, M.B., B.C., Military Hospital, Harrismith, O.R.C.

1905 SPENCER, CHARLES GEORGE, M.B., Kent Lodge, 65, The Avenue, Kew Gardens, S.W.

1903 TAYLOR, SIR WILLIAM, K.C.B., M.D., C.M., Queen Anne's Mansions, St. James's park, S.W.

Corrected to June 14th, 1907.

ANNUAL MEETING

OF THE

ROYAL MEDICAL AND CHIRURGICAL SOCIETY OF LONDON,

HELD IN THE SOCIETY'S HOUSE, No. 20, HANOVER SQUARE

Friday, March 1st, 1907, at 5 p.m.

J. WARRINGTON HAWARD, F.R.C.S., President.

HOWARD H. TOOTH, M.D., C.M.G., } Hon. Secs.
STEPHEN PAGET, }

J. Y. W. MAC ALISTER, Secretary.

Present—31 Fellows.

The PRESIDENT nominated Dr. Clifford Beale and Mr. Laurence Read as scrutineers of the ballot, and declared the ballot open until 6 o'clock.

The SENIOR HONORARY SECRETARY read the Report of the Council (see page lxxxvii).

The SENIOR HONORARY TREASURER submitted the Audited Statement of Accounts, together with the Report of the Treasurers thereon (see page xcii).

RESOLVED on the motion of the PRESIDENT:

"That the Report of the Council, together with the Treasurers' Statement of Accounts be adopted."

The PRESIDENT read the Annual Address (see page xcviii).

On the motion of Sir THOMAS SMITH, seconded by Mr. KIALLMARK:

RESOLVED:

"That the best thanks of the Society be given to the President for his Address, and that he be requested to allow its publication in the 'Transactions.'"

On the motion of Sir DYCE DUCKWORTH, seconded by Dr. SIDNEY COUPLAND.

RESOLVED:

"That the best thanks of the Society be given to the retiring Vice-Presidents, Dr. Thomas Buzzard and Dr. J. F. Payne, for their valuable services during their respective terms of office."

On the motion of Dr. EASTES, seconded by Dr. RICE:

RESOLVED:

"That the best thanks of the Society be given to the retiring Members of Council, Dr. Sidney Martin, Dr. William Pasteur, Mr. Clinton Dent, Dr. H. R. Fuller, for their valuable services during their respective terms of office."

The Scrutineers of the Ballot reported that the following officers and other members of Council had been elected:

President.—J. Warrington Haward.

Vice-Presidents.—Sir Thomas Lauder Brunton, M.D., F.R.S.; T. Henry Green, M.D.; W. Harrison Cripps; Herbert William Page, M.C.

Hon. Treasurers.—Sir William Selby Church, Bart., K.C.B., M.D.; Alfred Pearce Gould, M.S.

Hon. Secretaries.—Howard Henry Tooth, C.M.G., M.D.; Stephen Paget.

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

Members of Council.—Theodore Dyke Acland, M.D.; Edwin Clifford Beale, M.B.; Dudley Wilmot Buxton, M.D.; Philip Frank, M.D.; George Ogilvie, M.B.; John Bland-Sutton; Andrew Clark; Walter H. H. Jessop, M.B.; Bilton Pollard, M.B., B.S.; James J. MacWhirter Dunbar, M.D.

REPORT OF THE COUNCIL

TO THE

ANNUAL GENERAL MEETING,

Held 1st March, 1907

NINETEEN HUNDRED and SIX, the hundred and first year of the Society's existence, was fully equal to any previous year in the steady progress made by the Society, both in scientific work and in material strength. At the present moment the numerical strength of the Society is represented by—

29 Honorary Fellows.
835 Ordinary ,,

Making a total of 864 Fellows.

The Council, as last year, again desire to remind Fellows that the regulations provide for the exhibition and demonstration of interesting cases, new apparatus, and, in fact, anything likely to be of interest to the Fellows at the Ordinary Meetings before the reading of papers. There are no formalities to discourage such exhibitions and demonstrations; all that is necessary is to inform the Honorary Secretaries beforehand, and if this information be given in sufficient time the demonstrations will be announced in the journals. The Council feel that it would add greatly to the interest of the meetings if such demonstrations were more frequent, and they urge Fellows to avail themselves as often as possible of the privilege.

On the suggestion of Dr. Payne, the Council have approved of holding meetings for the discussion of his-

torical and antiquarian papers, and the following regulations have been drawn up. Already a highly interesting paper has been offered, and the date fixed for reading will shortly be announced.

REGULATIONS FOR HISTORICAL MEETINGS.

1. One Ordinary Meeting in each session may be set apart for papers or discussions on the history and antiquities of medicine, and for the exhibition of books, MSS, works of art, instruments, or other objects illustrating the above-named topics.
2. All such papers shall be previously submitted for the approval of the Council.
3. Objects for exhibition must be approved by the Honorary Secretaries, and shall be on view half an hour before the beginning of the Meeting.
4. A record of all objects exhibited, with the descriptions given, shall be preserved by the Secretaries, and papers, after being read, shall be published in the 'Transactions.'

Redemption of Debentures.—Since the last Annual Meeting the Council have formed a Debenture Redemption Fund, to be used from time to time, as may be advisable, for the redemption of the Debenture Debt. Into this fund will be paid all composition fees, and such sums as can be spared out of each year's income. All these sums are invested in trustee securities, and each dividend as received is also invested, so that the Fund is practically invested at compound interest.

Valuation of the Society's property.—Owing to questions that arose during the course of negotiations for amalgamation, the Council gave permission to the Medical Society to have our freehold and leasehold property valued by an independent valuer, with the gratifying result that he reported that the property in question was worth considerably more than the sum at which it stood in the Society's accounts, and, in addition, he stated that the whole of the property was in good and substantial repair.

Meetings.—On June 12th a Special General Meeting

was held to approve an alteration in the By-laws made by the Council, which had the effect of doing away with the February ballot for the election of Fellows, and substituting for it one to be held in October. There were several disadvantages attaching to election at the February ballot, and it was believed that a ballot held at the beginning of the new session in October, to dispose of all proposals sent in subsequent to the June ballot, would prove a great convenience.

The high standard of the papers read at the Ordinary Meetings has been well maintained. A special discussion on "The Operative Treatment of Non-malignant Ulcer of the Stomach, and its Indications, Limitations, and Ultimate Results," was opened on Tuesday, November 13th, 1906, and was continued over three meetings, the discussion, of which a full report is almost ready for publication, being of great value.

Union of Medical Societies.—The negotiations for effecting the proposed Union of Medical Societies have made considerable progress.

The Council feel that nothing could be more fitting, for the end of this Report, than the acknowledgment of their indebtedness to the Secretary, Mr. MacAlister, for his anxious and prudent service of the interests of the Royal Medical and Chirurgical Society; and they desire to record here their gratitude to him and to the other servants of the Society.

Report of the Honorary Treasurers:

> "In presenting the annual financial statement to the Council, the Honorary Treasurers find little calling for special remark. The total income of the Society was slightly less than that for 1905, which is mainly accounted for by one quarter of the Old Berners Street rent being included in that year. The total expenditure for the year was also slightly less,

being £4427 9*s*. 2*d*. against £4478 15*s*. 8*d*. for 1905.

"The volume of 'Transactions' for the year was unusually costly, and accounts for the balance of excess of income over expenditure for the year being less than the Treasurers hoped.

"£200 has been invested in New South Wales 4 per cent. stock to form the nucleus of a sinking fund, the interest on which is to be added to the capital as it falls due.

"The freehold and leasehold property of the Society, which has stood in our accounts since 1896 as worth £51,566 11*s*. 6*d*., has been lately valued by an independent valuer, who placed its value in excess of that sum, and reported that the whole of the premises were in good substantial repair."

Report of the Honorary Librarians:

"The new card catalogue is now complete and in working order, and gives general satisfaction to those who consult the Library.

"The number of books and pamphlets added to the Library for the past year, 1906, has been 590, of which 181 were presented by Fellows and others.

"The number of Fellows visiting the Library was 3630.

"The total number of books and journals borrowed was 3679.

"From Lewis's Library 405 volumes were borrowed.

"The Library has been well used both for reference and circulating purposes during the past year.

"It has been suggested that the income arising from the Gant Bequest should be spent on the purchases of medical, surgical, and other works of permanent interest.

"The design for a simple book-plate to be placed in the books thus purchased will shortly be decided upon."

Report of the House Committee:

"It is satisfactory to find that the statement made in our last Report, as to the general condition of the Society's house, has been confirmed by an independent surveyor.

"Your Committee instructed the London Sanitary Protection Association to make a thorough investigation of the drainage system and of the whole of the sanitary apparatus of the house. The Report stated that the work that was done by the original Building Committee in 1889 is still in excellent condition; but some additional work was recommended in order to bring the system up to the latest standards in sanitary science. This work is now almost completed."

Statement of Liabilities and Assets, 31st December, 1906

Liabilities.

	£	s.	d.
3 *per Cent. First Mortgage Debentures* . . [The Debenture Debt has been reduced by £100 since the previous statement, by the Redemption of One Debenture.]	33,200	0	0
Sundry Liabilities	1,438	18	7
Balance, being Surplus of Assets over Liabilities, viz.—			
Balance, 31st Dec., 1905 £29,167 4 0			
Add Appreciation of Investments 28 12 0			
£29,195 16 0			
Add Excess of Income over Expenditure for the year 1906 180 11 9			
	29,376	7	9
	£64,015	6	4

Assets.

				£	s.	d.
Freehold and Leasehold Property as per valuation of Messrs. Giddy and Giddy, 2nd June, 1896	51,150	0	0			
Subsequent additions . .	416	11	6			
				51,566	11	6
Fixtures, Fittings, and Furniture	1,393	3	7			
Additions during year . .	44	17	8			
	£1,438	1	3			
Less 5 per cent. written off for depreciation of Fixtures, etc.	69	13	3			
				1,368	8	0
Engravings . . . . (as per Valuation of Mr. F. B. Daniell, Aug. 19, 1896).				555	0	0
Contents of Library (as per Valuation)				9,092	14	7
Investments in New South Wales 4 per Cent. Inscribed Stock Debenture Redemption Fund, £185 8*s.* 10*d.* valued at	199	6	9			
Permanent Endowment Fund, £330 16*s.* 3*d.* valued at	355	12	6			
				554	19	3
Sundry Debtors for Rents and Outstanding Subscriptions .				518	0	7
Cash at Bank and in hand .				359	12	5
				£64,015	6	4

Audited and approved,

NEWSON-SMITH, LORD & MUNDY,
Chartered Accountants.
5th February, 1907.

W. S. CHURCH,
A. PEARCE GOULD, } *Hon. Treasurers.*

INCOME AND EXPENDITURE ACCOUNT FOR THE YEAR ENDING 31ST DECEMBER, 1906

Expenditure	£	s.	d.	£	s.	d.
Rent, Rates, Taxes, and Insurance ...	179	12	10			
Salaries of Staff and Accountant ...	706	5	0			
House Servants, Cleaners, etc. ...	361	11	0			
Lighting, Warming, and Cleaning ...	186	17	10			
Printing, Stationery, Stamps and Telegrams	290	0	1			
Meeting Expenses	53	9	10			
Miscellaneous Disbursements ...	64	7	9			
				1842	4	4
Repairs, Alterations, etc. ...				226	17	2
Depreciation of Fixtures, Fittings, etc.				69	13	3
Library Purchases and Expenses ...				454	6	0
Interest on Debentures				995	15	3
'Transactions'				771	8	6
Audit Fee				10	10	0
Telephone				17	8	11
Lift Charges				39	5	9
Balance, being excess of Income over Expenditure during the year ...				180	11	9
				£4608	0	11

Income	£	s.	d.	£	s.	d.
449 *Annual Subscriptions at* £3 3s.	1414	7	0			
150 *do.* £1 1s.	157	10	0			
				1571	17	0
Composition Fees				39	18	0
Entrance Fees				81	18	0
Rents Receivable				2602	7	0
Sale of 'Transactions'				54	7	3
„ 'Climates and Baths' ...				11	7	11
Interest: Permanent Endowment Fund				12	11	4
Miscellaneous Receipts:						
Interest on Deposit ...				6	11	0
Fees for use of Epidiascope ...				27	8	0
Westminster Fire Office on transfer of Policy				191	5	7
Gant Bequest: Interest ...				8	9	10
				£4608	0	11

Audited and approved,

NEWSON-SMITH, LORD & MUNDY,
Chartered Accountants.

5th February, 1907.

W. S. CHURCH,
A. PEARCE GOULD, } *Hon. Treasurers.*

PERMANENT ENDOWMENT FUND.

[Established and contributed to by the Donations of Fellows.]

From Sir Richard Quain			£100	Forward	£250
„ Mr. Edward Law Hussey			50	From Mr. Edwin Unwin Berry	124
„ Sir Richard Douglas Powell			100	„ Dr. Fitzpatrick	5
			£250		£379

Invested in £330 16*s.* 3*d.* (par value) New South Wales Inscribed 4 per Cent. Stock "A" a/c.

MARSHALL HALL MEMORIAL FUND.

[Being the amount subscribed for a Memorial to Marshall Hall. Every five years the accumulated interest is given as a Prize for the best published work on Nervous Diseases.]

	£	*s.*	*d.*		£	*s.*	*d.*
Consols in trust on Dec. 31st, 1905 (*par value*)	615	11	1	Consols in trust on 31st Dec., 1906 (*par value*)	631	14	5
Interest for 1906 (added to Fund) „	16	3	4				
	£631	14	5		£631	14	5

GANT BEQUEST (£500).

[Being a sum of £500 bequeathed to the Society by the late Fredk. James Gant (sometime Vice-President of the Society) to be invested and the income to be spent on books to be marked "Gant Bequest."]

Invested in £510 17*s.* 1*d.* (*par value*) Southern Nigeria (Lagos) 3½ per Cent. Inscribed Stock.

DEBENTURE REDEMPTION FUND.

(New South Wales Inscribed 4 per Cent. Stock "B" a/c.)

1906.		£	*s.*	*d.*	£	*s.*	*d.*		£	*s.*	*d.*
July 18th.	1st Investment to purchase (*par value*)	182	4	8	200	0	0	Amount of Stock on 31st Dec.	185	8	10
	Interest for 1906, to purchase (*par value*)	3	4	2	3	9	3	Interest accrued and invested	3	9	3
								Costs of purchase and difference between "par" and purchase price	14	11	2
					£203	9	3		£203	9	3

LIST OF PAPERS

N.B.—The Council of the 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

J. WARRINGTON HAWARD

PRESIDENT

AT THE

ANNUAL MEETING, MARCH 1st, 1907

During the past year the Society has lost by death 1 Honorary Fellow and 14 Fellows, of whom it is my duty to give a brief record.

Sir Michael Foster, K.C.B., M.D., LL.D., F.R.S. The death of Sir Michael Foster deprived the Society of a greatly-respected Honorary Fellow, the scientific world of a distinguished ornament, and the medical profession of one who, although only for a short time engaged in actual practice, gave, by his teaching and influence, an immeasurable stimulus towards a higher standard of scientific attainment.

Michael Foster was born on March 8th, 1836, at Huntingdon, where his father, Mr. Michael Foster, F.R.C.S., practised as a surgeon. He was educated at University College, London, and, after taking the degree of M.B.London in 1859, he commenced practice in Huntingdon. But his enthusiasm, and the desire for a wider sphere of work, soon led him to relinquish the drudgery of country practice, and to devote himself to the advancement and teaching of physiology.

In 1869 he was appointed Professor of Practical Physiology at University College, London, and, in succession to Huxley, Fullerian Professor at the Royal Institution. In the following year, at the recommendation of Huxley, he began teaching physiology at Cambridge as Prælector of Physiology at Trinity College, and in 1883 was appointed by the University to the newly-endowed chair of Physiology, which he held till 1903. Here he devoted himself to the development of the biological school, in the methods of which he effected, to use the words of one of his most distinguished pupils, "a veritable revolution." To quote Dr. W. H. Gaskell's appreciative letter in the 'Times,'[1] "No student who fell under his spell ever desired to leave him, and so, having gathered round him a band of enthusiastic pupils, he began to select from them teachers of various branches of physiology, recognising always that physiology was part of a great scientific subject, and not merely a branch of medicine." . . . "These young and enthusiastic teachers, imbued with his ideas, and following his methods, soon produced a most remarkable effect, and the University, to its astonishment, woke up one day to find that, without any effort of its own, it possessed a school of biology second in reputation to no other in the British Empire."

In 1872 Foster was elected a Fellow of the Royal Society, and from 1881 to 1903 was one of the Secretaries. Besides his work at Cambridge and at the Royal Society he took part in much public scientific work. He was a member of the Committee of the Lister Institute of Preventive Medicine, of the Lawes Agricultural Trust, and of the Royal Commissions on Sewage Disposal, on Vaccination, and on Tuberculosis. In 1899 he was President of the British Association, and was made a K.C.B. In 1900 he was elected Member of Parliament for the University of London in succession to Sir John Lubbock, who was raised to the Peerage. In the House of Commons he was not a frequent speaker, but was always listened to with

[1] The 'Times,' Saturday, February 9th, 1907, p. 5.

respect as one whose attainments entitled him to speak with authority. Elected as a Liberal Unionist, his opinions led him eventually to change his side and to sit on the Liberal benches. It cannot be said that his political conduct was accepted with the same satisfaction as his scientific work, and at the General Election in 1906 he lost his seat to Sir Philip Magnus.

Sir Michael Foster was a clear and impressive lecturer, a sympathetic teacher, and a willing helper to the industrious student. Besides the charm of his personal character, and the example of his scientific enthusiasm, Foster displayed the rare power of discovering, stimulating, and encouraging the talents of others. He was connected with many learned societies, possessed many academic distinctions, and to the end of his life was an energetic advocate of the claims of science.

Foster published in 1876 his 'Text-book of Physiology,' a most attractive guide to a difficult subject, the popularity of which is shown by its having reached a seventh edition. He also took part in the production of several elementary books on physiology, embryology, and histology, besides editing with Professor Ray Lankester the scientific memoirs of Huxley. Other works were the 'Life of Claude Bernard,' and a volume of 'Lectures on the History of Physiology.' But far beyond the influence of his books, great as that has been, will be the ever-widening effects of the knowledge and methods which he transmitted to his pupils, and which they have carried far and wide.

Sir Michael Foster died suddenly in London on January 29th of the present year. His funeral at Huntingdon, and the memorial services at St. James's Church in London and at Trinity College, Cambridge, were attended by a large number of distinguished men of science and representatives of learned societies.

Sir Michael Foster was twice married, and leaves a son, a member of the medical profession.

Lionel Smith Beale, M.B.Lond., F.R.C.P., F.R.S., Consulting Physician to King's College Hospital; Honorary Fellow of, and Emeritus Professor of Medicine at, King's College; died March 28th, 1906, aged 78 years.

He was born on February 5th, 1828, in the Parish of St. Paul, Covent Garden, and was the only son of Mr. Lionel John Beale, a Member of the Royal College of Surgeons, who practised in Long Acre. He was educated at King's College School, and in 1845 entered the medical department of King's College, having been apprenticed in 1841 to Mr. Joseph Rose, a surgeon at Islington. In 1847, after matriculating at the London University, he became assistant to the late Sir Henry Acland in the museum at Oxford, a post which he held for two years. In 1849 he obtained the licence of the Society of Apothecaries, and during the same year he conducted for the medical department of the Privy Council an investigation into an epidemic of cholera at Windsor. In 1851 he graduated Bachelor of Medicine, and was appointed Resident Physician in King's College Hospital. The following year he established a private laboratory for microscopical and physiological research, wherein he also gave instruction in physiological chemistry and histology, and showed his capacity for that kind of work. In 1853 he was appointed Professor of Physiology in King's College, in 1869 Professor of Pathology, and in 1876 Professor of Medicine. For forty years Professor Beale was physician to King's College Hospital, where he added fame to an already distinguished staff.

He was said to have been especially skilful in reading the signs of disease in the facies of the patient, a study which is perhaps at present somewhat neglected for more recondite methods not always more reliable.

His industry was extraordinary as a teacher, an investigator, a writer, and a practical physician.

As a teacher he did much to popularise medical microscopy; as an investigator he greatly improved the

methods available for histological research; as a writer he made very numerous and valuable contributions to scientific literature; and as a physician he was much valued both in public and private practice.

Besides his medical writings, which included works "On the liver," "On renal and calculous disorders," "On slight ailments," and "On the structure and growth of the tissues," he wrote much upon "The origin of vital action," "On protoplasm or matter and life," and "On life theories and religious thought," and vigorously combated the purely physical and mechanical doctrines of life.

Professor Beale never proceeded to the degree of Doctor of Medicine, but he received many other distinctions. He was elected a Fellow of the Royal Society at the early age of twenty-nine; he was made a Fellow of the Royal College of Physicians in 1859, where also he became a member of the Council, Censor, Curator of the museum, Lumleian Lecturer and Baly Medallist. He delivered the Croonian Lectures to the Royal Society on "The ultimate Nerve-fibres distributed to Muscle and some other Tissues, with Observations upon the Structure and probable Mode of action of a Nervous Mechanism." He was President of the Royal Microscopical Society, and a fellow of many foreign medical and scientific societies.

He was, moreover, keenly interested in gardening, and even in London grew ferns and other plants; while at his home at Weybridge his garden was famed for its beauty.

Professor Beale married in 1859 Frances, daughter of the Rev. Peyloe Blakiston, M.D., F.R.S., of St. Leonards, who died in 1892. His son, Mr. Peyton Beale, is surgeon to King's College Hospital.

Professor Beale was elected a Fellow of this Society in 1862. He served on the Council and as a referee, and contributed one paper to the 'Transactions' on "The Diminution of the Chlorides in the Urine in Cases of Pneumonia, and on the Chemical Constitution of the Sputa in that Disease."

Dr. John Clarke was elected a Fellow of the Royal Medical and Chirurgical Society in the year 1848, but he had for so long given up practice and lived in retirement that he was probably known to but few of our present Fellows.

Dr. W. H. Dickinson has kindly furnished me with the following notice:

"Dr. John Clarke, M.D.St. Andrews, F.R.C.P., was the second son of Sir Charles Clarke, the first baronet of that name. The subject of this notice was born in August, 1823, and died last Easter, having not quite completed his 83rd year.

"He was educated at Eton and at St. George's Hospital, to which his father had been attached as Lecturer on Midwifery. He adopted the line of practice in which Sir Charles had been eminent, and became physician to the Lying-in Hospital, York Road, and in 1866 succeeded Dr. Robert Lee at St. George's Hospital as Obstetric Physician and Lecturer on Midwifery. In 1873 he was made Examiner in Midwifery and the Diseases of Women at the College of Physicians.

"In 1875 an abrupt and complete breakdown in health compelled him, greatly to the regret of his colleagues, his class, and his patients, to retire from all his appointments and professional work, and that at a time when a considerable and increasing practice gave promise of much more.

"What laid him up was a severe and persistent attack of thrombosis, or, as it used to be called, phlebitis, which involved both legs, and soon became complicated with some intra-cranial mischief thought to be of the same nature, added to which he became afflicted with eczema, which was widely diffused, and which continued to trouble him to the end of his life. Both affections were attributed to gout, from which his father had also suffered, and which in the view of some of his relations was aggravated by the loss of walking exercise when he set up his carriage. From this combination of ills he

never completely recovered, nor was he ever able to resume practice. Thus at the age of fifty-three a successful and promising professional career was brought to an end. The immediate cause of his death was a fracture of the thigh caused by a fall which seemed insufficient to produce it.

"Dr. John Clarke had all the characteristics which are associated with the term *gentleman*, in whatever sense it be applied. The word is used, as Dr. Johnson tells us, with two different meanings, one implying "exterior grace, the other honour."[1] Dr. Clarke had both. His refined face, his tall and graceful figure, always well but never conspicuously dressed, together with his courtesy of address, could not fail to impress, in the former sense, the most casual acquaintance; while as regards the latter, those who knew him well were aware of the sterling excellencies, uprightness, and trustworthiness which lay beneath the attractive exterior."

John Henry Bryant, M.D.Lond., F.R.C.P., was Assistant Physician and Lecturer on Materia Medica and Therapeutics at Guy's Hospital, and his untimely death at the age of thirty-eight removed from the staff of that Hospital and Medical School an able Physician and brilliant teacher whose loss was greatly regretted by all who knew him.

Dr. Bryant was born at Ilminster in Somersetshire, and was educated at the Grammar School of that place, and at Sherborne School. He entered Guy's Hospital in 1886, where he won numerous prizes, among which were the Treasurer's Gold Medals in Medicine and Surgery. He graduated M.B. and B.S. at the London University in 1890, obtaining First Class Honours and the Gold Medal in Medicine. He became M.D. in 1891, M.R.C.P. in 1895, and F.R.C.P. in 1901; he was also a M.R.C.S., and was elected a Fellow of this Society in 1898. At Guy's Hospital he held the offices of House

[1] Boswell, vol. ii, p. 328.

Physician, Resident Obstetric Assistant, and Medical Registrar, and in 1898 was appointed an Assistant Physician to the Hospital. In the School he was Demonstrator of Morbid Anatomy and Lecturer on Materia Medica and Therapeutics. He was also one of the Editors of the 'Guy's Hospital Reports.'

Dr. Bryant contributed many valuable papers to the 'Guy's Hospital Reports,' and to the medical journals; among the more important being those on "Suppurative Pylephlebitis," on "Functional Pulmonary Incompetence and Atheroma of the Pulmonary Arteries," on "Bacteria in Thrombi," and on "A case of Obliterative Endarteritis in a Child."

He also wrote for 'Allchin's System of Medicine' the article on "Diseases of the Abdominal Blood-vessels," in which he made good use of the abundant material furnished by the Guy's Hospital records.

Dr. Bryant was greatly esteemed as a teacher, sparing no pains to make the subject intelligible and attractive to the student, and taking the greatest care to ensure accuracy of statement.

His clinical work was also characterised by the most systematic thoroughness, combined with a sympathetic insight into the patient as well as the disease. His charm of manner, sincerity, and broad-mindedness combined to make a most attractive character.

Surgeon-Major John William Trotter, M.R.C.S., L.S.A., was the eldest son of the late Dr. John Trotter, of Durham. He was Resident Medical Officer at St. Mary's Hospital from 1851 to 1854, joined the Coldstream Guards in 1854 as Assistant Surgeon, served in the Crimean campaign, and was present at the fall of Sebastopol.

In 1874 he was promoted Surgeon-Major, and transferred to the Scots Fusilier Guards. A few years later, having met with a severe injury the result of a fall from his horse, he retired from the army.

In 1882 Mr. Trotter married Mary Ovington, only

daughter of Mr. Robert Chilton, of Billingham Grange, Stockton-on-Tees, who survives him. After his marriage he lived in York, where he died on June 14th, 1906.

Eldred Noble Smith, F.R.C.S.Edin., L.R.C.P., died at his home in Queen Anne Street, on July 20th, 1906, in the fifty-ninth year of his age. He was the son of Mr. Thomas Smith, of Hertford, a well-known architect, from whom he inherited a mechanical skill which proved very useful to him in his orthopædic practice, and also a facility of drawing which enabled him to illustrate numerous medical publications.

He received his medical education at St. Mary's Hospital, where, after obtaining the M.R.C.S. and L.R.C.P., he held the office of House Surgeon. He was afterwards Resident Medical Officer to the Lock Hospital, and House Surgeon to the Bristol Hospital for Children.

After engaging for some years in general practice he obtained, in 1880, the F.R.C.S. of Edinburgh, and thenceforward devoted himself to Orthopædic Surgery. He was for many years Surgeon to the City Orthopædic Hospital and to the All Saints' Children's Hospital, and also Orthopædic Surgeon to the British Home for Incurables.

Mr. Noble Smith published a variety of works on Orthopædic Surgery, and he was associated with Dr. E. Klein in the production of the 'Atlas of Histology,' published in 1880, for which he executed with much skill the numerous elaborate illustrations.

He was for three years the Honorary Secretary to the Metropolitan Counties' Branch of the British Medical Association, of which he was also the Representative on the Central Council.

David Henry Goodsall, F.R.C.S., L.R.C.P., died suddenly from heart disease at his home in Devonshire Place, on September 14th, 1906. Mr. Goodsall was well known as an authority on diseases of the rectum, and had been for

many years Surgeon to St. Mark's Hospital for that class of diseases.

He was born on January 4th, 1843, studied at St. Bartholomew's Hospital, where he held the office of Midwifery Assistant, was admitted a Member of the Royal College of Surgeons in 1868 and became a Fellow of the College in 1872; he was also a Licentiate of the Royal College of Physicians.

In 1871 he became Assistant Surgeon, and in 1888 Surgeon to St. Mark's Hospital, and in 1872 Surgeon to the Metropolitan Hospital, Kingsland Road. He was also Surgeon to St. Saviour's Hospital.

Mr. Goodsall took great interest in all these institutions, and did much excellent work in connection with them. He was elected a Fellow of the Royal Medical and Chirurgical Society in 1889, and was also a Fellow of the Medical Society, of the Hunterian Society, and of the Statistical Society, and a Member of the Clinical Society. At the Medical Society he was for nineteen years Chairman of the House and Finance Committee, and for ten years Honorary Treasurer, in which capacity he took an active part in the negotiations with regard to the proposed union of Medical Societies.

Mr. Goodsall published, with Mr. W. E. Miles, a work on 'Diseases of the Anus and Rectum,' and he contributed numerous papers on allied subjects to the journals. He was a man of much energy and vigour, and his sudden death came as an unexpected blow to his friends and colleagues.

William Spencer Watson, M.B., F.R.C.S., died on September 17th, 1906, at the age of seventy. He was the son of Dr. John Watson, who practised for many years in Bloomsbury. He entered as a medical student at King's College, London, where he obtained an Entrance Scholarship, and subsequently the Warneford Scholarship in Medicine.

He was elected an Associate of King's College in 1857,

graduated M.B. with Honours at the London University in 1860, and obtained the F.R.C.S. England in 1862. He was for many years Surgeon to the Central London Ophthalmic Hospital, to the Royal Eye Hospital, and to the Throat Department of the Great Northern Hospital, and was Medical Examiner to the Westminster and General Life Assurance Association.

Mr. Spencer Watson was elected a Fellow of this Society in 1861. He served on the Council in 1883–4, and contributed a paper to our 'Transactions' on "A case of Acute Inflammation of the Vitreous Humour of both Eyes." He was also a member of the Medical and of the Ophthalmological Societies, and was the author of works on 'Diseases of the Nose and Accessory Passages,' on 'Abscess and Tumours of the Orbit,' on 'Keratitis,' and on 'Eyeball Tension,' besides papers on allied subjects contributed to the journals.

Mr. Watson retired from practice a few years since owing to failing health, and unfortunately soon afterwards became blind. He was a member of the Court of the Sadlers' Company, in which he took great interest, and of which he had been Master.

Brigade-Surgeon-Lieut.-Colonel Alexander Crombie, C.B., M.D.Edin., L.R.C.S.Edin., and L.S.A., who died on September 29th, 1906, was a distinguished member of the Indian Medical Service, which he entered in 1872, and in which he served till 1899.

Dr. Crombie, who was the youngest son of Mr. David G. Crombie, of Kilmenning, Fifeshire, was born in December, 1845. He was educated at St. Andrews and Edinburgh, and graduated with honours at the University of Edinburgh.

In India Dr. Crombie held many important offices. He was appointed Lecturer on Surgery at the Calcutta Medical College in 1873, Superintendent of the Medical School at Dacca in 1877, and Surgeon-Superintendent of the European General Hospital at Calcutta in 1888; he

was also a Fellow of the University of Calcutta. Dr. Crombie engaged in consulting practice in Calcutta and subsequently in London; he was a member of the Medical Board at the India Office, was Physician to King Edward VII's Hospital, and Lecturer on Tropical Diseases at the London School of Tropical Medicine. He was made a Companion of the Bath in 1902.

Besides many other contributions to the literature of Medicine Dr. Crombie wrote the articles on "Sprue," "Hill Diarrhœa," and "Abscess of the Liver" in 'Allchin's Manual of Medicine.'

Dr. Crombie was a Fellow of the Medical Society of London, and was elected a Fellow of the Royal Medical and Chirurgical Society in 1898.

Henry Walter Syers, M.A., M.D.Cantab., M.R.C.P., died on October 10th, 1906, at the age of 54, the victim of a poisoned wound contracted in the post-mortem room. He was born at Kenilworth in October, 1852, entered at Gonville and Caius College, Cambridge, graduated in the Natural Science Tripos, and proceeded M.A. and M.D. He studied medicine at Birmingham and at Westminster Hospital, and in 1883 was admitted a member of the Royal College of Physicians. He held the offices of Medical Registrar and Tutor at Westminster Hospital, and Resident Clinical Assistant at Bethlem Hospital; in 1888 he was elected to the staff of the Great Northern Central Hospital, to which he became Physician in 1905.

Dr. Syers was elected a Fellow of this Society in 1890, was also a Fellow of the Medical Society, a member of the Neurological Society, and President of the North London Medical and Chirurgical Society.

Dr. Syers published in 1901 a treatise on the 'Theory and Practice of Medicine.' He was the author of 'Statistical Reports of the Westminster Hospital for 1885–7,' and of other contributions to the medical journals, and of translations of Morat's 'Physiology of

the Nervous System,' and of Laumonier's 'New Methods of Treatment.'

He was an enthusiastic worker both at the bedside and in the post-mortem room, and interested in all the modern scientific developments relating to medicine. He was, moreover, an excellent linguist, was well versed in archæology and in music, and was a skilful pianist.

William Sedgwick, M.R.C.S.Eng., L.S.A., died at Acton on October 21st, 1906, at the age of 85. He was born in Islington, and was one of the fourteen children of Mr. Thomas Sedgwick, a West India merchant, formerly of Dent, in Yorkshire. William Sedgwick was, I believe, related to the famous geologist, Adam Sedgwick, who came from the same Yorkshire dale, and he displayed many of the remarkable intellectual qualities which characterised his distinguished relative.

He began his medical education by apprenticeship to Dr. J. Challice, of Bruton Street, and afterwards entered as a student at University College Hospital. Here he was dresser to Mr. Liston when that great surgeon performed for the first time in London an operation during anæsthesia.

Having qualified as M.R.C.S. in 1845, he made several voyages to India and China, having the medical charge of troops. He then studied for a short time at the medical schools of Paris, and having in 1848 obtained the Diploma of the Society of Apothecaries, he commenced practice in Marylebone, where he was actively engaged during the cholera epidemic of 1854. This afforded him the opportunity of studying the disease from various points of view, especially in connection with the chemical changes involved, and led to the publication in 1856 of a book on the 'Nature of Cholera as a Guide to Treatment.'

In 1871 he contributed to the 'Lancet' an article on "Some Physiological Errors concerning Cholera," and in the volumes of our 'Transactions' for 1868, 1871, and

1886 are three elaborate papers in which Mr. Sedgwick dealt with the "Suppression of Urine in Cholera," with "Temporary Glycosuria as a Sequel to Cholera," and with "The Chemical Pathology of Respiration in Cholera," subjects to which he recurred in his Presidential Address to the Harveian Society in 1889. Mr. Sedgwick at one time devoted much attention to the study of heredity, and he published between the years 1861 and 1867 a series of most suggestive and instructive articles in the 'British and Foreign Medico-Chirurgical Review' upon the "Influence of Age and Sex in Hereditary Disease."

He also read at the meeting of the British Medical Association in 1882 an interesting paper on "Atavism," in which he quoted cases showing that the influence of atavism on the hereditary transmission of disease can be maintained for four, five, seven, or more generations; "and that when, as not unfrequently happens, two, three, four, or more children in the same family are born with the same congenital defect; or, later on in life, suffer from the same disease at the same age; that the absence of any evidence of an ancestor having been similarly affected, may be due to the remoteness of the source from which the inheritance has been derived; and that when, moreover, in such cases there is also limitation by sex, that the evidence is then very strongly in favour of the extended, but undetected, influence of atavism."

Philosophical work of this kind does not always meet with the recognition that it merits, and I suspect that these papers of Mr. Sedgwick's are now but seldom referred to, although they received appreciative notice from Mr. Darwin.[1] Like many other good pieces of work, they filtered unperceived into the scientific knowledge of the time, lying there—

"like seed,
Silently buried toward a far-off spring."

Mr. Sedgwick was elected a Fellow of this Society in

[1] 'Variation of Animals and Plants under Domestication,' 1868, vol. i, p. 404; vol. ii, pp. 4, 7, 9, 13, 14, 17, 22, 24, 34, 36, 72, 78, 326, 401.

1863, and served upon its Council in 1884–5. He was President of the Harveian Society in 1889. He was for many years Surgeon to the Marylebone Dispensary, to St. Saviour's Hospital for Women and Children, and to St. Cyprian's Orphanage.

Mr. Sedgwick married in 1869 Norah, daughter of Dr. Challice, of Bruton Street, who survives him, and he leaves two sons and a daughter.

For the last ten years Mr. Sedgwick had retired from practice and lived at Acton, where he ended a life characterised by great intellectual activity, scientific enthusiasm, and beneficent work.

Henry Howard Hayward, M.R.C.S., L.D.S., died at the age of 72, on November 13th, 1906. He was educated at University College School and St. Mary's Hospital, became M.R.C.S. in 1858, was for twenty years Dental Surgeon and Lecturer on Dental Surgery at St. Mary's Hospital, was also Dental Surgeon to the Brompton Hospital, and Assistant Surgeon to the Dental Hospital.

Mr. Hayward became a Fellow of the Royal Medical and Chirurgical Society in 1860; he was also a Fellow of the Harveian Society. He wrote on "Dental Practice in Relation to Medical Science," and also contributed to Pye's 'Surgical Handicraft.' Since about 1890 he had retired from practice and lived at Ealing.

John George Douglas Kerr, M.B., C.M., who died at Bath, November 14th, 1906, was a well-known and very popular practitioner at Bath. He was educated at Glasgow University, where he won the Gold Medal in Surgery, and graduated in 1880. After serving as House Surgeon to the Glasgow Western Infirmary, and spending some time in foreign travel, he joined the late Mr. T. G. Stockwell in practice at Bath. Here he soon became known as an authority upon the use of the waters, and his advice was sought by large numbers of those who went to Bath for treatment. Dr. Kerr was Hon. Physi-

cian to the Hospital for Women at Bath, and had been President of the British Balneological and Climatological Society and of the Pathological and Clinical Society of Bath. He was elected a Fellow of the Royal Medical and Chirurgical Society in 1890, and was also a Fellow of the British Gynæcological Society. Besides a popular guide to the Bath waters, he wrote on rheumatoid arthritis, the Nauheim treatment, and other allied subjects. Dr. Kerr was an enthusiastic sportsman, spending his holidays in deer stalking, mountain climbing, and yachting. He took an active part in municipal affairs, and was a Justice of the Peace for Somerset. In the autumn of 1905 Dr. Kerr suffered a severe attack of influenza, which left him with much impaired health, and in November, 1906, he succumbed to another attack of that disease at the early age of forty-nine. Dr. Kerr was not married.

Edmund Symes Thompson, M.D., F.R.C.P., died on November 24th, 1906. He was the son of the late Dr. Theophilus Thompson, F.R.S., was educated at St. Paul's School and King's College, and graduated M.B. of the London University, taking honours in surgery, obstetrics, and physiology, as well as the University Scholarship in Medicine.

In 1860 he became M.D., and in 1862 Member of the Royal College of Physicians, of which he was elected Fellow in 1868.

In 1860 he was elected Physician to King's College Hospital, but resigned that office in 1865 that he might devote himself specially to the study of diseases of the chest.

In 1863 he was appointed Assistant Physician, and in 1871 Physician to the Brompton Hospital, engaging there with much energy in work in which his father had been previously distinguished. This office he held till 1889, when he was made Consulting Physician.

Dr. Symes Thompson gave much attention to the

study of climate in relation to disease, and published articles on that subject. He also edited and added to the "Lectures on Pulmonary Consumption" by Dr. Theophilus Thompson.

Dr. Symes Thompson was for nearly forty years Gresham Professor of Medicine; he acted for many years as medical adviser to the Equity and Law Assurance Society; he had been President of the Harveian Society and of the Balneological and Climatological Society, and Physician to numerous charitable associations. He was elected a Fellow of this Society in 1862, and served the Society on the Council, as Secretary, and as Referee. He contributed one paper to our 'Transactions' on "The Elevated Health Resorts of the Southern Hemisphere," besides giving valuable assistance in the preparation of the 'Report on the Climates and Baths of Great Britain and Ireland.'

Dr. Symes Thompson was interested in, and gave valuable help to, many religious institutions, especially those concerned with the welfare of the poor and with the promotion of temperance. He died at his country house at Fenmere, near Buckingham, and his funeral was attended by numerous friends and colleagues, by whom he was much esteemed.

George Bagot Ferguson, M.A., M.D., M.Ch., F.R.C.S., died suddenly while performing an operation at the Cheltenham Hospital, on November 27th, 1906. He was the only son of Mr. William Bruce Ferguson, was born on January 13th, 1843, and was educated at Cheltenham College, at the University of Oxford, and at St. Bartholomew's Hospital. In 1865 he graduated B.A. with First Class Honours in Natural Science, and proceeded to the M.D. in 1875. After holding the office of House Surgeon to St. Bartholomew's Hospital he commenced practice in Cheltenham, and was soon after elected to the staff of the General Hospital, to which he was Senior Surgeon at the time of his death.

Dr. Ferguson soon attained a high reputation both among the public and his professional colleagues; his skill, his energy, his wide culture, and his unfailing kindness conducing to great popularity. He was especially esteemed as a consultant and operating surgeon. In 1892 he became a Fellow of the Royal College of Surgeons, and in 1893 M.Ch. of the University of Oxford.

Dr. Ferguson was Surgeon to Cheltenham College and other local institutions, he was a Fellow of the Royal Medical and Chirurgical and Obstetrical Societies, and had formerly been Examiner in Natural Science at Oxford. In 1901 he was President of the British Medical Association, and greatly aided the success of the meeting which was held in Cheltenham that year.

Dr. E. T. Wilson, Physician to the Cheltenham Hospital, and for many years one of Dr. Ferguson's colleagues, in an appreciative notice contributed to the press, writes:—"Coming to Cheltenham with a brilliant reputation won at Oxford and St. Bartholomew's Hospital, George Bagot Ferguson joined the Dispensary Staff in 1872, and soon displayed that indefatigable aptitude for work which has characterised him to the close of his life.

No opportunity for improving his surgical knowledge and skill was ever neglected, and during the many years of his active life the chief centres of medical activity were visited, not only in Europe, but in America; the best methods of surgical procedure were carefully studied and afterwards put into practice at home, while the knowledge acquired by wide reading and a very remarkable memory were brought to bear on the daily work of his profession —indeed, no greater proof of the freshness of his intellectual ardour could be found than the fact of his qualifying for the M.Ch. at Oxford in 1903, when the Fellowship of the Royal College of Surgeons had been already conferred upon him in the previous year. But Dr. Ferguson was not merely a surgeon; his interests extended widely into the general field of science, and each new discovery in turn claimed his special attention and study, whether

it were the X ray, or bacterial infection, or wireless telegraphy, or photography in colour, the subject would first be thoroughly mastered, and then communicated to others in a lecture of singular eloquence and perspicuity. Dr. Ferguson was a ready speaker, an admirable *raconteur*, with a keen appreciation of humour which made him welcome in any society, and no one who was present at the great gathering of the British Medical Association in Cheltenham in 1901 can forget the grace and urbanity with which he occupied the position of President, and the complete success of the meeting, due in large measure to the infectiousness of his genial and kindly personality. To his many patients his loss is simply irreparable, and many among his brethren in the profession will miss the skilled counsel on which they were wont to rely in times of difficulty and doubt, while among his immediate colleagues for so many years at the hospital it is felt that a gap has been made which it will be difficult indeed to fill."

His address to the British Medical Association was greatly admired, and I quote the following remarks from an article in the 'Times' giving an account of the meeting:—"On the whole, perhaps the most remarkable feature of the meeting has been the inaugural address delivered on Tuesday evening by the President, Dr. Ferguson, who seized the opportunity as an occasion for dealing with questions of a wider scope than those which have usually been discussed on similar occasions, and for endeavouring to impress upon his hearers, and through them upon the public, the importance of striving after a more systematic cultivation of science than has hitherto been attempted in this country, not only in order to add precision to the art of healing, and to increase our power of destroying the causes of disease, but also as a source of supply of some of the most important elements of national greatness and prosperity. . . . Dr. Ferguson made an urgent plea for such endowments of scientific education in Great Britain as would afford to our young

men at least as great facilities for study and investigation as those now to be found in nearly all other civilised countries."

Dr. Ferguson published papers upon traumatic fever, cholera, and other subjects, and contributed articles on various surgical subjects to the 'St. Bartholomew's Hospital Reports' and the medical journals.

You have heard the Report of the Council, including the Reports of the Honorary Treasurers and of the Honorary Librarians, and I think you will agree that it shows the Society to be in a condition of satisfactory activity and financial stability.

There have been 14 Fellows elected into the Society during the past year, the number of candidates for the Fellowship having, no doubt, been diminished by the known probability of an union of the Medical Societies of London.

With regard to our finances, you will observe that in spite of the heavy cost of the last volume of the 'Transactions,' our expenditure for the year was well under our income, and that we have the satisfactory surplus of assets over liabilities of more than £29,000.

In connection with the proposed union of Medical Societies some doubt was expressed as to the accuracy of the estimate of our assets, and the Medical Society of London asked to be allowed to have, at their expense, an independent valuation of the Society's premises, the valuer to be nominated by the Presidents of the two Societies. Your Council acceded to this request, and the President of the Medical Society proposed and I agreed that the valuation should be made by Messrs. Elliott, Son and Boyton, a firm well known to be experienced in the estimation of property in this neighbourhood. By the courtesy of the Medical Society we have been furnished with a copy of this report, which is eminently satisfactory, inasmuch as the value of our freehold and leasehold property is estimated at £53,275, which exceeds, by £1709,

the amount which appears in our statement of assets. Moreover, the report states that the buildings are maintained in "good substantial repair," and are "good for many years to come."

The papers read before the Society have been of great interest and value, and a discussion upon "The Operative Treatment of Non-malignant Ulcer of the Stomach and its Chief Complications, with Indications, Limitations, and Ultimate Results," introduced by Dr. Hale White and Mr. Mayo Robson, was maintained during three evenings.

This discussion was largely attended, and showed the advantage of considering this important subject from both the Medical and Chirurgical points of view.

Dr. Payne's suggestion adopted by the Council "that one ordinary meeting in each Session may be set apart for papers and discussions on the 'History and Antiquities of Medicine,' and for the exhibition of books, MSS, works of art, instruments, or other objects illustrating these topics" will doubtless lead to the production of much interesting material.

It will be remembered that Sir Richard Douglas Powell, in his Presidential Address at the Annual Meeting in 1905, raised again the question of a possible union of the Medical Societies of London.[1] The proposal was very favourably received, and a resolution was carried "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."[2] In accordance with this resolution the President of the Royal College of Physicians, Sir William Church, was asked to convene a meeting at the Royal College of Physicians to consider the proposal. This meeting was held on April 10th, 1905,[3] and, as a result, a Committee

[1] See 'Transactions,' vol. lxxxviii, p. cxxvi.

[2] See 'Transactions,' vol. lxxxviii, p. xcvii.

[3] See 'Transactions,' vol. lxxxviii, p. clxxxvii.

was appointed to make the necessary inquiries, and to draw up a scheme of union to present to the various Societies for their consideration.

The report of this Committee[1] was considered at a Special General Meeting of our Society on November 23rd, 1905,[2] and with some modification was adopted, and the Council was authorised to take the necessary steps for carrying into effect the proposed union of other Societies with the Royal Medical and Chirurgical Society.

I am glad to say that the arrangements for effecting this union and for the constitution of the new Society are approaching completion.

The course advised by Mr. Palmer, K.C., to whom the question was submitted, is that the Royal Medical and Chirurgical Society should petition the King for a supplementary Charter changing the name of the Society to the desired fresh name, modifying its regulations so as to suit the new circumstances and providing for the union with it of the other Societies. This advice has been acted upon and a Petition in due form has been sent to the office of the Privy Council.

It must, I think, be admitted that in consenting to take part in the union we are giving up some of our privileges for the welfare of the larger body which will form the new Society, but I need hardly assure the Fellows that every care has been and will be taken by your representatives on the Organising Committee to safeguard the rights of the Fellows of the Royal Medical and Chirurgical Society.

The chief matter remaining for settlement is the transference of the security for the Debenture Loan to the proposed new Society, upon which the interest of the Loan will, of course, be a first charge. The security is ample and remains unchanged, so that it would seem that there should be no insuperable difficulty as to the transference.

The Trustees for the Debenture-holders have, moreover,

[1] See 'Transactions,' vol. lxxxviii, p. ccvii.

[2] See 'Transactions,' vol. lxxxviii, p. ccxv.

been authorised by the Council to obtain a separate legal opinion as to their responsibilities in this matter.

"Alteration," says Hooker,[1] "though it be from worse to better, hath in it inconveniences, and those weighty," but I think you will agree that we ought not to grudge some trouble or temporary disturbance if the result is the furtherance of the objects for which our Society was founded.

One important difference between our profession and an ordinary trade is that while in trade competition often appears to be a healthy and beneficial stimulus, in our profession infinitely greater advantages are to be obtained from friendly co-operation.

Moreover, now that our work has become so complex and extended that some sub-division is inevitable, there is the increasing danger of contentment with a narrow and restricted view; as an antidote to which it is much to be desired that in whatever department of practice we may be engaged, we should be in touch with the work of our colleagues in other directions.

These advantages, it may reasonably be expected, will be promoted by the contemplated union. And so, gentlemen, although this may be the last Annual Meeting of the Royal Medical and Chirurgical Society as such, we shall hope that, strengthened by our association with its other constituents, widened by the incorporation of bodies of diverse interests, and invigorated by the infusion of fresh energy, we shall add to the caution and experience of age, the enthusiasm and activity of youth, and take a part in the advancement of our art not unworthy of the record of our Society during the past hundred years.

I have only further to add the expression of my indebtedness to all the Honorary Officers of the Society for their constant assistance and support, and to our Secretary, Mr. MacAlister, for his skilful and indefatigable care of all the interests of the Society.

[1] 'Eccl. Polity,' B. iv, § 14.

SPECIAL GENERAL MEETING

OF THE

ROYAL MEDICAL AND CHIRURGICAL SOCIETY OF LONDON,

HELD AT THE SOCIETY'S HOUSE, No. 20, HANOVER SQUARE,

Friday, June 14th, 1907, at 5 o'clock.

WARRINGTON HAWARD, F.R.C.S., President.

HOWARD H. TOOTH, M.D., C.M.G., } Hon. Secs.
STEPHEN PAGET, }

J. Y. W. MAC ALISTER, Secretary.

Present—45 Fellows.

The PRESIDENT.—Gentlemen, you are asked to attend this meeting to-day for the purpose of considering, and, if thought fit, of passing the following resolution, which I will read to you:

> "That the Supplemental Charter graciously granted to the Society by His Majesty the King be and the same is hereby dutifully accepted."

You will remember that we were advised by Mr. Palmer, the King's Counsel whom we consulted, that the simplest way of effecting the proposed union of societies was to petition the King to grant us a Supplemental Charter conferring the necessary powers for that purpose. We did so, and, as you are aware, the King has been graciously pleased to grant this Charter, which I have now the pleasure of presenting to you. And what I

ask you now to do is to consider, and, if you please, to pass the resolution, the terms of which I have read to you.

I move from the Chair that this resolution be accepted and passed.

The resolution was then put, and carried unanimously.

The President then continued—

And now, Gentlemen, having passed that resolution dutifully accepting the Supplemental Charter granted by the King, our Society, by virtue of the powers therein conferred, becomes the

ROYAL SOCIETY OF MEDICINE,

with new powers.

And thus the idea, first started in 1808, revived in 1860, and again in 1870, of an union of this with other Medical Societies of London has at last been realised.

Of the origin and development of the present scheme I spoke in my address to you at the Annual Meeting in March. I will only, therefore, now remind you that the question of a possible union of the Medical Societies of London was raised by my predecessor in this Chair, Sir Richard Douglas Powell, in his Presidential Address in 1905.[1] The suggestion was very favourably received, and a resolution was carried "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."[2] In accordance with this resolution the President of the Royal College of Physicians, Sir William Church, was asked to convene a meeting at the Royal College of Physicians to consider the proposal. This meeting was held on April 10th, 1905,[3] and, as a result, a Committee was appointed to

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make the necessary inquiries, and to draw up a scheme of union to present to the various Societies for their consideration.

The report of this Committee[1] was considered at a Special General Meeting of our Society on November 23rd, 1905,[2] and with some modification was adopted, and the Council was authorised to take the necessary steps for carrying into effect the proposed union of other Societies with the Royal Medical and Chirurgical Society.

We thereupon petitioned the King for a Supplemental Charter changing the name of the Society to the desired new name, modifying its regulations so as to suit the new circumstances and providing for the union with it of the other societies, and this the King was graciously pleased to grant.

Fourteen other societies eventually agreed to join, and a committee was formed of representatives of these societies, with Sir William Church as Chairman, to arrange the terms of union, the new constitution of the Society, and the by-laws by which it should be governed.

This Committee has recently completed its labours, and the resolutions for carrying its recommendations into effect will be submitted to the meeting of Fellows to be held immediately in the next room.

This will therefore be the last meeting of the Royal Medical and Chirurgical Society, under the existing constitution.

Gentlemen, there would be something a little melancholy in contemplating the dissolution of a society which had an honourable record. But our Society is not dying, it is only joining hands with a number of other societies to pass into a fresh stage of existence, wherein we may hope that its good work will still be recognisable and long maintained.

Two years ago, when the Society attained its hundredth year, you received the admirable record of its

[1] See 'Transactions,' vol. lxxxviii, p. ccvii.

[2] See 'Transactions,' vol. lxxxviii, p. ccxv.

history and work produced by the joint labours of Dr. Norman Moore and Mr. Stephen Paget, so that there is no need for me to attempt any such retrospect.

But a perusual of that volume will afford you convincing evidence of the usefulness of our Society, for you will see that our 'Transactions' contain the record of many of the most important contributions made to medicine and surgery during the last hundred years.

It seems to me, indeed, that those ninety volumes will form the best possible memorial of our Society's life.

Gentlemen, I shall always look upon it as the greatest honour of my professional life to have been elected to serve as your President, and I desire to acknowledge most gratefully the courteous assistance which I have invariably received from the Honorary Officers and Council, the kindly support accorded me by the Fellows of the Society, and the ever ready help of our able and enthusiastic Secretary, Mr. MacAlister.

[*Note.—At the close of the meeting the President and Fellows passed into the adjoining room to take part in the First General Meeting under the new constitution.* See page cxlviii.]

OBITUARY

OF FELLOWS FROM 1ST MARCH TO 14TH JUNE, 1907.

(Prepared by the President.)

Since the Annual Meeting in March, 1907, there have died two Honorary Fellows and six Fellows, of whom a brief record follows.

Geheimrath Professor Ernst von Bergmann, a Foreign Honorary Fellow of the Society, died on March 25th, in his seventy-first year.

A notable figure in the medical circles of Berlin and Professor of Surgery in the University, Professor von Bergmann was the most distinguished representative of the medical profession in the German Empire. He was born in Riga and educated in the Russian University of Dorpat, where in 1864 he obtained a teaching appointment. In 1866 he volunteered as surgeon with the Prussian army in the Bohemian campaign. He was actively engaged in the German medical service during the Franco-German war of 1870, and in 1877 he served with the Russian army of the Danube in the war with Turkey; so that he had altogether an extensive experience of military surgery, in which he acquired a great reputation. He held the Professorship of Surgery at Dorpat, and subsequently at Würzburg, and on the death of Langenbeck was appointed to the chair of surgery in the University of Berlin. Early in his career he had been greatly interested in the study of wound infection, and on the publication of Lord Lister's work he appreciated at once its great importance, and advocated and practised Listerian methods in military surgery. Subsequently he adopted the purely aseptic treatment of wounds, relying upon the exclusion of infection by the careful sterilisation

of instruments and dressings and the precautions necessary for surgical cleanliness.

Professor von Bergmann made many valuable contributions to the literature of surgery, especially that connected with the treatment of wounds, and the surgery of the brain and skull.

When in 1887 the Emperor Frederick, then Crown Prince, developed symptoms of malignant disease of the larynx, Professor von Bergmann was summoned to take charge of the illustrious patient, upon whom he proposed to operate. Sir Morell Mackenzie was, however, called in, and threw some doubt upon the diagnosis, whereupon a painful controversy ensued. The correctness of von Bergmann's opinion was entirely confirmed by the subsequent course of the disease.

von Bergmann was the recipient of many honours, both in his own and other countries, besides many academic distinctions, and was a member of the Upper House of the Prussian Diet. When last year he celebrated the seventieth anniversary of his birthday, the congratulations of his many friends and admirers were striking evidence of the high esteem in which he was held.

He was an Honorary Fellow of the Royal College of Surgeons of England, and also of the Royal Medical and Chirurgical Society.

Robert Barnes, M.D., F.R.C.P., F.R.C.S., an Honorary Fellow of the Society, died at his home in Eastbourne on May 12th, 1907, in his ninetieth year.

Dr. Barnes was born in Norwich on September 4th, 1817. He was the son of Philip Barnes, an architect, who was one of the founders of the Royal Botanic Society of London.

His early education was at Bruges, where he was at school from the age of nine to thirteen. When fifteen he was apprenticed to Dr. Richard Griffin, of Norwich, and subsequently entered as a medical student in London, at University College, St. George's Hospital, and the Great

Windmill Street School. In 1842 he became a Member of the College of Surgeons, and after a year spent in Paris he entered upon practice at Notting Hill. In 1848 he obtained the M.D. of London, and in 1859 was elected a Fellow of the College of Physicians. He soon became known in the department of obstetrics, to which he thenceforth devoted himself, and he subsequently had the distinction of holding the office of Obstetric Physician and Lecturer on Midwifery at three of the great London hospitals—first, at the London; secondly, at St. Thomas's; and lastly, at St. George's Hospital.

Dr. Barnes was an admirable teacher, emphatic, clear, and definite. His lectures, which were based upon a large and maturely considered experience, displayed also a profound knowledge of anatomy and pathology; his delivery was fluent, his language well chosen, and his material carefully arranged. The lectures were subsequently published, and the volume on obstetric operations was translated into many other languages, bringing him a world-wide fame.

Dr. Barnes advocated and practised obstetric surgery as well as medicine, and in 1883 was made an Honorary Fellow of the Royal College of Surgeons of England.

He took an active part in the foundation of the Obstetrical Society of London and of the British Gynæcological Society, and he made numerous valuable contributions to the 'Transactions' of these and other Societies.

Dr. Barnes was elected a Fellow of the Royal Medical and Chirurgical Society in 1861, and served the Society on the Council, as Referee, and as Vice-President; at the Centenary of the Society in 1905 he was elected an Honorary Fellow, a distinction which he highly valued.

He was also a Fellow, and the recipient of Honorary distinctions, of many scientific societies both British and foreign.

Dr. Barnes was a man of great energy, both physical and intellectual; he learned Spanish and studied Spanish literature when over eighty-five, and until within a year

of his death was accustomed to row out to sea and dive from the boat for his morning swim. He had many and wide interests; he formed decided opinions and expressed them vigorously; he was a genial companion and a generous friend.

Dr. Barnes was twice married, first to Miss Eliza Fawkener, and secondly to Miss Alice Maria Hughes, who survives him.

Allan MacFadyen, M.D., C.M.Edin., M.R.C.S.Eng., died March 1st, 1906, aged forty-six.

Dr. MacFadyen was well known as a distinguished bacteriologist, and held until recently the office of Director of the Lister Institute of Preventive Medicine, to which he was appointed on its completion in 1891. He took great interest in the foundation of what was then called the Jenner Institute, and prepared the plans for the laboratories after studying similar institutions on the Continent.

As a result of his investigations into the life history of bacteria and the conditions of their growth, he made many valuable contributions to this branch of science and contributed numerous papers to the Royal and other Societies.

Dr. MacFadyen was born at Glasgow, and educated at Edinburgh, he had also studied at Göttingen, Berne, and Munich.

He held the Fullerian Professorship of Physiology at the Royal Institution from 1901 to 1904, and was Sanitary Research Scholar of the Grocers' Company from 1889 till 1902. He was elected a Fellow of the Royal Medical and Chirurgical Society in 1898.

Arthur Ernest Sansom, M.D.Lond., F.R.C.P., Consulting Physician to the London Hospital, and to the North-Eastern Hospital for Children, was born at Corsham, Wiltshire, in 1838; he received his medical education at King's College, London, where, besides being Warneford

Scholar, he obtained other distinctions. He also studied in Paris.

After graduating at the University of London Dr. Sansom was appointed to the Staff of the London Hospital, and also to that of the North-Eastern Hospital for Children; he was elected a Fellow of the Royal College of Physicians, a Fellow of King's College, a Fellow of the Royal Medical and Chirurgical Society, and of many other English and foreign scientific societies. He was President of the Medical Society of London in 1897; and had brought before that Society, in 1870, a paper in which he maintained that putrefaction and fermentation were due to the influence of living, growing, and multiplying material; in this paper, and in a book on the "Antiseptic System," published in 1871, he drew attention to the importance of the researches of Pasteur and Lister.

Dr. Sansom also wrote on the action and administration of chloroform, but his best known works were upon diseases of the heart, on which he delivered the Lettsomian Lectures.

Dr. Sansom was greatly respected as a physician, and his charming manner and genial kindness made him a general favourite with both the profession and the public.

He retired from practice two or three years before his death, which occurred at Bournemouth on March 10th, 1906.

Willian Henry Ransom, M.D., F.R.C.P., F.R.S., died on April 16th, 1907, aged eighty-three.

Dr. Ransom was Consulting Physician to the Nottingham General Hospital, an institution in which he had taken great interest, and which he had served for thirty-six years as Physician. He had been for many years one of the leading physicians of Nottingham, but had retired from practice, and was, during the last three years, somewhat of an invalid.

He was born at Cromer, educated at Norwich, and,

after an apprenticeship at King's Lynn, pursued his medical studies at University College, London, and in France and Germany.

In 1850 he obtained the M.D. of the University of London, and commenced practice in Nottingham. In 1854 he was elected a Fellow of the Royal Medical and Chirurgical Society, in 1869 a Fellow of the Royal College of Physicians, and in 1870 a Fellow of the Royal Society.

Dr. Ransom was a man of great energy and intellectual power, and in addition to the labours of a large practice he engaged in biological and geological researches. He contributed a paper to our Society upon "Actinomycosis of the Vermiform Appendix," and he wrote an article on "Intestinal Worms" for 'Reynolds' System of Medicine.' His investigations in embryology were communicated to and published by the Royal Society.

In 1892 Dr. Ransom was President of the Medical Section of the British Medical Association which met at Nottingham, when he delivered an address upon "Vegetable Pathology," a subject to which he had given much attention.

In 1905 he published an interesting essay on "The Inflammation Idea in General Pathology," in which again he illustrated the subject by studies of the processes of inflammation and repair in plants and animals, and maintained that inflammation "is a casualty, a disease, and not an evolved faculty, as is repair."

He took, moreover, an active interest in politics and education, and was concerned in the government of the University College of Nottingham, and of the High School. He had also served in the Volunteer Corps.

Dr. Ransom was held in high regard both as a Physician and as a citizen, and Nottingham and its Hospital owed much to him.

In 1860 Dr. Ransom married Miss Elizabeth Bramwell, who died some years since; one of his sons is now Senior Physician to the Nottingham General Hospital.

Jonathan Forster Christian Horace Macready, F.R.C.S., died on April 26th, 1907.

Mr. Macready, who was born in 1850, was a son of the celebrated actor, William Charles Macready. He received his professional education at St. Bartholomew's Hospital, where he was distinguished by his industry and ability.

In 1878 he was elected to the staff of the Great Northern Hospital, where for many years he did valuable work, both as Surgeon and as Member of the Managing Committee. He was also Surgeon to the City of London Hospital for Diseases of the Chest, to the Cheyne Hospital for Children, and to the Truss Society.

Mr. Macready wrote a comprehensive and admirably illustrated 'Treatise on Ruptures,' and papers on "Contraction of the Palmar Fascia."

He was elected a Fellow of the Royal Medical and Chirurgical Society in 1881, and was a Fellow of the Medical Society and a Member of the Clinical Society of London.

Kind, courteous, and sagacious, Mr. Macready was held in high regard by his colleagues and all who knew him.

Sir Joseph Fayrer, Bart., K.C.S.I., M.D.Edin., LL.D. Edin. and St. And., F.R.C.P.Lond., F.R.C.S.Eng. and Edin., F.R.S.Lond. and Edin., Physician Extraordinary to the King.

This distinguished member of the medical profession and of the Indian Medical Service died at his home in Falmouth on May 21st, 1907, at the age of 82.

He was the second son of Robert John Fayrer, a Commander in the Royal Navy, and was born at Plymouth on December 6th, 1824. He was educated in Ayrshire and at Liverpool, and, after a short period of service at sea, he began the study of medicine in 1844 at Charing Cross Hospital, where he obtained a scholarship. In 1847 he became a Member of the Royal College of Surgeons, and shortly afterwards was given a commission

in the Navy. During the next year he travelled over Europe with Lord and Lady Mount-Edgcumbe, and, in 1849, while in Rome, he, with characteristic energy, took the opportunity of attending the hospitals and the University, of which he obtained, after examination, the degree of M.D. Here he also made acquaintance with Garibaldi, and saw some of the fighting in the memorable defence of Rome against the French. Soon after returning to England in 1850 Fayrer was sent to India in charge of troops. This was the beginning of the career in India in which he carried out the splendid work which brought him a great reputation and many honours, and which made him one of the heroes of the Mutiny.

After some active service in Burmah Fayrer was appointed Residency Surgeon at Lucknow, where, and elsewhere during the Indian Mutiny, he rendered splendid service. He took an active part in the heroic defence of Lucknow, and attended Sir Henry Lawrence when he received his fatal wound.

In 1859, while on furlough in England, he took the M.D. of the University of Edinburgh, and shortly afterwards was appointed Professor of Surgery in the Medical College, Calcutta, and Surgeon to the Hospital. In 1861 he was elected a Fellow, and in 1863 a Member of the Senate of the University, and a President of the Faculty of Medicine.

In 1868 Dr. Fayrer was made a Companion of the Star of India, and in 1870 he travelled on the staff of the Duke of Edinburgh through the North-West Provinces. He was subsequently appointed Physician to the Duke and Honorary Physician to the Queen. In 1872 Dr. Fayrer returned to England, when he was elected a Fellow of the Royal College of Physicians, and was appointed a Member of the Medical Board of the India Office, of which he became President in 1874.

In 1875 he accompanied the Prince of Wales during his visit to India, when he was made a Knight Commander of the Star of India.

On his return to England he settled in London in consulting practice, and was appointed a Consulting Physician to Charing Cross Hospital, where he lectured on tropical diseases. He was also an Examiner for the Army Medical Service, and a Member of the Senate of the Army Medical School, in which he took great interest.

Sir Joseph Fayrer was elected a Fellow of the Royal Society in 1877; he was already a Fellow of the Royal Society of Edinburgh, and had received many other academic honours. He was a member of many learned and scientific societies, British and foreign; he joined the Royal Medical and Chirurgical Society in 1872; he also held the Presidency of the Medical Society, of the Epidemiological Society, and of the Asiatic Society of Bengal.

He delivered the Croonian Lectures at the College of Physicians in 1882 on the "Climate and Fevers of India," and published numerous papers and articles on tropical diseases.

Fayrer had, moreover, been known in India as an accomplished surgeon and brilliant operator, and had written valuable articles on surgical subjects, notably on osteo-myelitis, and on thrombosis after surgical operations. He was elected a Fellow of the Royal College of Surgeons of England in 1878.

He was a keen naturalist as shown by his splendid works on the 'Thanatophidia of India,' and on the 'Natural History of the Tiger,' and he was also a fine sportsman.

Besides his many other accomplishments Fayrer was an excellent linguist, and had wide literary knowledge.

In 1895 he retired from the India Office, and his portrait was painted by subscription and presented to Netley Hospital. In 1896 he was made a Baronet.

Sir Joseph Fayrer was a man of most attractive character. His varied experience and knowledge made him a delightful and interesting companion; his strong sense of right and justice, his contempt for all affectation,

and his kindness of heart, made him a generous and reliable friend. He was a brave soldier, a dexterous surgeon, a sagacious physician, and an excellent administrator. He was held in high regard by all who knew him.

Sir Joseph Fayrer was married in India in 1855 to Bethia Maria, daughter of General Andrew Spens. His three surviving sons are all in the service of the country; the present Baronet, Lieutenant-Colonel Sir Joseph Fayrer, R.A.M.C., is Medical Officer to the Duke of York's Royal Military School.

In 1900 Sir Joseph Fayrer published an interesting volume entitled 'Recollections of my Life,' which contains an excellent and characteristic portrait. The book is a modest record of a life full of varied interests and beneficent work, and includes an account of the memorable siege and defence of Lucknow, besides many other autobiographical details.

Julius Dreschfeld M.D., F.R.C.P., B.Sc., Professor of Medicine in the Victoria University, and Consulting Physician to the Royal Infirmary, Manchester, died June 13th, 1907, aged sixty-two.

Dr. Dreschfeld occupied a distinguished position at Manchester as a physician whose advice was greatly valued and in much request, and he had also a wide reputation in the various sciences connected with medicine.

He was born at Niederwerm, in Bavaria, in 1845, and after receiving his early education at Bamberg he went to Manchester and became a student at Owens College and the Manchester Royal School of Medicine, where he displayed great industry and ability.

In 1864 he returned to Bavaria and studied at Würzburg under Kölliker and von Bezold, taking the degree of M.D. in that University in 1867. During the Austro-Prussian War he served as an Assistant Surgeon in the Bavarian Army.

In 1869 he returned to England, obtained the L.R.C.P.,

and entered upon practice in Manchester. In 1875 he became a member and in 1883 was elected a Fellow of the Royal College of Physicians where, in 1888, he delivered the Bradshaw Lecture on "Diabetic Coma." In 1873 he was elected Assistant Physician to the Manchester Royal Infirmary, to which he became Physician in 1883, and Consulting Physician in 1905.

Dr. Dreschfeld arranged and catalogued the pathological specimens in the Museum of Owens College, where he lectured on pathology, and in 1891 he was appointed Professor of Medicine in the Victoria University.

He also held the office of Examiner at the Royal College of Physicians and at the University of Cambridge.

Dr. Dreschfeld was an active member of the medical societies of Manchester, and was elected a Fellow of the Royal Medical and Chirurgical Society in 1898. He was President of the Medical Section of the British Medical Association at its meeting in Manchester in 1902.

Dr. Dreschfeld, besides being a most successful teacher and lecturer, made many valuable contributions to the literature of medicine. In addition to papers on pathology, especially that of the nervous system, he wrote clinical articles on affections of the heart, on Graves' disease, on locomotor ataxy, and other important subjects. He also furnished many admirable articles to 'Allbutt's System of Medicine.' Dr. Dreschfeld was to have delivered the Lumleian Lectures at the Royal College of Physicians next year. He may be said to have died in harness, working hard to the last; and the high esteem in which he was held was manifested by the very large attendance at his funeral, at which, besides his colleagues, friends, and pupils, there were many representatives of the University, of scientific societies, of medical charities, and of other public bodies.

Dr. Dreschfeld was twice married, and leaves a widow and four children.

UNION OF MEDICAL SOCIETIES

In further continuation of the record of proceedings and negotiations begun in *Med.-Chir. Trans., vol. lxxxviii*, continued in *vol. lxxxix*, and now happily concluded, the Hon. Secretaries of the Committee of Representatives (Dr. Latham and Mr. Pendlebury) have kindly furnished the following:

REPORT OF THE COMMITTEE OF REPRESENTATIVES.

(1) The first meeting of the representatives, appointed by the Amalgamating Societies under the terms of the Report of the Organising Committee adopted in July, 1906, was held at 20, Hanover Square, on January 18th, 1907.

The Societies who agreed to amalgamate on the lines laid down in the Report were:

(1) Royal Medical and Chirurgical Society.
(2) Pathological Society.
(3) Epidemiological Society.
(4) Odontological Society.
(5) Obstetrical Society.
(6) Clinical Society.
(7) Dermatological Society of London.
(8) British Gynæcological Society.
(9) Neurological Society.
(10) British Laryngological, Rhinological, and Otological Association.
(11) Laryngological Society.
(12) Dermatological Society of Great Britain and Ireland.
(13) British Electro-Therapeutic Society.

Subsequently the Therapeutical Society decided to join the amalgamation. The Otological Society will decide whether it will join on June 10th.

(2) The following delegates of societies formed the Committee:

Sir William Church . .	Royal Medical and Chirurgical Society.
Mr. Warrington Haward .	
Dr. Garrod . . .	Pathological Society.
*Dr. Whitelegge . . .	Epidemiological Society.

* After March 15th the Epidemiological Society was represented by Dr. Bulstrode.

Mr. Mummery . . .	Odontological Society.
Dr. Champneys . . .	Obstetrical Society.
Mr. Makins . . .	Clinical Society.
Dr. Galloway . . .	Dermatological Society of London.
Dr. H. Macnaughton-Jones	British Gynæcological Society.
Dr. Henry Head . .	Neurological Society.
Mr. Chichele Nourse . .	British Laryngological, Rhinological, and Otological Association.
Sir Felix Semon . .	Laryngological Society.
Dr. Stowers . . .	Dermatological Society of Great Britain and Ireland.
Dr. Lewis Jones . .	British Electro - Therapeutic Society.
Dr. T. E. B. Brown . .	Therapeutical Society.

Dr. Frederick Taylor was appointed by the Royal Medical and Chirurgical Society as their financial representative; and Mr. Lloyd Williams was appointed by the Odontological Society as their financial representative. These gentlemen were co-opted as members of the Committee.

In addition, it was decided to co-opt as members of the Committee:

The President of the Royal College of Physicians,
The President of the Royal College of Surgeons,
The Dean of the Faculty of Medicine of the University of London,
Dr. Allchin,
Mr. T. Laurence Read.

Sir William Church was elected Chairman of the Committee; Dr. Latham and Mr. Pendlebury were elected Honorary Secretaries; Dr. Nachbar was appointed Secretary.

(3) A general meeting of members of the amalgamating societies was held at the Royal College of Physicians on February 19th to consider the name of the new Society, and it was decided to petition for a Royal Charter under the title of "The Royal Society of Medicine."

(4) HIS MAJESTY THE KING has graciously granted a Charter to The Royal Society of Medicine.

(5) A Sub-Committee, consisting of the Chairman and the Honorary Secretaries, was appointed to frame the By-laws of the new Society. These were subsequently considered and amended by the whole Committee at successive weekly meetings, and afterwards submitted to the solicitor, Mr. Lithgow. The changes recommended by the solicitor were then considered by the Committee, and the By-laws finally revised and adopted as now printed.

(6) A Sub-Committee on Finance, consisting of Mr. Haward,

Dr. F. Taylor, Dr. Head, and the Honorary Secretaries, was appointed to draw up a schedule of the assets and liabilities of the new Society, and an estimate of income and expenditure. These were adopted by the Committee, and are appended to this Report.

(7) The Honorary Secretaries sent a circular letter to each member of the amalgamating Societies, asking in what capacity he wished to join the new Society, whether as a Fellow or as a Member of one or more Sections, and enclosed a printed post-card for the reply.

An analysis of the answers received is given in the Report of the Finance Sub-Committee.

(8) An Advisory Committee, consisting of the Chairman, Dr. Head, and the Honorary Secretaries, was appointed to make such arrangements as they might deem necessary to meet the working requirements of the new Society.

(9) It was decided to hold the first meeting of the Royal Society of Medicine on Friday, June 14th:

(*a*) To pass a resolution constituting the Royal Society of Medicine.

(*b*) To pass a resolution concerning the Charter.

(*c*) To approve the By-laws.

(*d*) To appoint the President and Honorary Officers of the Society and such members of the Council as require election, not being Presidents of any Section.

(Signed) W. S. CHURCH, *Chairman.*
ARTHUR LATHAM, } *Honorary*
HERBERT S. PENDLEBURY } *Secretaries.*

May 31st, 1907.

REPORT OF THE SUB-COMMITTEE ON FINANCE.

Estimated Income.

		£ s. d.	£ s. d.
1.	*From Subscriptions		5094 9 0
2.	From Investments:		
	R.M.C.S. Gant Bequest (Library) ...	16 19 8	
	R.M.C.S. Permanent Endowment Fund	12 11 4	
	Gynæcological Society	10 9 2	
	Clinical Society ... ... (say)	7 0 0	
	Obstetrical Society	134 17 3	
	Pathological Society ... ... (say)	15 0 0	
	Odontological Society	123 10 0	
			320 7 5
3.	Rents R.M.C.S., at present	2830 0 0	
	(*vide* Reports, July, 1906)		2000 0 0
4.	Entrance Fees, at present	208 19 0	say 150 0 0
5.	Composition Fees, at present	97 13 0	„ 50 0 0
6.	Sale of Proceedings, at present	210 14 0	„ 100 0 0
			£7714 16 5

* This sum of £5094 represents subscriptions actually promised, in

Estimated Expenditure.

	£	s.	d.
Debenture Interest, R.M.C.S.	996	0	0
Maintenance and Salaries (Report of 1906)	2150	0	0
Library	454	6	0
Proceedings	1773	0	0
Extra Printing and Meeting Expenses, at present £368 ...	500	0	0
Addition to Maintenance and Salaries	550	0	0
Addition to Library Expenses	350	0	0
Balance	941	1	5
	£7714	7	5

Analysis of Answers received to Circular Letter.

Number of post cards issued, 2866:

Wish to become Fellows	1443	
Wish to become Members of one or more Sections ...	443	
Have Compounded	104	
Dead, refusals, no address, etc.	122	
Not answered	754	
	2866	
1443 Fellows at £3 3s.	£4545	9 0
443 Members of one or more Sections	476	3 6
104 Compounders	72	16 6
	£5094	9 0

Assets.

	£	s.	d.
1. R.M C.S.			
*Freehold and Leasehold Property, 20, Hanover Square, valuation 1896	51,150	0	0
Engravings, 1896	555	0	0
Contents of Library, 1896	9,092	14	7
2. Investments.			
(*a*) R.M.C.S.			
Redemption of Debenture Fund £185 8s. 10*d*. New South Wales 4 per cent. Stock	199	6	9
Permanent Endowment Fund £330 6s 3*d*. New South Wales 4 per cent. Stock	355	12	6
Gant bequest £510 17s. 1*d*. 3½ per cent. Southern Nigeria (Lagos)	500	0	0
(*b*) Gynæcological Society.			
£270 Grand Trunk 4 per cent. Debentures ...	289	0	0
£5 Caledonian Railway 4 per cent. Debentures	6	0	0
(*c*) Clinical Society.			
After expenses paid will have in Consols ...	200	0	0

response to the 2866 post cards issued. About 750 individuals have sent no answer; the majority of these will probably join as Members at £1 1s. per annum. In addition the Society is certain to attract a considerable number of new Fellows and Members from amongst those who do not belong to any of the Amalgamated Societies; especially as no entrance fee is to be imposed before March, 1908. It would appear probable that the balance of income over expenditure will be some £1500.

* Valued in 1906 at a higher figure.

			£ s. d.
* (*d*) Obstetrical Society.			
Consols £600	£528		
Midland Debentures £1641	1,287		
L.C.C. 3 per cent. £807	759		
Victoria 3 per cent. £580	504		
		3,078	0 0
(*e*) Pathological Society.			
After expenses, say £550 Consols		450	0 0
* (*f*) Odontological Society.			
£5,200 Consols		4,500	0 0
		£70,375	13 10

The above figures are exclusive of Cash at Bank (£1075) at end of 1906.

Liabilities.

	£ s. d.
£33,200 3 per cent. First Mortgage Debenture	33,200 0 0
Balance of Assets over Liabilities, excluding Cash Balance and Libraries of Obstetrical and Odontological Societies	37,175 13 10
	£70,375 13 10

* Exclusive of value of Library and Museum.

Concluding the record of the Amalgamation Movement it will probably be of interest to supplement the Official Report, and other documents, with the following which provided the first draft scheme for the consideration of those present at the first meeting of the Society at which the question was first considered and of the Committee of Representatives.

Sussex Hotel, St. Leonards-on-Sea,
February 18*th*, 1905.

To Sir Richard Douglas Powell, Bart., K.C.V.O., M.D., etc.,
President of the Royal Medical and Chirurgical Society.

Dear Mr. President,—At your request I have endeavoured to put on paper my ideas as to the possibility of forming in London a Confederation, or rather an amalgamation of the existing Medical Societies.

I know that in spite of many difficulties the thing is perfectly feasible, if all the contracting parties instead of dwelling on the difficulties, or the purely fanciful loss of prestige, will approach the subject with a determination to secure for British Medicine the great advantages which would undoubtedly be conferred by the establishment of such a powerful organisation.

But if any scheme of amalgamation is to be a success all questions, not only of individual wealth or pre-eminence, but also of exclusiveness, should be ignored, and one aim alone be kept in view, viz. the promotion of Medical Science by the banding together into one harmonious whole all the available talent to be found in the various societies now existing.

Some of my suggestions may appear at first sight merely arbitrary, but for some years I have, from time to time, carefully weighed and considered almost every point which is likely to arise in practice, and although I have endeavoured not to burden this letter with unnecessary detail, I think I am prepared to give good reasons for every suggestion I have made.

Without further preface I now offer for your consideration my—

ROUGH DRAFT OF A SCHEME FOR AMALGAMATING THE PRINCIPAL MEDICAL SOCIETIES OF LONDON INTO ONE SOCIETY, WHICH SHALL INCLUDE SECTIONS FOR THE SPECIAL STUDY OF THE SUBJECTS NOW DEALT WITH BY DIFFERENT SOCIETIES.

General.

For my purpose I have dealt with the following Societies only, not from any intention of excluding any omitted, but simply because in the short time at my disposal I have been unable to get the necessary figures in reference to others. But the lines upon which I have based my calculations admit of the list being either diminished or increased without greatly affecting the proportionate result—that is, assuming that in any case the Medical Society and the Royal Medical and Chirurgical Society remain in the final list. Indeed, it should be borne in mind that these two Societies are the ones that would bring the most substantial financial contributions to the amalgamation, and that most of the others would reduce the *average* wealth of the new body. These two Societies are able, by themselves, to form a strong and wealthy academy, which could, in itself, find all the material necessary for useful working sections.

The Societies are the—

Medical.	Laryngological.
Royal Medical and Chirurgical.	Dermatological.
Pathological.	Otological.
Clinical.	Diseases of Children.
Obstetrical.	Odontological.
Epidemiological,	Life Assurance Medical Officers.
Physiological.	Balneological.
Neurological.	Anæsthetists.
Medico-Psychological.	Hunterian.
Ophthalmological.	Harveian.
Gynæcological.	Medical Officers of Health.

These Societies represent a membership of about 7000, but a hasty analysis and comparison of the various lists show that this membership is made up of about 4750 *persons*.

If these Societies formed themselves into a new body, it should begin with a membership of (say) 4000 persons, and upon this estimate my calculations are based.

SUBSCRIPTION.

One of the chief difficulties in any scheme of amalgamation of the London Societies is the varying subscription for membership,

and unless it is agreed at the outset that those paying such a subscription as half a guinea (as is the case with more than one society on my list) are willing to pay a higher subscription in return for the greatly increased privileges they will enjoy, they must be dropped from the scheme.

On the other hand, it is impossible to expect that any considerable number of the members of those Societies which have a guinea subscription would be willing to pay *Three guineas* (the subscription of the Royal Medical and Chirurgical); and, as it would not only be invidious, but unfair that the old members of that body should pay a higher subscription than their fellow members of the New Society, they should benefit by a reduction of their subscription. I propose, therefore, that the subscription difficulty be met by a compromise.

Those members of the New Society who should desire to enjoy all the advantages it can offer should be elected "FELLOWS" and pay a subscription of TWO GUINEAS, while those members of existing Special Societies who would be satisfied with the privileges of one special section, should be elected as "MEMBERS" and pay an Annual Subscription of ONE GUINEA. Members might have the use of their sectional library,[1] and be entitled to receive the Fasciculi of the 'Transactions' belonging to their sections.

(It might be worth considering whether to permit a "Member" to join a second section on payment of an additional subscription of HALF A GUINEA.)

While dealing with the question of subscriptions one point should be emphasised. There should be an end for ever of the inconceivably unfair and unbusinesslike arrangement prevailing in some Societies (until recently even in the Royal Medical and Chirurgical Society) of electing members who pay no subscription. They cost their society a proportion of the working expenses, and by so much diminish the funds provided by their subscribing *confrères* for publication and other useful work; and even in some instances are elected to office, and dispose of funds to which they do not contribute. The value of a member's adhesion to a society which he is willing to join *on the free list* is rather less than the value of his subscription! My scheme provides for no "deadheads."

The only Fellows or Members of the New Society who should pay no subscription are those who have paid a Life Composition Fee to the Society or Societies to which they already belong.

[1] It should be remembered that under existing conditions only three of the Special Societies possess libraries.

Headquarters and Property.

Only two of the Societies on my list possess houses of their own. That of the Medical Society is a valuable property, but is not large enough to house such an institution as is now contemplated. There remains, therefore, only the House of the Royal Medical and Chirurgical Society, which in its present condition would provide enough accommodation for the new Society. When more room is required it will be the result of an increased membership, and that will mean an increased subscription income, which will enable the Society to dispense with some of its non-medical tenants, until at length the whole of these magnificent premises shall be in the exclusive enjoyment of a powerful and wealthy medical institution in every way worthy to represent British Medicine.

[Parenthetically, however, I would suggest that as soon as possible the building site should be utilised to its utmost extent, for before long neighbours will acquire ancient lights, which will seriously depreciate the value of the property. I would suggest—

(1) To build, over the existing meeting-room and library-room, a new meeting-room capable of seating at least 500 persons, with two or more smaller rooms suitable for Committees and sectional library-rooms. The present meeting-room could then be utilised as an additional reading-room, and the North Room become the Council Room. Or, if that be found to be impracticable—

(2) To build, over the North Room, corridor, and part of Library, two or three useful rooms for Committees and Sectional Libraries.

I have already made arrangements to provide the capital required for such an operation, and I believe the additional income which could be obtained through these additions would provide interest and sinking fund to pay it off.]

The House of the Medical Society would provide a valuable income producing asset of the new Society. It already brings to its owners a net profit rental of about £600, and when that Society becomes absorbed in the new one and its members are housed in Hanover Square it should, if properly developed, produce from £1200 to £1500.

The Societies possess invested funds to the value of nearly £5000, and these, put into the common fund and wisely invested, should produce an income of at least £200.

Transactions.

For the combined 'Transactions' of the new body several changes would have to be made. At present all the Societies

publish octavo volumes, and if this *format* be retained it would mean publishing some twelve or fourteen volumes every year. I suggest that both for economy and convenience the *format* of the Royal Society's 'Philosophical Transactions' be adopted. The quarto page in double column would effect considerable economy in space, and consequently in cost, while for illustration purposes a quarto plate is infinitely superior to an octavo.

Instead of an annual volume, I would suggest the publication of a monthly fasciculus during the nine working months of the year only, for medical publications in July, August, and September are apt to be lost sight of. There should be no attempt at uniformity of size in the fasciculi, but each should contain just those papers that are ready for press when publication day arrives. Prompt publication is of paramount importance in medical literature, and it is absurd that (as often happens now) an early paper of the session should be kept back because some later one, owing to dilatoriness of the author or for any other reason, is not ready for press.

To enable members of sections to receive their sectional papers only, each section should have a separate pagination and each paper a distinctive number, which would give its place in the final volume. This would involve no difficulty in the general index. Instead of a simple page number, a reference would be given thus, "24^{16}." The large figure would indicate the paper, and the small figure the page of the paper. I submit a specimen of the new format.

Published in this form—*punctually* each month—I firmly believe that your 'Transactions' would soon be regarded as a periodical of the highest value, which every medical man of any standing throughout the English speaking world must possess, and that it would command a large and profitable sale far in excess of the sum given in my budget, which I have conservatively fixed at the amount realised under existing conditions.

Management.

There should be a General Council, in which should be vested the control of the Society at large and the management of its property. The Council should consist of at least fifteen *Fellows* (including the President, two Hon. Treasurers, two Hon. Secretaries, two Hon. Librarians, two Hon. Editors, and six non-official Councillors), and to these should be added the Presidents of Sections, who should rank as Vice-Presidents of the Society. Each section should annually elect its own President and other officers, and manage its own affairs, except in the matter of expenditure, which should be subject to the control of a general Finance Committee. There should be a Library Committee; a broadly representative Editorial Committee, who

should decide by ballot as to acceptance or rejection of all papers; a Finance Committee; and a House and Property Committee.

Staff.

Some of the Societies included in the list have permanent paid officers, and they would probably stipulate for the continued employment of these. Obviously it would be to the advantage of the new body to secure the services of a staff already trained.

The increased work and detail would render it necessary to make further additions to the existing staff of the R.M.C.S., viz.:

An assistant clerk for the Secretary, a typist, a bookkeeper and cashier, additional servants to attend at meetings (say two), and one or more Call Boys for the Library.

The entire Staff would therefore consist of:

Secretary and Clerk.
Librarian, with additions to the Staff of the Library taken from other Societies (probably three).
Library Assistant and Call Boys.
Bookkeeper and Cashier.
Typist.
Porters and house servants as at present, with possible addition as above.

Incorporation.

In case of difficulty, and in any case of delay in obtaining a new Charter, it probably would be wise to form the new Society under the existing Charter of the Royal Medical and Chirurgical Society, which is comprehensive enough to cover such a scheme; and it would then only be necessary to defer temporarily the necessary alterations in the Constitution of the Council and in some minor details. Once formed, there should be no difficulty in obtaining a *Supplementary* Charter, conceding a change of name and the other alterations as to Council, and other details.

Budget.

In this estimate of Income and Expenditure of the new body *I have left out of count the existing income and expenditure of the Royal Medical and Chirurgical Society except as to two items specially alluded to*, for, as that Society now stands, its income shows a substantial annual surplus. I therefore *assume* its accounts as a foundation, and merely deal with probable *additional* expenditure and revenue under the proposed new conditions.

Additional Expenditure.

	£	s.	d.	£	s.	d.	£	s.	d.
Cost of combined 'Transactions' published in 4to monthly fasciculi for Fellows, with separate sectional fasciculi for Members of Sections...				3500	0	0			
Less cost of R.M.C.S. portion (already provided for)	400	0	0						
By Sales as at present	350	0	0						
				750	0	0			
							2750	0	0
Cost of increase of staff (for details see Appendix)							720	0	0
Loss from R.M.C.S. income by reduction of subscription to £2 2s.				450	0	0			
Loss of rent from "meeting" tenants...	500	0	0						
Loss of rent from Odontological ...	270	0	0						
				770	0	0			
							1220	0	0
Cost of increased purchases for library							300	0	0
Additional printing and stationery ...							500	0	0
Contingencies (say)							510	0	0
							6000	0	0
Estimated surplus							697	0	0
Total							£6697	0	0

Additional Income.

	£	s.	d.
Rents from Chandos Street and income from investments of various societies	1500	0	0
Entrance fees—to be paid by new Fellows and Members *after* the formation of the new Society, say 100 at £2 2s. ...	210	0	0
Subscriptions of 1000 Members of other Societies who already belong to more than one, and who it may be assumed would desire to become "Fellows" at a subscription of £2 2s.	2100	0	0
Subscriptions of 2500 "Members" at £1 1s. (I deduct 500 subscribing Members allowed for in R.M.C.S. budget)...	2625	0	0
Estimated sectional subscriptions from Members who might desire to belong to two sections, say, 500 at 10s. 6d. (*say*)	262	0	0
	£6697	0	0

There are many details which will require careful consideration, but it would be better to deal with them after discussion of the main features, and if I can be of use to you in this discussion, I need scarcely say that I am heartily at your service.

Very faithfully yours.

J. Y. W. MacAlister.

THE ROYAL SOCIETY OF MEDICINE

THE FIRST GENERAL MEETING OF FELLOWS UNDER THE NEW CHARTER

HELD AT

THE SOCIETY'S HOUSE, 20, HANOVER SQUARE, LONDON, ON FRIDAY, JUNE 14TH, 1907, AT 5.30 P.M.

Mr. WARRINGTON HAWARD (President of the Royal Medical and Chirurgical Society), said—Gentlemen, I think Dr. Latham has a few letters expressing regret for not being able to attend this meeting.

Dr. LATHAM (Honorary Secretary of the Amalgamation Committee) said he had received letters from Docteur R. Wybauw, of Spa; Dr. T. E. Lloyd, of Abergavenny; Dr. J. J. Cox, of Manchester; Dr. G. A. Gibson, of Edinburgh; Sir James Sawyer; Sir John Byers, Sir Constantine Holman; Dr. W. F. Somerville, of Glasgow; and Dr. Douglas Stanley, of Birmingham.

The CHAIRMAN.—Gentlemen, I take the chair, that we may proceed without delay to the proper business for which you have been called together this afternoon, and I will not detain you from that business for more than a moment. But I think we shall all agree that it is a matter for mutual congratulation that the efforts which have been made to promote the union of the Medical Societies of London have been crowned with success—(applause)—and that we are called together

to-day to take the necessary steps for establishing the Royal Society of Medicine under its new constitution. You will doubtless remember that when the question of the union of the Medical Societies was raised two years ago by Sir Richard Douglas Powell in his Presidential Address to the Royal Medical and Chirurgical Society, the suggestion was received very favourably; and that at the meeting of members of the various Medical Societies shortly afterwards, held at the Royal College of Physicians, it was resolved that a scheme of union should be drawn up, to be presented to the various societies for consideration. As a result, there was formed an organising committee of representatives of the societies proposing to join, with Sir William Church as chairman, and it is by the labours of that committee that the matter has been brought to a successful conclusion. The King, as you know, has been graciously pleased to grant a charter conferring the necessary powers for enabling the uniting societies to join the Royal Society of Medicine. And so the idea first started in 1808, and revived unsuccessfully in 1860, and again in 1870, is at last realised. I do not doubt that this union will be to the advantage of all concerned—(hear, hear)—that it will widen the outlook, add to the strength, stimulate the vitality, and consolidate the interests of all the constituent societies; that it will facilitate co-operative work, and favour economy of management. But, Gentlemen, I need hardly say that for the attainment of this union there has been necessarily a great deal of careful negotiation; and all those who have been concerned in the matter must, I am sure, feel that the success of the Committee's work has been very largely due to the patience, the courtesy, the tact, and the skill of Sir William Church—(applause)—and of the Honorary Secretaries of that Committee, Dr. Latham and Mr. Pendlebury to whom indeed, we owe a large debt of gratitude. (Applause.) I will now ask the President of the Royal College of Physicians to propose the first resolution.

Sir RICHARD DOUGLAS POWELL.—Mr. President and Gentlemen, I have the honour to move—

> "That at this meeting of Fellows of the Royal Medical and Chirurgical Society, with Fellows and Members of the following Societies—
>
> Pathological Society of London; Epidemiological Society; Odontological Society of Great Britain; Obstetrical Society of London; Clinical Society of London; Dermatological Society of London; British Gynæcological Society; Neurological Society; British Laryngological, Rhinological, and Otological Association; Laryngological Society of London; Dermatological Society of Great Britain and Ireland; Otological Society of Great Britain and Ireland; British Electrotherapeutic Society; Therapeutical Society—
>
> which have agreed to unite with the Royal Medical and Chirurgical Society under the name of the Royal Society of Medicine, we, who are here present, do hereby constitute ourselves the First General Meeting of the said Royal Society of Medicine, and do now proceed to do such things and acts as are necessary for the carrying on of its work under the new constitution."

In proposing this resolution, I think perhaps a very few words may be fitting, inasmuch as this is the resolution which inaugurates and affixes the seal of accomplishment upon the scheme of union of the great Medical Societies of London, in bringing about which, as our chairman has just observed, so much time and labour and patience have been expended over a series of years. And apart from the usefulness of such a union, apart from the support and the increased strength which such union gives to each constituent member, and apart from the economy and, I hope, the increased efficiency of work which such a union must effect, I have great hopes, and I think we all believe that the effect of such a union into a great central Society, will serve more closely to co-ordinate the many ramifications into which our profession has divided. It was inevitable, and, indeed, for the progress of medicine, that branches of specialisation, both of research and practice, should arise. And the tendency, naturally, has been for those branches to lose touch some-

what with one another, and continuity with the parent stock, to become isolated and separated, and to lose that interest and that sympathy of communication which we hope may now be restored between the several branches. No man, of course, can acquire and maintain an expert knowledge of the special branches of medicine which are enumerated in this resolution, and still remain equipped with the requirements for the teaching and practice of general medicine. But I do believe that it is very possible to keep in touch with the most advanced teaching of such sections of medicine, and to utilise them in harmony and due perspective with the requirements of general medicine. And I think that this kind of co-ordination is more likely to be brought about now that these various sections are associated—happily associated—in a great central institution, of which the sections become part of a united medical science, each special department adding to the general stock of knowledge, and becoming in its turn broadened in outlook by its association with the general body. I therefore move this resolution, which I will not read again. (Applause.)

Mr. Henry Morris (President of the Royal College of Surgeons).—Mr. Chairman, the proposer of this resolution, Sir Richard Douglas Powell, has told us that very few words were necessary to introduce this subject to the notice of the meeting. As the seconder of this resolution I beg to remark that I think no words from me at all, as seconder, are required; except that I should like to say I cannot help feeling that we all owe a great debt of gratitude to the various members of the Committee, and to Sir William Church, the Chairman—(Hear, hear),—who, after so long a time, and after having given so much care and attention to the matter, have been able to bring this great object to a successful termination, especially in view of the fact that all previous efforts have failed to do so. (Applause.) I therefore have very great pleasure in seconding the resolution which has been submitted to you by Sir Richard Douglas Powell. (Applause.)

The CHAIRMAN.—Does anybody wish to speak on this resolution? Then I put it. *Carried by acclamation.*

I declare the resolution unanimously carried. (Applause.)

Sir WILLIAM CHURCH.—I now rise, Sir, in accordance with your wish to move—

> "That the By-laws which have been prepared by the Committee of Representatives of Societies, and now submitted to the meeting, be and the same are hereby adopted and confirmed as the By-laws of the Royal Society of Medicine."

It is necessary that this general meeting should proceed at once to the adoption of the By-laws. To remove any possible misapprehension which may be in the minds of any of the Fellows, I wish to say that these By-laws, if you pass them to-day, of course, are binding only until they are abrogated or amended. All these By-laws may be abrogated or amended, according to the constitution of the Royal Society, due notice, of course, being given. I mention this because already I have heard that amendments are desired. Of course, any Fellow of the Society is at liberty to take such steps as he thinks are necessary for the amendment of the By-laws, but for the purpose of our being able to do legally the remainder of the business which is down on the agenda paper to-day, it is necessary, in the first place, that these By-laws should be passed. I therefore, without any further words, ask you to be good enough to accept the resolution that these By-laws be adopted. (Applause.)

Mr. MAKINS.—I rise, Sir, with pleasure, but with a sense of some responsibility, to second the proposition which has been submitted by Sir William Church. This is a momentous resolution since, in the main, these By-laws are those which are to steer the new ship on what we hope and believe will be a successful voyage in the future. At the same time, anybody standing in my position is able to second this resolution with the greatest confidence, because a very unusual amount of trouble has been taken in the preparation of these By-laws. Without

untiring industry, tact, and patience on the part of the Chairman and Secretaries of the late Committee of Representatives, they could never have been produced in the form in which they are to-day; and it must be remembered that in their production the Chairman and Secretaries have been aided by representatives from each of the other Societies, and that each of the representatives from these bodies has been cognisant of the difficulties and troubles which arise in the management of Medical Societies. Consequently, I think, we have every reason to believe that these By-laws, subject to possible alterations such as Sir William Church has foreshadowed, will be a successful guide to the work of the Royal Society of Medicine in the future. I beg, Sir, to second this resolution. (Applause.)

The CHAIRMAN.—I may mention that in addition to the care which you have already heard has been taken in connection with these By-laws by the Committee of Representatives, legal advice has been taken as to their correctness. So I think you may safely confirm them. Does anyone wish to speak on this resolution? Then I will put it to you. *Carried unanimously.*

The CHAIRMAN.—Then, Gentlemen, having passed these By-laws, we can now proceed to the election of the Council and Honorary Officers of the Royal Society of Medicine. On page 7 of the By-laws you will find there is a special provision for the election of the Officers and Council. You will see this enactment: "All the members of the first Council (other than the *ex-officio* members)" that is to say, the Presidents of each Section of the Society who are thereby Vice-Presidents of the Royal Society of Medicine, "and all the Honorary Officers of the Society, shall be elected by show of hands at the meeting of the Society at which these By-laws are passed, or at a meeting of the Society to be held as soon as possible after the passing of these By-laws. It shall be no objection to such meeting or to the election of the Council and Officers that no notice convening such meeting shall

have been given under these By-laws or the previous By-laws of the Society. The persons so elected shall hold office until the first Annual General Meeting. The Council so elected shall be at liberty forthwith to exercise all the powers conferred upon the Council by the Charters or the By-laws, notwithstanding that the *ex-officio* members or any of them shall not have been appointed." That is the provision, and I will now ask you to proceed to the election of the Honorary Officers and Council. I will ask Sir Thomas Smith to propose the first resolution, which concerns the President.

Sir THOMAS SMITH.—Before reading the resolution, Sir, which I have the honour to have in my hand, I cannot forbear repeating the congratulations which have been uttered by others on the success attending the labours of all you gentlemen who have worked at the amalgamation of these Societies. And I may be excused for that, as, thirty-seven years ago, in a subordinate position, I very laboriously, but futilely, worked at the amalgamation of these Societies. You have been good enough to mention that, and the President of the College of Surgeons has been good enough to rub it in—(laughter),—but it was not my fault. I was only the Secretary; Sir George Burrows was President, and therefore, luckily, I cannot take a very large share on my own shoulders. However, the Society being now constituted, I think you will agree with me that the next most important step to take is to elect a President. And I also think we shall all agree that the President should be intellectually a strong man; that he should be a man of independent character, of business habits, of scientific attainments, and that he should command the respect of the members of this Society. Now, the terms of the resolution which I shall read to you abundantly fulfils all those conditions. It is to the effect:

> "That Sir William Church, Baronet, K.C.B., be and is hereby elected the first President of the Royal Society of Medicine."

(Loud applause.) We all know that for the last two years Sir William Church has worked hard at this amalgamation, and I am informed that he has never omitted to attend any meeting, either of the General Committee, of which he was Chairman, or any of the Sub-committees, to many of which he was summoned to attend. Indeed, he has never failed to attend a single meeting in the last two years. (Applause.) I have very great pleasure, Sir, and I feel honour in doing so, in proposing that Sir William Church be elected first President of the Royal Society of Medicine. (Applause).

Dr. OSLER.—I have much pleasure, Mr. Chairman, in rising to second this resolution. I cannot add anything to what Sir Thomas Smith has said in regard to the qualifications of our future President. He is known to you all, he occupies a distinguished position in the profession, but, above all, Sir, he is that "rare bird" in the profession, a good business man, and that is what we need to steer the ship safely. May I, Sir, with such an audience before me, say one word which is not quite pertinent to the subject? I think, Sir, that what is needed now for this Royal Society of Medicine is money for a new building and for the development of the Library, and I think the profession in this great metropolis can get it. They can get anything they want if they go to their friends, and no profession has such friends as we have. (Hear, hear). I would ask you to remember that "He who asketh much getteth much." (Laughter.) I have much pleasure in seconding the resolution. (Applause.)

The CHAIRMAN.—It is evident by your reception of this resolution that it is hardly necessary to put it. But, as a matter of form, it must be put. Therefore, I ask those who are in favour of it to signify it in the usual way. ("All, all.") *Carried by acclamation.*

The CHAIRMAN.—You will all agree, Gentlemen, that we are extremely fortunate, as Sir Thomas Smith has already stated, in obtaining as the first President of the

Royal Society of Medicine one who will preside over it with such dignity and wisdom as we know Sir William Church will. And I will ask him now to take the chair, which I gladly vacate in his favour. (Loud applause.)

Sir WILLIAM CHURCH then took the chair.

Mr. HAWARD.—Mr. President, I have the pleasure to present to you a symbol of your presidential authority, the Master-key[1] to this building, which will henceforth be the home of the Royal Society of Medicine. (Applause.)

The PRESIDENT.—Fellows of the Royal Society, I have to thank you, and I do thank you very deeply and sincerely for the honour which you have just paid me in electing me the President of the Royal Society of Medicine. It is a very great honour indeed, and I appreciate it most highly. To be elected the first President of this Royal Society is, I feel, an historical event, not only in my life, but in the history of the Society. I feel assured that this Society has a great future before it, and that it will be representative of all that is best and highest in the ethical, the scientific, and the practical work of medicine. (Hear, hear.) I feel that by your favour I really have been placed in a position to which I not only have no claim, but for which I am scarcely fitted. When I look around upon those who surround me to-day I see many who have very much higher claims, from the advancement which they have made in their various branches of medical study, than I have. And if I consider that your vote that places me in the chair to-day has been in any way due to the fact that you think that I have worked hard in endeavouring to effect this union, I again feel that I have not a right claim to be in the chair, for I can assure you that the greater part of the labour should be attributed to others. And, in connection with that, I cannot refrain from saying that the bulk of the labour—the heat—if I may say so—of the

[1] The gift of the Secretary, Mr. MacAlister, to the Royal Medical and Chirurgical Society, on the opening of the building.

day fell upon the two Honorary Secretaries, who have been the Honorary Secretaries of the Organising Committee and of all the Committees, which have had many meetings in this and the contiguous rooms. To them I looked for advice and assistance, and to be prompted in the work, and I never looked in vain. And it is to them, and to the advice and assistance that we have received from others, that our labours have been brought to the satisfactory end that I think they have to-day. I had intended to ask your permission to say a few words as to the steps by which this union has been formed, and also about the objects which the promoters of the idea had in their minds, but much of that has been so well said by Mr. Haward and by the President of the Royal College of Physicians, that I will not trouble you with the remarks which I was going to make on the subject. But allow me now to congratulate Sir Richard Douglas Powell on having succeeded where so many have failed before him. (Applause.) To him it is due that we are here to-day. It was he who asked me, from the accident of my being at the time President of the Royal College of Physicians, to call the societies together, and, but for his action, I certainly should not have taken the step myself. Therefore it is primarily due to him, and to the wise words which he let fall in his Presidential Address to the Society in 1905. It is primarily due to him that we are here as the Royal Society of Medicine to-day. (Applause.) I would, however, just like, if you will allow me, to express my own views, telling you what were the objects present in my mind which made me desirous that this union of societies should take place. They were the same that, I believe, influenced former generations, and they may be summed up in very few words: the advancement of British medicine. (Applause.) The remarkable increase in our knowledge which has taken place during quite recent years, and the necessity for the application of scientific methods for the attainment of such knowledge, has rendered a division of

labour absolutely necessary. It has become impossible for any one person to be cognisant of all the subjects which surround medicine, and upon which medicine to a great extent depends. Neither can any one society profess to offer opportunities for the demonstration and discussion of the very diverse subjects which stand now in more or less close relationship with medicine. Hence it is certain that, just as numerous special societies have already been formed, other special subjects will require societies or sections in the future. And it was with the desire that their work should be co-ordinated, and to keep the sections more in touch both with one another and with what I may call medicine and surgery taken in the whole, which made me take so much interest in the union of the societies when first this scheme was mooted. The promoters of the union also believe that by this means the members of the old societies as Sections will obtain greater facilities of intercourse with other sections, and this at much less expense. Further, it places, at a comparatively small cost, what I trust will soon become the principal medical library in the Kingdom, at the convenience of not only the Fellows of the Society, but also of members of sections, most of which, as separate societies, had no library of their own. In fact, I believe, and I think most of those who work with me believe, in the old proverb, "In union is strength," whilst in separation, even if it be separation without discord, there is weakness. I would like to add that, although we are somewhat disappointed that the oldest of our London medical societies has not seen its way to join our union, they no doubt have good reasons for remaining aloof, and will continue to fill a useful position in the medical life of the metropolis, and we wish them well. (Hear, hear.) In conclusion, let me again thank you for the honour which you have done me. You have placed me in a position the responsibility of which is great, for I believe that by our action to-day we have established a Society which is destined to have a great and powerful

influence on the medical profession, as well as on the development and furtherance of medical science. With your assistance and forbearance I trust that I may be enabled to fill the chair to the satisfaction of the Society, and to maintain in it the traditions and principles which have been held by the many distinguished men who have, in former years, occupied the Presidential chair of the old Royal Medical and Chirurgical Society. (Applause.)

The Royal Society of Medicine will now proceed to the election of its Honorary Officers and Council. Lists of names have been placed in the room, and it is in the power of any Fellow to substitute other names, and demand that a ballot be taken. It was thought desirable that some names should be placed before you, in order that you might have the means of knowing those gentlemen who some of us thought would take great interest in the Royal Society of Medicine, and would be of much assistance and use upon this Council. I need hardly remind the Royal Society of Medicine that the labours of this Council will be very arduous, and those who have not taken part at all in the organisation of the Society can have little idea of the amount of detail which has yet to be arranged before we can get so complex a Society as the Royal Society in a condition in which its work will go on without difficulty or friction. I trust, therefore, that the Fellows of the Royal Society of Medicine will not be impatient. I fear that there must be difficulties and some little friction at first, but I trust that the labours of the Council may bear such fruit that when the first session of the Royal Society of Medicine commences next October things may be in a condition to work easily and smoothly. I have ventured to make those few remarks, because I thought some of you might be under the impression that everything would be ready to be started from to-day. But that is really impossible; some time must be given for the Council to get things into working order.

I now call upon Dr. Pye-Smith.

Dr. PYE-SMITH, F.R.S.—Gentlemen, I have been asked to move the following resolution:

> "That the following nominations of the Committee of Representatives of Societies for the posts of Honorary Treasurer, Honorary Librarian, and Honorary Secretary, and for such Members of Council (other than *ex-officio* members) as require election be and the same are hereby adopted and confirmed."
>
> *Honorary Treasurers.*—F. H. Champneys, M.D.; A. Pearce Gould, M.S.
>
> *Honorary Librarians.*—Rickman J. Godlee, M.S.; Henry Head, M.D., F.R.S.; Norman Moore, M.D.
>
> *Honorary Secretaries.*—Arthur Latham, M.D.; Herbert S. Pendlebury, M.B., B.C.
>
> *Members of Council.*—Sir Richard Douglas Powell, Bt., K.C.V.O., M.D. (*President of the Royal College of Physicians*); Henry Morris, M.B. (*President of the Royal College of Surgeons*); J. Warrington Haward; G. H. Makins, C.B.; Sir Shirley F. Murphy; Thomas Laurence Read; Sir Felix Semon, K.C.V.O., M.D.; Frederick Taylor, M.D.; Thomas J. Walker, M.D.

We feel, Sir, I think all of us, that on this auspicious day there is no need for prolonged speeches; and I will only say that I trust and believe most heartily that the amalgamation and union which we have accomplished this day will be of the utmost use, not only to the benefit of each Society which has joined in it and for the progress of medical science, but to the universal benefit of ourselves and the whole profession. And it has this great advantage, that it is a striking instance which we can show to all the outside world, that after all our profession is one—(Hear, hear)—that we have all the same education, the same scientific basis, the same practical experience, and that the art which we practise is one and the same art; that it will flourish as our efforts flourish, and will be always to the good of the whole nation. I beg, Sir, to move that resolution. (Applause.)

Mr. CLINTON DENT.—I have great pleasure, Sir, in seconding the resolution. But I think that no words are needed from me; the names here are thoroughly repre-

sentative, they are all those of trusted and tried men, most of them having held the several offices which they are now going to adorn by the hard work which you, Sir, have already foreshadowed for them. I will say no more, but will second this resolution, for I resolved to myself that in this Royal Society of Medicine any speeches I may make shall, if it lies in my power, be to the point, and that, at any rate, they shall be characterised by brevity. (Applause.)

The PRESIDENT.—If no Fellow wishes to add to or alter any of these names I will put the resolution to the meeting.

The resolution was then put and carried unanimously.

The PRESIDENT.—The next point upon the agenda paper is a technical one, and I venture to move it myself from the chair, and that is—

> "That Messrs. Newson Smith, Lord and Mundy, Chartered Accountants, be appointed Auditors of the Society's accounts."

Many of you know that that firm has for many years audited the accounts of the Royal Medical and Chirurgical Society, and I think they have always given the Treasurers and other officers complete satisfaction. I venture to hope that you will nominate them as the first auditors of the Royal Society of Medicine.

The resolution was carried.

The PRESIDENT.—I call upon Sir Thomas Barlow to propose the next resolution.

Sir THOMAS BARLOW.—Mr. President and Fellows of the Royal Society of Medicine,—The resolution which I will ask you to carry is that a vote of thanks to the members of the Committees and Sub-committees who have conducted the necessary negotiations from April, 1905, be accorded. Sir, this is one of the resolutions which only needs to be read to commend itself to the hearty acceptance of everybody in this room. I am sure that many Fellows have little conception of the amount of labour that has been

taken up in the constitution of this Society. (Hear, hear.) It has involved weekly meetings during a great part of the period which has elapsed since the subject was first accepted by the profession, and the skill, the tenacity, the good temper, and the kindliness which have been shown by the members of these Committees and Sub-committees are beyond all praise. The Royal Society of Medicine, Sir, has shown its appreciation of the valuable work of the Chairman of these Committees by electing you as President of this Society, and I can only say that you, Sir, and your colleagues have not only earned the gratitude of English medicine in this generation, but for many generations to come. I have great pleasure in moving this resolution. (Applause.)

The PRESIDENT.—I will ask Dr. Habershon to second it.

Dr. HABERSHON.—I have been asked, Sir, to say a word or two in seconding this resolution. I feel that no words can be added to the graceful speech of Sir Thomas Barlow, nor to the expression of the feeling which we have heard in the various speeches to-day. We all owe the very greatest debt of gratitude to those gentlemen who formed the Committees and Sub-committees which have been practically the builders of this ship which is now launched. And I think I may reiterate that since at every meeting the President, Sir William Church, has been present, and also the Honorary Secretaries, we owe them a double debt of gratitude—(applause)—for the care, the courtesy, the patience which they have shown, and the arduous labours they have had. I beg to second this resolution. (Applause.)

The resolution was unanimously carried.

The PRESIDENT.—Dr. Latham asks me, for the Honorary Secretaries and the other members of the Committees, to return to you their very grateful thanks for the resolution which you have passed. And that, I believe, concludes the meeting.

REPORT OF THE HON. TREASURERS

ON THE

FINANCIAL POSITION OF THE SOCIETY AT THE DATE OF AMALGAMATION.

In presenting our Financial Statement up to June 30th, 1907, we would particularly draw attention to the fact that the item of "Sundry Liabilities," which stood at the end of 1906 at £1438 18*s*. 7*d*., has been reduced to £78 17*s*., which is the amount of interest accrued (but not yet due) upon the Debenture Bonds since May 31st.

Having paid all accounts due against the Society up to date, and having added more than £100 to the "Debenture Redemption Fund," there is a balance in hand of £923 13*s*. 1*d*.

It is a great satisfaction to us that the closing Financial Statement of the Royal Medical and Chirurgical Society, under its old constitution, is so eminently satisfactory.

W. S. CHURCH.
A. PEARCE GOULD. } *Hon. Treasurers.*

2nd August, 1907.

[*For Accounts see over.*

Statement of Liabilities and Assets, 30th June, 1907

Liabilities.	£	s.	d.	£	s.	d.
3 per Cent. First Mortgage Debentures				33,200	0	0
Proportion of Interest on Debentures accrued to date but not payable until the 30th November. (N.B.—*The Interest on Debentures is payable 31st May and 30th November. The last payment was on 31st May, and the amount accrued is calculated to 30th June*)				78	17	0
Balance, being Surplus of Assets over Liabilities, viz.—						
Balance, 31st Dec., 1906	£29,376	7	9			
Add						
Excess of Income over Expenditure for the half-year	923	13	1			
	30,300	0	10			
Less						
Depreciation of Investments to *Par*	42	6	1			
				30,257	14	9
				£63,536	11	9

Assets.	£	s.	d.	£	s.	d.
Freehold and Leasehold Property as per valuation of Messrs. Giddy and Giddy, on 2nd June, 1896	51,150	0	0			
Subsequent additions	416	11	6			
				51,566	11	6
Fixtures, Fittings, and Furniture	1,368	8	0			
Less 5 per cent. written off for depreciation	34	4	0			
				1,334	4	0
Engravings (as per Valuation of Mr. F. B. Daniell, Aug. 19, 1896).				555	0	0
Contents of Library (as per Valuation)				9,092	14	7
Investments in New South Wales Inscribed Stock—						
Debenture Redemption Fund	288	11	11			
Permanent Endowment Fund	330	16	3			
				619	8	2
Sundry Debtors for Rents, Outstanding Subscriptions, etc.				319	13	4
Cash at Bank and in hand				49	0	2
				£63,536	11	9

Audited and approved,

NEWSON-SMITH, LORD & MUNDY,
Chartered Accountants,
37, Walbrook, E.C.,
2nd August, 1907.

W. S. CHURCH, M.D.,
A. PEARCE GOULD, M.S., } *Hon. Treasurers.*

J. Y. W. MAC ALISTER, *Secretary.*

Income and Expenditure Account for the Half-year ending 30th June, 1907.

Expenditure	£	s.	d.	£	s.	d.
Rent, Rates, Taxes, and Insurance ...	61	19	2			
Salaries of Staff and Accountant ...	355	15	0			
House Servants, Cleaners, etc. ...	185	1	2			
Lighting, Warming, and Cleaning ...	159	6	3			
Printing, Stationery, Stamps, and Telegrams	79	17	3			
Meeting Expenses	50	13	10			
Miscellaneous Disbursements ...	31	0	2			
				923	12	10
Repairs, Alterations, etc. ...				145	18	4
Depreciation of Fixtures, Fittings, etc.				34	4	0
Library Purchases and Expenses ...				313	2	2
Interest on Debentures				498	0	0
'Transactions'				60	16	9
Audit Fee				10	10	0
Telephone				4	3	10
Lift Charges				24	12	0
Amalgamation Expenses ...				45	12	2
Balance, being excess of Income over Expenditure during the half-year				923	13	1
				£2984	5	2

Income	£	s.	d.	£	s.	d.
435 *Annual Subscriptions at* £3 3s.	1370	5	0			
149 *do.* £1 1s.	156	9	0			
				1526	14	0
Composition Fees				22	1	0
Entrance Fees				22	1	0
Rents Receivable				1321	18	6
Sale of 'Transactions'				54	11	5
,, *'Climates and Baths'* ...				2	10	8
Interest: Permanent Endowment Fund				6	5	8
Miscellaneous Receipts:						
Interest on Deposit ...				2	9	1
Fees for use of Epidiascope ...				17	4	0
Gant Bequest: Interest ...				8	9	10
				£2984	5	2

Audited and approved,
NEWSON-SMITH, LORD & MUNDY,
Chartered Accountants,
37, Walbrook, E.C.
2nd August, 1907.

W. S. CHURCH, M.D.,
A. PEARCE GOULD, M.S., } *Hon. Treasurers.*
J. Y. W. MACALISTER, *Secretary.*

[*For Investments, etc., see over.*

PERMANENT ENDOWMENT FUND.

[Established and Contributed to by the Donations of Fellows.]

		£
From	Sir Richard Quain	100
,,	Mr. Edward Law Hussey	50
,,	Sir Richard Douglas Powell	100
,,	Mr. Edwin Unwin Berry	124
,,	Dr. Fitzpatrick	5
		£379

Invested in £330 16*s.* 3*d.* (par value) New South Wales Inscribed 4 per Cent. Stock "A" a/c.

MARSHALL HALL MEMORIAL FUND.

[Being the amount subscribed for a Memorial to Marshall Hall. Every five years the accumulated interest is given as a Prize for the best published work on 'Nervous Diseases.']

	£	*s.*	*d.*		£	*s.*	*d.*
Consols in trust on 31st Dec., 1906 (*par value*)	631	14	5	Consols in trust on 30th June, 1907	640	2	5
Interest for six months (added to Fund) ,,	8	8	0				
	£640	2	5		£640	2	5

GANT BEQUEST (£500).

[Being a sum of £500 bequeathed to the Society by the late Fredk. James Gant (sometime Vice-President of the Society) to be invested and the income to be spent on books to be marked "Gant Bequest."]

Invested in £510 17*s.* 1*d.* (*par value*) Southern Nigeria (Lagos) 3½ per cent. Inscribed Stock.

DEBENTURE REDEMPTION FUND.

(New South Wales Inscribed 4 per cent. Stock "B" a/c.

	£	*s.*	*d.*	£	*s.*	*d.*		£	*s.*	*d.*
In trust on 31st December, 1906	185	8	10				Amount of Stock on 31st December, 1906	185	8	10
costing				203	9	3	Since purchased	103	3	1
Investment (including interest for half year) to purchase	103	3	1				Cost of purchase and difference between "*par*" *and purchase price*	25	8	10
costing				110	11	6				
				£314	0	9		£314	0	9

Audited and approved,

NEWSON-SMITH, LORD & MUNDY,
Chartered Accountants,
37, Walbrook, E.C.,
2nd August. 1907.

W. S. CHURCH, M.D.,
A. PEARCE GOULD, M.S., } *Hon. Treasurers.*

J. Y. W. MACALISTER. *Secretary.*

ON SOME ASPECTS OF DILATATION OF THE HEART

BY

ALFRED M. GOSSAGE, M.B.Oxon., F.R.C.P.

Received March 30th—Read October 23rd, 1906

THE application of the results of physiological research by Wenckebach (1)[1] to the explanation of clinical phenomena has thrown a fresh light on many of the problems connected with disease of the heart, while accurate instrumental methods of study have afforded a number of new facts requiring explanation. Cardiac dilatation is the most important factor in heart disease, and, as might be expected, novel ways of research have opened out a variety of new problems here as elsewhere. Among other points, it has been shown that the old explanation of the mode of production of dilatation of a cardiac chamber is insufficient and to a large extent inaccurate. That explanation supposed that the dilatation was a purely mechanical process depending on the pressure within the chamber forcing the walls out and on the weakness of the walls themselves. While intra-ventricular or intra-auricular pressure undoubtedly has considerable influence on the production of dilatation, this pressure is not the only cause and indeed not the most important one, for we frequently find the heart dilating without any increase of pressure. Weakness of the cardiac walls, too, is not a sufficient explanation; for, as Mackenzie (2) has recently pointed out, we may find a ventricle working against a pressure sufficient to rupture its walls without any dilata-

[1] *The numbers in brackets refer to Bibliography at end of paper.*

tion. In several cases of rupture of the heart there has been no dilatation of the cavity although the walls have been abnormally thin.

The pressure is greatest on the cardiac walls during systole, but we cannot suppose that dilatation of a chamber occurs at this time. Increase of systolic pressure, however, may prevent the ventricle emptying itself properly and the blood left will tend to increase the diastolic pressure. Roy and Adami (3) have shown that the ventricles are never completely emptied during systole. The diastolic pressure in the ventricle, however, does not only depend on the quantity of blood left after systole and the quantity entering, but still more on the resistance to dilatation offered by the cardiac walls. At the commencement of diastole when the walls are lax the pressure may be negative—*i. e.* the ventricle may exert a suction action on the auricles and the auricles on the great veins.

The resistance of the cardiac walls to distension depends partly on their native elasticity, but still more on the tone of the muscle-fibres. We thus find that the main factor in the diastolic distension of the cardiac chambers beyond the normal is not an increase of pressure within the chambers, but a diminution of the resistance offered by the cardiac walls owing to loss of muscular tone. Doubtless an increase of pressure inside the cavity in time injuriously affects the muscle tone and thus may give rise to dilatation, but a moderate and temporary increase of pressure has a stimulating effect on contractility and probably also on tonicity. Loss of tone and dilatation may take place without increase of pressure, and increase of pressure is only accountable for dilatation after having injuriously affected tone. The potent action of regurgitation through a valve in the production of dilatation of the recipient chamber is obvious, though even here dilatation does not result until the tone of the muscle is diminished. There is no need to further enlarge on the importance of the tonicity of the muscle-fibres in the prevention of dilatation of the cardiac chambers; the arguments in favour of

the above view have already been convincingly put forward by Colbeck (4).

It has been assumed that the muscle-fibres possess the function of tonicity, and the facts connected with cardiac dilatation afford one of the stronger arguments in favour of attributing this quality to heart-muscle. The experimental proof is mainly concerned with the action of drugs on the heart. Gaskell (5) has shown that certain drugs, such as sodium hydrate, antiarine, veratrine, and digitalin, increase tonicity and ultimately bring the heart to a standstill in a condition of excessive tone; incidentally it has been found that they also diminish the rate of the cardiac rhythm (*vide* Gaskell's tracings). Other drugs, such as lactic acid and muscarine,[1] diminish tonicity and ultimately bring the heart to rest in a flaccid condition; these increase the rate of the cardiac rhythm.

As a result of dilatation of the heart we find that the particular chamber or chambers affected tend to work less efficiently. Roy and Adami (3) have pointed out that for the same amount of blood to be thrown into the aorta a dilated ventricle has to contract less (*i. e.* its muscle-fibres have to shorten less) than a normal ventricle. This mechanical advantage of the dilated chamber is likely to be more than counterbalanced by the fact that it has to work against greater resistance—in other words, it has to do more work to throw the same amount of blood into the aorta. The work done by the ventricle depends on the amount of blood discharged and on the intra-ventricular pressure. According to Roy and Adami (3 and 6) the pressure on a unit of surface remaining the same, the total intra-ventricular pressure will vary approximately as the square of the radius of the cavity if the cavity is taken as approximately spherical. With a constant pressure in the aorta, therefore, the intra-ventricular pressure will increase with increasing dilatation. Even

[1] The recent work of Rhodius and Straus (Pflüger's 'Archiv,' 1905, 110, p. 492) shows that the action of muscarine on the heart is not due to any influence on the muscle tone.

with a normal discharge of blood much more than the usual residuum will be left after the systole of a dilated ventricle and with any failure of contractility the usual amount will not be discharged. The large amount of blood left in the ventricle at the end of systole will render it difficult for the auricle to discharge itself completely into the ventricle, and then lead to loss of tone in the auricle and dilatation. Dilatation of the auricles will tend to be followed by congestion of the veins, with all the attendant phenomena that are so well known in heart failure.

These aspects of cardiac dilatation need detain us no longer, but there are other aspects which require a more detailed study. Previous to entering on this it is necessary to consider the various functions of cardiac muscle on which the cardiac rhythm is dependent.

The work of Gaskell (5), Engelmann (7) and others has conclusively proved that the rhythmical beating of the heart comes from the innate qualities of the muscle-fibres and is quite independent of the central nervous system or of the intrinsic ganglia. This does not mean that the rhythm cannot be influenced by stimuli that are brought to the heart by the cardiac nerves; it only insists that the heart-muscle is capable of beating rhythmically when entirely separated from all nervous structures and that under normal circumstances it does beat rhythmically without any activity of the nervous system. The properties of cardiac muscle-fibre are, then, as follows: rhythmicity, which, as Engelmann has shown, means the building up of a stimulus, excitability, conductivity, contractility, and tonicity. These various properties are not possessed by all the muscle-fibres to the same extent; for instance, the muscle-fibres at the sinus venosus (great veins in humans) possess but poorly the property of contractility, but have rhythmicity to a higher degree than any other fibres (see below). All these several properties are possessed by all the muscle-fibres, and even small strips are capable of rhythmically beating—*i. e.* are capable of building up the stimulating substance, of being excited,

of conducting the excitation to all parts, and of contracting. Since the stimulating substance is formed most rapidly at the sinus venosus (great veins) the ordinary cardiac contraction starts there and is conducted thence through the auricles to the ventricles. The muscular connection of the auricles and ventricles in man is confined to a small band in the septum between the two sides of the heart, the bundle of His (8), and since in this band conductivity is probably less highly developed than elsewhere an appreciable time is required for the stimulus to pass from auricle to ventricle. The occurrence of the contraction destroys the stimulating substance, destroys excitability, destroys conductivity and contractility. After a contraction, therefore, a period of rest is required in which the stimulating substance is re-formed and excitability, conductivity, and contractility are gradually recovered. When the proper relation between stimulus and excitability is reached a fresh contraction takes place.

After excitability, conductivity, and contractility are sufficiently restored it is possible to artificially excite a contraction before the normal impulse descends from the sinus venosus. Such an artificial contraction is called an extra-systole, and, like a normal contraction, temporarily destroys the properties of cardiac muscle. The stimulus to produce an extra-systole may be applied at the sinus venosus, on the auricles, or on the ventricles. If applied at the sinus venosus, the stimulating substance would, of course, be destroyed, and would require the usual time for re-formation, so that the rhythm of the heart would be altered in that the beats would now take place at regular intervals after the extra-systole. If the stimulus were, however, applied on the auricles or ventricles, it might not reach the sinus before the normal stimulus had been fully formed. In this case the normal stimulus would be ineffective, since the auricles and ventricles would not have recovered their properties sufficiently to respond (they would be in the refractory stage). Since the stimulating substance is formed faster at the sinus than

elsewhere and the sinus dominates the rhythm, the auricles and ventricles would have to await the next fully formed stimulus from the sinus, and so the interval between two normal ventricular systoles would be double the ordinary interval, the extra-systole occurring in this prolonged interval. If the artificial stimulus reached the sinus before the normal stimulus had been completely re-formed the stimulating substance would be destroyed too early, but would at once start re-forming again, and the interval between two normal contractions would be less than double the normal interval. The extra-systole, since it occurs before contractility has fully recovered, is smaller than a normal contraction, although it is always the full contraction of which the heart is at that moment capable. The earlier artificial stimulus is applied in diastole the smaller the resulting contraction and the stronger the necessary stimulus to produce contraction.

The various properties of cardiac muscle can be separately influenced by various extraneous circumstances—*e. g.* nervous influences, drugs, etc.; but although they can all of them be thus separately influenced, any change in any one of them probably to some extent alters all the others. As an example of this one may instance an increase of excitability, which quickens the rate of the heart and so allows less time for the other functions to recover, leading to a diminution of contractility, conductivity, etc.

Wenckebach (1) has applied these physiological conclusions to the clinical study of patients, and he has shown that we can explain many of the types of arhythmia of the heart by the occurrence of extra-systoles or by alterations in certain of the properties of cardiac muscle. Wenckebach's views have been amply confirmed and extended by James Mackenzie (9). This observer has shown that by taking simultaneous tracings of the radial pulse and of the pulsations in the jugular veins, which appear in a considerable number of cases, it is possible to arrive at conclusions as to what is occurring in the different chambers of the heart.

The appearances presented by a graphic record of the

venous pulse in the neck vary very considerably. If, however, the record be made simultaneously with one from the radial pulse or from the apex beat, it is possible to exactly time the various waves shown in the venous pulse and to interpret their significance fairly accurately. In the various tracings given here the jugular pulse and the radial have been recorded simultaneously. By keeping the clockwork still for a moment upright lines are obtained in both the venous and arterial records which exactly correspond with one another in time, and therefore afford standards for measuring off corresponding points on the two curves. In the radial curve we can mark off the period during which the aortic valves are open. This

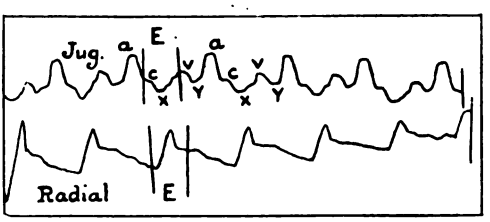


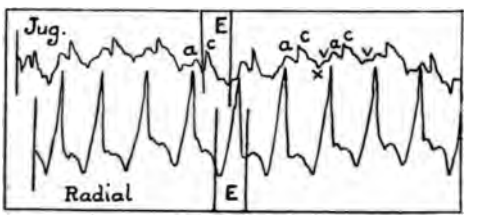
FIG. 1.

period is called the sphygmic period, and is marked with an *E* in all the curves. An exactly corresponding space, the sphygmic period *E* can, therefore, be marked out on the venous tracing, and thus we are enabled to exactly time the waves in that curve. In most of the tracings the sphygmic period is preceded by a wave *a*, which is evidently due to the contraction of the right auricle; this is usually followed by a fall *x* during the sphygmic period, which must be caused by the emptying of the veins by the dilatation of the auricle.[1] When present this fall *x* affords one of the most certain signs of the previous contraction of the auricle, since the auricle cannot dilate unless it has

[1] Keith (10) has shown that this is mainly caused by the ventricular systole pulling down the auricular floor. In Figs. 1 and 2 the fall *x* coincides with the ventricular systole. With a very dilated auricle the ventricular systole can have no such effect, and hence the fall *x* disappears from the venous pulse.

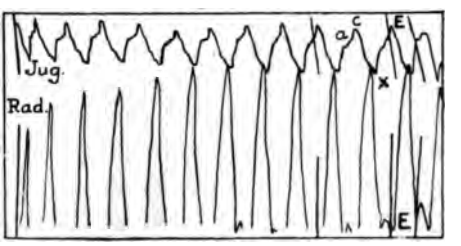
previously contracted. The fall *x* is nearly always broken by a wave *c*, which, as on Fig. 2, may be higher than the auricular wave *a*. From its early position in the sphygmic period this wave is evidently due to the pulsation in the carotid artery. Only under exceptional circumstances is

FIG. 2.



it possible to obtain a tracing of the jugular pulse without this wave due to the carotid, but its presence is rather an advantage than otherwise, as it helps in the accurate timing of the other waves. The fall *x* is often followed by a wave *v*, which lasts beyond the sphygmic period and is due to the passive filling of the veins. This wave can

FIG. 3.



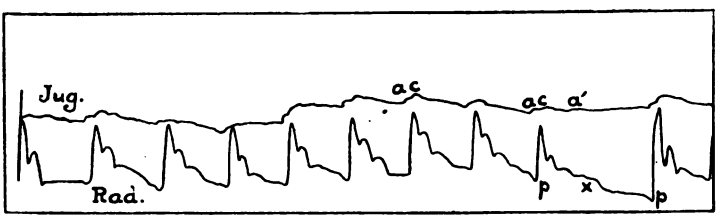
also be seen in pressure curves taken in the auricle itself. The wave *v* is followed by the fall *y*, which is evidently due to the diastole of the ventricles. In some cases, as in Fig 3, the wave *v* is large, and no trace of the fall *y* is obtained; here the auricular contraction *a* merely appears as a small wave joined to this large wave. Other variations of the venous pulse will have to be remarked on

later. The jugular pulse thus gives us information as to variations of pressure in the right auricle and, in cases of incompetence of the tricuspid valve, of variations of pressure in the right ventricle; the radial pulse similarly tells us about the left ventricle, and a careful study of both throws much light on the action of the heart in the varying circumstances of disease. It is not uncommon with dilatation of the heart to get enlargement of the liver, and this may pulsate owing to the right auricle or ventricle throwing blood forcibly back. Tracings can also be obtained from the liver which are simpler in character than those obtained from the jugular vein but which show exactly the same essential characteristics, and fully confirm the conclusions drawn from the study of the venous pulsations in the neck. It is only in exceptional cases, of course, that any pulsation occurs in the jugular veins, the prevention of regurgitation into the veins being partly due to their valves, but still more, as Keith (10) has pointed out, to the tone of the muscle-fibres surrounding the opening of the veins into the auricle.

It is probable that the heart is constantly exposed to a number of stimuli apart from the stimulus it rhythmically builds up for itself. These stimuli may arise from nervous impulses, from alterations in the composition of the blood-supply, or from foreign substances contained in the blood, and from tension of the muscle-fibres themselves. Under ordinary circumstances these stimuli are not sufficiently powerful to excite a contraction, but with an increase of the stimulus, or an increase of excitability of the muscle, an extra-systole would be produced. Of these two, an increase of excitability is the most frequent cause of extra-systoles in human beings. Such extra-systoles may start from the auricles or from the ventricles, and in cases showing a venous pulse it is easy to ascertain their point of origin. An example of an extra-systole is shown in Fig. 4. Here the regular contraction of auricle and ventricle is shown in the jugular and radial pulses, but at the point x in the latter an abortive systole has occurred

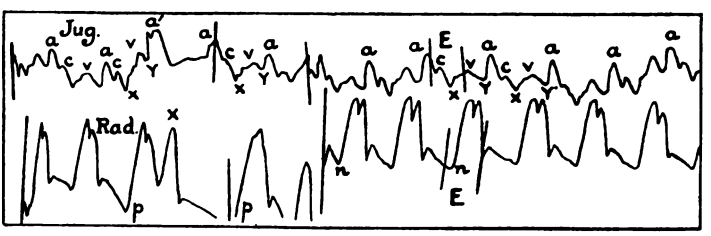
too early, and in the jugular tracing an early contraction of the auricle at *a′* is also to be seen. The interval *p—p* is less than that between two ordinary pulse-waves, which means that the contraction had reached the great veins

FIG. 4.



before the normal stimulus had been fully re-formed. After the long interval *x—p*, the compensatory pause, the succeeding contraction is larger owing to the longer time given to the contractility for recovery. An extra-systole starting from the ventricle is exemplified in Fig. 5 at *x*. In the latter part of this tracing the heart is found to be

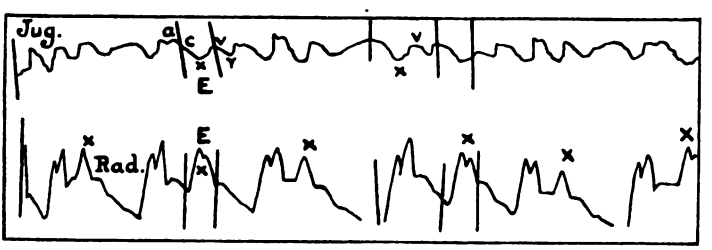
FIG. 5.



beating quite regularly, and the auricular beat *a* occurs at regular intervals. In the early part of the tracing, however, the large wave *a′* is found at the exact spot where the auricular wave would be expected with regular action of the heart. This large wave *a′* must evidently be due to the auricle, which is beating quite regularly, but it also corresponds in time with the extra-systole *x* which

has occurred at this point in the ventricle. Hence the systole of the auricle here takes place while the ventricle is contracting. The large size of the wave a' is explained, according to Mackenzie (11), by the fact that the auricle on its contraction finds the ventricle in systole, and being, therefore, unable to send its blood into the ventricle, is compelled to throw it all backwards into the veins. Where the extra-systole starts from the ventricle the contraction has not time to reach the great veins before the normal stimulus is completed, and therefore, as explained above, the interval between two normal beats is practically double the normal interval (actually it is a little less than double

FIG. 6.

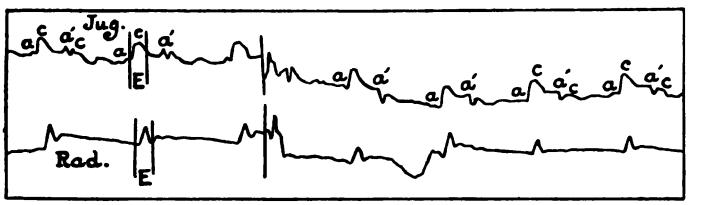


the normal interval, as the contraction succeeding the long rest is conducted more rapidly to the ventricles, and so occurs a little early there). In Fig. 5 the interval p—p is equal to the double interval n—n.

At times, particularly in cases of valvular disease with somewhat dilated hearts, extra-systoles tend to occur regularly, giving rise to the so-called bigeminal and trigeminal pulses. An example of this condition is given in Fig. 6, where it will be seen that the extra-systoles are of auricular origin, the rhythm of the auricles exhibiting the same irregularity as that of the ventricles. When the extra-systole of the ventricle takes place very early and so is very feeble no pulse may be felt at the wrist and no wave may even be shown in the sphygmogram. In this way a false bradycardia may result, the correct interpreta-

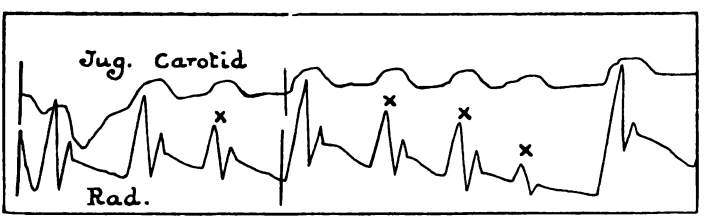
tion of which may at times be difficult. For instance, in Fig. 7 the pulse as felt at the wrist was 48 and the radial tracing shows the same number of beats per minute; the jugular pulse is obviously beating at double this rate.

FIG. 7.



Owing to extensive bronchitis and very considerable dyspnœa the heart sounds were very difficult to make out, and at first I considered that here only every other beat of the auricles was followed by the ventricles owing to an abnormal lowering of excitability, as in the case recently reported by Hay (12). Subsequent tracings, however,

FIG. 8.

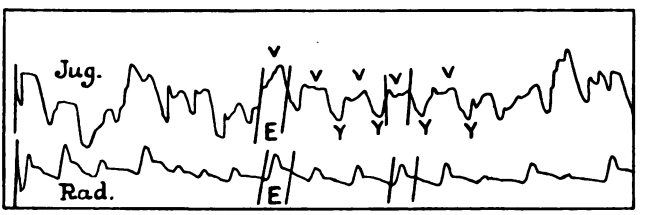


when the general condition of the patient had greatly improved and the venous pulse had disappeared, showed the remarkable collection of extra-systoles which can be seen in Fig. 8. This means that the excitability was heightened instead of lowered, and that the correct interpretation of Fig. 7 is that an extra-systole had started from the auricles after each normal beat, but that the

ventricular beat was not of sufficient strength to send a wave to the radial pulse.

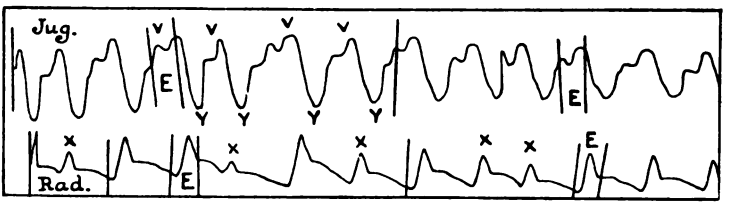
When the excitability is greatly increased the number of extra-systoles may be so great as to completely obscure

FIG. 9.



the normal regular cardiac rhythm. An example of this is given in Fig. 9, where all the beats appear as if they were extra-systoles of ventricular origin and it is impossible to recognise any rhythm. The administration of digitalis rendered the heart in this case less rapid and less irregular, and in Fig. 10 from the same patient, after three days'

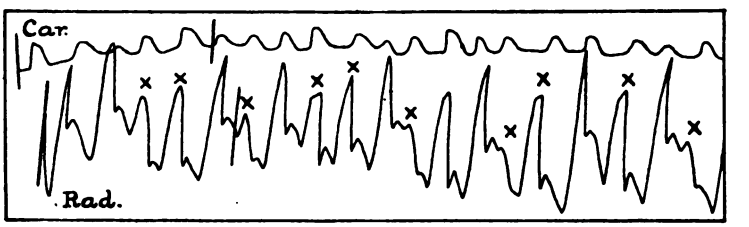
FIG. 10.



treatment with ʒii of infusion of digitalis every 6 hours, it is easy to recognise a rhythm and to distinguish between extra-systoles and normal contractions. Extra-systoles are, of course, not confined to dilated hearts, but may be found with hearts that are perfectly healthy, but increased excitability is one of the most prominent features of cardiac dilatation, so that it is not surprising to meet with extra-systoles more frequently in this condition.

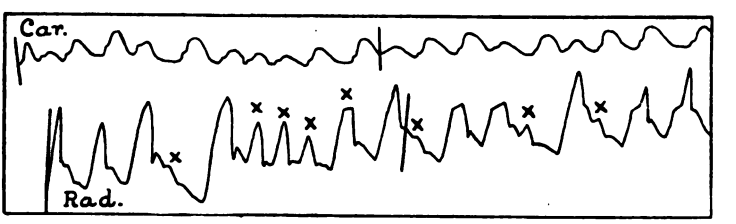
Wenckebach[1] (13) is not satisfied that the theory of extra-systoles is sufficient to explain the "delirium cordis" shown in such figures as Figs. 9 and 17. He lays stress on the absence of the compensatory pause after the presumed extra-systoles, and asks how one is to say which

FIG. 11.



are extra-systoles and which normal contractions. To explain this excessive irregularity he, therefore, supposes that the stimulus is irregular in its formation, taking longer to form for one beat than another. In the tracings given above I have demonstrated how with increasing excitability we get more frequent extra-systoles, giving

FIG. 12.



all gradations up to the "delirium cordis" in Fig. 9. How complex an irregularity may result from numerous extra-systoles is well illustrated in Figs. 11 and 12, which are taken at the same sitting from a patient with mitral stenosis and marked irregularity in the pulse felt at the wrist. The tracing from the neck does not show any

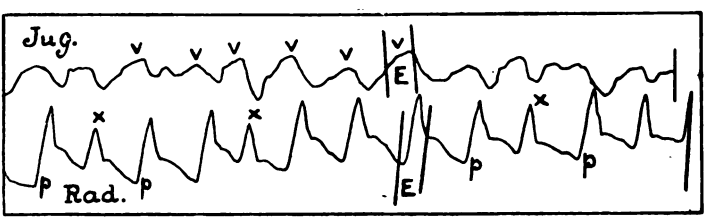
[1] Professor Wenckebach has altered his views since the publication of 'Die Arhythmie.'

venous pulsation, but the carotid pulse exhibits the extra-systoles even where hardly any pulse appears in the radial tracing. In the tracing from the wrist extra-systoles are obviously shown at the points marked *x*. In these tracings, and in Fig. 8, where several extra-systoles follow one another, the compensatory pause is only found after the last one. It is a small step from the condition of this case, where the extra-systoles are as numerous as the ordinary contractions and occur irregularly, to the condition shown in Fig. 9, where all or nearly all the contractions have been presumed to be extra-systoles. With excessive excitability, too, we should expect most or all of the contractions to be extra-systoles, unless the rate of formation of the normal stimulus be also very greatly increased.

There is a further point with regard to the irregularity of the rhythm in "delirium cordis." As will be pointed out later, the inception of the rhythm in these cases is assumed by the ventricles. It has been shown that under ordinary circumstances when an extra-systole starts from the ventricle, the compensatory pause is the longest possible, since the extra-systole does not reach the starting-point of the normal rhythm (viz. the great veins) until the full development of the normal excitation, and the ventricles have to await the next normal excitation before their next normal systole can occur. Where, however, the rhythm of the heart starts from the ventricle, the place of its origin will be close to, or identical with, the place of origin of a ventricular extra-systole, and hence the compensatory pause will be short, as in cases of extra-systoles arising near the great veins, or absent, as in cases where an extra-systole arises from the great veins themselves. When the ventricles take on the inception of the cardiac rhythm, there is usually so great an irregularity that it is difficult to get two normal systoles together so as to ascertain the normal interval, but in the tracings given in Fig. 13 extra-systoles obviously occur at x^1 and *x*, while the other contractions are normal ones. The tracing was

taken from a woman aged 66, in whom for the past three months the rhythm had been ventricular in origin and invariably irregular. Each extra-systole occurs too early after the preceding normal beat, and in the case of x^1 is followed by a compensatory pause which is so short that the interval p—p is less than double the interval between two normal contractions—*i. e.* the extra-systole must have started near the point of origin of the normal rhythm. The interval after x is only of the same length as the interval between two normal contractions, so that the

Fig 13.



In this figure the first and third × signs should be ×′ instead of ×.

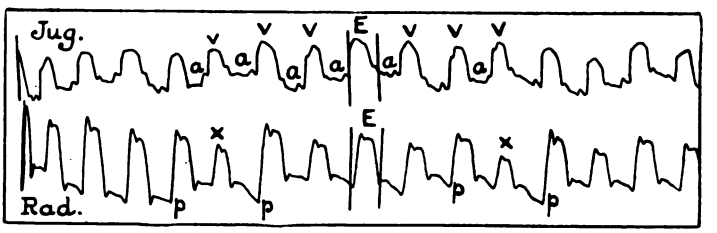
extra-systole must have started from the same point as the normal beats (compare also Fig. 10). It is thus evident that an increase of excitability and the consequent occurrence of extra-systoles affords an amply sufficient explanation of the irregularity that is found in dilatation of the heart, even of an irregularity so excessive as to justify the name of "delirium cordis." This explanation is, too, a much simpler one than that afforded by the supposition of an irregularity in the time of formation of the stimulus.

One of the striking alterations in the cardiac rhythm associated with dilatation is the increase in rate which we invariably find. An increase in rate may be due to two causes, (1) increased rate of production of the stimulus, and (2) increased excitability. In most cases both these factors are probably at work, especially where the increase of rate is very considerable. With a very rapid heart

and so a greatly increased rate of production of the stimulus little opportunity is afforded for the occurrence of extra-systoles. We, therefore, usually find that with great rapidity of the heart the rhythm is regular and not infrequently with reduction of rate irregularity makes its appearance. This point will be referred to again when considering the action of digitalis on the heart.

The significance of the various waves that are obtained in a tracing of the usual type of venous pulse has been already explained; *a* the wave from the auricular contraction, *c* the carotid wave, *x* the fall due to auricular diastole, *v* the wave due to diastolic filling of the veins,

FIG. 14.



and *y* the fall due to diastole of the ventricle. Regurgitation through the tricuspid valve naturally alters the appearance of the venous tracing very materially. Slight regurgitation shows itself by an increase in the wave *v* and by its earlier occurrence. With greater incompetence of the valve this wave from the ventricle will occupy the whole, or nearly the whole, of the period of the ventricular systole, obscuring the wave *c* due to the carotid. For instance, in Fig. 14 the whole of the sphygmic period *E* is occupied by the great wave *v*, which is preceded by the small auricular wave *a*. In Figs. 9 and 10 the wave *v* is seen to extend even beyond the sphygmic period and to last throughout the ventricular contraction, since the small resistance offered by the veins allows the ventricle

to force blood into them after the increased aortic pressure has closed the semilunar valves.

Should the auricles lose their tone in cases of incompetence of the auriculo-ventricular valves, they would be violently distended by the blood forcibly thrown into them by the ventricles and hence would become extremely dilated. In consequence of this extreme dilatation the contraction of the thin-walled auricles would be very inefficient and the refilling of the ventricles would mainly depend on the quiet flow of blood into them during diastole. Mackenzie (14) has discovered that there is a further very remarkable phenomenon associated with that more extreme dilatation of the heart which we are accustomed to call heart failure. This is the transference of the inception of the cardiac rhythm from the great veins to the ventricles. One might reasonably suppose that this was caused by the progressive dilatation of the auricle eventually paralysing it. That this is not the correct explanation is proved by the fact that in some cases of acute dilatation (*e. g.* in paroxysmal tachycardia) the starting of this peculiar type of cardiac rhythm is quite sudden, and with recovery the auricles and ventricles again beat in their normal relation to one another. Further, it can be shown that in some cases the auricles are not paralysed, but continue to beat although their systole takes place at the same time as that of the ventricles. The fall *x* in the jugular pulse (*vide* Fig. 1) is due to the diastole of the right auricle and occurs, as a rule, early in the period of the ventricular systole. It is impossible to imagine any other cause for a marked fall in the venous pressure during the ventricular systole except the diastole of the auricle. The regular repetition of such a fall in the venous tracing becomes, therefore, one of the most certain signs of the occurrence of auricular systoles. I have been fortunate enough to meet with two cases showing the simultaneous contraction of auricle and ventricle. The girl from whom I took the tracing in Fig. 15 had chronic disease of the mitral valve with a regurgitant murmur and

was suffering from a recent attack of rheumatic pericarditis and some dilatation of both the right and left sides of the heart. In this tracing a wave *a* occurs regularly in the early part of the sphygmic period followed by the fall *x* then the wave *v* and the fall *y* due to ventricular diastole. The fall *x*, as pointed out above, must be due to the diastole of the auricle, and the only trace of the necessary preceding contraction of the auricle is the wave *a*. The systole of the auricle requiring so much less time than that of the ventricle, we find both the systolic rise in the veins and the diastolic fall from suction of the auricle completed during the sphygmic period.

FIG. 15

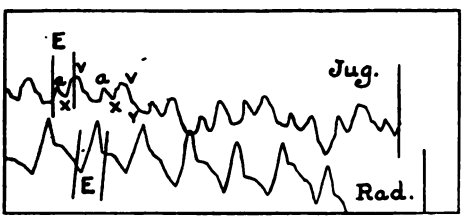
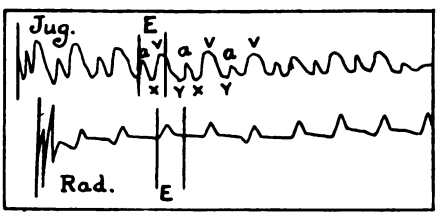


FIG. 16.

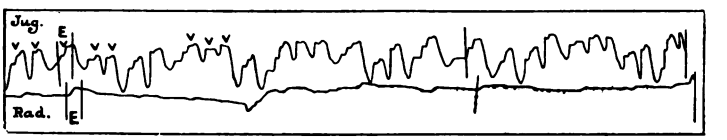


Mackenzie (15) has stated that this type of cardiac rhythm may be permanent or may disappear with recovery of muscle tone. Fig. 16 from the same patient a week later than Fig. 15 shows that the same type of rhythm was still present, as did another record, ten days after the first. Fourteen days after the first tracing the venous pulse had disappeared and the chambers were probably beating in their proper order. The patient made a progressive improvement during these fourteen days and the cardiac dilatation became less.

It is more usual in cases where the rhythm starts from the ventricles to find no evidence of an auricular contraction, as in Figs. 9, 10, 13, 17, and 18, where in the jugular pulse there is only a single wave *v*. It is noticeable that this wave *v* starts before the sphygmic period *E* and lasts

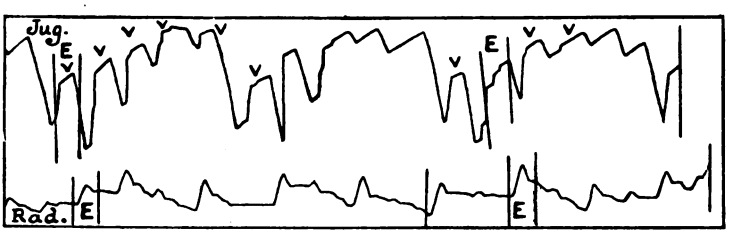
beyond it. The ventricle, however, is in systole for an appreciable period before the aortic valves open, and for an appreciable period after they close—*i. e.* before and after the sphygmic period, and it would be quite possible

FIG. 17.



for the ventricle to force blood into the veins where the resistance is low when the high arterial pressure closes the aortic valves. It is probable that the contraction of the auricles where the cardiac rhythm takes its start in the ventricles is a hindrance to the circulation, since it would hamper the flow of blood towards the heart from the veins

FIG. 18.



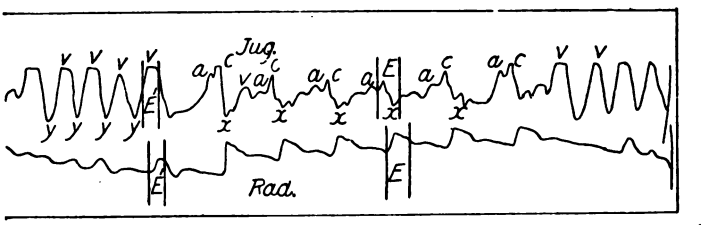
without itself sending any blood through the auriculo-ventricular valves into the contracting ventricles, but this condition would be more easily recovered from than that where the auricles are merely passive. It can well be imagined that this latter condition when once established tends to become permanent, and it is remarkable how well the circulation can be maintained even for years without

the auricles working. The patient from whom Figs. 9 and 10 were taken improved greatly while in the hospital, and was able to get up and move about fairly well, although there was, of course, considerable dyspnœa on exertion. Nevertheless on all of the numerous occasions that she was examined the jugular pulse was of the same character, showing permanent paralysis of the auricles. The patient from whom Fig. 17 was obtained never showed any sign of an auricular contraction, but this patient died after some weeks in the hospital from heart failure. Mackenzie (15) has been able to examine the hearts post mortem of several patients who during life exhibited this evidence of paralysis of the auricles, and in all he found a very extreme degree of auricular dilatation, and this was also found in the patient from whom Fig. 17 was taken. Although the venous pulse only gives information as to the condition of the right auricle, it is probable that the left is paralysed in these cases as well. Mackenzie (16) has noticed in cases of mitral stenosis that with the onset of heart failure and the inception of rhythm by the ventricles the pre-existing presystolic murmur disappears, being sometimes replaced by a diastolic bruit, best heard at the apex. Personally, I have not heard a presystolic bruit in any of the four cases recorded here, nor in the one or two others with the same type of venous pulse that I have come across. The patient whose pulses are recorded in Figs. 9 and 10 had a diastolic murmur at the apex, and no doubt had some stenosis of the mitral valve, and at the post mortem on the patient who gave Fig. 17 stenosis of both mitral and tricuspid valves was found.

When fully established this type of cardiac rhythm starting from the ventricles tends, as stated before, to become permanent; but in the short and temporary attacks of heart failure which are not uncommon during the course of valvular disease the rhythm, after a temporary derangement, may return to the normal order of great veins, auricles and ventricles. This is perhaps best

illustrated in cases of paroxysmal tachycardia of which some excellent tracings are given by Mackenzie (17) and Hay (18). The obvious indication for treatment is to try to get the normal rhythm restored as soon as possible in cases of heart failure, or, in other words, to restore the muscle tone as soon as possible. Mackenzie does not find the ventricular inception of the rhythm common in the chronic myocarditis of older people without valvular disease, whereas Hay (18) finds it common. Personally I have taken tracings from about a dozen patients in whom I had diagnosed chronic myocarditis, and in two I found the rhythm permanently starting from the ventricle (see

Fig. 19.

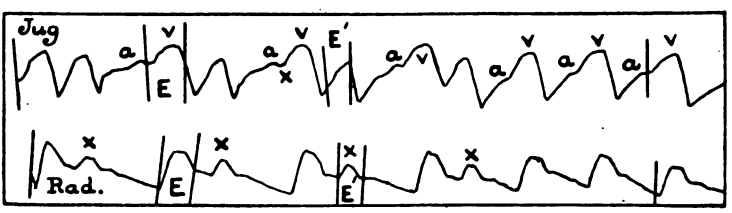


Assumption of the inception of rhythm by the ventricles.

Fig. 13). I have not yet had an opportunity of seeing the actual development of this type of cardiac rhythm, but Fig. 19, which I reproduce with the kind permission of Dr. Mackenzie, shows the change from the normal to this abnormal type. At *E* is the normal rhythm, *a* the auricular contraction before the sphygmic period *E* and in the latter *c* due to the carotid and the fall *x* due to the auricular diastole, while at *E'* there is no sign of an auricular systole, the wave *v* being entirely caused by the ventricular systole. Previous to a change from the normal inception of the rhythm at the great veins to this abnormal inception by the ventricles it is apparently usual to meet with a number of extra-systoles starting from the ventricles. Gaskell (5) has shown that in animals it is possible to make the rhythm start from the ventricles

by stimulating them sufficiently rapidly. What actually causes the auricles to cease contracting in these cases is apparently firstly the loss of tone of the auricular muscle and then on the top of this the excessive distension of the chambers by the ventricles forcing blood through the incompetent auriculo-ventricular valves before the arrival of the time for the auricular contraction. This is well illustrated in Fig. 20. It will be noticed that at the commencement of the tracing an extra-systole of ventricular origin occurs after each normal beat, producing the so-called bigeminal pulse. At *E*, which marks the sphygmic period of a normal contraction, the large wave *v* due to

FIG. 20.



tricuspid regurgitation is preceded by the small auricular wave *a* while at *E'*, the sphygmic period of an extra-systole, there is no trace of any contraction of the auricle, the early ventricular systole forcibly distending the auricle before the end of its diastole and preventing the systole. Mackenzie has pointed out that the ventricular inception of the rhythm is invariably associated with irregularity unless the action is very rapid, a fact that one may express otherwise by saying that it is always accompanied by a great increase of excitability and the development of frequent extra-systoles (*vide* Figs. 9, 10, 13, 17, and 18).

Certain of the phenomena associated with cardiac dilatation are secondary to the impaired working of the circulation —*e. g.* the dyspnœa, the dropsy, the congestion of the lungs and liver, etc., while others are more directly concerned with the heart itself such as pain, the actual dilatation of

the chambers, the rapid irregular action, and the assumption by the ventricles of the post of starter and controller of the cardiac rhythm. The subject of pain associated with disease of the heart has been fully dealt with by Head (19) and Mackenzie (20) and need not be entered into here. The rapid and irregular action has usually been referred to the influence of the nervous system, and much ink has been spilt in discussing whether it is due to loss of vagus control or to excitation of the accelerator nerves. There is no doubt that impulses can be conveyed to the heart by the nerves exciting or inhibiting cardiac contraction or influencing the functional activities of the cardiac muscle. Tonicity would as certainly be subject to these influences, either in a positive or a negative sense, as the other properties of the muscle-fibres; and it is because of the nervous influence on tonicity that we meet with the remarkable instances of sudden dilatation of the heart, so well known to all clinicians, from emotional disturbance, pain, etc. (4). Without, however, some permanent alteration in the central nervous system or some permanent irritation to produce reflex phenomena, such nervous influences on the heart would be expected to be but temporary, the only evidence we have of a constant action of the nervous system on the heart being the tonic effect of the vagus in slowing the rhythm. In cases of chronic heart disease there is a permanent increase of rate, and frequently a permanently irregular action, and yet we have no evidence of there being any organic lesion of the nervous system. On the face of it, therefore, the probable source of these alterations of function would be likely to be found in the heart itself, which is demonstrably changed.

We have seen that the main factor in the causation of dilatation of the heart is a depression of the tonicity of the muscle-fibres, and yet dilatation is associated with a considerable increase in at least two of the other properties of the muscle, excitability and the rate of formation of the stimulus. It is possible that another property is also increased, viz. contractility, since the dilated heart has

often to do more work than the normal one. The question of contractility is, however, a complicated one. The heart-muscle is like an after-loaded skeletal muscle, and may do more work the greater the load, within limits, the greater tension of the fibre causing it to possess greater contractility; or, as Engelmann would put it, the tension has a positive inotropic influence. On the other hand, the dilated heart beats more rapidly, and thus less time is afforded to the contractility to recover, so that it would tend to be diminished. Nevertheless, allowing for all this, a real increase of contractility is not improbable. An alteration of conductivity is still more difficult to estimate, but there is evidence that with an increase of tone in some cases this property is diminished and therefore it is possible that it is increased with loss of tone. It has seemed to me that the whole of these phenomena, as well as certain other peculiarities of cardiac rhythm, may be explained by the alteration of the tone of the muscle (21).

It has been already pointed out that an alteration in any one of the properties of cardiac muscle will affect to some extent all the others. Alterations in tone are even more likely to influence the other properties than changes of any other function, since tone has a considerable resemblance to an actual contraction. Now, a contraction destroys all the other properties, and we would therefore expect tonicity to diminish these other properties either directly or by delaying their recovery after a contraction. On the other hand, diminution of tone should tend to augment the other properties, or to hasten their recovery after a contraction. With lessened tone, therefore, we should expect an increased rate of production of the stimulus, increased excitability, heightened conductivity, and contractility.

Gaskell (5) writes: "It would not be unreasonable to expect that different parts of the heart would vary in their tonicity, just as they vary in their rhythmicity." There seems to be a general assumption that tone is greater in the ventricles than in the auricles; and considering the greater amount of work that the ventricles

have to perform, this assumption probably accords with the facts. The different appearance of the muscle-fibres in different parts of the heart has been noticed by Gaskell (5), the most characteristic and best striated fibres being found in the ventricles, the least characteristic in the sinus venosus, the fibres that join auricles and ventricles, and the conus arteriosus. It is not unlikely that the special properties of heart-muscle are present to the greatest extent in the most highly developed fibres, special structure and special function going together, this applying even more to the important property of tonicity than to the other properties of cardiac muscle. The greater relative tonicity of the ventricular muscle would keep the other functions in subjection, so that under ordinary circumstances the stimulating substance would be built up more slowly there, excitability would be less, as also would contractility and conductivity. The ultimate relation of these various properties to one another would naturally depend on the proportions in which they were originally present, which need not necessarily be the same all over the heart. Where tone is least developed the stimulating substance would be built up most rapidly, as at the sinus venosus, at the auriculo-ventricular junction, and at the conus arteriosus, places from which Gaskell (5) has shown an intrinsic rhythm is particularly prone to start, but where conductivity and contractility are much less developed than elsewhere. With loss of tone there would be a general exaltation of the other functions, which would especially affect the ventricles where these functions are the more highly developed. With increasing dilatation, or loss of tone, the sequence of events would be expected as follows: an increase in the heart rate, the occurrence of extra-systoles, at first auricular in origin, but later arising from the ventricles, and at last the transference of the starting-point of the cardiac contraction from great veins to ventricles. With the transference of the control of the rhythm to the ventricles extra-systoles and so great irregularity would be conspicuous

owing to the great increase of excitability, except where the cardiac rhythm is very rapid. This sequence of events is precisely what we actually find in experience of dilating hearts. Contractility should be heightened, and I have pointed out that it probably is heightened in some cases, but the changes in contractility and conductivity are difficult to judge owing to the marked influence on them of alterations in the cardiac rate. These functions may be, and indeed sometimes are, injuriously affected by the same cause that depresses the tonicity.

Experimentally it has been shown that certain drugs increase tonicity until the heart is stopped in systole, and, as would be expected from the above theory, these drugs diminish the rate of the heart. Similarly drugs that decrease tonicity increase the rate. The action of one of these drugs, digitalis, has been extensively studied in the therapeutics of the human heart. That it increases tone has been shown experimentally; clinically we find that it diminishes dilatation, decreases the rate, and renders the heart less irregular (*i. e.* diminishes excitability), all of which actions would be expected from its influence on muscle tone. It is true that in some cases an irregularity appears after the administration of digitalis, as in the case recently reported by Fauconnet (22), where digitalis reduced the pulse from 120 per minute to 48, this infrequency being found by the sphygmogram to be due to the regular occurrence of an extra-systole unfelt by the finger after each normal beat, causing a bigeminal pulse, so that the heart was really beating at 96 per minute. Such an irregularity does not mean an increase of excitability from the digitalis, but is merely a consequence of the reduction of rate, allowing an opportunity for the occurrence of extra-systoles in a heart that was always over-excitable.[1] Contractility and conductivity are

[1] This view is borne out by Hering's ('Deut. Archiv f. klin. Med.,' 79, p. 175) observations on the disappearance of a bigeminal pulse from the administration of atropine, in which he found that the heart only became regular when its rate was so increased that the normal impulse arrived

usually favourably affected by the increased rest afforded by the diminished rate, this being more potent than the depressing effect of increased tonicity, but under certain circumstances they can be shown to be injured by digitalis.

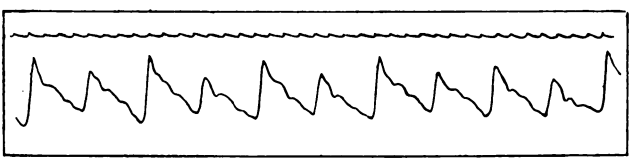
It has been found that where the contractile power of the muscle is lowered in experiments with excised hearts a peculiar alternating type of cardiac rhythm is prone to set in where the heart is regular in time, but the beats are alternately larger and smaller in size. Hoffmann (23) demonstrated that with depressed contractility the beats are not only smaller, but last a shorter time. Wenckebach (24) explains the alternating action of the heart by the longer pause after such a small short contraction allowing the contractility to better recover, with the result that the next contraction is larger and lasts longer; the next pause is, therefore, shorter and is followed by a smaller and shorter contraction, which is succeeded by a longer pause and then a larger contraction, and so on. Depression of contractility results from a number of causes—*e. g.* action of water (25), vagus irritation (23), poisons, etc. Among the latter may be especially mentioned antiarin and digitalis (26), both of which have been shown to depress contractility and to induce an alternating action of the heart. For instance, Straub (27), working with antiarin, quickly induced an alternating action of the heart and ultimately before the heart stopped in systole the smaller beats disappeared, reducing the rate of the heart to half. Although excitability and conductivity were both also depressed, this halving of the frequency seems definitely to have been due to the damage done to the contractility. It is noticeable that in Straub's experiments the effect on the ventricles was more marked than on the auricles and on the auricles than on the veins, so that the ventricles at one time were beating at half the rate of the auricles and at another the

as soon as, or sooner than, the regularly recurring extra stimulus which caused the bigeminal pulse; as the effect of the atropine wore off, and the action became less frequent the bigeminal character returned.

auricles at half the rate of the veins. The ventricles, too, were first stopped by the drug and then the auricles and lastly the veins. These remarkable results are explainable on the supposition that the tonicity is relatively greatest in the ventricles and that antiarin acts on the other functions of cardiac muscle through tonicity, but are difficult to account for on any other hypothesis.

An exactly similar alternating action of the heart has been described several times in human beings since the original case described by Traube in 1872. By the kind permission of Dr. Mackenzie I reproduce a tracing of his showing this condition excellently (Fig. 21). It is probable that all these cases, or nearly all, have resulted from the administration of digitalis, and that in all of them the

FIG. 21.

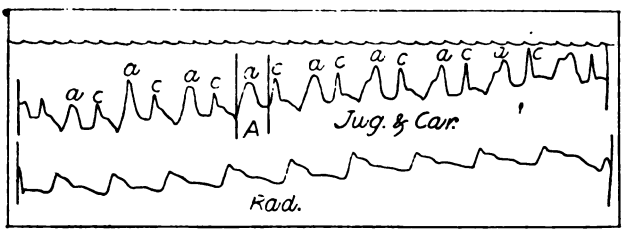


contractility was already depressed before the exhibition of the drug.

The jugular pulse when present gives us, as Mackenzie (28) has shown, a very accurate method of estimating the condition of conductivity. Under ordinary circumstances the contraction takes a certain time to pass from auricle to ventricle, which can be estimated by the difference in time between the wave *a* and the wave *c* in Fig. 1 or 2. Of course other factors help to cause this interval, viz. the systole of auricles and the presphygmic period of the ventricular systole or that portion of it before the aortic valves open. These factors, however, occupy a constant time and so do not cause any variation in the *a*—*c* interval. Depression of conductivity will lengthen this interval and if extreme will prevent some beats passing to the ventricle. Where conductivity is already depressed the administration of digitalis may depress it further, leading, as in Figs. 22

and 23, to every other beat being dropped. In Fig. 22 the *a*—*c* interval, as shown by the time-marker, is two fifths of a second instead of the normal one fifth, whereas in Fig. 23 after the administration of digitalis every second beat of the auricles is not followed by a ventricular beat. It is thus seen that increase of tone, as induced by drugs,

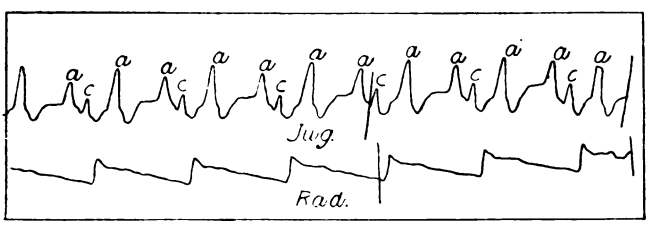
FIG. 22.



is associated with diminution of all the other properties of cardiac muscle, while loss of tone in dilatation is accompanied by an increase certainly of some, and probably of all, the other properties.

It is difficult, then, to avoid the conclusion that increase and diminution of tone stand in causal relationship to diminution and increase of the other properties.

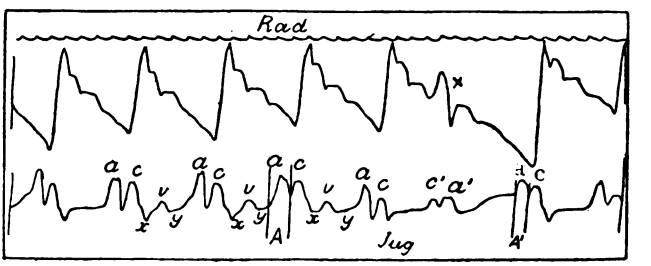
FIG. 23.



Mackenzie (14) has expressed the opinion that where the ventricles take on the inception of the cardiac rhythm the real place of origin of the contraction is in the fibres joining the auricles and ventricles. In support of this view he points to Gaskell's (5) observation that when the sinus venosus has been prevented from starting the

cardiac rhythm, as by vagus stimulation, the ventricle may take on a rhythm starting from the canalis auricularis. In some of his own observations where conductivity was depressed Mackenzie has found a remarkable recovery after an extra-systole as in Fig. 24 ,where the ordinary *a*—*c* interval is double the usual time, but the interval *A'* after the extra-systole *x* is only the normal length. This extra-systole *x* is ventricular in origin, the auricular wave *a'* occurring after the carotid wave *c'*, which corresponds with the wave *x* in the radial. From this striking recovery of conductivity he concludes that the *A*—*V* fibres must have been stimulated some time before the auricular contraction *a'* in order to give sufficient time for this

FIG. 24.



recovery. This explanation which is a probable one for the particular extra-systole quoted, does not afford, as Mackenzie himself acknowledges, an absolute proof that all contractions start from the *A*—*V* fibres in cases where the ventricles start the rhythm. On my theory of cardiac contraction, where tonicity plays such an important *rôle*, in cases of the ventricular inception of the rhythm the contraction would be expected to start from the highly-developed ventricular fibres, although the theory does not absolutely preclude the *A*—*V* fibres, as the place of origin. It is to be remembered that an explanation has to be found, not only for the ventricular inception of the rhythm, but also for the increased rate and for the irregularity. The increased rate is the more remarkable as the normal ventricular rhythm, as shown in cases of complete "heart block,"

where the ventricular beat is quite independent of the auricles, is only 28—36 per minute.

Where, however, the patient recovers from the attack of heart failure, or, in other words, there is some restoration of muscle-tone, in cases in which the auricles are so far paralysed that they are incapable of contracting again so that the rhythm remains ventricular, it would be expected that the inception of rhythm would be taken on by the least highly developed fibres which have the smallest amount of tonicity. Such fibres are the $A—V$ fibres, and it is, I think, probable that in such tracings as in Figs. 10 and 13 the rhythm starts from these $A—V$ fibres. This opinion is borne out by the different types of extra-systoles found in Fig. 13.

The hypothesis that loss of tone leads to the ventricular inception of the rhythm requires certain assumptions; these assumptions are, however, in themselves not improbable, and they afford an explanation of the selective action of antiarin and digitalis on the contractility of the ventricles.

Mackenzie ('Brit. Med. Journ.,' 1906, vol. ii, p. 1007) has recently shown that in chronic myocarditis with failure of contractility the ventricular rhythm may start without dilatation. Here another explanation than I have offered must be found, but the same result may follow from different causes, and my explanation may be the true one in dilatation of the heart.

In conclusion, I have to thank my colleagues, Drs. de Havilland Hall, Murrell, and Hebb for their kindness in allowing me to make use of the patients in their wards.

Particulars of the Tracings.

Fig. 1.—Simultaneous tracing of the radial and jugular pulses from a young man, aged 20, with mitral stenosis. There was dilatation of the left ventricle, some cyanosis, marked pulsation of jugular veins, and considerable limitation of the area of cardiac response.

Fig. 2.—Radial and jugular tracing from T. A—, a man aged 34, who had marked tremor of the hands and tongue, and slight dyspnœa on exertion. The heart's action was excited and rapid, and there was a systolic murmur at the bottom of the sternum. Slight pulsation of the jugulars could be seen. Patient had never had rheumatism and denied excess of alcohol or tobacco.

Fig. 3.—Radial and jugular tracing from M. S—, a woman aged 43, suffering from cirrhosis of the liver and ascites. Physical examination of the heart showed nothing abnormal, but there was marked pulsation of the jugulars.

Fig. 4.—Radial and jugular tracing from E. F—, a woman aged 40, with mitral stenosis and regurgitation. Shows an extra-systole of auricular origin, the beat *a* in the jugular being before the beat *x* in the radial. The extra-systole is followed by the long pause *x–p*, and the interval *p–p* is less than double the normal interval.

Fig. 5.—Radial and jugular tracing from M. B—, female, aged 28. A patient of mine for the past three years with heart disease. Her face was congested and there was considerable limitation of the area of cardiac response as well as a good deal of pain over the heart. The heart was much enlarged, the apex beat being in the fifth space about an inch external to the nipple, diffuse and heaving. There was a presystolic thrill at the apex and presystolic and systolic murmurs, while at the base over the aorta was a marked systolic thrill and a rough systolic murmur and a soft diastolic bruit. The pulse was small, quick, and compressible, and it was sometimes markedly irregular. There was evidently stenosis and incompetence of both the aortic and mitral valves, but the general aspect of the patient was that of mitral disease. The venous pulse was not very conspicuous to the eye but gave a marked tracing. In this record the heart is beating quite regularly except for the extra-systole shown at *x*, which, as explained in the text, is of ventricular origin, the venous tracing showing that the auricles are beating quite regu-

larly. The wave *a'* occurs during the systole *x*, and is, therefore, much larger than the other auricular waves *a*.

Fig. 6.—Radial and jugular tracing taken a month previously from the same case as Fig. 5. Shows a well-marked "bigeminal pulse." The extra-systoles are marked *x* and are evidently of auricular origin, since the jugular tracing exhibits an exactly corresponding irregularity to that of the radial. The compensatory pause after each extra-systole is well marked.

Fig. 7.—Radial and jugular tracing from Mrs. G—, a patient of Dr. Murrell's. She was suffering from cirrhosis of the liver and about a year previously had had a Talma-Morrison operation performed for the relief of ascites. The ascites had returned and there was general œdema, much dyspnœa, and signs of dilatation of both left and right ventricles. There was a mitral regurgitant murmur. The tracing shows that with every other beat the auricular wave *a* is too early, an extra-systole starting from the auricle. No corresponding wave is seen in the radial to this auricular extra-systole, but a wave is shown from the carotid, so that the ventricle was beating.

Fig. 8.—From the same patient as Fig. 7, six days later. There is no pulse in the jugular, the tracing from the neck only showing the carotid pulse. Extra-systoles are shown at the points marked *x* in the radial tracing. Three extra-systoles follow one another, the compensatory pause only occurring after the last. This patient improved steadily and left the hospital fairly well. She was re-admitted a month later with fresh ascites. The jugular pulse was now ventricular—*i. e.* the ventricles had taken on the inception of the rhythm.

Fig. 9.—Jugular and radial tracings from Mrs. C—, aged 42, a patient of Dr. Hebb's. She had had rheumatism at the age of 18. For six years she had had dyspnœa on exertion, and for four years had suffered from a cough in the winter. For the past two years the dyspnœa had been worse and there had been occasional attacks of palpitation. Swelling of the feet came on three months before admission.

When first seen she was slightly cyanosed and was unable to lie down flat in bed. Her pulse was 90, small, feeble, and irregular, but the heart was evidently beating more rapidly. Both sides of the heart were dilated, the apex being just external to the nipple and the superficial cardiac dulness extending to beyond the right border of the sternum. There was a musical systolic murmur at the apex and a diastolic murmur, also best heard at the apex. There was marked pulsation of the jugulars synchronous with the pulse. The record shows no trace of an auricular beat, there being only the large wave *v* present, due to the ventricular systole. The radial pulsations are probably all extra-systoles.

Fig. 10.—From the same patient as Fig. 9, after three days' treatment with digitalis. The venous pulse is of the same character as before. In the radial it is easy to recognise a rhythm broken by extra-systoles which are marked by *x*. The patient improved steadily and was able to leave the hospital fairly well, though there was naturally great limitation of the area of cardiac response. The venous pulse was taken frequently and was always of the same character. It is noticeable that the interval after an extra-systole is only of the same length as that after a normal beat.

Fig. 11.—Radial and carotid tracings from E. H—, aged 54, female, an out-patient of mine for the past five years with mitral stenosis. The area of cardiac response was limited, but otherwise her chief complaint was recurring attacks of gout. In the autumn of 1905, however, she was laid up for three months with "congestion of the lungs," and when she returned to me last March her pulse exhibited the striking irregularity shown in the tracing. This irregularity is evidently due to the occurrence of the extra-systoles marked *x*.

Fig. 12.—Same as Fig. 11.

Fig. 13.—From Mrs. W—, aged 66, a patient of Dr. Hall's. For the past year she has frequently been an in-patient for the relief of dyspnœa and ascites, requiring

to be frequently tapped. The liver was large, irregular, and evidently cirrhosed. There was considerable dyspnœa and the heart was rather dilated, the apex being in the nipple line. At the apex a systolic murmur was sometimes heard. The vessels were much thickened and tortuous, and the blood-pressure was decidedly raised (160 to 180 mm. of mercury by the sphygmometer). There was usually a trace of albumen in the urine. The case was evidently one of cirrhosis of liver, granular kidney, and extensive vascular degeneration, including fibrosis of the heart. The venous pulse was taken frequently and was always ventricular in character as in the tracing. Extra-systoles were always a conspicuous feature of the records. Unfortunately, a time-marker was not used in taking this tracing, so that the deductions as to the length of the compensatory pauses are not founded on absolutely certain data.

Fig. 14.—From an out-patient of mine, E. H—, aged 45, who had suffered from dyspnœa on exertion for some years. She came to the hospital on January 16th, 1906, and on examination the heart was found to be considerably dilated on both right and left sides. A systolic murmur could be heard at the apex and another systolic murmur at the bottom of the sternum. The liver was considerably enlarged and there was marked pulsation of the jugulars. It was not possible to admit this woman and she did not come to the hospital again. The tracing shows the large wave *v* from regurgitation through the tricuspid valve. This wave is preceded by the small wave *a* due to the auricle. The tracing also shows two extra-systoles at *x*, each followed by a compensatory pause. These extra-systoles are each of auricular origin but evidently arise from a spot at a considerable distance from the great veins since the interval *p*—*p* is in each case double the usual interval between two normal contractions. It is noticeable that with each extra-systole the interval between the auricular wave *a* and the ventricular wave *v* is shorter than normal, so that the extra-systole has probably started

from the auricular end of the auriculo-ventricular fibres or near them. The larger size of the wave after the long pause is well shown.

Fig. 15.—Radial and jugular tracing from a female patient under Dr. Hebb, aged 15, who had had an attack of rheumatism three years ago and has had mitral regurgitation since. She was re-admitted into the hospital with rheumatism, pericarditis, and dilatation of left and right sides of the heart. The jugular pulse shows that the auricles and ventricles are beating at the same time.

Fig. 16.—From the same patient a week later. The venous pulse is of the same character as in Fig. 15 and still shows the auricle and ventricle beating simultaneously. The patient has since improved very much and gets up. There is now no sign of dilatation of the right heart and the venous pulse has disappeared.

Fig. 17.—From a female, aged 52, under Dr. Hebb, who had been in the hospital several times with heart disease. In 1905 she was an in-patient from July until September with mitral and tricuspid regurgitation. The heart was then enormously dilated, especially towards the right, and there was general œdema and enlargement and pulsation of the liver. The jugular bulb formed a large pulsating swelling in the neck. It was found necessary to bleed her several times, on each occasion with considerable benefit. The patient gradually improved and was able to leave the hospital at the end of the year. In February, 1906, she returned with a fresh attack of heart-failure, there being great dyspnœa and general dropsy. The heart was again much dilated on both sides and the liver much enlarged. The jugular bulb again formed a large pulsating swelling above the clavicle, the pulsations being synchronous with the pulse. The radial pulse was very small and irregular, and this, together with the œdema of the arms, rendered it impossible to get a satisfactory tracing. The venous pulse only shows the great wave *v* from the ventricle, with no sign of any auricular contraction. This type of venous pulse continued although the general condition of the

patient somewhat improved. After temporary improvement she became worse and died of heart failure.

Fig. 18.—From Mrs. R—, aged 61, who had been an out-patient of mine on several occasions with chronic disease of the mitral valve and a regurgitation murmur. Admitted February 17th, 1906, under Dr. Murrell, with cyanosis and great dyspnœa. The heart was dilated and the liver enlarged and there was marked pulsation of the jugular veins. The radial pulse showed numerous extra-systoles, producing great irregularity, and the venous pulse was purely ventricular in origin, there being no sign of any auricular beat. Improvement took place in this patient very rapidly, but the jugular pulse when obtained was of the same type. On March 9th, however, the jugular pulse had entirely disappeared, and it is possible that the chambers of the heart were again beating in their normal order.

Fig. 19.—After Mackenzie. Shows the transition from the normal type of cardiac rhythm to that where the ventricles undertake the inception. This patient had previously shown numerous extra-systoles, starting from the ventricles.

Fig. 20.—From Mrs. B—, aged 50, who had been an out-patient of mine for several years suffering from tertiary syphilis (periosteal nodes) and from stenosis and incompetence of the mitral valve. She has had several attacks of heart failure, with œdema of the feet and enlargement of the liver, but at the present time is fairly comfortable. The tracing shows a marked bigeminal pulse with extra-systoles x following the normal beats. In the jugular pulse a wave a, due to the systole of the auricle, is shown before the sphygmic period of each normal beat and this wave is followed by the fall x, due to auricular diastole, but with the extra-systoles no trace of an auricular beat is to be discovered.

Fig. 21.—Pulsus alternans after digitalis. After Mackenzie.

Fig. 22.—Tracing showing depression of conductivity.

The *a–c* interval is two fifths of a second instead of the normal one fifth. After Mackenzie.

Fig. 23.—From the same patient as Fig. 22, after administration of digitalis, showing that every other beat is not conducted to the ventricles. After Mackenzie.

Fig. 24.—Tracing showing a ventricular extra-systole at *x*. Conductivity is depressed as shown by the *a–c* interval being two fifths of a second, but shows a striking recovery at *A'* after the extra-systole. After Mackenzie.

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DISCUSSION.

Dr. ALEXANDER MORISON remarked that it would be agreed that Dr. Gossage had, in his interesting paper, supplied much food for thought and discussion. The speculative and practical questions he had raised were, however, too numerous to be dealt with in detail by every speaker. Personally, he had little difficulty in agreeing with him that cardiac dilatation was due to loss of tone in the muscle fibres of the heart. But he was curious to learn more of this same "tone," or "tonicity," to use a term in the language of myogenists implying an independent active quality, and he would content himself in the remarks he proposed making to considering the probable nature of the active properties of visceral muscle, as the conception we formed of them doubtless influenced our views as to the nature of abnormal conditions affecting the action and power of the heart. It had been interesting during recent times to note the trend of thought in the explanation of cardiac action, and the revival with recent views of opinions entertained, combated, and again revived in much earlier times, but now considered by many to be more firmly based than of yore on physiological observation. They were, he presumed, all agreed that there were more factors than one in organic action—that there were certainly three, namely, the blood, the nerves, and the cell, and that these three were merely general terms, the intimate nature of which must certainly be divisible into many more, and the full knowledge of which would give us the key to the explanation of life itself, the obtaining of which, he understood, was regarded as possible by some, among

whom he had the misfortune not to be able to include himself. In the meantime, and as affecting cardiac action, the muscle-cell, as the dominant factor in such action, took precedence of the others in the opinion of many whose authority was, with justice, regarded as great. To reassert its predominance, this myogenic theory had to displace its predecessor in predominance—the neurogenic theory, and much patient work had been undertaken to show that certain muscular structures manifesting rhythmical and involuntary action were free from nerves. But this argument had been largely abandoned, for further research had in most instances shown the presence of nerves in situations once regarded as nerveless, and with this demonstration, naturally, theories based upon such an assumed absence fell to the ground. They had now reached a point at which the denial of the *existence* of nerves in any given muscular structure had been abandoned, and conceptions of the character of their influence had been modified; they were now considered to regulate and control, they no longer initiated, any form of visceral motion. He did not know whether the notion had ever been entertained that visceral muscular contractility was, in its elementary form, dependent upon the influence of a nerve-supply, and he was rather under the impression that the organic activity, the quality or purpose of a particular cell, be it muscular, or peptic, or excretory, had always been regarded as a thing apart both from the influence upon it of blood-supply and of nerves. The cell was not blood, nor was it nerve, whatever primal unity in the genesis of life might be concerned in binding them together. But, once the organic cell had come into full development, he presumed it would be admitted that its sustained or persistent activity was dependent both upon its blood- and nerve-supply. The view, therefore, which was taken in some quarters, that changes in the condition of the visceral muscle-cell *alone* were primarily responsible for abnormalities in its action, and with which the hæmic and neural factors were only remotely connected, was, he confessed, one which, to his mind, had not yet been established. That blood, nerves, and cell, by their concerted action, produced persistent functional activity would not, he supposed, be disputed, and it would not in any way surprise him if the neurogenic theory again displaced the at present prevalent myogenic. Digestion, for example, was certainly effected by the peptic cell, not by the blood or nerves of such cell directly; and muscular motion, he admitted, was likewise the essential property of the muscle-cell, provided they defined what they meant by this essential motion. It was, nevertheless, a property the persistence and quality of which was surely as much dependent upon the influence of the blood and its nerve-supply as was the persistence of the functional activity of the peptic or any other cell which was essential to the exercise of a particular function. This, how-

ever, he imagined, they must all admit, although the matter was at times so treated as to leave the fact in doubt. The phenomena manifested by cardiac muscle, he remarked, were motions of a rhythmical or repeated kind. This would manifestly be impossible without qualities in the cell which permitted of its contracting and not contracting. It might contract powerfully or feebly, or its contraction might have a certain average force. This average force for the sustenance of effective action might be termed "tone," or anything else, so long as they were agreed to connote certain phenomena by the term chosen. The motion in the organ, he said, might travel from one point to another, and under ordinary circumstances in a given direction. Motion might thus be "conducted" from one point to another, and the agency of conduction could not, of course, exclude the muscle-cell, but need it necessarily exclude the general innervation of the organ? They had now anatomical and experimental reason to believe that certain fibres of peculiar character lay in the track of normal auriculo-ventricular contraction, and served to associate auricular with ventricular contraction. These fibres, he presumed, were admitted to contract themselves, but he also knew that, like all other muscular fibres, they were endowed with nerves. Did these nerves, it might be asked, control or regulate their contraction, or did they merely serve nutritional purposes? He was not aware that this point had been settled, and if unsettled, the autonomy of the cells was surely also unsettled. These conducting fibres, while they appeared in a measure to be essential to the orderly sequence of auriculo-ventricular contraction, were not, of course, responsible for the contractile property of the cells either in the auricles or ventricles. For the ventricular cells might contract in such a manner as to suggest an independent origination of contractile action. These cells, therefore, were assumed to build up their own "stimulus," —stimulus being a term used to denote an invisible cause assumed to arise in the cell during a given observable phenomenon, namely muscular movement, or rather just preceding it. This assumed cause being then also assumed to induce this observed phenomenon, argued, of course, or led to the further assumption that the mobile muscle-cell was *receptive* of such influence, and was therefore "excitable," and that in this excitability the nerve supply of the cell had no essential place, an excitability according to some which might even amount to pain. Hence they came to the conclusion that the muscle-cell was stimulable or excitable, that it was contractile, yea rhythmically contractile, and contractile with a certain average force or tone, that it possessed "tonicity." The peptic and salivary cells, he supposed it was admitted, were similarly stimulable, and stimulable as often as the material which called forth their secretional action was presented to them, until by a repetition of stimulation they ceased

to respond, their average force or tone was exhausted, and they ceased to respond in a characteristically normal or usual manner to stimulus—they thus lost "tone." What, he asked, was exhausted under these circumstances—the cell, the blood, or the nerves? The cell certainly, but what of the blood and the nerves? As regards the *blood*, functional activity necessitated and was characterised by an increased blood-flow, so long as the cell was functionally active. With the subsidence or diminution of cellular activity this vascular supply was also diminished. It seemed scarcely necessary that the effective power of the blood to aid secretion should be materially altered with the normal waning of cellular activity. But, on the other hand, it would, he thought, not be denied that the quality of the blood had a very considerable influence on the functional activity of the cell. It was a common clinical experience that the quality of the blood had a noteworthy influence, especially upon the quality of the action of that perpetually active cell, the cardiac muscular cell, and that a correction of the nutritive power of the blood might be all that was necessary to restore "tone" to an enfeebled heart. As regarded the *nerve-supply* of the cell, they were dealing with something more akin to the cell itself—something in more intimate connection with it. In any case the intimate relation of nerve to cell was well known, and the influence of the nerves on the action of the cardiac muscle cell, both by way of the emotions and by that of non-emotional reflex, was so frequently observed a phenomenon that neurogenists might be excused if they had at times attributed more than its due share to the influence of the nervous system upon cardiac action. The nature of this intimate connection it was not necessary to speculate upon, whether by the suggestion of an intramuscular and incorporated nerve distribution or by any other mode of connection. In the higher animals, however automatic the action of the cardiac muscle cell might be regarded as being, its force and persistence could not long survive a disseverance from the neural and hæmic sources of its life. The cardiac muscle cell was a mid-point, a term common to its nerve-supply, and the properties of the latter were, as they knew, like the various properties attributed to muscle-cells, also various, and indeed somewhat the same in kind. Thus they were accelerant or augmentative, depressant, inhibitory, and trophic. There was, however, this difference between those who perhaps unduly exalted the *rôle* of the nervous system in visceral muscular action and those who, it might be, unduly depreciated it, that the effects of nerve-stimulation were observed in the mobile cell acted upon, while the myogenist imputed an independent activity to certain phenomena, the essential cause of which was assumed to be such or such and resident in the cell itself. That was to say, cause and effect were resident in the self-same structure; effect

was not registered outside the seat of cause. It was admitted, he continued, that the heart might be quickened and slowed through the agency of the nervous system. He did not know that it was actually disputed that it might also be rendered irregular in action by agency of the nerves, but it was certainly now maintained that the regular sequence of auricular and ventricular action was secured, not by nervous influence, but by muscular conduction. The latter conclusion was based upon experimental injury to the heart in a certain situation. It was admitted that nerves were also divided during this operation, but it was contended that inasmuch as nerve and muscle injury elsewhere did not produce this dissociation, it was not nerve but muscle injury which caused this result in this particular instance. It was to be admitted that a good deal was to be said for this view, but it should nevertheless be remembered also that not muscle only but also nerve was destroyed in the experiment, and at the central point of junction of the auricles and ventricles. The unquestioned fact that even these associative muscular fibres were nerve-endowed suggested that irregularity of the heart or dissociated action of the chambers might be due, at least in some cases, to the influence of the nervous system upon the muscular fibres in question. In the production, therefore, of the stimulus which caused the muscle to contract, in the excitability of the muscle-cell which caused it to be receptive of the influence of such stimulus, in the conduction throughout the organ of contraction from point to point, in the force and character of cardiac action, he conceived that the direct influence of the nervous system could not be excluded. Was there, then, he asked, anything exclusively cellular in cardiac or other visceral muscular action? Contractility, it appeared to him, might be so regarded, but the various phases of its manifestation he thought it was more difficult to regard as purely cellular. The embryologist told them that to this rhythmicality must be added, because this phenomenon had been observed before nerves were seen to reach the muscle. But nerves were in process of growth. Marconi (or his predecessors) had taught them that messages might be transmitted without telegraph wires, and the nerve in process of generation, though separated from the nascent muscle, might quite conceivably through the medium of the nutritive fluid in which they both grew respond to one another without gross contact. Embryological facts were anatomically observable, but they had not come within the sphere of experimental physiology, and it was difficult to suppose they ever would. Hence a certain argument by analogy might be permitted. The temporary palpitation of eviscerated organs did not necessarily invalidate such a supposition or disprove a lingering influence of the nervous system in a detached organ, for our knowledge of the visceral nervous system was far from complete. Dr. Gossage had spoken

of the varying degrees of rhythmicality in various portions of the heart and vessels. But this varied, as they knew, in other organs also, and with this variation he had found anatomical differences in the structure of the intrinsic nervous system. Thus on the trunks of nerves running through the uterus he had altogether failed to find any ganglion-cells, while in those constantly rhythmical organs, the heart and the intestines, they knew that these bodies were very numerous. Even in the rhythmical spleen they were fairly abundant. He would, therefore, with all diffidence suggest that they were not yet in a position to accept as established the full conception of muscular automatism ascribed by myogenists to the action of the heart.

Dr. E. I. Spriggs said that such experimental investigations were of the greatest value to medicine and were the most efficient means by which we could hope to improve our knowledge of the appropriate treatment of such morbid conditions as were being discussed before the Society that evening. He did not think it was justifiable to assume from the evidence brought forward that the stimulus for the extra systoles shown in the tracings originated in the ventricle in every case. Too much reliance could be placed upon venous pulse tracings. These did not present by any means an accurate picture of events in the auricle. In many cases the auricular systole was well shown and in some the fall of auricular pressure which occurred during the ventricular systole. The rest of the cycle of the auricular pressure changes was usually obliterated by the beat of the carotid, or still more by tricuspid regurgitation when this was present. From Hürthle's published curves of auricular pressure it could be seen that there were other variations of pressure not indicated in such tracings as were shown. This being so, it must be borne in mind that the tracings could only be regarded as giving partial evidence. When an auricular wave was definitely shown, that could be regarded as a positive result, but the absence of such a wave did not prove that the auricle had not contracted. What effect could be expected on the tracing if the auricle contracted when it was only partly filled with blood? Yet they knew that experimentally in the excised heart auricular beats which were barely perceptible could be transmitted to the ventricle and were capable of initiating a ventricular beat. For these reasons he did not think that an interpolated radial pulse beat which was not accompanied by a corresponding auricular tracing should be necessarily put down as of ventricular origin only, neither should the presence of a pure ventricular rhythm be too rapidly assumed upon such evidence. They knew that extra-ventricular systoles could be produced experimentally, independent of the auricle, also a true ventricular rhythm; but, without prejudice to such evidence, he did not think the tracings shown could be regarded as a proof of this occurrence in all of these cases. He thought that in the

discussion of tone and tonicity too little attention had been paid to the actual mechanical conditions determining dilatation of the heart, especially terminal dilatation. When the walls of a viscus such as the heart were distended they acted at an enormous disadvantage. The tension in the muscle necessary to produce any given pressure would have to be increased eightfold if the radius of the chamber were doubled. With a load too great for it to move, a muscle relaxed instead of contracting, and in this way a dilated chamber was brought to a standstill. The final failure of a ventricle was probably often due to the preceding failure of the corresponding auricle.

Dr. JOHN HAY thanked the Society for the honour of being invited to be present and to take part in the discussion. He said that the paper was full of suggestion and that it dealt with the subject of heart disease from a somewhat new point of view. The tendency to-day was to inquire more into the functional power of the myocardium in heart disease and to lay less stress on the existence of valvular defect. One of the methods used at the inquiry was to take simultaneous tracings of the radial and jugular pulses, and from these tracings much of value could be obtained. He had used this method for three years and found it of great service. The investigations of Gaskell and Engellmann were now being applied by Wenckebach and Mackenzie to clinical medicine, and to-night Dr. Gossage had given the result of his own work. The importance of the function of tonicity in its relationship to dilatation of the heart had long been recognised. Dr. Gossage in his paper suggested that tonicity had a definite relation to the other functions of the cardiac muscle, and that when tonicity was increased contractility, rhythmicity, conductivity, and excitability were diminished and *vice versâ*. He concluded that if this theory were sound conditions which increased tone should diminish frequency. The tracings published by Gaskell in his paper on "Tonicity," in Schäfer's text-book on 'Physiology' were of interest in this connection. One tracing showed a great increase of tonicity in the heart as the result of perfusion of the organ with a weak solution of sodium hydrate. With this rise in tonus, however, the frequency was markedly increased. He pointed out that Dr. Gossage referred largely to the action of digitalis as an example of a drug which increased the tone and therefore diminished the frequency; but some experimental work done recently by G. T. Haynes demonstrated that the preliminary slowing produced by digitalis was almost entirely the result of its action on the terminal portions of the vagus, and that following on this stage there was a simultaneous increase of both tone and frequency. He suggested that the resemblance between tone and a contraction was superficial only. He considered that this matter might with advantage be referred to the physiologists. He remembered that Bottaggi

and Grünbaum viewed tone as due to changes in the sarcoplasm, a contraction as due to variation in the sarcostyles—in other words, that the two functions of tonicity and contractility were fundamentally different in character. In conclusion, he said that though the theory was very ingenious he could not accept it without more evidence in its favour.

Professor WENCKEBACH (Groningen) expressed his gratitude to the Society for having sent him an invitation to be her guest on that evening and to join in the discussion. He was greatly pleased to see that the method of analysing heart-action in man along physiological lines was spreading throughout England, Dr. Gossage's paper giving proof of that fact. However devoted to the myogenic theory of heart-beat, he agreed with Dr. Morrison, who said that a coming time might see another change in general opinion and the neurogenic theory adopted. Theories might change but facts would remain, and what they were finding in man by analysing the various pulse-tracings was not theory but facts. The question whether the stimulus to contraction was produced by the muscle-fibre itself or was brought to the heart by nerves had no influence upon their views on the other qualities of heart-muscle—excitability, conductivity, contractility—so they might discuss freely the facts Dr. Gossage reminded them of that night. As to the remarks of Dr. Spriggs that in jugular-pulse tracings there might be a lack of evidence of actual contractions of the heart, he was quite right in saying that they should not say a thing was not there when they did not see it. They had to keep this in their mind and to be very careful indeed in the analysis of their tracings. Still, they might say that they, practical men, more, perhaps, than the experimenter, were fully convinced of this law, that they should not say a thing was not there when they did not see it; daily practice taught them so. Dr. Hay discussed the question whether they were on the sound basis of knowledge if they regarded tonicity as a quality of the heart-muscle comparable with the other qualities of the heart. He was sorry to say that he could not give any evidence in that matter, and could only point to the fact that Engelmann, who had studied these qualities so splendidly and shown that they were *independent* of each other in the muscle-cell, did not say a single word on that quality. Gaskell, who first of all dealt with their qualities, and other English investigators, had given them many beautiful experiments on tonus of the heart-muscle. Still, what tonus was in its relation to contractility and the other qualities of muscle was not well understood, and this statement held good, not only for the heart, but for the voluntary and involuntary muscles as well. So they wanted further evidence and information from the physiologist. Now this was a very important conclusion. They saw here, as Dr. Gossage had shown them so ably, that here was a question of so eminent a *practical* importance as dilatation of

the heart, which only could be solved by further physiological investigation. This fact was eloquent in showing that they were on the right track when they applied their physiological methods at the bedside. In that way only would they be able to put definite questions, and to do so in a form so as to be understood by the experimenters, and that the experimenters might be able to give a clear and useful answer to their questions.

Dr. GOSSAGE, in reply, said that two complaints had been made against his paper, one that it was too long, and the other that it was too short, and he was compelled to plead guilty to both counts of the indictment. He would like to point out, however, that the title of the paper was "Some Aspects of Dilatation of the Heart," and that it was not intended to be a complete treatise on the subject. He had touched on the mechanical side of cardiac dilatation at the commencement of his paper, and he entirely agreed with Dr. Spriggs in his insistence of its importance. The concurrence of an increase of tone with an increase of rate when the heart was acted on by sodium hydrate and by digitalis, as pointed out by Dr. John Hay, was a strong argument against the theory that increase of tone led to a diminution of the other properties of cardiac muscle. It must, however, be remembered that the action of these drugs on the heart is probably a complex one, and that although the main action is on tonicity the other properties may be directly affected as well as indirectly through tonicity. He was very conscious that he had presented the subject in an incomplete form, and that much work required to be done on tonicity and its possible influence on the other properties of muscle. This work, however, was physiological and required to be done in an experimental laboratory. He himself was merely a clinician, and had neither time nor opportunity for experimental research, and he had brought the matter forward in the hope that the physiologists might investigate it thoroughly. On the clinical side it was important to keep in mind that the loss of tone did not explain everything in cardiac dilatation even if his theory should prove to be well founded. Dr. Spriggs had urged the importance of mechanical considerations, and he himself wished to point out that the various properties of the cardiac muscle might be affected in certain cases quite independently of any alteration of tonicity, so that the phenomena of dilatation might be complicated by marked decrease of one or more of the other properties —*e. g.* a loss of conductivity or of contractility.

OBSERVATIONS UPON CHEYNE-STOKES' RESPIRATION

BY

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A case of Cheyne-Stokes' respiration with incontinence of urine due to a cerebral lesion lasting for four months.

PART I.—OBSERVATIONS UPON THE RESULTS OF THE CLINICAL AND POST-MORTEM EXAMINATION. BY G. NEWTON PITT.

Thomas G—, aged 43, admitted October 3rd, 1904, on account of pain in his chest and of shortness of breath.

He was abroad in the army from 1881 to 1887. He had typhoid fever in 1884, but he has never had rheumatic fever. In May, 1904, he was under treatment for bronchitis and pain over his heart. He developed an attack in which he temporarily lost the use of his left arm, it became so feeble that he had to lift it up with his right

hand when he wished to move it. He himself attributed the weakness in his arm to over-use during his work as an electric car driver. He says there was also some slight difficulty in his speech, but these symptoms were only temporary. About ten days before admission he complained of pain in his chest and was very short of breath on walking or going upstairs, and there was some slight œdema of the legs.

Condition on admission.—He is a well-developed, healthy-looking man. On examination his lungs are found to be normal. The apex beat is in the fifth and sixth spaces one inch external to the nipple. The deep cardiac dulness begins above with the third rib. There is no dulness to the right of the sternum. A well-marked diastolic bruit is audible at the base of the heart traceable down the sternum. It is loudest in the second right space and up towards the neck. A systolic thrill is also palpable at the base. The urine is normal and is of sp. gr. 1020.

October 7th.—At 7.30 a.m. he felt faint and fell back on his pillow but did not completely lose consciousness. It was noted shortly after that his left arm and leg were paralysed, and to a less extent, the muscles on the left side of the face. The knee jerk on the left side disappeared, and the plantar reflex was in type extensor, while that on the right was flexor. The left lower extremity lay rotated outwards. There was incontinence of both urine and fæces, and he developed a Cheyne-Stokes' type of respiration. In the afternoon the paralysis diminished so that he was able to move his toes and foot on the left side and he regained some use in his left arm, having a fair grip. His mental condition is very dull, he lies in an indifferent semi-somnolent state.

10th.—The patient has improved considerably; he has regained the use of his left arm and leg, but he complains of abnormal sensation in them. His temperature has been normal, the grip of his hand is fair, and his mental condition has also improved.

12th.—The patient is restless and tries to get out of bed

at night. Sometimes there is considerable trouble in managing him. He has incontinence of urine two or three times a day, and also of fæces. He complains of some pain in the abdomen. He has been taking a mixture of 15 minims of Tincture Digitalis and 7 grains of Citrate of Caffein. The heart's action is feeble, the left ventricle dilated, and the compensation imperfect. The pulse is water-hammer in type. The paralysis was considered to be due to a small embolus in the right middle cerebral artery, with only a very small patch of softening. For some time it has been noticed that his hands are very livid, and apt to be cold.

26th.—The rhythmical character of the breathing remains very marked, but the power in the limbs of the left side is better. There is great mental torpor. He answers questions very slowly and deliberately in a sleepy sort of way, but accurately, and he moves his hands slowly. His pulse has become more irregular, the diastolic bruit is somewhat musical in character, and at the base of the heart there is a systolic bruit. The apex beat can be felt six inches from the sternum. Three grains of iodide of potassium were added to the mixture. The evidence pointed to the aortic incompetence being probably associated with atheroma of the aorta. During the variations in respiration it was noted that the size of the heart varied greatly. During dyspnœa there was no dulness to the right of the sternum, but with development of apnœa the right auricle became distended, so that on more than one occasion there was over an inch of dulness in the fourth right space. The characters of the Cheyne-Stokes' respiration are described later on by Dr. Pembrey and Mr. Allen.

November 4th.—His mental condition has slightly improved. There is still a little imperfection in sensation in the left leg, and a very slight paralysis persists.

17th.—The Cheyne-Stokes' respiration has persisted throughout, the depth of the respiration varying in a most marked way during the course of each minute. He has periods of fairly rapid deep-laboured respiration, which

gradually waxed and increased in intensity, their intensity then gradually diminishes, and this is followed by a period of apnœa of considerable duration in which there is but little or no respiratory movement. The respiration again starts gradually increasing in intensity to be again vigorous for a short time. He remains in a drowsy condition, paying but little attention to his surroundings, and his talk is very often quite irrelevant; he can, however, be roused when he is able to answer a question properly. At times he is noisy and restless, especially at night. The incontinence persists, he is rather more anæmic than he was. A leucocyte count was 7000 per c.mm. The evidence has always been in favour of an atheromatous aorta, rather than of a fungating endocarditis. The temperature remains normal.

The right side of the heart became dilated during the periods of apnœa, and we were able on many occasions to demonstrate that, while in dyspnœa there was no dulness to the right of the sternum, during apnœa an area of dulness could be demonstrated extending as much as one inch and a quarter in the fourth space to the right of the sternum. Repeated observations were made with regard to the variation in his mental activity with the alterations in respiration; sometimes the latent period which elapsed before he said he felt a cutaneous stimulus was longer during apnœa than during dyspnœa. It was more difficult also to make him answer during apnœa, but on many occasions we were not able to satisfy ourselves of any appreciable difference in his mental activity during the two periods, and what difference there was, was more marked some days than it was others. No oscillatory movements of the eyes were noticed. The rate of pulse varied, but there was no marked difference in muscular power.

24th.—The urine contains a faint trace of albumen, having hitherto been free from it. The specific gravity varies from 1014 to 1028; a systolic thrill is often palpable at the base of the heart.

December 1st.—His condition has markedly changed, and is very grave to-day; his breathing is much more laboured, his hands and face are markedly cyanosed, and he is quite unconscious. The Cheyne-Stokes' respiration is more marked during sleep; he is unable to swallow food, which tends to pass into the trachea and set up choking. He is therefore fed with a nasal tube every eight hours. His temp. is 96°, his resp. 16, his pulse 70. While he was asleep it was noticed that the apnœa lasted about thirty-five seconds, and the dyspnœa about twenty-five, and these periods have been fairly constant. During the dyspnœa he took fifteen breaths; the thoracic movements commenced feebly, rose up to a maximum at the seventh, and then gradually died away again. The pupils are equal, and react to light both during the dyspnœa and the apnœa.

2nd.—The pupils remain equal in size. All day yesterday he did not speak; he was drowsy and unable to swallow. There were trachea râles. He was very helpless, and the paresis in the left arm and leg were quite marked. About 2 p.m. he improved so that he was able to answer questions and to swallow. He was only able to pick up a sixpence with his left hand very clumsily and with difficulty. His left thigh was rotated outwards, there was an extensor plantar reflex on that side, and the knee-jerk was brisker than on the right. The soft palate does not move when it is touched, but it does with phonation. His mental condition is not so good as previously, and he wanders more in his talk. He has the idea that he has been getting up for some days although he has not left his bed. The dyspnœa is very marked, and he is very restless. The pulse tracing is typical of aortic incompetence, with the vessel almost empty during diastole. His condition is feeble, so that he is unable to sit up in bed, and incontinence of urine and fæces still persist. He is still fed through the nose, and is taking a mixture containing digitalis, nitro-glycerine, and acetate of potash, and also a stimulating mixture

with some strychnine. The periods of dyspnœa and apnœa remain very fairly constant, as shown by the following table :

Period of Dyspnœa.			Apnœa.
No. of respirations.		Duration.	Duration.
20	...	38 secs.	32 secs.
23	...	40	30
23	...	35	35
22	...	35	35
22	...	35	35
21	...	35	38
22	...	37	40
21	...	40	33
21	...	37	38

8th.—The nasal feeding was stopped on the 6th as he has been able to swallow since then, otherwise his condition remains unchanged, and he is still very dull mentally. His tongue is dry and furred.

15th.—He is taking his food better, and is able to use his left hand a little, and mentally he is so far improved that he can recollect events of two years ago. He still remains cyanosed. The systolic bruit at the base is still very rough.

18th.—He was able to be lifted on to a couch in the afternoon for a short time.

27th.—His mental condition has improved slightly, he has had less incontinence lately, and there is less cyanosis of his face and hands.

January 3rd, 1905.—The patient is more sensible, and talks rationally. There is a musical diastolic bruit audible and palpable over the cardiac area. The urine is sp. gr. 1010, and free from any abnormal constituents. The Cheyne-Stokes character of the breathing is less marked. The grip of his left hand is stronger, but it is necessary for him to concentrate his attention if he is to hold anything in it, as directly his attention is diverted the grip is relaxed.

20th.—The dyspnœa is greatly increased by fog. There is a slight trace of albumen in the urine.

On the 24th he was apparently in his usual condition, when at 1.30 p.m. he became extremely low and dyspnœic, the periods of dyspnœa greatly increasing in length. Towards the end he had a period of apnœa for ninety-seven seconds, followed by a period of dyspnœa, to die away again finally. The heart-sounds towards the end were more feeble and rapid, he became very cyanosed, and died after an attack which lasted half an hour in spite of treatment by strychnine and oxygen.

Inspection.

Old adhesions on the right side between the lobes of the lung, with general thickening of the pleura. The lungs were emphysematous.

Heart weighed 751 grammes. The left ventricle was considerably dilated, and its wall hypertrophied, the enlargement of the organ being mostly due to the change in this part.

The posterior aortic cusps were much atrophied, and the free edges thickened and retroverted, so as to allow an extreme amount of regurgitation. The endocardium immediately below the valves was thickened by the repeated impact of the regurgitant stream. The mitral valves, as also those of the right side, were normal.

The mitral orifice measured four inches, and the tricuspid five inches in circumference. The heart muscle appeared healthy.

The aorta was very atheromatous, chiefly in the ascending arch, but the change was less marked in the transverse portion. There was nothing specially characteristic of syphilis about it. The liver and alimentary canal were normal, the spleen weighed 280 grammes, and contained an old infarct. All the viscera were tough from chronic cardiac failure; the kidneys, weighing 368 grammes, appeared normal, excepting for the presence of three or four old shrunken infarcts. The testicles were healthy.

The brain weighed 1350 grammes. On inspection there was a large depression, as shown in diagram, on the right side practically limited to the temporo-sphenoidal lobes, due to shrinking and softening of the cortex and subjacent tissues. The surface of the brain otherwise appeared normal. The whole of the second convolution

FIG. 1.

A transverse section through right half of brain, showing area of softening shaded horizontally. The oblique shading indicates the site of a cavity where the softening had been extreme.

the anterior part of the first and only a small anterior portion of the third were softened.

The softening extends deeply, involving besides the cortex, part of the caudate, lenticular and optic thalamus ganglia, and the subcortical fibres; but only a small area in the anterior part of the internal capsule was involved. The rest of the brain was free from any softening, the left side being quite normal.

A series of transverse vertical sections were made with the following results:

(A) 6·5 cm. from the front (No. 3 Daniell's sections). This was just anterior to the tip of the temporo-sphenoidal lobes, and was free from disease.

B) 7·5 cm. (No. 5). The greater part of the lenticular nucleus and a small portion of the internal capsule are

FIG. 2.

A similar section, 2 cm. posterior, through corpora mammillaria.

so extensively softened that their site is now occupied by an irregular cavity, which, however, is very much smaller than the area indicated in the diagram because the affected tissue has been greatly reduced in volume. The insular cortex and the outer part of the caudate nucleus are only partially softened. The lower corner of the lenticular nucleus is the only part of it which has escaped.

(c) 9·5 cm. (No. 9). Through the corpora mammillaria. In this section the tissues are soft but have not broken

down into cavities. The parts mainly affected are the first and second temporo-sphenoidal lobes, with the insula, the external capsule, and, to a less extent, the upper part of the lenticular nucleus. The first temporal convolution was less involved than the second. The corpus callosum, the optic thalamus, and the third temporal convolution, and the gyrus Hippocampus were unaffected.

The cerebral vessels showed some atheroma but no clot was detected in the right middle cerebral artery. There is no doubt the lower branches of the right middle cerebral artery were blocked, but whether due to an embolus or a thrombus still remains uncertain.

The result of the microscopical examination of the medulla is given later on.

Remarks.—The case presents several very remarkable features which may be discussed in turn. The patient, who had well-marked aortic incompetence, was seized on October 7th with an attack of left hemiplegia with incontinence of urine and fæces, and within a very short time development of a well-marked Cheyne-Stokes' respiration. The hemiplegia rapidly diminished so that on many occasions it was difficult to demonstrate it, although when he became very feeble it again became more obvious. The incontinence of urine, however, persisted more or less permanently, and was quite out of proportion to the amount of mental torpor which he exhibited and this raised the question as to whether it was due to a lesion of some cerebral centre or centres for the regulation of the function of the bladder. Bechterew,[1] in 1888, thought that there was a centre for micturition on the inner side of the anterior part of the optic thalamus. Czylharz and Marburg[2] in 1901 concluded that there were three cerebral centres for micturition—1. In the motor cortex at the juncture of the centres regulating the movements of the upper and the lower extremities. 2. In the

[1] Bechterew, 'Neurolog. Centralblatt,' 1888, p. 505.

[2] Czylharz and Marburg, 'Jahrbuch f. Psychiatrie,' 1901.

corpus striatum : both of these serving for conscious automatic micturition. There is a third centre in the optic thalamus which is actuated by the emotions over the bladder, and possibly also the cerebellum has some control. Lesions in the cortex and motor tracts produce retention, while those in the sub-cortical and cerebellar regions produce incontinence. The impulses are conveyed mainly by the pyramidal tracts.

Homburger,[1] who in 1903 re-investigated the whole subject, does not agree with them, but came to the conclusion that a unilateral patch of softening in the basal ganglia only causes a transitory incontinence, but the patient soon learns to keep clean in the daytime although the act of micturition was often precipitate; incontinence would, however, often occur in the night. With the development of a second patch of softening in the ganglia on the opposite side with or without a lesion of the direct cortical capsular fibres, the incontinence became permanent and was indistinguishable from a spinal incontinence, the urine constantly dribbling away. There would also generally be some residual urine, and micturition was always so precipitate that although there might be a sensation of the need to micturate there was usually not time to obtain a vessel to catch the urine. In addition to incontinence, bilateral lesions of the ganglia produced spasmodic paraparesis of the lower extremities, with retention of the movements of the feet and inability to pass from the recumbent to the sitting posture, exaggerated reflexes, an absence of Babinski's reflex and spasmodic attacks of laughing and weeping. When the patient was sat up on a chair, his legs would be stretched out stiffly in front of him, his head would be sunken on his chest and he would slip off the chair if not held. When he tried to walk it was as if on stilts with his legs wide apart, and the power in the arms was not perfect. Homburger collected ten cases of multiple softening of the ganglia, in six of which the lesions were bilateral and the incontinence was persistent.

[1] A. Homburger, 'Neurolog. Centralblatt,' 1903, p. 199.

He selected cases in which the cortex and internal capsule were unaffected. Lesions below the floor of the ganglia did not give rise to these symptoms. He came to the conclusion that superficial cortical lesions did not produce a permanent incontinence of urine, whereas bilateral lesions of the sub-cortical fibres and basal ganglias will do so. The lesion in the present case was in the ganglia on the right side and extended out to the surface of the brain below the Sylvian fissure, the damage above this being extremely minute. No lesion whatever could be discovered on the left side of the brain. The incontinence, although somewhat persistent, was not as marked and continuous as in the cases Homburger described when there were bilateral lesions. The incontinence was undoubtedly more marked and persistent than we have met with in other cases of hemiplegia, especially bearing in mind that although he was slow his mental condition was often very fair and the amount of paralysis very slight. Still while it might be brought forward as evidence supporting Czylharz and Marburg it cannot be said to really controvert Homburger's conclusions.

The cases of multiple softening which Homburger considered were due to degenerate arteries, hence the frequency with which the lesions were bilateral; but in the present case the area of softening was a very extensive one and was due to an embolus, the opposite side of the brain being healthy. Although it was noted that the patient was unable to sit up unless supported, he presented no other evidence suggestive of a bilateral softening of the ganglia.

PART II.—EXPERIMENTAL OBSERVATIONS, ESPECIALLY IN RELATION TO THE CAUSATION OF PERIODIC BREATHING. BY M. S. PEMBREY AND R. W. ALLEN.

The phenomena of Cheyne-Stokes' respiration were presented by the patient in so pronounced a form that it seemed important to determine, if it were possible, the

cause or causes of the periodic breathing. The problems which needed investigation were the apnœa, the period of waxing and waning respiration, and the relationship of these to each other, and to any changes in the circulation and nervous system. The apnœa might be due to the removal of so much carbon dioxide from the blood by the previous deep and rapid breathing that the partial pressure of the gas was depressed below its stimulating value for the respiratory centre; on the other hand apnœa might arise from the absorption of sufficient oxygen during the period of breathing to maintain the pressure of oxygen in the blood above the value at which deficiency of oxygen arouses the nerve-cells of the respiratory centre. Further, it might be that both of these changes were involved in the production of apnœa. The typical waxing of the respiratory movements during the period of breathing might be due to an increase of carbon dioxide, a decrease of oxygen, or to both of these factors.

Such were the causes to be sought on the theory that apnœa and breathing are due to changes in the tensions or partial pressures of the gases in the blood which supplies the respiratory centre in the medulla. There are, however, other possible factors. The periodic breathing might be due to intrinsic rhythmic changes in the activity of the nerve-cells of the centre for respiration. The rapid and deep ventilation of the lungs during the dyspnœa might, by impulses sent up the vagus nerves, so alter the excitability of the centre that respiration would be inhibited for a period. Rhythmic changes in the contraction of the heart and the circulation of the blood might cause alternating periods of activity and inactivity in the nerve-cells of the respiratory centre. These views were kept in mind, but attention was chiefly directed to the regulation of the respiratory movements by the gaseous constituents of the arterial blood for experiments upon men and animals show that the partial pressures of the carbon dioxide and oxygen are the most important factors.

The respiratory movements.—The Cheyne-Stokes' breath-

ing was observed fifteen weeks before the death of the patient, and, apart from a few days on which continuous or irregular respiration was noted, it persisted during that time. The period of apnœa lasted twenty to forty seconds, and during the phase of breathing there was a typical waxing and waning of the respiratory movements. The first breath which followed the apnœa was so slight as to need careful observation for its detection; it was succeeded by a series of respirations increasing in depth and culminating in deep gasping breaths; these gradually decreased in amplitude and died away into the next period of apnœa. The phase of breathing lasted about forty seconds; the cycle, therefore, occupied about one minute.

The alternation of breathing and apnœa persisted for hours or even days; changes, indeed, were observed in the duration of the respective periods on different days, but on any given occasion the time relations were often very constant. The character and duration of the respiratory movements are well shown by the graphic records (Plates Figs. I, II, IV, VI, XI), and the following determinations, which are samples of the numerous ones given in the appendix to this paper or recorded graphically in the curves.

Date.	Duration of apnœa in seconds.	Duration of breathing in seconds.	Number of respirations.
Nov. 26, '04	27	45	29
	25	47	27
	26	47	28
	26	47	27
	26	45	26
	30	47	27
Dec. 2, '04	36	34	20
	35	37	22
	36	37	20
	32	31	21
	38	35	20
	37	36	20
Jan. 11, '05	18	50	26
	21	43	28
	22	44	22

The ventilation of the lungs during the Cheyne-Stokes' breathing was determined by allowing the patient to breathe through a mask provided with inspiratory and expiratory valves, and connected with a gas-meter. The volume of air expired was between six and seven litres per minute, and a similar value was obtained when the patient was feeling better in health, and was breathing continuously. It follows, therefore, that an excessive ventilation of the lungs was effected by the forcible respirations of the periodic breathing; a larger quantity of oxygen than normal would thus be brought into the alveolar air, and a larger quantity of carbon dioxide would be removed. This effect, however, would be of a temporary nature. At the end of apnœa the composition of the alveolar air would show an opposite condition, and thus there would be, during a complete cycle, an oscillation between two extremes. This alternation will be demonstrated later by the analyses of the samples of alveolar air which were taken at different phases of the Cheyne-Stokes' breathing.

The variations in the volumes of the expirations during the waxing and waning respiration are shown by the following figures: 10, 20, 40, 250, 340, 100, 100, and 150 c.c. These were recorded when the patient breathed 21·2 litres of air in three minutes and twenty-nine seconds, that is at the rate of 6·1 litres per minute.

The heart, pulse, and blood-pressure.—The influence of the respiratory movements upon the pulmonary circulation is well shown by the difference in the area of cardiac dulness on the right side during the alternating periods of breathing and apnœa; the respiratory movements favour the passage of the blood from the right to the left side of the heart, and their cessation during apnœa was accompanied by an increase of cardiac dulness to the extent of an inch or more in the fourth intercostal space to the right of the sternum. The rate of the pulse was irregular, but, as the following determinations show, was generally more rapid during the period of apnœa.

Date.	Apnœa, duration in seconds.	Pulse rate, actual.	Pulse rate per minute.	Hyperpnœa, duration in seconds.	Pulse rate, actual.	Pulse rate per minute.	Number of respirations.
	33	48	87	37	52	84	—
	31	47	91	40	52	78	—
	32	48	90	35	50	86	—
	33	50	91	38	50	79	—
	34	50	88	40	53	80	—
Nov. 26, '04	27	36	80	45	60	80	29
	25	37	89	47	59	75	27
	26	38	88	47	61	78	28
	26	33	76	47	60	77	27
	26	32?	74?	45	60	80	26
	30	39	78	47	62	79	27

In each of the above series the periods are consecutive.

The blood-pressure in the brachial artery was determined by C. J. Martin's modification of the Riva Rocci sphygnometer,[1] and it was found to rise during the period of breathing. At the end of apnœa and the beginning of breathing the pressures observed in three determinations were 175, 175, and 170 mm. of mercury. At the end of the period of breathing and the beginning of apnœa the respective values were 195, 195, and 190 mm. of mercury. These figures represent the maximum pressure, for, owing to the incompetence of the aortic valves, the mean pressure was low.

The composition of the air in the alveoli of the lungs during the different phases of the period of breathing.— There is no doubt that the gaseous composition of the blood, which supplies the brain, regulates the depth and frequency of the respiratory movements; other factors there may be, and probably are, but the condition of the arterial blood is the most important. An increase in the partial pressure of the carbon dioxide increases the depth and frequency of respiration; a decrease in the pressure of oxygen will have a similar effect, but one which is relatively not so marked. It is impossible, except in the

[1] The apparatus used gave for the blood-pressure of healthy men at rest values of 100 to 110 mm. of mercury.

case of animals, to obtain samples of the blood flowing to and from the brain, or to measure the rapidity of the circulation. A determination, however, of the composition of the alveolar air of the lungs may give some idea of the pressures of the gaseous constituents of the blood which is flowing into the left auricle, and thence through the left ventricle, aorta and its branches to the brain. The blood in the pulmonary capillaries is separated from the air in the alveoli by a layer of tissue only 0·001 mm. thick, and the total area of the capillaries is so enormous that the blood may be considered as a vast sheet of fluid exposed to the alveolar air over a surface of 90 square metres. Equality of pressure between the gases of the blood and alveolar air would thus be facilitated, and is, indeed, believed by many physiologists to occur.

The exactness of such an indirect estimation of the pressures of carbon dioxide and oxygen in the arterial blood cannot be stated. If, as Pflüger, Zuntz, Loewy, and others maintain, the gaseous transference between the blood and the air in the alveoli of the lungs depends upon the differences in the partial pressures of the component gases in the blood and alveolar air, then an analysis of the alveolar air will yield a measurement of the pressures of the gases in the blood flowing in the pulmonary capillaries; if, on the other hand, as Bohr and Haldane believe, the pulmonary epithelium can secrete oxygen from a lower pressure in the alveolar air to a higher pressure in the blood, it follows that such results for oxygen at least may be misleading. A deficiency of oxygen, according to Bohr, Haldane, and Lorrain Smith, stimulates the pulmonary epithelium to greater secretory activity. The discharge of carbon dioxide is, according to Bohr, an active excretory process, for he finds that the pressure of the gas in the arterial blood may be below the partial pressure of the gas in the alveoli. This difficult question cannot be decided, for there is at present no evidence which is accepted as conclusive; it is unnecessary to discuss it here, especially since the evidence for

and against the rival theories has been recently given elsewhere.[1]

Samples of the alveolar air at different stages of the waxing and waning respiration were taken by the method employed by Haldane and Priestley; a modification was necessary in the shape of a mask, which fitted in an air-tight manner over the patient's nose and mouth. The results of the analyses are given in the following table.

Composition of the Alveolar Air.[2]

Early Period of Waxing Respiration.

Date.	Number of expiration.	Carbon dioxide vols. per cent.	Oxygen vols. per cent.
Nov. 25, '04	3rd	3·90	12·19
	3rd	3·61	12·84
Dec. 6, '04	2nd	3·56	13·61
	3rd	3·63	13·66

Period of Dyspnœa.

Date.	Number of expiration.	Carbon dioxide vols. per cent.	Oxygen vols. per cent.
Nov. 25, '04	5th	3·26	15·48
	10th	2·76	17·60
Dec. 6, '04	8th	3·22	15·67
	10th	2·97	16·66
	12th	2·74	16·75

Late Period of Waning Respiration.

Date.	Number of expiration.	Carbon dioxide vols. per cent.	Oxygen vols. per cent.
Nov. 25, '04	23rd or 24th	2·93	17·49
	24th	2·63	18·5
Dec. 6, '04	18th	2·00	18·86

In the cases of the early period of waxing and the late period of waning respiration the true values for the carbon dioxide are, no doubt, higher, and for the oxygen lower, owing to the shallow nature of the first and last expira-

[1] Article "Respiratory Exchange," by M. S. Pembrey, 'Recent Advances in Physiology and Bio-Chemistry,' edited by L. Hill, 1906.

[2] The alveolar air was measured saturated with water at 10°—14° C. Haldane's apparatus for gas analysis was used.

tions. The alveoli of the lungs are the effective seat of gaseous interchange; the remaining space, extending from the nose through the pharynx, larynx, trachea, bronchi, and bronchioles, is known as the "dead space." According to the estimations of Loewy, Haldane, and Priestley the "dead space" of the respiratory tract is about 140 c.c. The last portion of the air expired in a shallow expiration would thus be an admixture of air from the "dead space" and the alveoli, for it has already been mentioned that the volume of the air expired at different phases of the periodic breathing varied from 10 to over 340 c.c. The average volume of an expiration was 193 c.c. during a period of five minutes, when the patient took 179 breaths and expired 34·75 litres of air.

When the patient's general condition was better, and his breathing was of the continuous type, 31 per minute, the percentage composition of the alveolar air was 4·44 vols. of carbon dioxide and 16·34 vols. of oxygen; after the return of Cheyne-Stokes' respiration on this occasion the composition of the sample taken from the third expiration of the waxing breathing was 4·91 per cent. of carbon dioxide and 13·97 per cent. of oxygen.

The table of analyses shows that the partial pressure of carbon dioxide in the alveolar air was highest at the end of apnœa, and gradually decreased during the period of breathing; the partial pressure of oxygen showed a reverse relationship, and was highest at the end of the rapid breathing. The percentages of oxygen in the samples at the end of apnœa are so low that, according to Loewy's observations, the respiratory centre would be stimulated by the deficiency of oxygen. It would therefore appear that the carbon dioxide accumulates and the oxygen diminishes, until at last the nerve-cells of the respiratory centre are stimulated, the waxing respirations begin and culminate in very deep and rapid respirations, whereby much carbon dioxide is washed out and a large quantity of oxygen is taken in; apnœa then follows, due apparently to the absence of sufficient carbon dioxide to

stimulate the nerve-cells. The cycle would then be repeated.

It has long been known that apnœa can be readily produced in a healthy man by a series of rapid and deep breaths. By such an experiment an artificial condition of breathing, somewhat resembling Cheyne-Stokes' respiration, can be maintained for a time. The following analyses give the composition of the alveolar air at different phases of such experiments.

Experiments upon healthy men.—Subject: R. W. A—. Analyses of alveolar air, second expiration of waxing respiration—carbon dioxide 5·79 per cent., oxygen 12·24 per cent.; eighth expiration at height of deep and rapid breathing—carbon dioxide 4·16 per cent., oxygen 17·29 per cent.

Subject: M. S. P—. Breathed rapidly and deeply 18 times in 20 seconds, then breathed out a sample of alveolar air—carbon dioxide 3·22 per cent., oxygen 19·21 per cent.

Subject: M. S. P—. Breathed rapidly and deeply 17 times in 18 seconds. Sample of alveolar air from the last expiration—carbon dioxide 2·50 per cent., oxygen 19·23 per cent. Apnœa followed. The sample of the first expiration, when a desire to breathe was felt, had the following composition—carbon dioxide 5·59 per cent., oxygen 12·59 per cent.

Subject: A. B—. Breathed rapidly and deeply 21 times in about 20 seconds. Sample of alveolar air from the last expiration—carbon dioxide 4·10 per cent., oxygen 18·29 per cent. Apnœa followed. The sample of the first expiration, when a desire to breathe was felt, had the following composition—carbon dioxide 6·11 per cent., oxygen 11·26 per cent.

The results of the analyses of the alveolar air lead to the conclusion that the periodic breathing is probably due to changes in the partial pressures of the oxygen and carbon dioxide of the arterial blood which supplies the respiratory centre. In order to test this view observations were made upon the influence of various percentages of oxygen and carbon dioxide in the air which the patient breathed.

The influence of various percentages of oxygen upon the

respiration.—The observations were carried out by allowing the patient to breathe oxygen which was supplied through a mask provided with inspiratory and expiratory valves. The respiratory movements were recorded graphically for long periods upon a large kymograph, which carried a piece of smoked paper 280 cm. long and 25 cm. wide; such long records were necessary in order to control the observations. The procedure was as follows:—A stethograph, consisting of a small rubber bag, was fastened round the patient's waist at the level which gave the most movement during the weaker contractions of the diaphragm, and was connected with a recording tambour by means of flexible leaden tubing; it was thus possible to replace the bedclothes and, without any disturbance of the patient, to record the respiration for an hour or two; on some occasions the patient even went to sleep during the observations. The stethograph was sensitive enough to record, if it were required, the contractions of the heart as well as the respiratory movements. A record of the respiration of the patient was taken when he was lying quite undisturbed in bed; this part will be called in the description of the tracings "the control without mask and valves." During the continuation of the record the mask provided with inspiratory and expiratory valves was applied to the patient's face; the invariable effect was at first a shortening or abolition of the period of apnœa, but this soon passed off, and Cheyne-Stokes' respiration reappeared; this part of the observation will be designated "the control with mask and valves." Oxygen was then delivered from a cylinder into a rubber bag, which was connected by a tube with the inlet of the mask; the gas could thus be supplied under a pressure so slight as not to force the inspiratory valve during the phase of apnœa.

The following series of tracings will show that almost pure oxygen (90 per cent.), inhaled through a mask provided with inspiratory and expiratory valves, produced a very definite effect; in about half a minute or more the periods of apnœa became shorter, then were filled in by

shallow respirations, and in about three or four minutes were completely abolished; easy respirations of a continuous type were obtained during the further administration of the gas. The supply of oxygen was discontinued, but for the purpose of control the mask and valves were retained in position; the continuous type of respiration persisted for about a minute, then shallower respirations ushered in the return of periodicity, and finally the typical Cheyne-Stokes' respiration was obtained. The mask was removed, and then, after a further record, the observation was brought to an end. Variations in the promptness of the effect are shown by the different curves (Plates Figs. II, IV, V, VI).

On various occasions the following percentages of oxygen were administered, namely 89·97, 20·9, 17·43, 15·2, 14·38, and 8·67, and the tracings show that percentages above and below the amount of oxygen present in atmospheric air tended to abolish, or actually did abolish, the Cheyne-Stokes' respiration. When pure oxygen was inspired the respirations were easy, the patient felt more comfortable, and the dusky hue of the face was replaced by a more natural colour; when there was a deficiency of oxygen the respirations were deeper, but there was no distress (Plates Figs. III, IX, X).

The administration of oxygen in the ordinary clinical way by a glass funnel held in front of the patient's face did not abolish the periodic breathing; this was probably due to the dilution of the oxygen with air.

The total volume of air expired when the patient was breathing air and exhibiting Cheyne-Stokes' breathing was greater than the volume expired when he was breathing pure oxygen by means of the mask and valves. Thus during three whole periods of typical periodic breathing lasting 240 seconds 29·5 litres of air were expired, a rate of 7·4 litres per minute; oxygen was given, and in the first period of 90 seconds 9 litres were expired, a rate of 6·0 litres per minute; the respiration became continuous in type, and during 420 seconds 44 litres were expired, a rate of

6·3 litres per minute; the oxygen was replaced by air and Cheyne-Stokes' respiration returned, 18 litres were expired in 180 seconds, a rate of 6·0 litres per minute. A delay both in the appearance and the disappearance of the effect of oxygen upon the patient's breathing is shown in the tracings; this would be expected, for not only would several seconds elapse before the air in the alveoli became rich in oxygen, but time would also be required for the passage of the blood through the lungs and thence to the capillary vessels of the medulla oblongata. According to G. N. Stewart's calculations the blood passes from the pulmonary artery through the lungs to the pulmonary veins in about 15 seconds.

The influence of various percentages of carbon dioxide upon the respiration.—The effect of carbon dioxide in air or oxygen was investigated in a similar manner to that just described for oxygen. The curves show that the effect of carbon dioxide in amounts above 2 per cent. was very definite and prompt; with a low value, 2·26 per cent. in air, the apnœa was abolished but the waxing and waning respiration persisted; with higher percentages all tendency to periodicity was abolished (Plate Fig. XI). Carbon dioxide always yielded satisfactory results provided that it was above the minimum mentioned; in this respect it was more certain than oxygen. When the administration of the gas was discontinued the periodic respiration quickly returned. These facts are so clearly demonstrated by the graphic records that no further account is necessary.

The expired air of the patient contained 2 to 3 per cent. of carbon dioxide, an amount which was always sufficient to abolish apnœa (Plate Fig. VIII). The tolerance of the patient to high percentages of carbon dioxide is shown by Plate Fig. VII, and will be discussed later.

The influence of psychical conditions upon the respiration.—It may be stated briefly that anything which tended to increase the nervous excitability of the patient shortened or abolished for a time the period of apnœa,

and lessened the severity of the dyspnœa. It was often necessary to prolong the records owing to the disturbance introduced by some one coming to the bed and discussing the patient's condition and respiration. The influence of suggestion upon the mind of the patient was carefully watched; not only were the controls without and with the mask and valves made during each observation, but the supply of oxygen was several times stopped without the knowledge of the patient and replaced by air; on other occasions the oxygen was allowed unknown to the patient to escape into the air of the ward, so that the noise caused as it issued from the cylinder into the bag might still continue when the patient was breathing air. Oxygen and air were also given from large wedge-shaped gas-bags for the purpose of control. When mixtures of carbon dioxide and various percentages of oxygen or air were administered, one or two large gas-bags were used, and the mixture, whatever it might be, was known to the patient only as gas.

During sleep there was a tendency for the Cheyne-Stokes' respiration to be more pronounced, and on days when the breathing was continuous the periodic respiration often returned during sleep. Movement of the head from side to side during each period of breathing was sometimes seen in the patient when he was asleep.

The patient's mental condition appeared to be more active during the period of breathing than in the period of apnœa, and he often showed more inclination to talk during that time. Conversation with the patient would tend to abolish apnœa. When his general condition was better the respiration was continuous. He could at other times by an effort of the will breathe continuously for a short time, but soon relapsed into periodic breathing (Plate Fig. III).

The condition of the respiratory centre.—Stress has been laid upon the evidence of diminished nervous excitability, for all the observations tend to confirm the view that Cheyne-Stokes' breathing is associated with

a diminished excitability of the central nervous system. It is known that Cheyne-Stokes' respiration is present under perfectly natural conditions in mammals during some of the stages of hibernation, and in healthy infants and some adults during sleep; it can be produced in healthy animals by morphia and chloral. In these cases the general nervous excitability is diminished. During disease periodic breathing often follows the administration of morphia and chloral: in the present case such drugs were not given to the patient.

Confirmation of this view was found in the results obtained from the microscopic examination of the medulla oblongata. This was carried out by Dr. E. F. Buzzard, to whom we are greatly indebted for the excellent preparations and the following report:

Report on the Microscopic Examination of the Pons and Medulla oblongata. By E. F. Buzzard, M.A., M.D., F.R.C.P.

"The brain stem was hardened in 5 per cent. formalin. Three methods were employed in the examination of the pons and medulla.

(1) The Busch modification of the Marchi method for demonstrating recent degeneration in the nerve-fibres.

(2) A modified Nissl stain for investigating the condition of the nerve cells, and

(3) The ordinary hæmatoxylin-eosin stain for observing any morbid condition of the meninges or blood-vessels.

(1) *Busch method.*—A few sections from different levels of the pons and medulla were stained by this method and examined microscopically. A considerable number of fibres in the left, and a smaller number of fibres in the right pyramidal tracts were found to have undergone degeneration in all the sections, but the majority of the fibres in both were healthy.

With the exception of a few scattered degenerated fibres in the lateral regions of the medulla and in the tegmental region of the pons no other changes were observed.

(2) *Nissl method.*—The stain used was methylene blue, and a large number of sections were mounted serially from the posterior half of the fourth ventricle. It may be stated at once that the fixation and staining were very successful, and it was possible to study accurately the condition of the cells forming the nuclei of the 8th, 9th, 10th, and 12th cranial nerves.

(*a*) *The hypoglossal nucleus.*—The cells of this nucleus were of healthy size and shape on both sides. They

FIG. 3.

Nearly normal cell from the hypoglossal nucleus.

contained numerous chromatin granules which were rather larger than the normal, and in some instances tended to encroach upon the nucleus and render the latter less well defined than usual. The nucleolus was generally centrally situated, and rather prominent, perhaps slightly swollen.

On the whole the cells had a healthy appearance.

(*b*) *The dorsal nucleus of the vagus.*—In this case marked changes were seen in the cells, especially on the left side. In the left nucleus only about one third of the cells were of healthy appearance, and these were mostly situated in the ventral part of the nucleus. The remainder of the cells had undergone changes chiefly of

an atrophic character. Many were shrunken, greatly altered in shape, and contained only a few coarse granules with no recognisable nucleus or nucleolus. Others showed slight chromatolytic changes with excentric nuclei. Examples of the various types are represented in Figs. 4 and 5. It should be remembered that serial sections were examined, and it was possible therefore to examine the whole of each cell, and to exclude the possibility of the appearances being due to their incomplete presentation in any particular field. On the

FIG. 4. FIG. 5.

FIG. 4.—Healthy cells from the left dorsal nucleus of the vagus, near its ventricular aspect.

FIG. 5.—Degenerate cells from the deeper portions of the left dorsal nucleus of the vagus.

right side the changes were similar in character, but did not involve so large a proportion of the cells.

(*c*) *The nucleus ambiguus.*—The cells of this nucleus were, on the whole, natural in size and general appearance, although one or two showed chromatolytic changes. No difference was noted between the cells of the right and left sides.

(*d*) *The fasciculus solitarius.*—The cells situated around this bundle were scanty, and many of them had undergone morbid changes which are represented in Fig. 6. Excentration of nuclei and chromatolysis were noticeable and were often associated with a considerable amount of pigmentation.

(*e*) *The nucleus of the descending root of the eighth nerve.*—A large number of the cells of this nucleus showed swelling, chromatolysis, and excentration of nuclei, but there were no atrophic changes similar to what was seen in the case of the dorsal nucleus of the vagus. Two cells showing varying degrees of change are represented in Fig. 7.

(3) *Hæmatoxylin-eosin method.*—Only a few celloidin sections were stained by this method, and no very marked changes were observed in the blood-vessels. Slight

FIG. 6. FIG. 7.

FIG. 6.—Chromatolysis and pigmentation of cells of the descending glosso-pharyngeal root.
FIG. 7.—Slightly changed cells (chromatolysis) from the descending vestibular root.

thickening of the meninges with a small excess of round cells and numerous corpora amylacea were noted in some parts. No evidence of capillary thrombosis or hæmorrhage was forthcoming.

In conclusion it may be said that the most profound changes were seen in the dorsal nucleus of the vagus where a large proportion of the cells, especially on the left side, had undergone marked atrophy and degeneration.

The alteration in other nuclei were mostly of the chromatolytic type such as is commonly seen in toxic con-

ditions or as a reaction to lesions in the axis cylinder processes."

We may regard these changes in the pons and medulla oblongata, as well as those already described in the cerebrum, as due to defective supply of arterial blood. Experimentally such changes have been produced by Mott and Leonard Hill.

Discussion of the Results.

It is now necessary to discuss the results and explain, if it be possible, the cause of Cheyne-Stokes' respiration. The examination of the patient gave evidence of diminished nervous excitability, which was probably caused by a defective supply of blood to the brain due to the aortic regurgitation and atheroma. It could be easily understood that the respiratory centre would be still excited by a deficiency of oxygen, if the partial pressure of carbon dioxide were below its stimulating value, or the abnormal centre were less sensitive to carbon dioxide, the usual stimulus to respiration. The following may be the sequence of events. The partial pressure of the carbon dioxide increases and the oxygen decreases until the centre is stimulated, the respirations are feeble at first but gradually increase in vigour until they culminate in rapid deep respirations whereby a large quantity of carbon dioxide is washed out of the alveolar air and blood and a large quantity of oxygen is taken in. The respiratory centre is no longer stimulated owing to the reduction in the partial pressure of carbon dioxide and the increase in oxygen, apnœa therefore follows and persists until the cycle begins again. Owing to the time taken for the passage of the blood through the lungs to the medulla oblongata, there will be some delay before the effect of the pulmonary ventilation can be shown upon the respiratory centre; thus will arise the waxing and waning of respiration. The volume of the first breath or two

during the period of breathing is so small that it will have no effect upon the composition of the alveolar air and the strength of the stimulus to the respiratory centre will be increasing meanwhile.

If these views be correct, the administration of carbon dioxide by increasing the partial pressure of the gas in the alveolar air and arterial blood should supply a constant stimulus to the respiratory centre and apnœa should cease. The graphic records show that the inhalation of carbon dioxide in percentages varying from 2·2 to 11·2 produced all gradations from feeble respirations in the place of apnœa to continuous breathing of almost regular type. A small percentage of carbon dioxide 0·76, as would be expected, was not enough to raise the partial pressure of the gas to its stimulating value. Further, if the partial pressure of the carbon dioxide in the arterial blood be low during Cheyne-Stokes' breathing and the nervous excitability of the respiratory centre diminished, the patient should tolerate a greater dose of carbon dioxide than can a healthy man. This was found to be the case, air containing 11·2 per cent. of carbon dioxide produced regular breathing in the patient but great distress in ourselves. The administration of carbon dioxide would also probably improve the circulation by its stimulating action upon the heart.

The analyses of the alveolar air showed that the percentage of oxygen was sufficiently low at the beginning of the period of breathing to produce excitation of the respiratory centre. It is also necessary to consider why the administration of oxygen abolished the Cheyne-Stokes' respiration. It would seem that the breathing of the pure oxygen by the mask and valves maintained the pressure of oxygen in the alveoli and the blood at such a high level that, notwithstanding the aortic regurgitation, the respiratory centre was well supplied with oxygen, and being no longer stimulated by a deficiency of the gas, did not send out the vigorous impulses which had previously resulted in rapid deep breathing, whereby much carbon

dioxide had been washed out of the alveoli and blood at the same time that oxygen was taken in. The partial pressure of the carbon dioxide would thus be maintained at a more constant level, and would stimulate the respiratory centre to regular rhythmic activity. In order to test this view, samples of alveolar air were taken soon after the patient had breathed pure oxygen; the fifth expiration after the inhalation of oxygen gave 3·47 per cent. carbon dioxide, and 24·71 per cent. oxygen, the twenty-second expiration yielded a sample with 4·31 per cent. carbon dioxide, and 17·26 per cent. oxygen. Moreover, the natural continuous breathing of the patient yielded on different occasions samples of alveolar air with the following percentage compositions:—Carbon dioxide 4·27, 3·52, 4·44, oxygen 15·00, 15·7, 16·34.

The increase in the percentage of carbon dioxide is not very great, but, considered in relation to the percentage of oxygen, the difference between the alveolar air of continuous and periodic breathing is more accentuated.

It has been shown that the inhalation of a mixture of oxygen and nitrogen containing a smaller percentage of oxygen than that present in atmospheric air also abolished apnœa. The explanation is, doubtless, that there was a constant deficiency of oxygen, and thus a continual stimulus to the nerve-cells of the respiratory centre.

The continuous respiration produced by voluntary effort or slight excitement of the patient appears to have been due to an increase in the excitability of the respiratory centre caused by nervous impulses passing from higher centres to the neurons of the centre in the medulla.

Conclusions.

It would appear that the periodicity of Cheyne-Stokes' respiration in this case is due to a diminished excitability of the nervous system associated with a defective supply of arterial blood; the carbon dioxide accumulates, and

the oxygen diminishes until at last the nerve-cells are stimulated, the waxing respirations begin and culminate in marked dyspnœa, whereby a large quantity of carbon dioxide is washed out and sufficient oxygen is taken in; apnœa then follows, due, apparently, to the absence of sufficient carbon dioxide to stimulate the nerve-cells.

The inhalation of air containing more than 2 per cent. of carbon dioxide abolishes apnœa by maintaining the partial pressure of the carbon dioxide in the alveolar air and blood at its stimulating value.

The administration of pure oxygen by means of a mask and valves abolishes apnœa by maintaining the partial pressure of carbon dioxide in the blood at its stimulating value. The respiratory centre is no longer excited by lack of oxygen to send out the forcible impulses which had previously resulted in excessive ventilation of the lungs, whereby carbon dioxide had been washed out of the alveoli and blood.

Air containing a smaller percentage of oxygen than that present in atmospheric air abolishes apnœa; the constant deficiency of oxygen stimulates the respiratory centre.

References.

No attempt has been made to examine the extensive literature on Cheyne-Stokes' respiration. The following articles either refer to questions raised in this paper or serve as a guide to the subject.

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APPENDIX.

The data of observations not already given are collected here in order that the paper may not be overloaded with tables of results.

Table to show the duration of apnœa and breathing on various days, and the number of respirations during the periods of breathing.

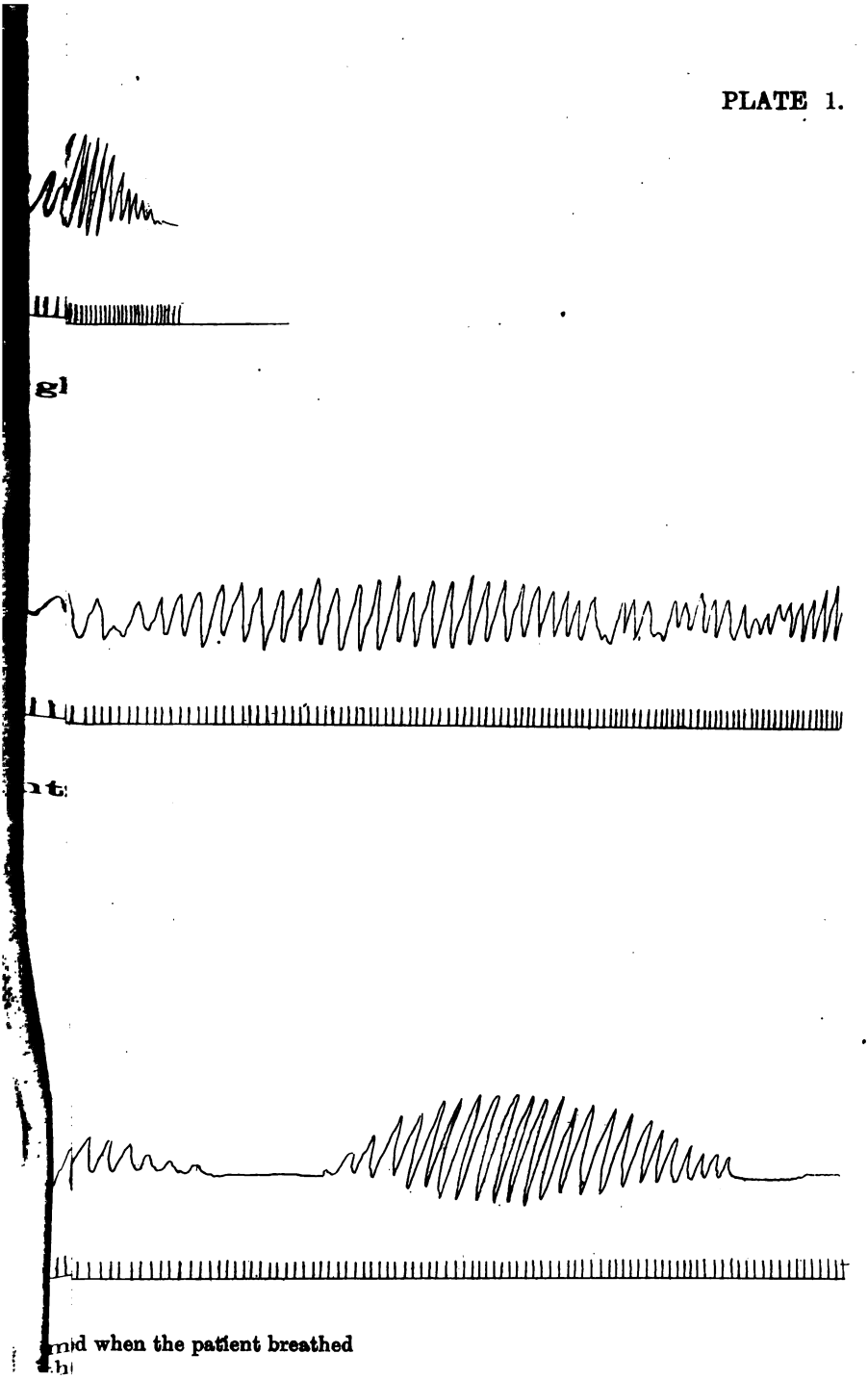
Date.	Duration of apnœa in seconds.	Duration of breathing in seconds.	Number of respirations.
Dec. 1, '04	33	37	—
	32	40	—
	33	35	—
	34	38	—
	33	40	—
Dec. 13, '04	43	41	18
	38	40	18
	43	46	20
Dec. 14, '04	37	49	17
	28	45	18
Dec. 15, '04	23	40	17
	22	38	16
	19	40	18
	21	38	16

Date.	Duration of apnœa in seconds.	Duration of breathing in seconds.	Number of respirations.
Dec. 17, '04	18	42	18
	22	47	18
Dec. 20, '04	20	52	20
Dec. 21, '04	20	44	19
	18	49	19
	18	40	18
	19	38	15
Jan. 5, '05	15	43	20
	16	48	21
Jan. 10, '05	25	46	23
	22	49	24

Note.—A preliminary account of the observations made by Pembrey and Allen was communicated to the Physiological Society, January 21st, 1905 ("Proc. Physiol. Soc.," 'Journ. Physiol.,' vol. xxxii, 1905, p. 18).

The expenses of the investigation were defrayed from a grant from the Royal Society.

PLATE 1.



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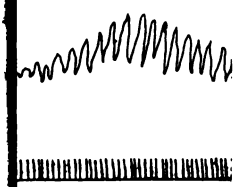

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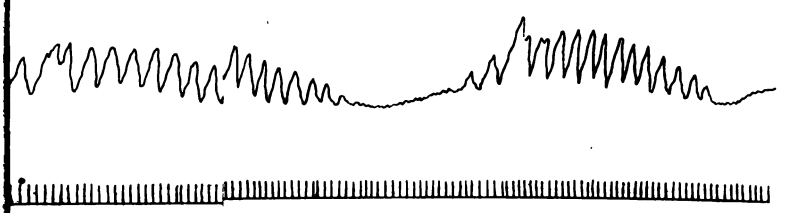

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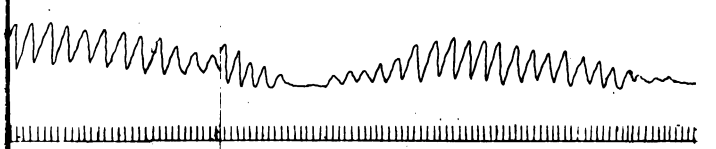

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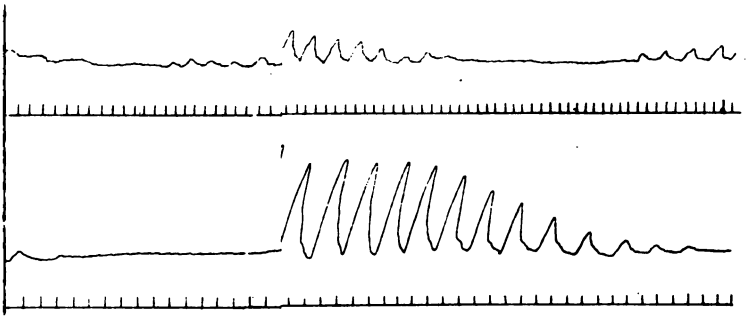
PLATE 3.



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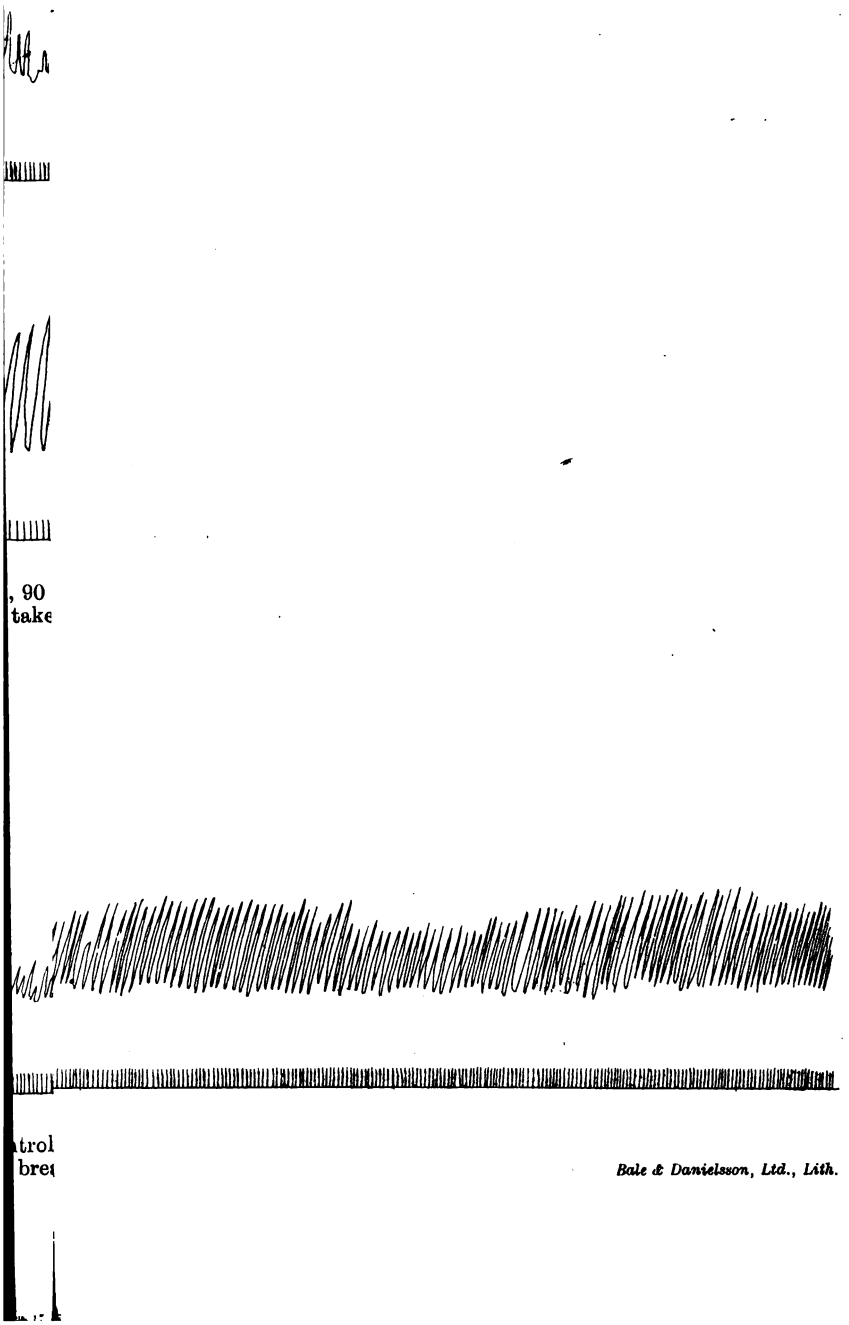

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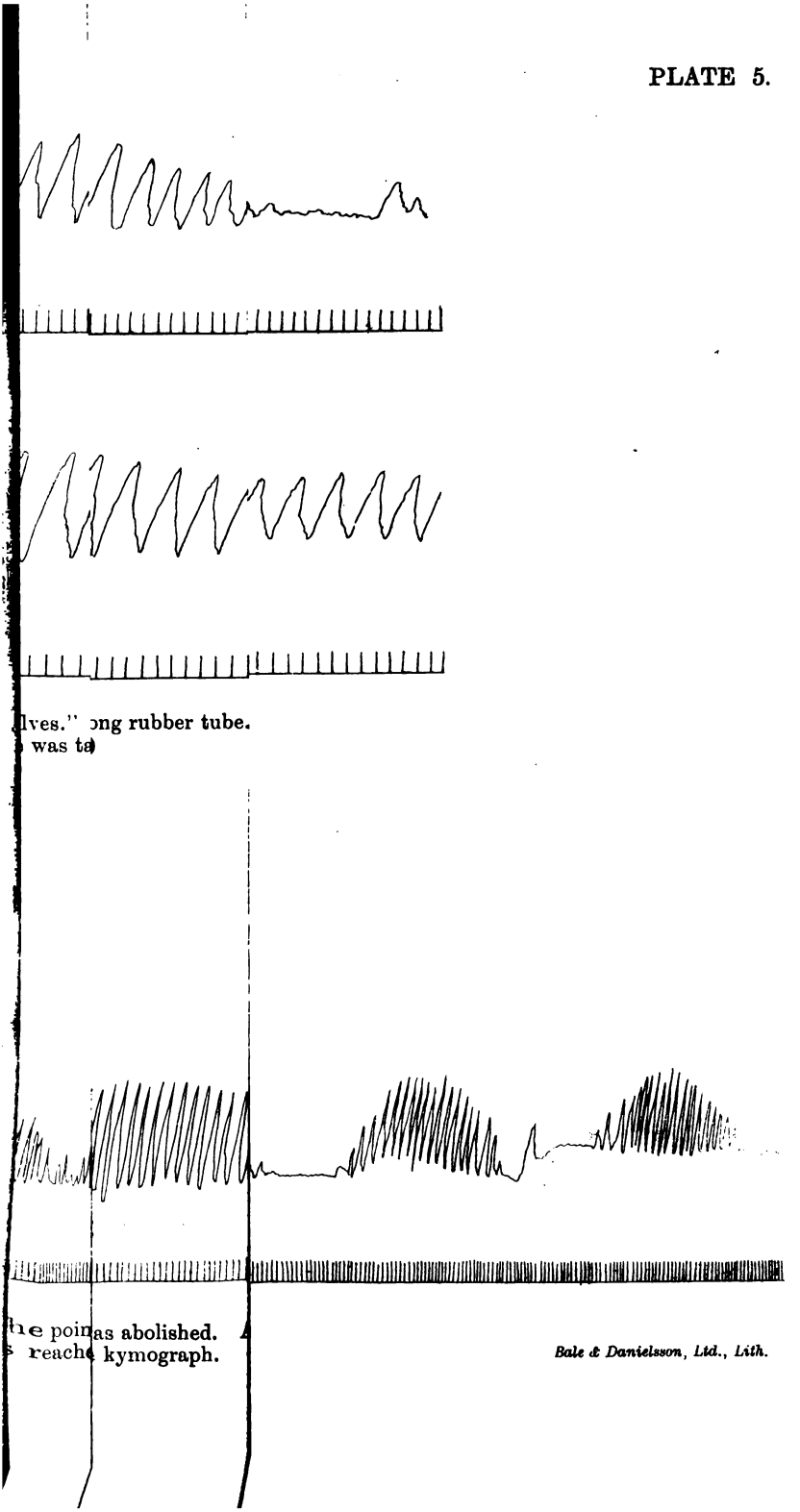

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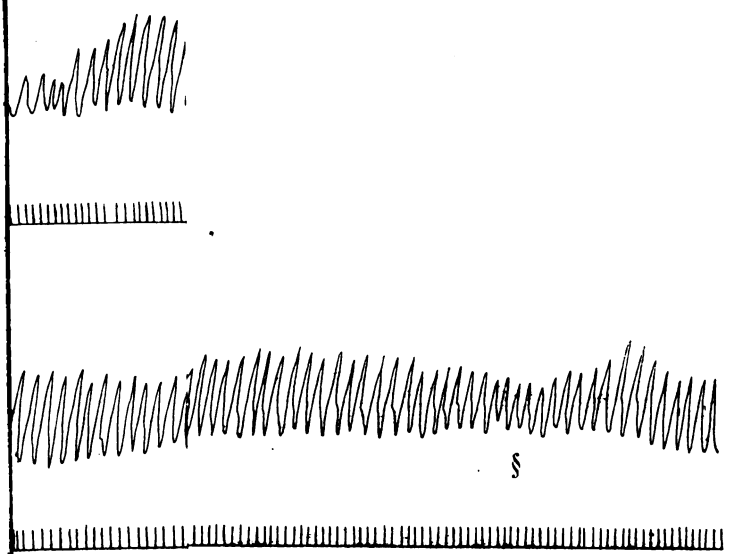

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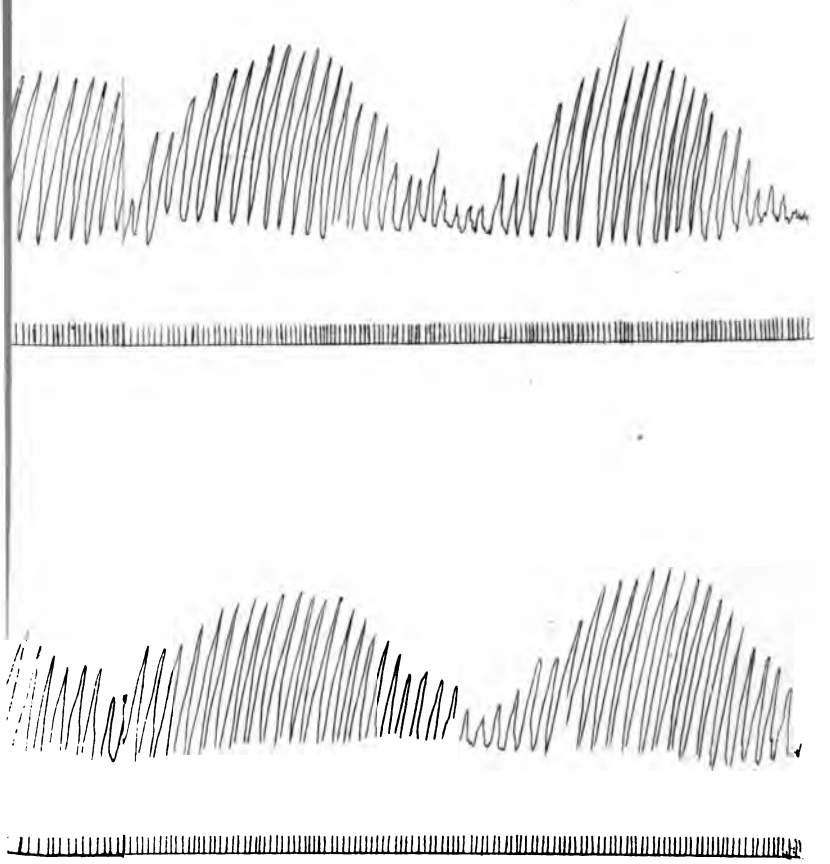

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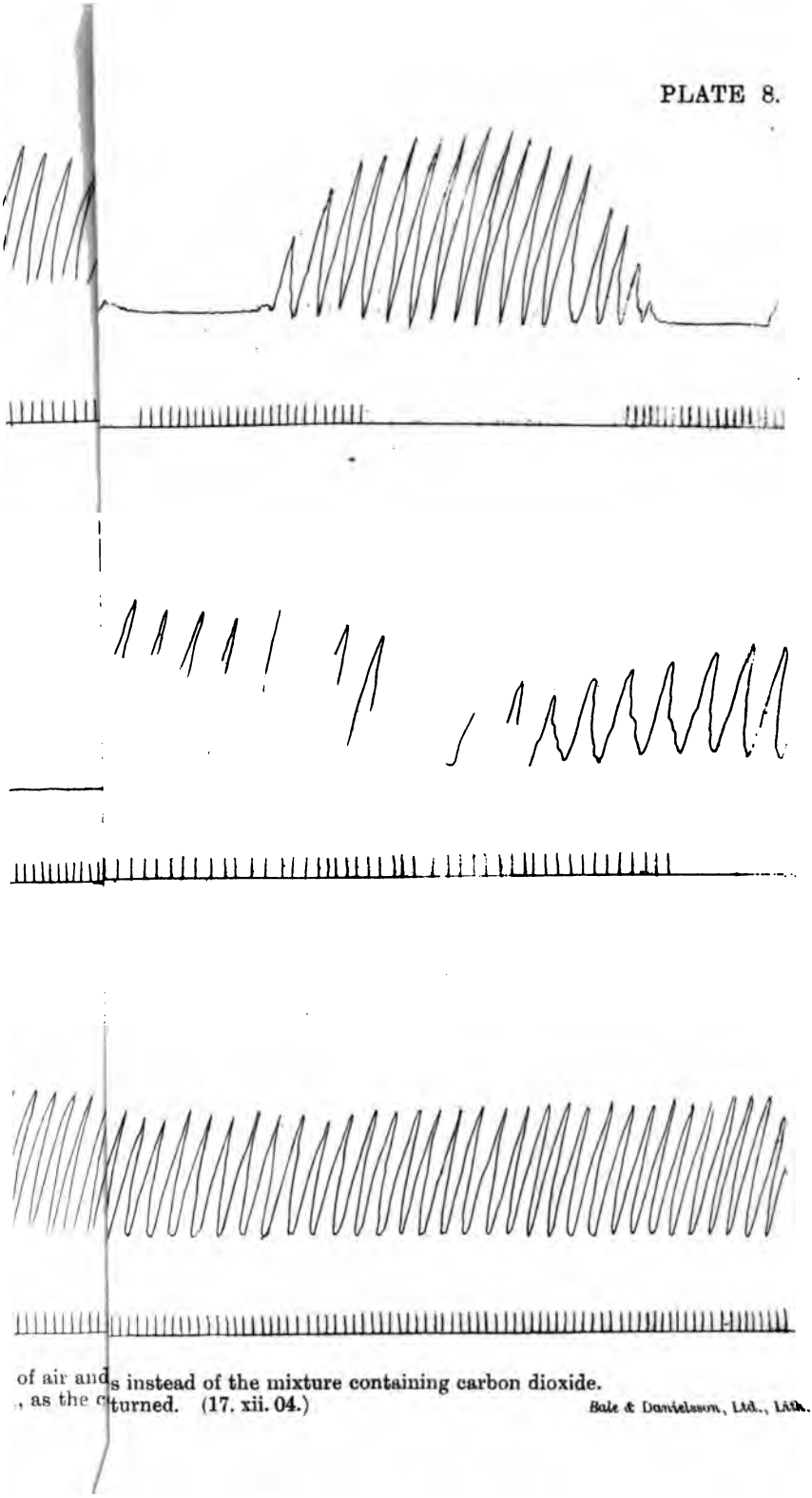

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OBSERVATIONS UPON TWO CASES OF CHEYNE-STOKES' RESPIRATION

BY

FREDERICK TAYLOR, M.D., F.R.C.P.,

M. S. PEMBREY, M.A., M.D.,

A. P. BEDDARD, M.A., M.D., F.R.C.P.,

AND

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Received November 22nd, 1906—Read January 8th, 1907

Two patients in Guy's Hospital presented the phenomena of Cheyne-Stokes' respiration in so characteristic a form that further observations were made to supplement those already recorded in the preceding paper.[1]

CASE 1.—*Observations upon the Results of the Clinical and Post-mortem Examination.* By Frederick Taylor and Herbert French.

George J—, aged 46, a gas-work stoker, was admitted into Guy's Hospital under the care of Dr. Frederick Taylor on July 17th, 1905, and died on February 4th, 1906. During the whole of this time, six and a half months, he had typical Cheyne-Stokes' respiration, except during the three days immediately preceding death.

He had been known to have kidney disease for at least ten years, and he was admitted for an exacerbation, with

[1] G. Newton Pitt, M. S. Pembrey, and R. W. Allen.

some œdema of his legs. The condition of his urine was variable. It always contained albumen, and moderate numbers of hyaline and granular tube-casts. The amount of albumen varied from a mere trace to 3 parts per thousand. The specific gravity was always low, and varied from 1006 to 1012, though on two occasions it rose to 1018. The quantity was above normal; even in the middle of August he passed as much as 92 ounces in 24 hours upon one occasion, and he frequently passed above 70 ounces.

The diagnosis was granular kidney, and this was confirmed by autopsy.

The heart was much hypertrophied, the impulse being in the fifth left intercostal space, three quarters of an inch outside the left nipple, heaving in character. There were no bruits; the first sound was prolonged at the apex, and in the aortic area the second sound was much accentuated. The pulse rate was usually between 60 and 70 per minute, and the maximum systolic blood-pressure, measured in the brachial artery by Martin's modification of the Riva Rocci apparatus, varied from 165 mm. Hg. to 178 mm. Hg. During hyperpnœa cardiac dulness could only just be detected to the right of the sternum; during apnœa this dulness increased a full inch further to the right.

The lungs were at first congested at the bases; the signs of this cleared up. Just before death there was dry pleurisy over the right lower lobe. There was no emphysema.

The gastro-intestinal viscera were natural, as were also the nerve reflexes.

There was well marked albuminuric retinitis in both eyes, with white patches of exudation, and hæmorrhages both old and recent. The patient, however, always said his eyesight was good.

The persistent Cheyne-Stokes' respiration is fully described below; it was very characteristic all the time the patient was under observation. In addition to this there

were several attacks of uræmic "asthma" during the first few days after admission. These disappeared with rest in bed, restricted proteid diet, purgatives, and digitalis.

When first admitted the man was muscular but thin. The initial œdema entirely disappeared, and relief was at first great. A month from admission the patient was able to be up and about, notwithstanding that he had Cheyne-Stokes' respiration all the time. A month later he took to his bed again, and remained there till his death. The observations upon his respiration were not begun until January, 1906, six months after he first came in.

He was always conscious, and, except for the "asthma" mentioned above, had no uræmic symptoms. He could read the newspaper with pleasure, but towards the end he took less interest in things, and when spoken to he would sometimes delay as much as half a minute before answering. This slowness of response was always more marked in apnœa than it was in hyperpnœa.

Though he took his food well he wasted markedly. His legs and body became mere skin and bone. Latterly he passed his urine and fæces into the bed, less from incontinence or unconsciousness than from simple lethargy. If a nurse were near he would ask for urinal or bed-pan; but if no nurse were near he would not be bothered.

After the first few days there was no œdema; the heart did its work well, without any of the recognised signs of failure.

Three days before death acute dry pleurisy set in, and simultaneously the Cheyne-Stokes' respiration disappeared. Death was very easy, the end coming during a deep sleep.

The autopsy showed the following organic changes:

Kidneys.—Both of the kidneys were small and granular in appearance. The capsules were adherent, the cortex thin, the arterioles prominent and thick-walled. In the right kidney there were also a series of sacculated, tuberculous abscesses. The ureters, bladder, and urethra were natural.

Heart.—The heart was much hypertrophied, more especially the left ventricle. There was no valvular disease beyond some atheroma of the aortic valves. The coronary arteries were tortuous and thick-walled, but quite pervious.

Lungs.—Small. There was recent pyogenic membrane over the right upper and lower lobes, and a recent infarct in the left lower lobe. There was no chronic lung disease.

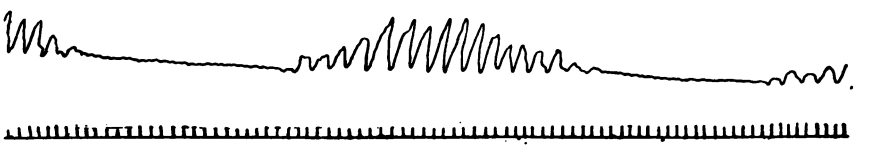
Brain.—The cerebral arteries were widely patent, thick-walled, and atheromatous. There was a small patch of softening in and around the left internal capsule. There was no hæmorrhage. The pons and medulla looked healthy to the naked eye, and were sent to Dr. Farquhar Buzzard for further examination. We are greatly indebted to him for the report given on page 90.

The remaining viscera showed no macroscopic abnormality.

Experimental Observations, especially in Relation to the Causation of Periodic Breathing. By M. S. Pembrey and Herbert French.

The patient exhibited a typical waxing and waning of the respiratory movements (Fig. 1) accompanied by a

FIG. 1.



Cheyne-Stokes' respiration. The curve reads from left to right, and the time is marked below in seconds. The small undulations during the period of apnœa are due to the beats of the heart (January 23rd, 1906).

general overflow of motor impulses; during this period he moved uneasily and complained of pain in the abdomen. During apnœa he remained conscious, but did not

speak of his own accord, and only answered questions after great delay. The duration of apnœa and breathing is shown by the tracing (Fig. 1) and the following consecutive determinations.

	Apnœa.			Breathing.			
Date.	Duration in seconds.	Rate of Pulse. Actual.	Per minute.	Duration in seconds.	Rate of Pulse. Actual.	Per minute.	Respirations.
13/1/06.	25	26	(62)	20	27	(81)	11
	22	28	(76)	20	25	(75)	10
	25	28	(67)	20	20	(60)	12
	25	25	(60)	20	33	(99)	11
13/1/06.	25	37	(89)	20	26	(78)	10
	22	32	(87)	25	27	(65)	11
	25	32	(77)	23	26	(68)	10
	22	28	(77)	21	25	(71)	10

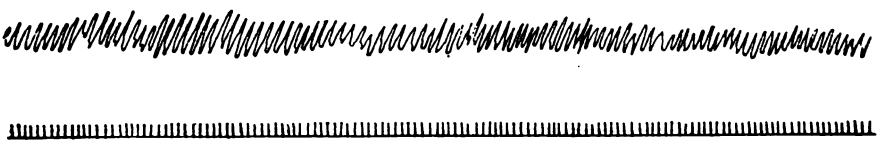
Three or four days before death (February 4th, 1906) his breathing lost the periodic type and became continuous; at this time there were signs of pleurisy, upon the right side. When the patient was asleep on January 29th his breaths were deep and regular, 30 per minute; after he awoke they were somewhat irregular, but still of the continuous type. The colour of his face was much fresher than it had been in the days when Cheyne-Stokes' respiration was present; he was, however, complaining of pain. Again on January 31st he was observed asleep; his respirations were regular and deep, and showed no periodic changes either in frequency or depth. After he awoke his respirations were still of a continuous type, but they undoubtedly showed a periodic change in depth; a series of deeper breaths followed shallower ones, and this alternation was repeated again and again. Percussion of the chest caused pain, especially upon the right side.

This disappearance of the Cheyne-Stokes' respiration is interesting; it would appear that, owing to the pleurisy, the patient was no longer able to take the very deep breaths which had characterised the height of the waxing respiration; carbon dioxide would therefore not be re-

moved so freely, and its mean partial pressure would be maintained at a higher level. It is possible that the pain caused by the pleurisy not only limited the expansion of the lungs, but also by impulses passing up the vagi and other nerves raised the excitability of the respiratory centre.

Our observations were made during the time when the Cheyne-Stokes' phenomena were at their height, and consisted chiefly in an investigation of the effects of oxygen and carbon dioxide upon the periodicity of the breathing.

FIG. 2.



The effect of pure oxygen administered by means of a mask provided with inspiratory and expiratory valves. Respiration is of the continuous type. (January 23rd, 1906.)

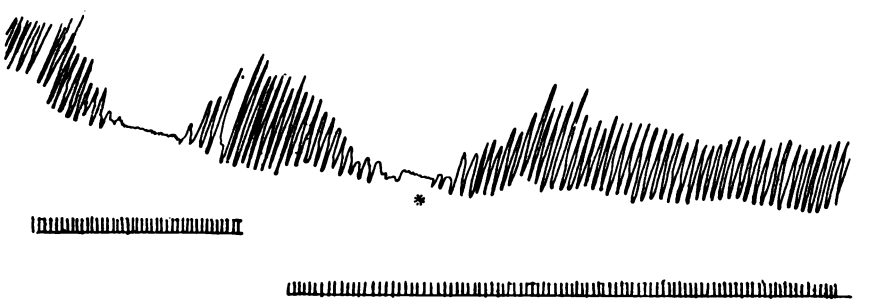
The administration of oxygen through a mask provided with inspiratory and expiratory valves caused the apnœic pause to disappear within one minute and a half. In one record, when the patient was breathing air through the mask and valves the period of breathing lasted thirty-nine seconds, and included twenty respirations; it was followed by apnœa for a period of nineteen seconds. During the breathing of pure oxygen the respirations were shallow, rapid, and continuous in type, 48 respirations in fifty-eight seconds (Fig. 2). Oxygen gave the patient much relief and removed the dusky ash-grey colour of the face.

Air containing 4·67 per cent. carbon dioxide and 20·48 per cent. oxygen was administered towards the beginning of a period of breathing; the tendency to apnœa was promptly abolished, the respirations became continuous in type, deep, and of a frequency of 48 in a minute (Fig. 3). The breathing of air containing 4 to 5 per cent. of carbon

dioxide caused the patient no distress, but several times made him cough.

Apnœa was also abolished when the patient breathed through a mask with a long rubber tube; the "dead space" was increased, and thus the inspired air, by admixture with some of the air just expired, contained a higher percentage of carbon dioxide and a lower percentage of oxygen than in normal air. Even the slight

FIG. 3.



The first portion of the tracing shows Cheyne-Stokes' respiration when the patient was breathing air through the mask and valves. At the point marked by the star a mixture of air and carbon dioxide, 20·48 per cent. oxygen and 4·67 per cent. carbon dioxide, was administered. The respirations became continuous in type. The small undulations during the period of apnœa are due to the beats of the heart. (January 23rd, 1906.)

disturbance of respiration produced by the application of the mask and valves tended at first to prolong the period of breathing and to shorten the period of apnœa, although in this case there was but a very small increase in the "dead space."

Samples of the alveolar air of the lungs were collected at different stages of the periodic breathing. The results of the analyses are given in the following table:

		Waxing Respiration.	
Date.		Carbon dioxide. Vols. per cent.	Oxygen. Vols. per cent.
13/1/06.	5th Expiration.	2·70	17·19
	„	2·01	18·01
23/1/06.	Height of Hyperpnœa.	1·87	18·51

Date.		Waning Respiration. Carbon dioxide. Vols. per cent.	Oxygen. Vols. per cent.
13/1/06.	10th Expiration.	2·05	18·89
	13th „	1·76	19·22

There is no doubt that the true percentages of oxygen were lower and of carbon dioxide higher, for the mask did not always fit absolutely tight to the patient's bearded face. The first few respirations of the waxing respiration were, moreover, so shallow that it was difficult to obtain reliable samples. The results do not, therefore, possess the same value as those obtained in the previous case.

Observations on the blood pressure of the brachial artery were made by means of Martin's modification of the Riva Rocci sphygnometer. At the height of hyperpnœa the readings for the maximum pressure were 178, 174, and 178 mm. of mercury, in the middle of the apnœic pause 178, 172, and 173, and at the end of apnœa 168, 168, and 165. Apart from the difference in blood pressure the influence of the periodic breathing was shown upon the area of cardiac dulness, which was increased by 18 mm. to the right of the sternum and 20 mm. outward to the left during the apnœic pause.

The Structural Changes in the Pons and Medulla Oblongata.
Report by E. Farquhar Buzzard, M.D., F.R.C.P.

"The pons and medulla were hardened in 5 per cent. formalin, and then cut transversely into a number of thin slices. With the naked eye these slices appeared normal, except for the presence of numerous small areas of softening, some of which were of a bright red and others of a dirty yellow colour. The largest of these areas was scarcely larger than a pin's head, and they were more numerous in the medulla than in the pons. In both regions they were more often seen in the neighbourhood

of the median raphe than in the more lateral parts. A number of slices were embedded in paraffin and cut; the sections were stained either with methylene blue or with logwood and eosin.

"*Microscopical.*—Although degenerative cell changes in the various nuclei of the medulla were striking, they could only be regarded as secondary to the vascular disease which was very marked throughout the sections. In the first place, nearly every arteriole and capillary in the substance of the medulla showed general thickening, chiefly of the media, with hyaline degeneration. Some of the larger vessels, especially those near the median line, presented, in addition to the hyaline change in the media, marked periarteritis, indicated by the presence of numerous round cells in the adventitia and perivascular spaces.

"In the second place, there were many recent and some older capillary hæmorrhages, especially just beneath the floor of the fourth ventricle, in the region of the dorsal nuclei of the vagus.

"Thirdly, evidence of numerous areas of softening was to be seen in the form of fibrous scars, remains of vessels surrounded by necrotic tissue and altered blood pigment. One of these involved and destroyed a considerable part of one of the inferior olives, and others were situated more dorsally near the median raphe.

"In addition to these conditions, small vessels with hyaline walls were occasionally to be seen lying unsupported in small patches of necrosed tissue, the appearance suggesting the origin of some of the capillary hæmorrhages.

"Changes in the nuclei were chiefly of a degenerative type, the cells being shrunken, misshapen, and homogeneously stained. Chromatolytic changes were also present in some parts. The hypoglossal nucleus was less involved than any of the others. Amylaceous bodies were fairly numerous around the periphery of the medulla."

Conclusions.

These observations upon Cheyne-Stokes' respiration confirm the results obtained in another case by Pembrey and Allen.[1] It would appear on the same grounds that the periodicity of Cheyne-Stokes' respiration is in great part due to "a diminished excitability of the nervous system associated with a defective supply of arterial blood; the carbon dioxide accumulates, and the oxygen diminishes, until at last the nerve-cells are stimulated, the waxing respirations begin and culminate in hyperpnœa, whereby a large quantity of carbon dioxide is washed out and sufficient oxygen taken in; apnœa then follows, due, apparently, to the absence of sufficient carbon dioxide to stimulate the nerve-cells.

"The inhalation of air containing more than 2 per cent. of carbon dioxide abolishes apnœa, by maintaining the partial pressure of the carbon dioxide in the alveolar air and blood at its stimulating valve.

"The administration of pure oxygen by means of a mask and valves abolishes apnœa by maintaining the partial pressure of carbon dioxide in the blood at its stimulating valve. The respiratory centre is no longer excited by lack of oxygen to send out the forcible impulses which had previously resulted in excessive ventilation of the lungs, whereby carbon dioxide had been washed out of the alveoli and blood."

Case 2. F. H—. Observations by M. S. Pembrey and A. P. Beddard.

F. H—, aged 45, was standing in a lift on January 3rd, 1906, when the wire cable broke, and he fell in the lift a distance of forty feet. Both his thighs and the fingers of his left hand were broken, but his head was apparently uninjured, and he was able to crawl out of the lift.

On admission to the Hospital he was in a dazed con-

[1] See the preceding paper.

dition, but was able to answer questions sensibly. Thirty-six hours after the accident he slowly passed into a delirious condition. His respirations were rapid, about 40 per minute, his pulse about 120, his temperature varying, but never rising above 100°. He then gradually became comatose, and, after remaining in this condition for two or three days, improved, and by January 9th was sensible at times, but often very restless, stupid, and semi-conscious. His mental condition slowly improved, and by January 13th was apparently normal.

The exact date of the onset of the Cheyne-Stokes' respiration is uncertain, but on January 12th it was well marked. Examination then showed no abnormality in the heart and lungs, no signs of fractured skull, no paralysis; his pulse was of good tension and regular; his urine was normal; his breathing was of the Cheyne-Stokes' type; a period of waxing and waning respirations was followed by a period of apnœa. During apnœa he either closes his eyes, as if he were asleep, or shows conjugate deviation of the head and eyes to the right, the eyes remaining open. He makes no attempt to answer questions. During the period of breathing he rapidly wakes up and makes a series of forcible movements, which are nearly constant in form at the corresponding stages of succeeding periods of breathing; this repetition is especially seen in the movements of the right arm and head, and the movement of the right hand over the top of the head. He slowly answers questions during this time. The following figures give the duration in seconds of several periods of apnœa and breathing.

Date.	Apnœa.	Breathing.
12/1/06. ...	15 ...	25
	15 ...	
	10 ...	25
	15 ...	25
18/1/06. ...	25 ...	17
	17 ...	21

The number of respirations in the periods of breathing varied from 10 to 20 at different times, and were not easily recorded owing to the movements of the right arm and head.

The following figures give the duration in seconds of consecutive periods of apnœa and hyperpnœa, when the patient was breathing air through a mask and valves.

Date.	Apnœa.	Breathing.		Apnœa.	Breathing.
18/1/06.	12	26	→	22	27
	16	22		8	123
	18	23		21	21
	20				

Pure oxygen was then given; the respiration in ten minutes became continuous, the rate of breathing being 14 per minute. Breathing through a mask and long tube abolished apnœa, and any disturbance of the patient tended to shorten the apnœic period.

Samples of the alveolar air of the lungs were collected, but they were obtained with difficulty owing to the forcible movements which, as already described, always occurred during the period of breathing. The patient, moreover, had a thick beard, which rendered difficult the adaptation of the mask in an air-tight manner.

		Waxing Respiration.	
Date.		Carbon dioxide. Vols. per cent.	Oxygen. Vols. per cent.
13/1/06.	2nd Expiration.	3·82	15·03.
	3rd „	3·97	15·75.
	4th „	4·13	15·75.
12/1/06.	5th „	2·63	17·99.

Samples taken on January 17th, when the patient's breathing was of the continuous type, gave results:— Carbon dioxide 3·60 and 4·29, oxygen 16·88 and 15·77 vols. per cent.

The Cheyne-Stokes' respiration was more marked during sleep; it disappeared on January 22nd, but as late as

February 18th it recurred from time to time in a well marked form, especially during sleep and after the patient had complained of a bad headache.

The blood-pressure in the brachial artery was determined on January 13th by Martin's modification of the Riva Rocci sphygmometer; for apnœa the readings were 152, 146, and 142 mm. of mercury, for the period of breathing 160 mm. It must be remembered that the latter period was accompanied by forcible movements of the arm and upper part of the body.

The exact lesion of the brain in this case is uncertain. There was no wound on the scalp, no evidence of fractured skull, and no sign of hæmorrhage on the surface of the brain. It seems likely that the cortex of the brain was bruised, and that the symptoms of cerebral irritation and compression were due to that cause. From the nature of the forced movements, it would seem likely that the frontal lobes had been contused. The question arises whether this cerebral injury had any connection with the Cheyne-Stokes' respiration, and if so, what was the nature of that connection. The symptoms of cerebral compression had passed off before the Cheyne-Stokes' respiration ceased, and this fact makes it unlikely that a gross change in the cerebral circulation was the cause of the periodic breathing. On the other hand, there is the possibility that the cerebral respiratory centre located by various observers in the cortex of the frontal lobe may have been injured, and thus brought about the periodic modifications in the excitability of the bulbar centre.

During the time of observation the patient received no drugs, such as chloral and morphia, to the action of which the periodic breathing might be attributed.

The patient, who was under the care of Mr. Lane, made a satisfactory recovery.

Conclusion.

In this case the condition of the nervous system could not be determined, and direct evidence of a defective

supply of arterial blood was wanting. The administration of pure oxygen abolished apnœa, but not so readily as in the two other cases recorded.

A preliminary account of these cases was communicated to the Physiological Society on February 24th, 1906 ("Proc. Physiol. Soc.," 'Journ. Physiol.,' vol. xxxiv, 1906).

The expenses of the investigation were defrayed from a grant from the Royal Society.

Our thanks are due to Mr. Lane for the facilities we received of observing the patient under his care.

DISCUSSION.

Dr. GOSSAGE pointed out that the waxing and waning of the respiration required some further explanation if it were due to the stimulation of the nerve-centres in the bulb by excess of CO_2 or deficiency of oxygen. Taking, for the sake of simplicity, the action of CO_2 alone, he drew attention to the following points: In the tracings of Cheyne-Stokes' respiration there were six respiratory movements recorded after the largest; these six movements successively diminishing in size. The authors had shown that the amount of CO_2 contained in the lungs reached its minimum a little later than the largest respiratory movement, say, at the second or third of the waning movements, and that after this it increased again. Theoretically, the waning respirations were due to the gradual decrease in the amount of the CO_2 occasioning a gradually decreasing stimulus to the nerve-centres, and yet three waning respirations took place while the CO_2 was increasing, and with a still further increase of CO_2 there was a complete pause. A similar phenomenon took place with waxing respiration since the maximum amount of CO_2 in the lungs occurred at about the third respiration, and yet three increasing movements up to the largest took place with a diminishing amount of CO_2. It had, of course, to be remembered that the condition of the blood in the lungs did not correspond with that in the medulla, and that it was the blood in the medulla that stimulated the centre. A certain lapse of time was required for the blood to pass from the lungs through the left side of the heart to the medulla. The real question was whether this time was sufficiently long to allow of at least three respiratory move-

ments. The minimum content of CO_2 in the medulla should correspond to the early part of the respiratory pause, the maximum content of CO_2 in the medulla to the largest respiratory movements.

Dr. A. E. Garrod asked for an explanation of the waxing and waning of the respiration. Why, for example, the dyspnœic phase did not begin suddenly?

Professor Cushny said that he had recently received from Dr. J. Mackenzie, of Burnley, several tracings taken in cases of Cheyne-Stokes' respiration. In one of these in which the pulse was also recorded there was no slowing of the heart during the apnœa, and the blood-pressure seemed to be lower than during the hyperpnœa, indicating a depressed condition of both the inhibitory cardiac and vaso-constrictor centres. The other tracing was of special interest in that the patient suffered from hiccough, and this persisted during the apnœa. The respiratory centre is involved in hiccough, and the persistence of this spasmodic movement during the pauses of the breathing seemed explicable on the view suggested by Dr. Taylor to account for the reappearance of normal respiration in his case when pleurisy supervened—namely, that while the respiratory centre was unable to respond to the normal chemical impulse, it could do so when excited by abnormal excitation reaching it by the nervous channels.

Dr. Bezly Thorne asked whether, from what had been said, it was to be inferred that lesions of vessels and parenchyma of the bulbar centres are a condition essential to Cheyne-Stokes' breathing, and whether any information could be given as to the therapeutic value of inhaled oxygen in that condition.

Sir W. H. Broadbent (*who was unfortunately unable to be present owing to illness, sent the following note*).—The departure from normal regular breathing, which has received the name of Cheyne-Stokes' respiration, has interested me from the earliest period of my professional career. I was very soon led to associate it with high arterial tension, and I have found no reason to depart from this conclusion. I have never seen a case of Cheyne-Stokes' respiration in which the arterial tension was not high, and there is nothing in the cases related and commented upon by Dr. Frederick Taylor and those associated with him in the investigation which conflicts with it, or is inconsistent with this view. When the blood-pressure has been estimated it has been high, and even in the case of aortic incompetence cited where the mean pressure was low, the initial pressure, the pressure required to overcome the resistance in the capillaries, which is the basis and criterion of high arterial tension, was high.

High arterial tension is a cardio-vascular phenomenon, and is dependent upon the presence in the blood of some constituent which provokes resistance in the capillaries and arterioles.

From this point of view, the fact brought out in their experiments that the Cheyne-Stokes' rhythm is at once replaced by regular breathing on the inhalation of either oxygen or carbon dioxide, is of extreme interest. It is the more significant that oxygen and carbonic acid are exactly opposed in their relations with respiration. It is necessary, then, to go further in search of an explanation of Cheyne-Stokes' breathing than some change in the blood, and to postulate some morbid condition of the nervous system other than such as would be brought about by an abnormal blood supply?

As regards the changes found by Dr. Farquhar Buzzard in the medulla pons they are exactly such as are produced by high arterial tension and the degeneration of the minute arterioles to which this gives rise. They cannot be necessary, since there may be complete and permanent recovery from Cheyne-Stokes' breathing. This is, perhaps, most frequent in acute tubular nephritis, but it may be suspended indefinitely in chronic renal disease by treatment which promotes elimination, and it is suspended when any complication attended with pyrexia supervenes, such as pneumonia or pleurisy when, of course, the arterial tension is relaxed. It may be present in apparent health. I have known a man go to the City regularly for six months who had Cheyne-Stokes' breathing whenever I saw him. No doubt conditions of the nervous system favour or resist the liability to Cheyne-Stokes' respiration. I have known it begin with an attack of hemipleglia and cease as the immediate effects of the lesion (hæmorrhage) subsided.

I have always hesitated to call Cheyne-Stokes' respiration dyspnœa. The patient, except in advanced stages, is not conscious of any difficulty in breathing, and it is very rarely that there is cyanosis. It is astonishing how often it is overlooked. The suspended respiration seen in infants during sleep does not seem to me to correspond with true Cheyne-Stokes' breathing when I have watched if respiration has been recurred suddenly. Such has been the case also whenever I have had the opportunity of observing the interrupted breathing of old people during sleep. The irregular respiration of cerebral disease or injury has sometimes been erroneously called Cheyne-Stokes.

Dr. PEMBREY, in reply, ascribed the delay in the response to the slow diminution of CO_2, the respiration being so shallow that the effect produced was slow. The respiratory centre might be acted on by other factors than changes in the blood. Cheyne-Stokes' respiration had no essential pathological significance seeing that it occurred in infants and in hibernating animals.

HUMAN AND BOVINE TUBERCULOSIS

WITH SPECIAL REFERENCE TO TREATMENT BY SPECIAL KINDS OF TUBERCULIN

BY

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AT the International Congress on Tuberculosis held in London in 1901, Professor Robert Koch made the following declaration: "That (1) tuberculosis of man and the cow were different, and that tuberculosis of the cow could not be conveyed to man; (2) that the regulations concerning milk, butter, and meat, made against tuberculosis of animals and its transmission to mankind were not necessary."

During the last six years I have devoted as much time as possible in collecting facts and making research into this difficult problem, with a view to completely controverting the second part of Koch's statement. The absolute proof can never be finally given, for the simple reason that deliberate experiment from animal to man can never be permitted. Hence it is, we must work by round about methods, making clinical observation our basis, and relying on inferences from analogy, pathological research, and minute and careful comparisons between the bacilli of various origins for our ultimate conclusions.

The position of this interesting, scientific problem at the

present time is briefly as follows: Koch holds that human and bovine tuberculosis are separate and distinct diseases, and that bovine tuberculosis, if conveyed to man, cannot set up human tuberculosis.

Koch, in addition, believes that phthisis is conveyed from one person to another by direct infection, especially from contact with an advanced case of the disease, and that this is by far the commonest cause of the spread of phthisis pulmonalis.

Von Behring, on the other hand, holds diametrically opposite views. He says that human and bovine tuberculosis are the same disease, and that nearly all tuberculosis is the result of infection during infancy by means of infected milk, and that direct infection from person to person is not proved.

During the last ten years I have had under observation over 4000 cases of phthisis pulmonalis, and over 1500 cases of what are called surgical tuberculosis. In addition to having had the opportunity of observing this large number of cases in the wards of a hospital, I have made over 800 post-mortem examinations on tubercular cases, the great majority of which were very carefully and minutely examined. In addition to this hospital work, for the last four years I have been conducting a special research into the distribution of tuberculosis in Liverpool, and have made a large number of experimental and other observations in the Pathological Department of the University of Liverpool.

The general results and impressions of that work are given in this paper, but in the short space of time at my disposal it is impossible to do more than touch generally on a few of the more important problems opened up by this research. I would like to follow up the lines of my original paper of 1903, since I have no reason to modify the view then set forth—namely, that human and bovine bacilli are divisible into two distinct types of a common species: first, *Typus humanus*, and secondly, *Typus bovinus*.

During the last four years I have made a very large number of pure cultures of tubercle bacilli from various sources—human, bovine, and avian—and after growing a very large number one is bound to admit the distinctive cultural differences on the same medium between human and bovine bacilli. All are agreed on the distinctive characteristics of avian bacilli. I will show you several pure cultures from both human and bovine sources, and will leave you to form your own conclusions.

Ravenel affirms "that the tubercle bacillus from bovine sources has in culture fairly constant and persistent peculiarities of growth and morphology by which it may be differentiated from that ordinarily found in man." Long-continued residence in a particular host has resulted in the bacilli assuming characteristics which serve to distinguish it from others, and the fact that tubercle bacilli have lived for thousands of years in human lungs would be ample evidence for assuming a difference between bacilli which had lived for centuries in cattle.

Another fact of the most profound importance in studying this problem is that for centuries man has been accustomed to feed upon cattle and their products —milk, butter, cheese, etc., and in this way the human body has become tolerant to bovine tubercle bacilli. Whilst I firmly believe that human and bovine bacilli are different types of parasites, yet I am convinced that bovine bacilli are freely communicable to humans, and are the cause of a large amount of tuberculosis in children.

Two Royal Commissions were appointed to investigate this question. The work of the Imperial German Commission was purely experimental, portions of tuberculosis material being used from different parts of the body with special reference to organs of children affected by tuberculosis. The Report is unanimously in favour of Koch's contention that the bacilli from human and bovine sources have distinct morphological and patho-

logical characteristics which are sufficient for their easy differentiation. In addition, the German Commission has clearly proved that bovine tuberculosis is conveyed to children, and also particularly emphasised the fact that tuberculosis is often primarily from the intestines and mesenteric glands. From three cases of miliary tuberculosis in children they found the disease to have been introduced through the intestines, and in one case the infection was bovine from milk.

My own clinical and experimental work is in entire agreement with these results excepting with regard to the amount of bovine tuberculosis conveyed to humans, which I consider is considerable.

I believe that man is attacked by two distinct varieties of tubercle : one conveyed by infection from one person to another, the other by receiving into the body bovine bacilli from infected food. In other words, the human body is susceptible to both forms of tubercle. The difficult problem to determine is—what particular lesions in the human body it is each variety of tubercle produces. With a view to settling this question of the relationship between human and bovine tubercle bacilli, Professor von Dungern and Dr. Henry Smidt have conducted a large series of experiments on the anthropoid apes. They point out that in all experimental animals the bovine bacillus has exhibited in a more or less marked degree a higher pathogenicity than the human bacillus. This has been found to be the case for bovines, goats, pigs, rabbits, dogs, cats, guinea-pigs, and monkeys. Why should man be assumed to differ in this respect from all the experimental animals hitherto investigated? The general conclusions at which they arrived are : that the gibbon is equally susceptible to infection with both types of bacilli, and owing to the near relationship of this animal to man it seems probable that a similar susceptibility to both varieties of tubercle bacillus may be attributed to man. There was, however, in their feeding experiments, a difference in the distribution of the lesions, to which

they called attention. In both animals infected with bovine bacilli the small intestine showed ulcers and the mesenteric glands were greatly enlarged and caseous; whereas in the two gibbons fed with human bacilli no tuberculous lesions could be found, either in the intestine or in the mesenteric glands. The primary lesions were in the lungs.

Phthisis Pulmonalis.

After observing over 4000 cases of phthisis pulmonalis, I have been particularly impressed with the fact that the tuberculous process is nearly always strictly confined to the lungs. The patient usually gives a history, first of cough, then of expectoration. The disease after progresses to death, and the necropsy reveals destructive lesions in the lungs and nothing more. In a fair percentage of cases the intestines show secondary tuberculous ulcers, but beyond this by far the great majority of cases of phthisis show no other tubercular lesion. Phthisis pulmonalis is essentially a disease of young adult life, the great majority of deaths taking place between the ages of thirty and forty. On the other hand, strumous or tuberculous joints, enlarged glands, spinal disease, and abdominal tuberculosis with tabes mesenterica are essentially diseases of infancy and childhood, and are only rarely seen in adult life. I have given particular attention to the clinical manifestations of these various tuberculous affections, and it is rare to see a patient with enlarged glands, strumous joints, or spinal disease develop true phthisis pulmonalis; in fact, clinically, they appear to be antagonistic to each other, attacking the body at quite different periods of life and exhibiting generally opposite symptoms.

At the present time I have 140 cases of phthisis pulmonalis in the hospital, and in no case is there any other gross tuberculous lesion evident, which would certainly be the case if they were caused by one identical

bacillus, which would be free to be conveyed all over the body.

As a primary disease phthisis seems to originate in the lungs as a result of inhalation of infecting tuberculous matter, and secondarily, to cause tuberculous ulceration of the intestine by swallowed sputum.

Other forms of tuberculosis, such as enlarged lymphatic glands in the neck, tuberculous peritonitis, tuberculous joints, and probably tuberculous meningitis and lupus, are most likely caused by bovine bacilli being absorbed from the digestive tract in milk and other food, such as butter, butter-milk, cheese, sausage, etc.

Enlarged glands in the neck are, in my judgment, a grave danger. In a great many instances the infection from the fauces and tonsils by infected milk is limited by the glands, which either suppurate or are removed by the surgeon.

If the infection is allowed to spread downwards the apex of the lung may be directly attacked, and an extensive bovine infection of the lungs may result. I have seen several instances where the glands in the neck have slowly been allowed to progress until the pleura was reached, the result being a general bovine phthisis. For this reason it is of the highest importance that all tuberculous glands should be excised as quickly as they appear. From my own observation I would say that human tubercle bacilli do *not* attack the ordinary lymphatic glands of the body. I have just completed a series of 140 carefully conducted autopsies on children dying of tuberculosis—many of them ordinary phthisis and several of them abdominal and meningeal tuberculosis. Of this number forty-seven showed tuberculous intestinal ulcers with caseating mesenteric glands, and twenty-nine showed caseating mesenteric glands with intestinal ulcers. From the nature of the pulmonary lesions I am inclined to think that the tuberculous ulcers were caused by human bacilli, and that in those cases where the mesenteric glands were infected without ulceration of the intestine the bacilli

were of bovine origin. In short, human bacilli, whether primarily swallowed or as a secondary infection in phthisis, will cause intestinal ulceration, whilst bovine bacilli, ingested with milk, etc., will readily pass from the intestine, leaving no trace behind, attack the mesenteric glands, and thence spread to the lungs and all over the body. It is possible that this may be a means in future of distinguishing the origin of an infection.

I have instituted inquiries in a great many foreign countries with a view to noting the distribution of phthisis pulmonalis and the other forms of tuberculosis, the results of which have been most interesting, and tend generally to show that in those countries where tuberculous cattle are absent or where milk is not drunk by children, the surgical forms of tuberculosis are not observed, whilst phthisis pulmonalis is rife. One letter of great interest is from Siam, and is from the Government medical officer. He states "that during eight years' practice in Bangkok I do not remember having ever seen, or ever having heard of any case of strumous joints, lupus or tabes mesenterica, or, in fact, of tuberculous affections of such nature in children. I have often spoken of this to my colleagues, and could formulate no reason until I read your paper. The Siamese practically never drink cow's milk, either children or adults. Ordinary pulmonary tuberculosis, however, in adults is very rife in Bangkok. The same seems to hold good for China." I have received similar confirmation from Jersey, Guernsey, Egypt, Malay, India, Persia, and North America. When the inquiry is complete I will publish it.

A very valuable contribution to this question has just been made by Dr. Mayo, of America. He says, speaking of abdominal tuberculosis: "Practising, as my brother and I do, in an agricultural community we have long noticed the relative infrequency of pulmonary tuberculosis, while abdominal, bone, joints, and lymphatic disease of tuberculous origin are common. One cannot help believing that much of the surgical tuberculosis had to do with

uncooked milk, which is a common article of diet in this country."

I have also been particularly impressed with the fact that children who suffer from the surgical forms of tuberculosis—such as tubercular glands, joint disease, and lupus—very rarely indeed are attacked, even in the later stages of their malady, by phthisis pulmonalis. The lungs may be affected in the course of a general or miliary tuberculosis, but it is rare to see phthisis pulmonalis associated with surgical tuberculosis. The cause of death in the great majority of these cases is exhaustion and waxy degeneration, so that I have come to the conclusion that these two varieties of tubercle are antagonistic to each other in the human body, and that children who have suffered from bovine tuberculosis in the form of strumous glands, spinal caries, strumous joints, and lupus are immune against phthisis pulmonalis. Very much the same result is produced by vaccination against smallpox.

This theory of mine has also received great support by the work of von Behring and Römer. The only strain of tubercle bacilli which they find produces complete immunity in cattle is the human strain, and by using human bacilli they have been able to immunise thousands of cattle against bovine tuberculosis, not one of which has contracted tubercle although repeatedly exposed to infection by living in the same stalls with infected cattle. If human bacilli will protect cattle, it is natural to suppose the converse, that bovine tubercle in children will protect them against human tubercle or phthisis.

To sum up my views on the distribution of tubercle in the human body, I would say, speaking provisionally, and without any final evidence of proof, that—

(1) Tubercle bacilli of the typus humanus produces phthisis pulmonalis, ulceration of intestines, and abdominal lesions.

(2) Tubercle bacilli of the typus bovinus produces tubercular peritonitis, tuberculosis of the lymphatic glands,

tubercular bones and joints, tubercular meningitis (probably), and lupus.

I am of opinion also that acute miliary tuberculosis is of bovine origin.

Working on this hypothesis that the human body is attacked by two varieties of tubercle which may be present in the body at the same time, and which, generally speaking, are antagonistic to each other, I have devoted my attention within the last few months to the preparation of tuberculin for the treatment of these different lesions. After a fairly complete experience, I have come to the conclusion that Koch's Tuberculin ℞ has little or no healing effect in phthisis pulmonalis, and when we remember that it is manufactured from human tubercle, if my theory is correct, it is exactly what we should expect. For this reason I am at present treating over seventy cases of surgical tuberculosis in the wards of this infirmary with Koch's Tuberculin ℞, commencing with very small doses and slowly increasing up to a maximum dose of 1 milligramme. The results, without any other accessory treatment, have been beyond all my anticipation. Enlarged glands, joints, and lupus have been immensely improved, whilst discharging sinuses have cleared up, and, in one case, the symptoms of tubercular meningitis associated with tubercular peritonitis entirely disappeared. The full results of this large number of cases treated by tuberculin will be published in due course.

With the same end in view I have had prepared from one of my own pure cultures from bovine source a special tuberculin for the treatment of phthisis pulmonalis. The tuberculin was made from a typical culture of Perlsucht, very carefully sterilised and standardised. Several guinea-pigs were inoculated, but without any bad effect. Up to the present time I have treated six cases of early phthisis pulmonalis, four of which were associated with hæmoptysis, and all of which showed tubercle bacilli in the sputum with distinctive physical signs at one apex. These cases are still under treatment, but up to the present

all of them have shown marked improvement with total disappearance of physical signs. It is too soon yet to speak of the final results of treatment, but I hope to publish them after one year. This tuberculin was prepared from a pure culture kindly supplied to me by Professor Calmette, of Lille.

With a view to producing immunity against tubercle in children, especially in those who have been exposed to infection from a consumptive father or mother, I have lately been working with the serum of tuberculous cattle. I have purchased several dairy cows suffering from tuberculosis of the udder and have obtained, with the kind co-operation of Professor Aunett, a large amount of the serum of these cows. This serum has been very carefully sterilised and injected into guinea-pigs without any ill effects. I believe that the serum of a cow which has suffered from bovine tuberculosis will confer sufficient immunity when injected into a child as will protect it against an attack of human tuberculosis.

Considering the large number of children who are attacked by phthisis pulmonalis as the direct result of contact with a consumptive parent, a protective serum would be an immense step in the direction of stamping out consumption. In any case the serum is quite harmless and will only be used with the full consent of those parents whose children have been exposed to infection.

Conclusions.

(1) Human and bovine tuberculosis are different varieties of a common species.

(2) The human body is susceptible to both forms.

(3) Bovine tuberculosis is frequently conveyed to humans, both by means of infected food and by direct infection.

(4) These two forms of tubercle are antagonistic to each other.

(5) A mild attack of bovine tuberculosis protects against phthisis pulmonalis.

(6) Tuberculin from human sources has a marked curative effect on bovine lesions.

(7) Tuberculin from bovine sources may have a curative effect on phthisis pulmonalis.

Note.—Since the foregoing paper was printed the Report of the Royal Commission on "Human and Bovine Tuberculosis" has been issued. Their findings entirely corroborate the views set forth in my paper, and emphasise the danger of infection with bovine bacilli, especially to children, through the agency of milk.

DISCUSSION

Dr. DAVID LAWSON said: Dr. Raw has placed before us opinions and conclusions founded upon an inquiry which has extended over a period of ten years. These opinions are, therefore, entitled to most careful consideration. And for one to offer a serious criticism, without having had an opportunity to think carefully over them, would be mere presumption. With that in view one ventures to submit the following opinions which may be considered, not in antagonism, but in connection with facts which we have had so clearly and ably placed before us by Dr. Raw. And the first point which occurs to one is the fact that my clinical experience of pulmonary form of tuberculosis and its relationship to their other forms—called by Dr. Raw surgical forms of the disease—harmonises with those of the writer of the paper. Thus, in 800 consecutive cases of lung tuberculosis I have seen only one case of lupus associated with the chest form. Glandular disease along with lung disease has been rare, and only in three instances has bone disease accompanied phthisis. Meningitis or tubercular cornea as a complication or termination to pulmonary phthisis I have never seen. Indeed, the association of those forms of tubercular disease with phthisis has not been greater than can be reasonably explained on the assumption that a double infection by perfectly different types of organisms has occurred. To this extent my experience drawn from clinical observation seems to support Dr. Raw's views. On the other hand there are certain clinical facts which seem to point in another direction. Firstly Dr. Raw separates entirely phthisis pulmonalis from miliary tubercle. Now, not

infrequently one has observed miliary tuberculosis terminate a case of pulmonary phthisis. We have been accustomed to believe that this phenomenon, when it occurred, was due to the passage of tubercle bacilli from the lung into the blood-stream and their conveyance to distant organs where they at once set up disease in a discrete form. Dr. Raw offers us another explanation when he says such a person has become the subject of a fresh infection, and the life in this case is terminated by the operations of an altogether different organism than that from whose action the host has suffered during, it may be, years of previous disease. Such a proposition seems so improbable that I feel we must have further evidence placed before us before we are justified in discarding our old views and accepting the new ones. Again, Dr. Raw points out that the result of inoculating T.R. tuberculin in phthisis is *nil*, whilst that of inoculating it in surgical forms of the disease is excellent, and quotes this fact in support of his views. Working, as I have been now, for over two years with inoculations of T.R. in lung cases, guided by Wright's methods, I cannot admit the accuracy of the statement. For my part, whilst recognising that the good achieved by the inoculation of T.R. in lung cases is not by any means so striking or so reliable as we had hoped for, or as is frequently to be seen in the surgical cases, still, I am perfectly convinced that in a great many instances it exerts a most beneficent action and materially assists the curative process in the lung lesion. Dr. Raw has placed a most thoughtful and valuable contribution before the fellows of this Society and I wish to express the opinion that by doing so he has placed the Society under a deep obligation.

Mr. J. P. Lord spoke as a medical officer of health in a district, the only one in England, where compulsory examination of milk for the tubercle bacillus was made. Milk now found to be contaminated with the *Typus humanus* was assumed to have been contaminated after it had left the cow. Was there any method of distinguishing whether a tuberculous deposit in milk was human or bovine?

Dr. G. A. Crace-Calvert: After listening to the very able paper that Dr. Raw has just read, and remembering his very large clinical experience in Liverpool, one is naturally rather diffident about taking a view which is not in exact accord with that expressed to-night. I have, however, carefully read the report just issued by the Royal Commission on Tuberculosis, and, so far as I can follow their numerous experiments and conclusions, it seems to me that they are in favour of the theory that the two types (human and bovine) of tuberculosis are identical, or at any rate simply varieties of the same disease. They divide the cases of human tuberculosis that they have examined into three groups. Now whilst one of these groups

has a bacillus which closely resembles—if it is not absolutely identical with—the bacillus of bovine tuberculosis, yet one of the others has a bacillus with different cultural and other properties. But the important point is that by certain "passage" experiments through calves they have been able to make the bacilli of one group pass into those of the other. If this be confirmed, it seems to me to point to the identity of the two groups, or at any rate to their common origin from which (under certain conditions which we do not at present understand) the one or the other variety may develop. Again, Dr. Raw mentioned the effects of inoculation of tuberculin, and suggested that it would be best to use a bovine tuberculin for human tuberculosis and *vice versâ*. He quoted some cases of early pulmonary tuberculosis which had improved after inoculations of bovine tuberculin, but I do not think that this is conclusive, because in my experience most early cases improve readily with better diet and open air. I have used tuberculin (Koch's T.R.) in advanced cases, after sanatorium treatment had ceased to benefit them, and the results seem to me to point to the identity of the two classes of disease. I have employed small doses of tuberculin (T.R.)—which is prepared from human bacilli—in cases which Dr. Raw puts in different groups, and the results have been equally good in each class. For instance, a case of advanced pulmonary disease (human type, Raw) had eight inoculations, a case of tubercular mesenteric glands (bovine type, Raw), which followed on early pulmonary disease, had six inoculations, and a case of tubercular cervical glands (bovine type, Raw) had eight inoculations. Now all these improved considerably with human tuberculin, apparently showing that the cases are only varieties of one disease. Again, the Royal Commission found that cases of tubercular cervical glands were caused by bacilli of each of the varieties that they found, a fact which seems to point to the bacilli being simply varieties of the one micro-organism.

Dr. TOOTH: The questions already asked Dr. Raw are so numerous and important that I hesitate to add to them. Nevertheless I should like to ask two more, of a practical nature. First, as to the method of inoculation. Does Dr. Raw satisfy himself as to the correct time to perform inoculation by the observation of the opsonic index, as urged by Professor Sir A. E. Wright, or has he some less intricate clinical procedure as his guide? This is a question which must interest all practical physicians. We should also, I am sure, be interested to know where the bovine tuberculin can be procured. I take this opportunity of expressing my appreciation of the admirable paper that we have listened to this evening.

Dr. F. M. SANDWITH: Like many others, I am fresh from reading the second interim Report of the Tuberculosis Commission, and we are all indebted to Dr. Raw for many of his

interesting suggestions. I quite agree that the combination of lupus with phthisis pulmonalis is hardly ever seen, but I cannot agree that post-mortem examinations on cases of phthisis in this country do not reveal tuberculous meningitis. In Egypt meningitis is seldom or never seen in the post-mortem room, though every other form of tuberculosis mentioned by the reader of the paper is quite common, excepting always tubercular ulceration of the cornea, which, so far as I know, is extremely rare in any country. We began using the original tuberculin in Cairo directly it appeared, and I had the advantage of showing all our original cases to Professor Koch and hearing his views on the matter. We gave up using it in hospitals when we found bad results from cases of surgical tuberculosis. I have seen several cases of pulmonary tuberculosis following tuberculous glands of the neck. The investigation of the disease in Egypt presents some interesting factors, for bovine tuberculosis is very rare there, whereas it is so common in this country where no means are taken to stamp it out. Moreover, Egyptian mothers are able and willing to suckle their children, and sometimes continue to do so for so long a period as two years, and again, when cow's milk is given to the native children it is practically always boiled, because of the difficulty of keeping milk in a hot country from becoming sour. It is therefore difficult to believe that the tuberculosis from which young Egyptians suffer is chiefly of bovine origin.

The PRESIDENT, after expressing his sense of the great value of the paper, said: I am in entire agreement with Dr. Raw as to the infrequency of the association of surgical tuberculosis with pulmonary phthisis. In a large experience, both clinical and pathological, of the joint diseases of children, I have observed that while such conditions are often associated with glandular disease and affections of the skin, pulmonary tubercle is very seldom found among them, and that when death occurs in these cases it is usually due either to albuminoid disease of the viscera, or exhaustion from long-continued suppuration and pain. Many years ago, when upon the staff of the Hospital for Sick Children, I published a series of cases of joint and bone disease, which showed how rarely they were associated with pulmonary tuberculosis. On the other hand, tubercular meningitis is extremely common among young children. Some fear has been expressed by a previous speaker lest Dr. Raw's teaching as to the dangers attending the consumption of infected milk should lead to the disuse of that valuable article of diet; but I should have no fear concerning the diffusion of such carefully acquired knowledge, and if the facts and reasoning adduced by Dr. Raw should only frighten people into boiling their milk, his paper will have done a great good.

Dr. RAW, in reply, said that among 800 necropsies in phthisis

he had never seen any distinct infection of the meninges, arguing that it was a bovine disease. In the sanatoria abroad there were thousands of cases of phthisis without any other tuberculosis. He distinguished between acute general tuberculosis and acute miliary tuberculosis. Secondary ulceration of the intestines was human, the primary intestinal disease was bovine. Culturally the two types of tubercle bacilli could be distinguished. Bacilli in milk were probably bovine; in London about 25 per cent. of the dairy cattle were tuberculous. The fact that tuberculin would cause a reaction, whatever the source of the tuberculosis, did not necessarily mean that all forms of tuberculosis were the same. He had found the opsonic work impracticable. Bovine tuberculin was made on the Continent from private cultures. Milk was a dangerous food if its source were unknown. All tuberculous dairy cattle should be eliminated.

OBSERVATIONS ON A CASE OF CHYLURIA OCCURRING IN ENGLAND,

WITH A REPORT OF THE POST-MORTEM EXAMINATION

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Cases of chyluria are exceedingly rare in England and other temperate countries. In the last nineteen years there has only been a single case in the medical wards of Guy's Hospital, and I have been able to find in the literature only forty-three other cases which were supposed to have originated in Europe. I was, therefore, glad to avail myself of the permission kindly given me by Dr. Newton Pitt to investigate a case of chyluria which was in Guy's Hospital under his care in October, November, and December of 1906.

The patient was a sailor, 66 years old. He was thus older than any of the previously recorded cases with the exception of Byrom Bramwell's (27), who was also sixty-

six, the ages of the others varying between thirteen and fifty-seven. His last voyage to the tropics was in 1886, when he spent a fortnight in India, during which he had a slight attack of "fever."

Since that time he had enjoyed good health, but for the last four years had been troubled with some dull pain in the loins. In March, 1906, he first passed milky urine. He continued to do this without intermission until his admission into Guy's Hospital in October. His appetite remained good, but it was not excessive. In June, 1906, he weighed 10 st. 10 lbs.; in November he weighed only 9 st. In spite of this he remained fairly active and vigorous.

His symptoms were typical of those generally observed in cases of chyluria. The aching pain in the loins and loss of flesh have been noticed in most cases. The majority have shown more marked weakness than this patient, but several have lived for many years without becoming completely incapacitated.

Prout (1) in 1841 noted that the disease occurs much more frequently in the tropics than in Europe. Nearly fifty years later the explanation of this was shown by Wucherer and Lewis to be the frequent association of chyluria with infection by filariæ.

Among the forty-two cases described as European, which I have collected (Table I), eight had been in some tropical country at periods varying between ten and thirty-six years before the onset of the disease. Of the remaining thirty-four patients twenty are specifically stated to have never been abroad. These statistics suggest that a number of the cases, including the present one, which apparently originated in Europe, may have been due to infection with filariæ during residence in some tropical country.

In ten of the cases, described since Lewis' discovery in 1872, filariæ were looked for but not found, though one had lived in Algeria twenty-six years before the onset of

TABLE I.—*Cases of non-tropical chyluria.*

No.	Author.	Reference.	Sex.	Age at onset.	How long before onset was patient in the tropics, and where?	Filaria in blood or urine.	Remarks.
1	Prout[1] . . .	Stomach and Renal Diseases, 3rd edit., 1841	—	—	—	—	—
2	Golding-Bird.	Lond. Med. Gaz., xxxiii, p. 110, 1843	F.	35	—	—	—
3	Bence Jones . .	Med.-Chir. Trans., xxxiii, 1850, and xxxvi, 1853	M.	32	Trinidad, 22 years	—	Slightly diminished by abdominal belt. Much improved by gallic acid 5j a day.
4	Beale . . .	Urine and Urinary Deposits, p. 269, 1852	F.	50	Never	—	—
5	Elliotson . .	Med. Times and Gaz., 1857, ii, p. 288	F.	38	India, 10 years	—	—
6	Begbie . . .	Edin. Med. Journ., viii, p. 132, 1862	M.	21	India, 17 years	—	—
7	Ackerman . .	Deutsch. Klinik., 1863, p. 221	M.	40	Brazil, 18 years	—	—
8	Eggel . . .	Deutsch. Arch. f. klin. Med., vi, p. 421, 1869	F.	57	Brazil, 13 years	—	—
9	Oehme . . .	Ibid., xiv, p. 262, 1874	M.	50	Never	—	Died. Carcinoma of stomach.
10	Pavy . . .	Personal communication. (Under observation in 1876)	F.	35	„	—	Disappeared during pregnancy.
11	Glasier . .	Lancet, 1877, i, p. 909	M.	57	„	—	—
12	Dickinson and Ord	Path. Soc. Trans., xxix, pp. 391 and 402, 1878	F.	20	„	None	Diminished by pressure over lumbo-sacral promontory; apparently also by intravesical injection of $FeCl_3$.

[1] Prout records fourteen cases, eight of which were natives of hot climates, or had lived for many years in the tropics; he does not say whether any of the remaining six had been for shorter visits to tropical countries, but one of them appears to be the same case as Elliotson's (No. 5).

TABLE I.—*Cases of non-tropical chyluria*—continued.

No.	Author.	Reference.	Sex.	Age at onset.	How long before onset was patient in the tropics, and where?	Filaria in blood or urine.	Remarks.
13	Morrison	Ibid., xxix, p. 394, 1878	F.	56	Algeria, 26 years	None	—
14	Brieger	Charité-Annalen, vii, p. 257, 1882	M.	23	Never	—	—
15	Siegmund	Berl. klin. Woch., 1884, p. 150	M.	45	,,	—	—
16	Roberts	Urinary and Renal Diseases, p. 363, 1885	M.	45	,,	—	Died. Pulmonary tuberculosis, dilated lymphatics in abdominal wall.
			F.	35	,,	—	—
17	Senator	Charité-Annalen, x, p. 307, 1885	M.	46	Various parts of America, but probably in no filarial district, 13 years	None	Chylous ascites.
18	Francotti	Ann. de la Soc. Méd.-Chir. de Liège, 1886. Quoted by Saundby, *loc. cit. infra*	F.	Young	Never	—	Appeared at each pregnancy, absent in intervals.
19	Huber	Virch. Arch., cvi, p. 126, 1886	M.	48	—	—	—
20	Lachowicz	Rozprawz der Akad. der Wissensch in Krakan. Quoted by Franz and Stejskal	F.	54	Never	—	—
21	Newton Pitt	Unpublished (in Guy's Hospital, 1887)	M.	51	,,	None	Died. Granular kidney and tubal nephritis.
22	Goetze	Die Chylurie, Jena, 1887	F.	21	,,	—	Well-compensated mitral insufficiency.
23	Berri	Speriment. Firenze, viii, 114, 1890. Quoted by Franz and Stejskal	F.	—	—	—	Began in 8th month of pregnancy, disappearing immediately after parturition.
24	Myers	Brit. Med. Journ., 1890, ii, p. 627	F.	27	Never out of U.S.A.	None	Dilated lymphatics in skin of left thigh.

TABLE I.—*Cases of non-tropical chyluria*—continued.

No.	Author.	Reference.	Sex.	Age at onset.	How long before onset was patient in the tropics, and where?	Filaria in blood or urine.	Remarks.
25	Moscato . .	1895. Quoted by Predtetschensky, *loc. cit. infra.*	—	—	—	*Eustrongylus gigas* in urine	—
26	Daggett. . .	Brit. Med. Journ., 1896, ii, p. 1706	M.	49	Never	—	—
27	Bryom Bramwell .	Ibid., 1897, ii, p. 261	F.	66	Mauritius, 36 years	None	—
28	Phillips . . .	Ibid., 1898, ii, p. 1431	M.	13	—	„	—
29	Vieillard . .	Journ. de Pharm. et de Chemie, p. 53, Paris, 1899. Quoted by Franz and Stejskal	F	40	—	—	Began after last pregnancy; lasted 20 years.
30	Predtetschensky .	Zeits. f. klin. Med., xl, p. 84, 1900	F.	16	Never out of Russia	Ova of *Tænia nana* in urine	—
31	Saundby . .	Lectures on Renal and Urinary Diseases, p. 202, 1900	F.	Young	—	—	Pregnant, *vide* footnote, p. 139.
32	Waldvogel and Bickel	Deutsch. Arch. f. klin. Med., lxxiv, p. 511, 1902	F.	39	Never	None	—
33	Franz and Stejskal	Zeits. f. Heilkunde, xxiii, p. 441, 1902	M.	23	„	—	—
34	Gabbi . . .	Policlinico, xi, M., p. 389, 1904	F.	34	—	—	Improved by urotropine.
35	Giordono . .	Riforma Medica, xxi, p. 1297, 1905	M.	21	Never	None	Tuberculous testis.
36	Port . . .	Zeits. f. klin. Med., lix, p. 455, 1906	M.	63	„	„	Died. Tuberculous mediastinal glands.
37	Theilemann .	Inaugural Dissertation, Halle, 1906	M.	32	Never	—	—
38	Hertz . . .	—	M.	66	India, 20 years	None	Died. Obstruction thoracic duct, purulent pericarditis.

the disease, and another in Mauritius thirty-six years before.

I made a very careful search for filariæ, both in the blood and the urine of this patient, with a negative result.

I also employed the method introduced by Stäubli,[1] by means of which he succeeded in finding trichina embryos in the blood for the first time. I drew 10 c.c. of blood from a vein by means of a syringe, and at once mixed the blood with 100 c.c. of 0·3 per cent. acetic acid, which dissolves red corpuscles, but has no injurious effects on leucocytes or parasites. On centrifugalising and preparing films from the deposit microscopical examination showed a field filled with leucocytes only. No parasites were found either in blood taken during the day or at 12.30 a.m.

A differential blood count showed the absence of eosinophilia—additional evidence that no living filariæ were present in the patient's body.

Physical and chemical properties of the chylous urine.—Golding-Bird was the first to point out that the fat of chylous urine is not present in the form of oil globules, but as exceedingly minute "molecular" particles, resembling the "molecular basis" of chyle. A similar condition was present in this case, thus distinguishing it from the lipuria occurring in renal disease and after fractures, in which obvious fat droplets are present.

Leucocytes were invariably present, as Morrison and most later observers have also recorded. Morrison found, in addition, a few red blood-corpuscles, which were sometimes present in Gabbi's (34) case in sufficient numbers to give the urine a pink colour.[2] I was never able to find any in this case. A small number of epithelial cells were generally present in the centrifugalised deposit.

A few fibrin shreds were always present, though whilst the patient was in the hospital his urine never

[1] 'Deutsch. Arch. f. klin. Med.,' lxxxv, 286, 1905.

[2] Thus the presence of blood is not confined to tropical cases, as some writers have maintained.

clotted into a gelatinous mass like blanc-mange—an event which he described as having sometimes occurred before his admission. The fibrin shreds were never large enough to cause any difficulty on micturition whilst the patient was under observation, but on several occasions before he came to the hospital intravesical coagulation appears to have occurred, a considerable effort having been required to get rid of the clots present in the bladder.

No casts were found; this is in agreement with the observations of all previous writers on the subject.

On standing no definite creamy layer formed on the surface of the chylous urine, and none separated after an hour's centrifugalisation. Golding-Bird (2) and several subsequent observers noted the formation of a creamy layer on standing, but in Brieger's (14) case, as in this one, none was present.

Warming the fluid led to the production of a film on the surface exactly similar to the "skin" of milk. This is just what would be expected from the observations made on the subject of the formation of the skin on warmed milk by Mr. R. Jamison and myself.[1] We showed that it was not due to a peculiar property of caseinogen, as had previously been believed, but to the universal property of proteid solutions containing fat in suspension.

The opacity of the chylous urine did not clear on shaking with ether. This, like the similar behaviour of milk, was probably due to the agglomeration of proteid molecules round the fat particles. As with milk, the opacity was completely removed by shaking with ether if the physical condition of the proteid was altered by means of an alkali. On evaporation of the ethereal extract, fat, solid at ordinary room temperature, staining with osmic acid and Sudan iii, and giving the acrolein test, was obtained.

Whenever fat was present in the urine proteid was also found; it was coagulated by heat and precipitated

[1] 'Journal of Physiology,' xxvii, 26, 1901.

by nitric acid and by alcohol. The nature of the proteids was determined in the fat-free "lymphous urine" obtained by means to be subsequently described, and the results of the determination will be described in the same place.

Dextrose was never found, though repeatedly looked for, especially after meals containing excess of carbohydrate. Morrison (13) is the only observer who has found it in chylous urine.

The specific gravity was, on the whole, greater the larger the quantity of fat and proteid present; it varied between 1·012 and 1·024. The urine was generally acid, occasionally neutral or alkaline, and the variations were quite independent of the quantity of fat present.

Spontaneous disappearance.—Many authors have recorded that the chyluria spontaneously disappeared for periods of various length, only to return again without any obvious cause.[1] In the present case, from October 14th to 21st, there was no chyluria, although no special treatment had been adopted. Exercise and a very fatty diet had not the slightest effect during this period, the urine being perfectly normal, with no fat, albumen, sugar, or formed constituents. No cause could be discovered for the return of the chylous urine on October 21st.

On December 3rd a cystoscopic examination was made, and a urinary segregator was passed with the object of determining at what point in the urinary passages the admixture with chyle occurred. The patient had a meal containing a considerable quantity of fat at mid-day. At 2 p.m. the urine was distinctly chylous; at 2.30 p.m., immediately before the examination, it was quite milky. During the examination, however, no chylous urine was passed, although observations to be presently recorded showed that the maximum excretion of fat should not have occurred before about 5 o'clock, and the last traces should not have disappeared until about 9 p.m. Consequently the desired information was not obtained.

[1] In Theilemann's case (37) there was an interval of nine years between the first and second attacks.

The passage of the segregator caused some hæmorrhage, and the patient had subsequently to be catheterised for retention of urine. The hæmorrhage continued to a slight extent for two days. The bladder was washed out and helmitol administered, as signs of slight cystitis developed. These quickly disappeared, the urine becoming perfectly normal by December 6th. On December 4th, however, the patient's temperature rose and signs of bronchitis, and later of pneumonia, were found; his pulse became very rapid and feeble, and the heart sounds were faint, but no pericardial rub was detected. He died on December 11th. During the eight days which elapsed between the passage of the segregator and the death of the patient he received a mixed diet containing the ordinary proportion of fat. In spite of this no trace of chyluria returned, the urine being clear and free from albumen after the hæmorrhage had ceased, and the cystitis had disappeared on December 6th.

Influence of diet.—As long ago as 1850 Bence Jones (3) observed that chyluria was more marked with animal than vegetable diet, and since then most authors have referred to the connection between the disease and the nature of the food. A diet containing much fat has been found by all except Goetze (22) to increase the amount of fat in the urine, and many have noticed that the chyluria disappeared when the diet contained as little fat as possible and during starvation. Goetze alone denied the existence of such a connection. Franz and Stejskal (33) found that the increase in the fat of the urine was always associated with an increase in the amount of proteid present.

In order to study the effect of diet on the production of chyluria I obtained a specimen of the patient's urine immediately before meals, and made him pass his urine at frequent intervals afterwards. I was thus able to trace the connection more accurately than had previously been done, as others had only compared the day and night

urine, or the urine passed several hours after a meal with that passed before it.

Each specimen of urine was measured, its reaction noted, and its specific gravity ascertained. Albumen was tested for, and in a few cases was accurately estimated. Part of each specimen was put in one of a series of test-tubes of exactly the same size as each other, and the relative opacity of each pair was found by comparing the brilliancy with which an electric light could be seen

Photograph of test-tubes containing hourly specimens of urine to show the relation of the chyluria to the fat in the food. On Nov. 9th breakfast with 20 grms. of butter was given at 6 a.m., and a dinner containing very little fat at 12 o'clock. On Nov. 8th a dinner with plenty of milk pudding was given at 12 o'clock, after which no more fatty food was eaten.

through them. The more opaque specimens had to be diluted two, three, or four times with water before the comparison could be made. The specimen showing the smallest trace of opacity was numbered 1, and the others were numbered up to 15, which represented the greatest degree of opacity observed. As the opacity depends upon the amount of fat present the numbers

TABLE II.—*Influence of Diet on Chyluria.*

Date.	Time.	Exercise, etc.	Food.	Reaction.	Sp. gr.	Degree of opacity (= fat).	Albumen.
Nov. 7	6 a.m.	Up all day	Full breakfast	Acid	1015	3	Very little.
	6.30 „			„	—	2	Trace.
	7 „			„	1012	5	+
	8 „			„	1016	7	+
	9 „			Neutral	1017	6	+
	10 „			„	1020	10	+ +
	11 „			Alkaline	1022	11	+ +
	12 mid-day		Full dinner	Neutral	1022	12 (0·664%)	+ +
	1 p.m.			„	1018	6	+
	2 „			Acid	1020	9	+
	3 „			„	1022	13	+ +
	4 „		Strict tea	„	1022	14	+ +
	5 „			„	1021	15 (2·170%)	+ +
	6 „		Strict supper	„	1021	13	+ +
	7 „			„	1021	8	+
	8 „			„	1020	4	Very little.
	9 „			Alkaline	1018	0	0
	10 „			„	1017	0	0
	11 „			Neutral	1018	0	0
	12 mid-night			Acid	1019	0	0
Nov. 8	6 a.m.	Up all day	Breakfast with milk in tea, but no other fat	„	1021	0	0
	7 „			„	1016	2	Trace.
	8 „			Neutral	1017	5	+
	9 „			Alkaline	1019	2	Trace.
	10 „			„	1018	0	0
	12 mid-day		Strict dinner with plenty milk pudding	Neutral	1020	0	0
	1 p.m.			„	1020	1	+
	2 „			Acid	1021	3	+
	3 „			„	1021	4	+
	4 „		Strict tea	„	1022	7 (0·284%)	+
	5 „			„	1024	10	+ +
	6 „		Strict supper	„	1024	6	+
	7 „			„	1019	4	+
	8 „			„	1019	2	Trace.
	9 „			Alkaline	1017	1	„
	10 „			„	1017	0	0
	11 „			Acid	1017	0	0
	12 mid-night			„	1016	0	0

TABLE II.—*Influence of Diet on Chyluria*—continued.

Date.	Time.	Exercise, etc.	Food.	Reaction.	Sp. gr.	Degree of opacity (= fat).	Albumen.
Nov. 9	6 a.m.	Up all day	Breakfast with 20 grms. butter	Acid	1019	0	0
	6.30 „			„	1017	0	0
	7 „			„	1016	5	+
	8 „			„	1016	7	+
	9 „			Alkaline	1017	7	+
	10 „			„	1016	6	+
	11 „			—	—	3	Very little.
	12 mid-day		Almost strict dinner	—	—	1	Trace.
	1 p.m.			—	—	2	Trace.
	2 „			—	—	4	+
	3 „			—	—	2	Trace
	4 „			—	—	1	„
	5 „			—	—	0	0
	6 „			—	—	0	0
	12 mid-night			—	—	0	0
Nov. 10	6 a.m.	In bed	Full breakfast	—	—	0	0
	6.30 „			—	—	0	0
	6.45 „			—	—	0	0
	6.55 „			—	—	0	0
	7.5 „			—	—	0	0
	7.15 „			—	—	0	0
	7.20 „			—	—	0	0
	7.25 „			—	—	Faintest trace	Faintest trace.
	7.30 „			—	—	1	Trace.
Nov. 12	12 mid-day	In bed	Strict dinner	—	—	0	0
	4 p.m.		Strict tea	—	—	0	0
	6 „		Strict supper	—	—	0	0
	12 mid-night			—	—	0	0

indicate which of any two specimens contains the most fat, though, of course, not the actual amount present. Evidence of the accuracy of this supposition is given by the actual analyses made; in three specimens which had opacities represented by numbers 6, 11, and 14 the percentage of fat present was 0·284, 0·664, and 2·170 respectively.

Table II shows the results obtained, and figure 1 is a reproduction of a photograph taken of two series of test-tubes containing hourly specimens of the urine. If as little fat as possible was taken after dinner at mid-day, and the urine passed at midnight, the night's urine contained no fat and no albumen. The observations made on November 10th showed that when the patient was in bed one hour and twenty-five minutes elapsed before the first trace of chyle appeared in the urine. A later observation, when the patient was up, showed that chyle appeared in the urine already thirty-three minutes after eating 25 grms. of butter with bread and biscuits, the patient having walked up and down immediately after the meal. Further experiments on the variation produced by exercise will be described later.

The amount of fat present in the urine increased steadily every hour up to the fifth after the ordinary breakfast, and the ordinary dinner on November 7th, and the only slightly restricted dinner of the 9th [1]; when the meal contained only a limited quantity of fat, as with the breakfast on November 8th, and dinner on November 9th, the maximum occurred already two hours after the meal. No more fat was given on November 7th or 8th after dinner; the disappearance of chyle from the urine occurred eight hours after the meal on both occasions. After the breakfast on the 8th and the dinner on the 9th, in both of which the fat was restricted, the chyluria disappeared

[1] This corresponds well with Hamill's observations on a patient with a lymphatic fistula in the groin. The maximum amount of fat was found to be present six hours after the principal meal. 'Journ. of Physiology,' xxxv, 151, 1906.

after three and four hours respectively. Numerous other observations confirmed these time relations. After meals containing as little fat as possible (as on November 12th), no chyluria occurred unless much exercise was taken, in which case traces of fat appeared in the urine, as it was found impossible to devise a diet absolutely free from fat. The following diet, however, contained so little that if the patient rested for an hour or two after dinner no trace of fat appeared in the urine all day: Certain fish (haddock, cod, whiting, turbot, or brill), jelly, potatoes, green vegetables and fruit, bread with jam, honey, or treacle; no milk, butter, cheese, or eggs. A single egg for breakfast was sufficient to produce well-marked chyluria, but lean beef had no effect if care was taken to avoid all the obvious fat. It is thus possible for a patient with chylous urine, who is troubled with difficulty in micturition due to intravesical clotting, to control the chyluria completely by resting after meals which are composed in the way described.

The next question to decide was whether the fat in chylous urine was the same as that taken in the food or was derived from the body stores of fat. The speed with which the appearance of the fat in the urine followed the taking of fat in the food suggested that the former was the case. This is confirmed by Huber (19), who detected the smell of cod-liver oil in the urine after giving it to his patient, and by Franz and Stejskal (33), who found that the fat in the urine had the same melting-point as the unusual fat (erucic acid) which their patient ate. They also found that the urine was coloured red after the patient had taken some olive oil stained with Sudan iii. The present patient took some butter stained deeply with Sudan iii, which did not affect its taste in the slightest degree, with his bread on two occasions; the urine passed after the meal was chylous and tinged faintly red. But as the only fat taken in the meal was deep red, and the urine was white with only a slight pink tinge, it seems probable that the compound of the fat with the stain

was dissociated in the intestines. This seems to be confirmed by the fact that none of the fat particles in the urine appeared to be stained when examined under the microscope.

Diets containing excessive amounts of carbohydrates and of proteid had, in no case, the slightest effect on the urine. The former, given as bread, never produced a trace of chyluria or lymphuria, confirming the generally accepted view that carbohydrates are absorbed by the capillaries and not by the lymphatics; moreover, the conversion of carbohydrate into fat in the intestinal epithelium, which Pavy believes to occur in rabbits, does not appear to occur in man. Proteid food also never produced albuminuria, showing that proteids are probably absorbed by capillaries and not by lymphatics.

Thus, as far as food is concerned, albuminuria never occurred independently of chyluria, the variations in the amount of albumen in the urine being approximately parallel with that of the amount of fat. The ratio of the amount of fat to that of proteid present on the two occasions when both were accurately determined on November 7th, gave results which fit in well with the assumption that it is actually chyle which is present in the urine. The amount of proteid in chyle is about 2 per cent.; hence when 0·885 per cent. proteid was present in the urine, 44 per cent. of the specimen was chyle and 56 per cent. urine, and when 1·885 per cent. proteid was present, 94 per cent. was chyle and only 6 per cent. urine. As the percentage of fat in the two specimens was 0·664 and 2·170 respectively, the percentage of fat in the original chyle must have been $(0{\cdot}664 \times \frac{100}{44} =)$ 1·5 and $(2{\cdot}17 \times \frac{100}{94} =)$ 2·3 respectively. The former gives the maximum percentage of fat in the chyle after the breakfast, the latter after the dinner; as the dinner contained considerably more fat than the breakfast the larger percentage in the chyle is explained.

Two tablespoonsful of oleic acid were given on two occasions, once alone and once with a teaspoonful of glycerine. On neither occasion did any chyluria occur; this is probably due to the fact that slight diarrhœa was produced, so that the fatty acid passed too rapidly out of the body for absorption to occur.

Influence of exercise.—A number of authors have observed that exercise increases chyluria, though generally no exact data have been given. Golding-Bird (2) stated that albuminuria was occasionally present during the fat-free periods, but was unable to give any explanation of this. The few subsequent investigators who have confirmed his observation made no statement as to the nature of the albumen, and seem generally to have regarded its presence as a sign that the kidneys were slightly diseased. I have been able to demonstate very clearly the effect of exercise on chyluria, and to show that the albuminuria without fat is really lymphuria and due to exercise. (Table III.)

Experiments 1 and 2 show that albuminuria is produced within an hour by exercise, and that if no fat has been eaten for many hours no fat appears with the albumen in the urine. Numerous controls showed that in the absence of exercise, if no fatty food had been taken, no albuminuria was present.

Microscopical examination of the urine showed that leucocytes were present in large numbers, varying with the amount of albumen, although none could be found in the albumen-free urine passed during rest with a fat-free diet. Chemical examination showed that an albumen which began to coagulate at 69° was present; this was probably serum-albumen, the coagulation-temperature of which is from 70° to 85°. In addition a small quantity of a globulin coagulating at 56°, and rather more of another coagulating at 75° were present; the latter was, no doubt, serum-globulin, and the former fibrinogen, as this coagulates mainly at 56°, and a number of minute fibrin flocculi were found floating in the urine. The

TABLE III.—*Influence of Exercise on Chyluria.*

No.	Date.	Time.	Food.	Exercise.	Fat in urine.	Proteid in urine.
1	—	6 a.m.	Fasting	—	None	None
		6–7 „	„	Walking	„	Much
2	—	10 „	Fat-free	In bed until 10	„	None
		11 „	breakfast at	a.m. Walk from	„	Trace
		12 midday	6 a.m.	10 to 12	„	Much
3	—	8·30 a.m.	Full break-	Rest since 7	„	None
		9 „	fast	Walk immediately after breakfast	„	A little
		9·15 „		Continue walk	1	Much
		9·30 „		„ „	4	„
4	—	4 p.m.	Dinner with	Rest since 12.	5	„
		5 „	milk pudding	Walk from 5 to 6	3	Less
		6 „	but no other fat at 12, nothing since		6	Much
5	Nov. 15th	11 a.m.	Fat-free breakfast at	In bed all the morning	None	None
		11·45 „	6 a.m.	Right arm 3 lb. dumb-bell for ¾ hour in bed	„	„
6	—	4 p.m.	Fat-free	In bed all day	„	„
		4·45 „	dinner at 12	Left arm 3 lb. dumb-bell for ¾ hour in bed	„	„

presence of the three proteids of lymph and of leucocytes affords very strong evidence that the urine was mixed with lymph. No ordinary cases of albuminuria are associated with fibrinuria or with such a large admixture of leucocytes.

Experiment 3 shows that walking after a meal containing fat gives rise to albuminuria in half-an-hour, which is greatly increased in three-quarters of an hour, when chyle also begins to be present; in an hour chyluria is well marked. On another occasion chyluria was observed as early as thirty-three minutes after the meal. With a precisely similar breakfast taken in bed and not followed by exercise, chylous urine was not passed until the elapse of one hour and twenty-five minutes.

Experiment 4 shows that exercise taken during the decline in the chyluria produced by a meal causes a secondary increase in the chyluria.

These observations can only be explained by supposing that there is an escape of lymph into the urine when the flow from the legs is increased by exercise. After a meal containing fat, chyle does not escape into the urine until a certain degree of distension of the lacteals is produced, so that if the patient remains at rest the onset of chyluria will be appreciably later than the beginning of fat absorption. But the necessary distension is already present when the absorption begins, if exercise is being taken, so that the first appearance of fat in the urine—in experiment 3 three-quarters of an hour after food—indicates the moment in which fat absorption begins.

An hour's exercise with a 3-lb. dumb-bell, first with the right arm and then with the left, had no effect on the urine whether the patient was lying down (experiments 5 and 6) or sitting up; this suggested that the obstruction to the lymphatic system was below the point at which the lymphatics of the left arm unite with the thoracic duct, and did not affect the right lymphatic duct. As will be seen later, the autopsy confirmed this supposition.

Influence of posture.—Several authors have stated that the erect position tends to produce more chyluria than the horizontal position, but they did not realise the important part played by exercise in its production, so that their results might have been due to exercise rather than posture.

Ackermann (7), however, clearly demonstrated that his patient could entirely prevent the appearance of chyle in the urine by lying on the right side.

My own observations showed that merely sitting or standing produced neither chyluria nor lymphuria if the patient had eaten no fat food for several hours previously. After a meal containing fat, chyle seemed to appear in the urine rather sooner if the patient was up, though not taking any exercise, than if he was in bed. The difference, however, was slight, and would alone be insufficient to prove definitely that a vertical position increased the tendency of the chyle to escape. Conclusive evidence of the importance of posture was obtained, however, when exercise was taken. When the patient, lying in bed, alternately flexed and extended the right leg on to the thigh continuously for an hour, and then performed the same movements for an hour with his left leg, no lymphuria was produced. The same movement of either leg at the same rate for only three-quarters of an hour, when the patient was sitting, produced well-marked lymphuria.

The effect of posture depends probably upon gravity, which would aid the back-flow of lymph, caused by obstruction in the thorax, when the patient is standing or sitting.

Autopsy.—I have been able to find only three published descriptions of *post-mortem* examinations of patients who had suffered from non-tropical chyluria. In Oehme's case the patient died a few days over a year after the onset of chyluria; he was a man of fifty, and had been troubled during the whole of the time with gastric disturbances. At the autopsy a carcinoma of the posterior wall of the pyloric end of the stomach was found. Nothing else ab-

normal was present. Though Oehme (9) does not himself suggest that the growth had any connection with the chyluria, it does not seem improbable that it pressed upon the larger lymphatics just below the receptaculum chyli, and that an obstruction to the flow of lymph out of the abdomen was produced in this way. In support of this view is the fact that the duration of the chyluria corresponded with the duration of the gastric symptoms, which were presumably the direct result of the growth.

Sir Wm. Roberts' (16) patient died of pulmonary tuberculosis. In the skin and subcutaneous tissue of the abdominal wall were some enormously dilated lymphatics from which lymph and chyle had escaped during life. No cause was found either for these dilated lymphatics or for the chyluria. But chylous urine had only been passed on two occasions—once six and once five months before death, and the lymphatics of the abdominal walls had become less distended as the phthisis advanced, and had ceased to discharge five days before death. It was therefore hardly surprising that the cause remained undiscovered, as the obstruction to the lymphatics appears to have almost disappeared by the time the patient died.

In the case reported by Port (36) the thoracic duct was obstructed by the pressure of tuberculous glands; occasional spontaneous disappearance of the chyluria was apparently due to relief of pressure brought about by the bursting of caseous glands into the œsophagus or left bronchus. Perhaps in Roberts' case the obstruction and the final relief may have been due to the same causes.

In addition to these three cases a post-mortem examination was made on a patient who was under Dr. Newton Pitt in Guy's Hospital in 1887. Granular kidney and tubal nephritis were found. The cause of the chyluria was not discovered, but seeing that the patient had passed no chylous urine since the symptoms of nephritis began to develop eight months before death, this is not surprising.

Dr. Fawcett kindly allowed me to investigate the con-

dition of the lymphatics in the present case. I injected mercury at a pressure of about 25 mm. into the lymphatic system at various points. I first introduced the cannula into the thoracic duct, just below the point where it joined the angle between the jugular and subclavian veins. After the mercury had run in, the duct could be traced to the posterior thoracic wall. As the vessel ruptured at this point, it was necessary to introduce the cannula at another point just below. The mercury passed down the next part of the duct with difficulty, and even under a higher pressure it could not be forced further than a point 7 cm. above the diaphragm. On injecting upwards from the receptaculum chyli the mercury did not pass higher than this same point. On subsequently removing the whole of the thoracic duct it was found that a fine probe passed either from above or below met with obstruction at the same point—7 cm. above the receptaculum chyli. By using somewhat greater force the probe was pushed through the obstruction from below, but even after this the resistance could still be felt when it was passed in either direction.

On injecting downwards from the receptaculum chyli the lymphatics behind the peritoneum were readily filled, although the injection was in the opposite direction to that of the normal lymph flow. These lymphatics appeared to be abnormally large. By injecting at two other points lower down in the abdomen the connection between the lacteals and the large lymphatics round the aorta could be traced, and two large vessels were seen passing from the lymphatics internal to the external iliac artery over the lateral pelvic wall on to the side of the bladder, where they divided into numerous branches. The lymphatics on the left side of the bladder could not be injected. There was thus a very free anastomosis between the lymphatics of the right side of the bladder and those of the intestines, which was sufficient to allow mercury to pass in the wrong direction from the receptaculum chyli

to the bladder. As much of the lymph from the leg ascends in lymphatics which run with the external iliac artery, the lymph from the right leg would have had easy access to the lymphatics of the bladder, and that from the left leg had presumably to ascend to the vessels round the abdominal aorta and then descend again to the right side of the bladder.

The lymphatics to the kidneys do not appear to have been abnormally large, and the valves were still competent, as I was unable to inject them with mercury from the plexus round the aorta.

These observations strongly suggest that the obstruction in the thoracic duct had led to dilatation of the abdominal lymphatics, and that a reflux of chyle from the intestines to the right side of the bladder, but not to the kidneys or ureters, had become possible. I was unable to inject any mercury from the bladder lymphatics into the bladder lumen; but for nine days before death no chyluria had been present, and it may be supposed that in some way the fistulous opening had become closed, as had also occurred between the 14th and 21st of October. The interior of the bladder showed no trace of cystitis; a minute bluish papilliform projection about 2 mm. long, 1½ mm. broad, and 1 mm. high was found on the mucous membrane at a point on the inside of the bladder corresponding to the position of the dilated lymphatics on the outside. On microscopical examination this papilliform projection was found to consist of unorganised lymph. From the position of this peculiar papilliform mass of lymph, which was closely adherent to the bladder surface, Mr. Targett, who kindly examined the section for me, thought that it was very probably produced by coagulation of chyle at the point where the escape into the bladder occurred.

The prostate was normal, and the urethra showed no signs of inflammation, and there was a little submucous bruising in the membranous urethra, but no trace of inflammation was found.

The kidneys were quite normal.

About two ounces of pus were found in the pericardium, and recent lymph was present on the surface of the heart. The coronary arteries were very calcareous, and well-marked atheroma was present in the thoracic and abdominal aorta.

Acute bronchitis and hypostatic pneumonia were present in both lungs.

Pathogenesis.—Two theories have been advanced to explain the origin of the European cases of chyluria. According to the older one, first propounded by Prout (1) in 1841, later supported by Eggel (8) and Brieger (14), and recently revived by Waldvogel and Bickel, the escape of fat and proteid into the urine occurs from the blood, so that "chyluria" is a misnomer. Waldvogel and Bickel (32) believe that the blood loses its power of converting the fat of chyle into soluble substances; consequently it accumulates in the blood, and is excreted by the kidneys. But excess of fat in the blood has been found neither by me nor by any previous observer, although several, the first of whom was Bence Jones, have examined the condition of the blood. Moreover, it is impossible to explain by this theory why proteid and leucocytes should always accompany the fat in the urine. Conclusive evidence against it is given by my observations on the lymphuria produced by exercise, where there is no question of passage of fat into the urine at all. Moreover, the "molecular" condition of the fat is strong evidence that the urine is actually mixed with chyle, as a similar molecular condition occurs also in chyle, but nowhere else.

The second theory, which appears to have owed its origin to Ackermann (7) in 1863, has been further developed in connection with tropical chyluria by Manson. It has received the support of all recent writers on the subject, except those mentioned above. An obstruction to the lymphatics, somewhere between those of the small intestines and the end of the thoracic duct, is supposed to

exist. As a consequence there is stasis and a rise of pressure everywhere below the point of obstruction. Owing to the dilatation of the vessels the valves become incompetent, and a rise of pressure drives the chyle from the intestinal lacteals in a retrograde direction to the pelvis, whence it passes by the lymphatics of the abdominal wall to their anastomosis with those of the upper part of the body, which join the right lymphatic duct or the thoracic duct above the obstruction. Where the dilated lymphatics are feebly supported one may rupture. If this occurs into the abdominal cavity chylous ascites results; if a distended lymphatic in the submucous tissue of the bladder bursts lymph and chyle will escape and chyluria result. Finally, the subcutaneous lymphatics may give way, and a lymphous and chylous discharge from the skin is the result. More than one of these events may occur in the same patient, as in Senator's, who had chylous ascites as well as chyluria, in Sir William Roberts' patient, who had leakage from dilated lymphatics under the skin of the abdomen, and in Myers' patient, who had had a similar condition in the upper part of her left thigh for eleven years before the onset of chyluria.

In the present case and in Port's the obstruction was actually found;[1] in the other two cases in which a post-mortem examination was made none was observed, but no special means appear to have been taken to investigate the condition of the thoracic duct.

Chyluria occurring in the tropics is always associated with infection with filariæ. According to Manson[2] the thoracic duct becomes occluded "in some way as yet unexplained, either by mechanical plugging by a bunch of intertwined parent filariæ, or in consequence of inflammatory conditions brought about by the presence of the filariæ in the vessel and leading to stenosis." The

[1] An obstruction of the thoracic duct has also been found in a number of cases of chylous effusions into the pleural or peritoneal cavity.

[2] Allbutt's 'System of Medicine,' ii, 1079.

parasites have never been found in the thoracic duct, but stenosis, apparently identical in character with that of the present case, was found to be present by Stephen Mackenzie[1] and various other authors, who found, moreover, considerable dilatation of the abdominal and pelvic lymphatics. When stenosis is once produced the obstruction will remain whether the filariæ live or not; hence, the absence of the parasite from the blood and urine in the present case, and in the others in which the patients had been in the tropics many years before the onset of the chyluria, is no evidence that filariæ were not the original cause of the obstruction. The long period which elapsed between the supposed original infection and the onset of chyluria is only analogous to the many years which may elapse before dilated hæmorrhoidal veins rupture.

In those patients who have never been abroad obstruction must be due to some other cause, such as pressure exerted from outside by caseous glands, as in Port's case, by the pregnant uterus, as in the cases recorded by Francotti (18), Berri (23), and Vieillard (29), or by artificial means as in Saundby's case.[2] Possibly in some European cases parasites other than filariæ may cause the obstruction. Thus Predtschensky (30) found on one occasion ova similar to those of *Tænia nana*, a minute tapeworm almost unknown in England, in the chylous urine of his patient. Though no other evidence of infection with the parasite was forthcoming, Predtschensky suggested that it might have been the cause of the lymphatic obstruction. He states, moreover, that Moscato (25) found *Eustrongylus gigas*, a worm 9 cm. long, on two occasions in the urine of a patient who was passing

[1] 'Trans. Path. Soc.,' xxxiii, 394, 1882.

[2] A young unmarried woman, who bandaged her abdomen so tightly in order to conceal her pregnancy that severe œdema of the legs, vulva, and lower part of the abdomen resulted, passed chylous urine for some days after labour. Probably the pressure exerted by the abdominal bandage produced temporary obstruction to the lymphatics (Ref. 31).

chylous urine, the chyluria on both occasions ceasing temporarily after the passage of the parasite.[1]

The chief argument against this theory is the almost constant absence of sugar from chylous urine. But Munk and Rosenstein found that human lymph obtained from a lymphatic fistula contained only 0·1 per cent. dextrose. Chylous urine must always contain less than this—and generally considerably less—owing to the admixture of the chyle with the urine. As the ordinary methods are only sufficiently delicate to recognise about 0·1 per cent. of sugar in the urine, the slight excess present in chyluria readily escapes recognition.

At the discussion in Berlin on the case reported by Siegmund (15), who believed that the chyle escaped through a fistulous opening in the bladder, Virchow argued against this explanation on the ground that no connection existed between the lymphatics of the intestines and those of the bladder. The ease with which in this case I was able to inject the lymphatics of the bladder from the upper part of the abdomen proves the error of Virchow's statement, and demonstrates that a reflux of chyle from the intestines to the bladder could have occurred.

In the present instance the fistulous connection between the lymphatic and urinary tracts appears from the post-mortem examination to have been in the bladder. Dr. Pavy, whilst the patient was still alive, pointed out to me that this was probably the case, as even when the patient was passing clots *per urethram* there had been no renal colic, though its occurrence might have been expected if the escape took place in the kidneys or their pelves. That the leakage rarely if ever occurs in the kidneys is rendered likely by the invariable absence of renal casts from chylous urine. Moreover, in this case,

[1] Stuertz found eight ova of this parasite in different specimens of chylous urine of a man, whose illness began at the age of 20, when he was living in Brisbane ('Deutsch. Arch. f. klin. Med.,' lxxviii, p. 557, 1903).

as in two of the three others in which an autopsy was made, the kidneys were perfectly healthy.

Franz and Stejskal (33), however, state that they could observe during a cystoscopic examination chylous urine escaping from both ureters; they conclude that there was leakage into the pelves of both kidneys. Moreover, the post-mortem examination in Mackenzie's case of tropical chyluria suggested but did not definitely prove that the leakage was into the left kidney, and Port (36), finding that the pelves of both kidneys contained chylous fluid though the kidney tubules, which were perfectly normal, did not, believed that escape occurred in his case into the renal pelves.

Conclusions.

1. Chyluria, whether associated with filariasis or not, is always due to the same anatomical lesion—an obstruction to the thoracic duct or some of its largest tributaries.

2. The obstruction may remain for many years before rupture of a dilated lymphatic into the urinary tract occurs and chyluria results, so that all traces of an original filarial infection may have disappeared before the onset of the chyluria.

3. Chyluria depends upon the distension of the abdominal lymphatics with the chyle produced during absorption of fat from the intestines. After an ordinary meal fat appears in the urine in half an hour if exercise is taken, but in about an hour and a half if no exercise is taken. The maximum amount is present five hours after food, and the last traces disappear in eight hours. These observations suggest that fat digestion and absorption occur more rapidly, and that the commencement of absorption is earlier than has generally been believed.

4. The increased flow of lymph from the legs produced by exercise gives rise to distension of the lymphatics, so

that escape of lymph into the urine with the production of lymphuria may occur. It appears probable that such a lymphuria, though observed for the first time in the present case, will always be found to accompany chyluria.

DISCUSSION.

Dr. J. Molyneux Hamill.—I have listened to Dr. Hertz's communication with much interest, the more so since quite recently I myself have been engaged in work on chyle obtained from a case of chylous fistula, the main results of which I have embodied in a paper in the 'Journal of Physiology' for December, 1906. From the point of view of the investigation of chyle itself my case was more suitable than Dr. Hertz's, since, of course, I obtained chyle quite pure and free from any admixture of urine. Both Dr. Hertz's case and mine showed absence of glucose in the chyle, and this is of interest in view of Munk and Rosenstein's statement that sugar was present in the chyle obtained from a case of chylous fistula which they examined. I noticed also that Dr. Hertz mentioned that he never observed a separation of the fat of the chyle into two layers, *i. e.* the formation of cream. The formation of a creamy layer, of course, depends on the nature of the fat ingested. Munk and Rosenstein showed that the fat separated into two layers only when the food contained a fat with a high melting point, such as mutton fat. The patient whom I studied had such an aversion to fat of all kinds that I could not induce him to eat sufficient to put Munk and Rosenstein's statement to the test. At all events, I never observed the formation of a creamy layer in any of the samples of chyle which I examined.

With regard to the origin of the fat in chyluria it seems definitely established that it comes directly from the ingested fat in the chyle and not from any other source. Erben brings forward conclusive evidence on this point in a paper on "Chyluria," published in the 'Zeitschrift für Physiologischen Chemie,' and most other observers are in agreement with him on this point.

Dr. Hertz has called attention to the extreme rapidity with which the fat in the food appears in the chyle. The point is an interesting one, and I might, in this connection, point out that Munk and Rosenstein have shown that the rapidity with which fat is absorbed depends on the nature of the fat ingested, that a low melting point fat is absorbed much more rapidly than one with a high melting point.

I do not think that Dr. Hertz was justified in making the deduction he did with reference to the experiment with fat stained by Sudan III. It does not follow that the Sudan III was not, for the most part, absorbed because the urine was only pinkish in colour. It is possible to account for the absorption of Sudan III, whether we believe that fat is absorbed as such in minute particles or undergoes hydrolysis before being absorbed; this point has been fully established in the controversy waged between Hofbauer and Pflüger on the mechanism of fat absorption. The chyle appears pinkish, only because the particles of fat are in such a state of extremely fine subdivision that light falling on them is scattered and gives rise to a less saturated red, *i. e.* to a pink colour. The production of a perfectly white colour from yellow olive oil is a very similar phenomenon. For these reasons, I think that, in the absence of any stronger evidence, Dr. Hertz is carrying his inferences further than he is warranted by the facts at his disposal.

With reference now to the case which I myself had under observation I should be glad if anyone present could inform me whether operative interference with a view to improving the patient's condition would be possible. I had thought that an anastomosis between the portions of the lymphatic system on each side of the obstruction might be effected. The difficulties would obviously be great. In the first place the obstruction itself would have to be located, and after this would come the operation, which would, I think, tax the technique of surgery to the utmost. Since, however, the patient suffers considerable discomfort and impairment of health, I should be glad to know if such an operation could be undertaken with a hope of success at all commensurate with the risk involved.

Mr. STEPHEN PAGET had been interested in the question of chylous fluid in the body cavities, especially in the thorax, but he knew of no operation recorded such as had been alluded to. He asked for further clinical facts as to feverishness and cause of death.

The PRESIDENT thought it remarkable that there was no bladder irritability. It seemed that the connection of the lymphatics was more free than had appeared from anatomical observations. The obstruction might occur long after the cause had departed.

Dr. HERTZ (in reply).—I made no observations with regard to the influence of the melting point of the fat in the food on the rate of its appearance in the urine. From what Mr. Hamill has said it seems very probable that the remarkably early appearance of chyluria after meals depends partly on the low melting point of some of the fats present in ordinary mixed diets. But what I wished especially to emphasise was the earlier appearance of fat in the urine if exercise was taken immediately

after the meal. Under such circumstances the first trace of fat appeared in the urine in half an hour; thus the onset of fat absorption is earlier than has generally been believed to be the case.

I cannot agree with Mr. Hamill's remarks with reference to the fat stained with Sudan III. The butter, which was the only fat taken in the meal, was stained a deep red colour, but the very slight red tinge present in the milky urine would have escaped notice had the urine not been compared with another specimen passed after an ordinary meal. Moreover, under the microscope the fat particles did not appear to be stained at all. I am, therefore, of opinion that the compound of fat with Sudan III was dissociated in the intestines, and only a small part of the stain was absorbed by the lymphatics.

With regard to the question of surgical treatment discussed by Mr. Hamill and Mr. Paget, I do not think that any useful operation could be devised. Moreover, by means of a diet poor in fat, the inconveniece caused by intra-vesical clotting can be prevented, and by increasing the supply of proteid and carbohydrate in the diet it should be possible to prevent the occurrence of loss of weight.

I cannot give any definite information in reply to Mr. Paget's question as to the cause of death. The patient developed signs of bronchitis and hypostatic pneumonia with a rapid weak pulse and a raised temperature very soon after the passage of the segregator. At the autopsy, in addition to the pneumonia, purulent pericarditis was found, but there was no inflammation of the urinary tract. It seems therefore probable that the shock of the passage of the segregator in an elderly man somewhat weakened from the rapid loss of weight which had occurred during the last few months, was the exciting cause of the changes which led to his death.

The absence of vesical irritability, to which the President referred, is due, I believe, to the fact that the fat is present in the chyle in the inert neutral condition with only minute traces of irritant fatty acids. Moreover, its finely-divided condition would prevent mechanical irritation.

The President also remarked upon the probable long duration of obstruction before the onset of chyluria. I do not think that gradual increase in the obstruction is the only explanation. More probably the obstruction reached its fullest development within a comparatively short period of the initial filarial infection. Dilated lymphatics will, therefore, have been present for a considerable time, and rupture will only have occurred owing to some recent accidental cause, such as a slight injury. Thus cases have been described in which dilated lymphatics, due to obstruction of the thoracic duct have been present for years without rupture ever occurring.

THE EARLY DIAGNOSIS AND CURE OF THE PRE-SUPPURATIVE STAGE OF AMŒBIC HEPATITIS

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No one who has had a large experience of the early stages of amœbic abscess of the liver in a tropical climate can fail to have been struck by the great frequency with which definite clinical symptoms of suppuration in this organ are preceded by weeks or even months of fever, during which there may be few or no symptoms pointing to the liver as the cause of the trouble, and which is commonly ascribed to and treated as "malaria." Moreover, even when marked symptoms of acute hepatitis are present it is frequently impossible to decide whether an abscess has already formed or not without resort to an exploratory operation, which only too often gives a negative result in cases where an abscess of the liver is found at a later date.

In a paper (1) published a year and a half ago I discussed the value of leucocytosis in acute hepatitis, and concluded that its presence in a marked degree was generally an indication that suppuration had already taken place, but in the slighter degrees it may be present in acute hepatitis without actual suppuration, and that this early stage

of amœbic hepatitis may sometimes be cured, and suppuration prevented, by the administration of large doses of ipecacuanha. Since then I have repeatedly confirmed this observation, and can now go a step further in advance of that position.

During the last two years I have examined blood slides of all the fever cases on admission into the open wards of the European General Hospital, Calcutta, amounting to 13,500, and possess notes and temperature charts of them. In the course of this comprehensive investigation I have met with a most interesting group of cases, which appear to me to throw much light on the early pre-suppurative stages of amœbic hepatitis, and indicate that this disease may frequently be recognised by the blood changes when in a stage which admits of rapid cure, and so prevented from drifting on into the much more serious suppurative stage, as is now so commonly their fate. Further, these cases constitute a definite class of fever, usually of a chronic intermittent type, sometimes with no very definite symptoms of hepatitis, and rarely with any dysentery. They may be recognised, or at least strongly suspected, by the presence of a moderate degree of leucocytosis, generally of the type which I have described (1) as common in amœbic abscess of the liver, namely one in which the proportion of polynuclears is either normal or only slightly in excess. Further—and this is the most important practical point—this kind of fever yields rapidly to large doses of ipecacuanha in the absence of symptoms of dysentery or even of hepatitis, and the formation of tropical abscess of the liver is thus prevented. If this proposition can be substantiated a great advance will be made in the prevention of one of the most dangerous diseases of certain tropical countries, and I wish to bring before you this evening notes and charts of some of the cases on which the foregoing statements are based.

For convenience of reference all the cases of non-suppurative hepatitis met with in the Calcutta European Hospital fever series during the year subsequent to those

dealt with in the previous paper already referred to (1), on blood counts in amœbic hepatitis, have been embodied in the accompanying table and classed in accordance with, whether they showed clinical symptoms of dysentery and hepatitis, hepatitis alone, or no definite signs of either at the time the blood examination was made. The patients were under the care of Drs. B. Chatterton, J. C. H. Leicester and J. G. Murray, all of the Indian Medical Service, to whom I am greatly indebted for permission to examine the blood and make use of the hospital notes.

I. Cases of Acute Hepatitis complicated with Dysentery.

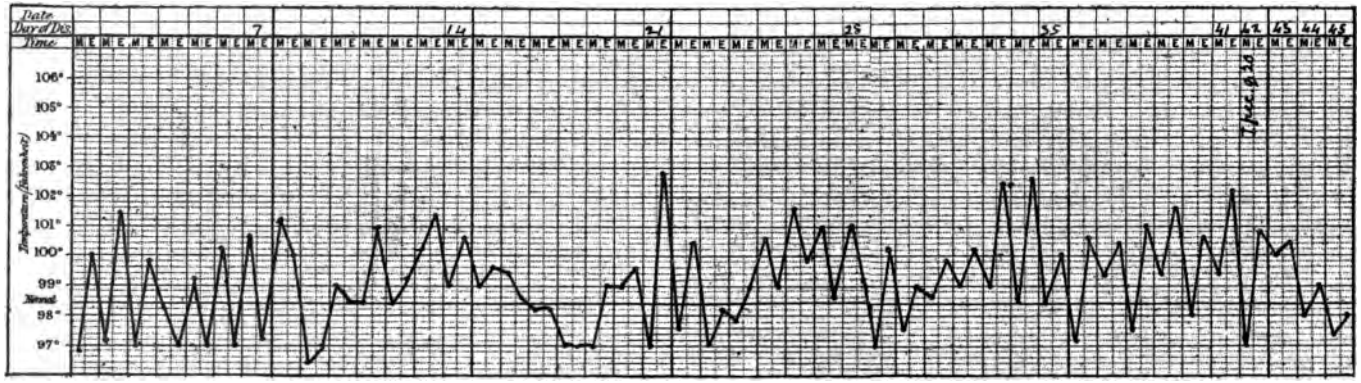
Beginning with the simplest and most straightforward cases, we first have Nos. 1 to 3, in which an acute hepatitis followed symptoms of dysentery, leucocytosis being present in each, while as the liver was also enlarged and very tender, liver abscess was suspected, and in No. 3 exploratory puncture of the liver in six places was done with a negative result. In all three cases the ipecacuanha treatment was followed by cessation of the fever and other acute symptoms within from two to four days, although they had been present for 15, 34 and 41 days respectively before this drug was given in large doses. The following are the principal points of interest in these cases, the temperature charts of which I show you on the screen.

Case 1.—A male, aged 38, who had been in hospital two and a half months previously for a slight attack of hepatitis, which yielded to ammonium chloride treatment in three days. He had suffered from dysentery on and off for seven months before this first attack of hepatitis, and on readmission was passing mucus and blood, but showed no signs of hepatitis. The dysentery improved under bismuth, but the irregular intermittent fever continued in spite of quinine, and one month after he came

Table of Blood Counts and Ipecacuanha Treatment of Pre-suppurative Amœbic Hepatitis.

No.	Sex.	Age.	Bowels.	Liver. Inches below costal margin.	Liver.	Days' fever before Ipecacuanha treatment.	Days' fever after Ipecacuanha treatment.	Red corpuscles.	White corpurcles.	Ratio of white to red.	Polynuclears.	Lymphocytes.	Large mononuclears.	Eosinophiles.	Remarks.
I. Cases with Dysentery and Hepatitis treated with Ipecacuanha.															
1	M.	38	Dysentery	1	Tender	41	2	3,600,000	15,250	1–236	83	13	4	0	—
2	M.	31	,,	2½	,,	15	4	—	Leucocytosis	—	78	16	2	4	—
3	M.	33	,,	1½	,,	34	3	—	,,	—	79	12	6	3	Aspirated.
II. Cases of Hepatitis without Dysentery treated with Ipecacuanha.															
4	M.	38	Normal	½	Tender	49	—	3,090,000	24,000	1–129	78	15	7	0	Aspirated.
5	M.	45	,,	2	,,	39	—	3,610,000	28,500	1–126	76	21	3	0	,,
6	M.	34	,,	½	,,	34	—	3,960,000	9,500	1–416	78	8	3	2	—
III. Cases of Hepatitis only without Dysentery treated with Ipecacuanha.															
7	M.	40	Irregular	1½	Tender	15	1	4,820,000	21,000	1–230	78	15	5	2	—
8	M.	38	Normal	1	,,	14	4	4,600,000	17,750	1–233	87	7	5	1	—
9	M.	31	Diarrhœa	0	,,	25	5	—	Leucocytosis	—	80	15	4	1	—
10	M.	35	Constipated	2½	,,	50	5	5,380,000	17,000	1–316	76	16	6	2	Aspirated.
11	M.	38	Normal	0	,,	13	6	4,910,000	9,500	1–517	79	16	4	1	—
IV. Cases without Dysentery or Liver Symptoms treated with Ipecacuanha.															
12	M.	40	Normal	1	Normal	6	—	4,290,000	15,250	1–272	81	11	7	1	Alcoholism.
13	M.	28	,,	¼	,,	53	15	—	Leucocytosis	—	—	—	—	—	Aspirated.
14	M.	24	Irregular	1	,,	35	2	4,660,000	20,750	1–225	—	—	—	—	—
15	M.	32	Constipated	0	,,	45	12	3,340,000	17,250	1–193	74	22	3	1	—

CHART I (Case 1).



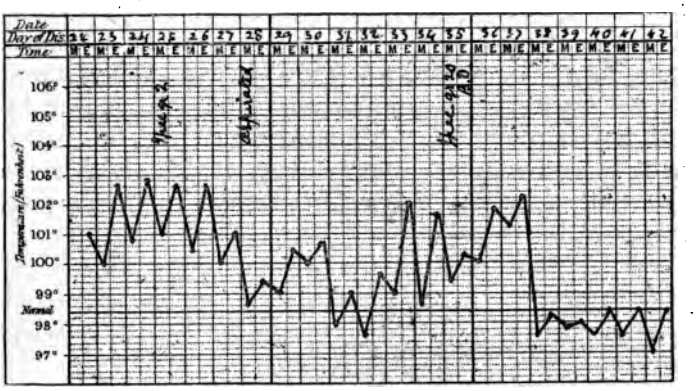
European, male, aged 38. Dysentery followed by hepatitis. Fever forty-one days before and for two days after ipecacuanha treatment.

into hospital I found slight increase of the leucocytes not amounting to an actual leucocytosis. Six days later, his liver having become tender, a second examination showed 15,250 white corpuscles per cubic millimetre, a leucocytosis having now developed. Although all active symptoms of dysentery had long ceased, I suspected a latent form of the disease as the cause of the hepatitis, and advised the ipecacuanha treatment, which was followed by a permanent cessation of the fever within two days as seen from Chart I, although the case was just one of those which ordinarily drift on into liver abscess.

Case 2.—This man was also admitted for dysentery, together with signs of acute hepatitis and a leucocytosis, but the remittent temperature fell to normal in four days under ipecacuanha, although there was a slight relapse of the dysenteric symptoms four days later, but at the end of sixteen days the fever finally left him. A Widal test, with Shiga's bacillus gave a negative result in a dilution of 1 in 20, so the dysentery was probably amœbic in nature. This case was a straightforward one of dysentery followed by hepatitis yielding to the well-known ipecacuanha treatment as advised, when dysentery is present, by Sir Patrick Manson and other authorities.

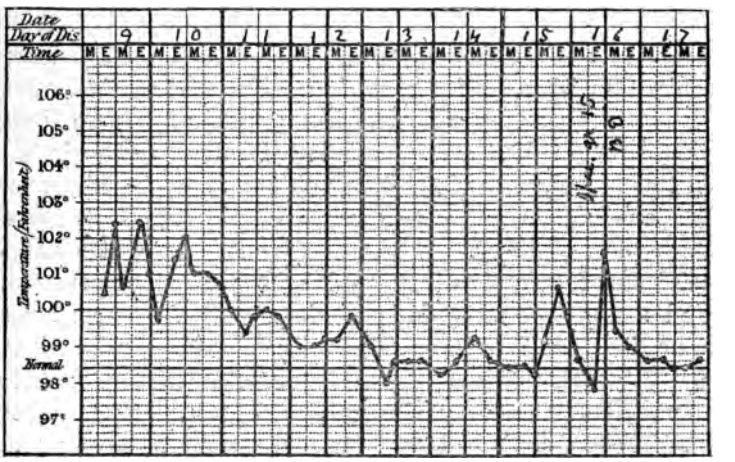
Case 3.—A male, aged 33, whose illness began three weeks before admission with dysentery, lasting for two weeks. He was passing loose greenish stools, while his liver was enlarged and tender, and leucocytosis was present. X rays showed loss of movement of the diaphragm on the right side, and, liver abscess being suspected, the organ was punctured in six places under chloroform with a negative result. He had been given ipecacuanha in 2-grain doses, with only some lessening of the pyrexia resulting, but two weeks after admission he was given 20-grain doses twice a day, and on the third day his temperature finally fell to normal, and he rapidly recovered. (Chart II.)

CHART II (Case 3).



European, male, aged 33. Hepatitis following dysentery. Aspirated for liver abscess with negative result. Fever and hepatitis ceased in three days under ipecacuanha treatment.

CHART III (Case 7).



European, male, aged 40. Hepatitis without dysentery for fifteen days. Symptoms disappeared in one day under ipecacuanha treatment.

Remarks on Group I.—These three cases illustrate the well-known treatment of cases of dysentery complicated by acute hepatitis by large doses of ipecacuanha, although the rapidity with which the hepatitis with a definite leucocytosis yielded to the drug in spite of the dysenteric symptoms being in abeyance in two of them is noteworthy, for it is just such cases which so commonly drift on into the suppurative stage of the disease if this treatment is neglected, as it too often is in the tropics at the present day.

II. Cases of Hepatitis without Dysentery not Treated with Ipecacuanha.

Next I come to cases of acute hepatitis without any recent dysentery, eight in number, which I have divided up in accordance with whether they were treated with large doses of ipecacuanha or not, and will first deal with the three cases not treated with this drug (Nos. 4 to 6).

Case 4.—A seaman, aged 38, admitted for acute hepatitis with a leucocytosis, but no dysentery. X rays showed reduced movement of the diaphragm on the right side, so eight days after admission the liver was aspirated in two places, only blood being obtained, so the abdomen was opened, the organ palpated, and punctured in three more places, also with a negative result. Irregular intermittent fever continued for twelve days after the operation, and a few days after the pyrexia ceased the leucocytosis was also found to have disappeared. He continued to get occasional slight rises of temperature for another month, but eventually left hospital apparently well after sixty-two days under treatment, and as he went to England I was unable to follow up his case further, so cannot say if he eventually developed a liver abscess or not. He was not treated with large doses of ipecacuanha while in hospital, and his recovery was a very protracted one.

Case 5.—A male, aged 45, admitted for low intermittent fever and hepatitis, his blood showing a very marked leucocytosis, 28,500 per cubic millimetre, and whose liver was also punctured in several places without any pus being found. The pyrexia ceased after thirty-nine days, and he left hospital at his own request much improved after fifty-seven days' stay. He was readmitted for another much slighter attack of hepatitis ten months later.

Case 6.—Admitted for mild hepatitis with low intermittent fever up to about 100° only, which lasted for thirty-four days under quinine treatment without any very acute symptoms appearing. There was no leucocytosis in this case which was probably alcoholic in origin, but is included, as I wish to bring before you a consecutive series of unselected cases.

Remarks on Group II.—The first two of this group are typical of the class of acute hepatitis without dysentery, in which leucocytosis pointing to liver abscess is present, and exploratory operation becomes necessary to decide if a liver abscess has formed or not. A negative result being not uncommonly followed by a slow improvement and ultimate recovery, although they are apt to relapse and an abscess is frequently found at a later date. They will serve for comparison with the next group of similar cases in which the ipecacuanha treatment was carried out.

III. Cases of Hepatitis without Dysentery Treated with Ipecacuanha.

Cases 7 to 11 all presented signs of acute hepatitis without any recent dysentery, four out of the five also showing marked leucocytosis. They were all treated with ipecacuanha.

Case 7.—Had been in hospital for a short attack of hepatitis four months before. No history of dysentery,

but had suffered occasionally from a watery diarrhœa alternating with constipation—a not infrequent occurrence in amœbic hepatitis. He suffered from fever for fifteen days of a low remittent and intermittent form, at the end of which he was put on the ipecacuanha treatment, and two days later his temperature finally fell to normal, and he made a good recovery, returning to work after thirty-two days in hospital. However, he returned five months later with a liver abscess, which was cured by aspiration and injection of quinine into the cavity as already reported (2), after which he went to work again and remained quite free from fever for eight weeks, when he returned with a second abscess in a different part of the liver to the one which had been injected with the quinine. A similar treatment was tried on the fresh abscess, but this time it failed, and eventually it had to be opened and drained, since which he has had no return of his trouble. (Chart III.)

In connection with this method of treating early cases of liver abscess by aspiration and injection of soluble bihydrochloride of quinine into the cavity to kill the amœba when it is found to be free from bacteria, I regret to have to report that in three further cases, treated by Captain J. G. Murray, I.M.S., at the General Hospital, Calcutta, it failed, and the open operation had to be resorted to, and I am informed that in Bombay a similar experience was obtained in two cases, so that it has not fulfilled my anticipation that it would succeed in most of the cases of tropical abscess. On the other hand, I have been informed of several cases in which it has proved successful in other hands than my own, so in view of the rapid and comparatively painless cures it sometimes effects, and its harmlessness in our Calcutta experience in those cases which have eventually to be cut open, I still think it is worthy of trial in early, deep-seated, amœbic abscesses of the liver.

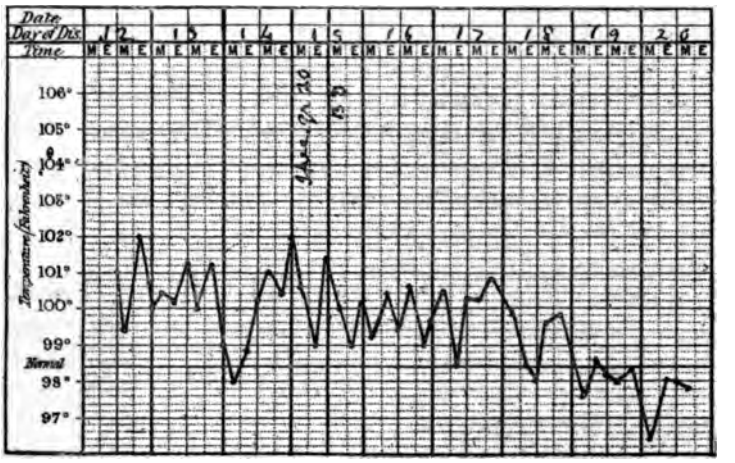
CASE 8.—A male, aged 38, admitted for acute hepatitis without any history of dysentery, and no present bowel trouble. His temperature showed the rapid oscillations produced by the frequent profuse perspirations so common in acute liver inflammation, a marked leucocytosis was present, and he had occasional rigors. Nevertheless, the pyrexia and all the acute symptoms yielded in four days to 20 grain doses of ipecacuanha morning and evening (Chart IV).

CASE 9.—A male, aged 31, admitted with a subacute hepatitis, with low intermittent fever, watery diarrhœa, and a slight degree of leucocytosis, all the symptoms disappearing after five days of the beginning of the ipecacuanha treatment.

CASE 10.—A male, aged 35, admitted for acute hepatitis with constipation and low intermittent fever, which improved for a time under quinine, only to return shortly after. A month after his admission I was asked to examine the blood, and found 17,750 leucocytes per cubic millimetre, so the liver was punctured with a negative result. He was then put on 5 grains of ipecacuanha three times a day, and four days later was injected with antistreptococcus serum, the temperature finally falling the next day, and he made a good recovery. In this case it is difficult to say whether the happy result was due to the ipecacuanha or to the serum, but I am inclined to think it was the former, because two and a half months later he returned with an amœbic abscess of the liver, which was opened and drained with an ultimately fatal result; so that his earlier attack was pretty certainly also amœbic in origin, and so likely to have yielded for a time to the small doses of ipecacuanha given (Chart V).

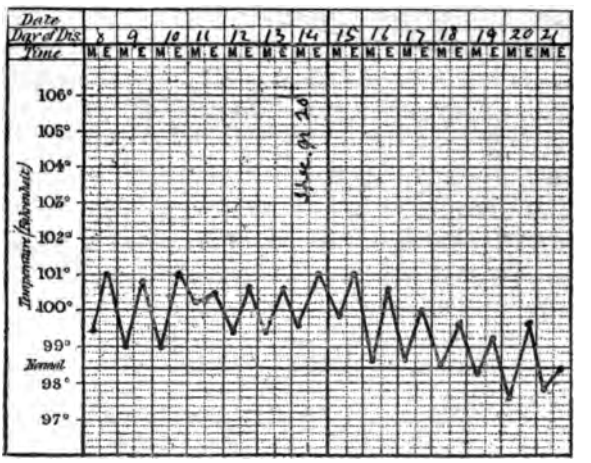
CASE 11.—Male, aged 38, who had suffered from dysentery twelve years before. Bowels now normal, but symptoms of acute hepatitis without leucocytosis and low

CHART IV (Case 8).



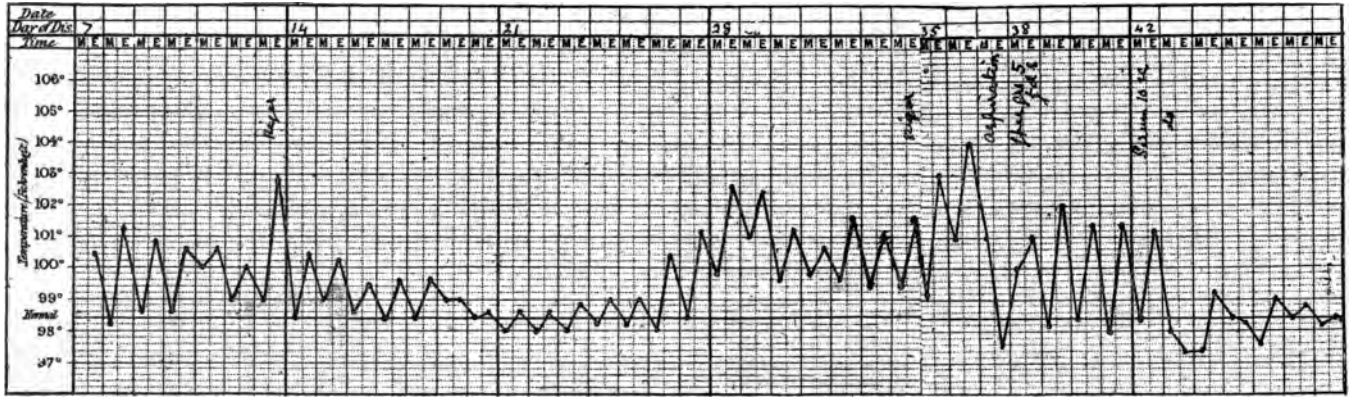
European, male, aged 38. Acute hepatitis without dysentery for fourteen days. Leucocytosis present. Hepatitis and fever ceased in four days under ipecacuanha treatment.

CHART VI (Case 11).



European, male, aged 38. Dysentery twelve years ago only. Acute hepatitis for thirteen days, ceasing in six days under ipecacuanha treatment.

CHART V (Case 10).



European, male, aged 31. Acute hepatitis without dysentery for thirty-seven days. Leucocytosis present. Liver aspirated with negative result. Fever and hepatitis ceased five days after ipecacuanha treatment and one day after antistreptococcus serum.

remittent fever, which declined when the ipecacuanha treatment was commenced, and finally ceased after six days, the case being a somewhat mild one (Chart VI).

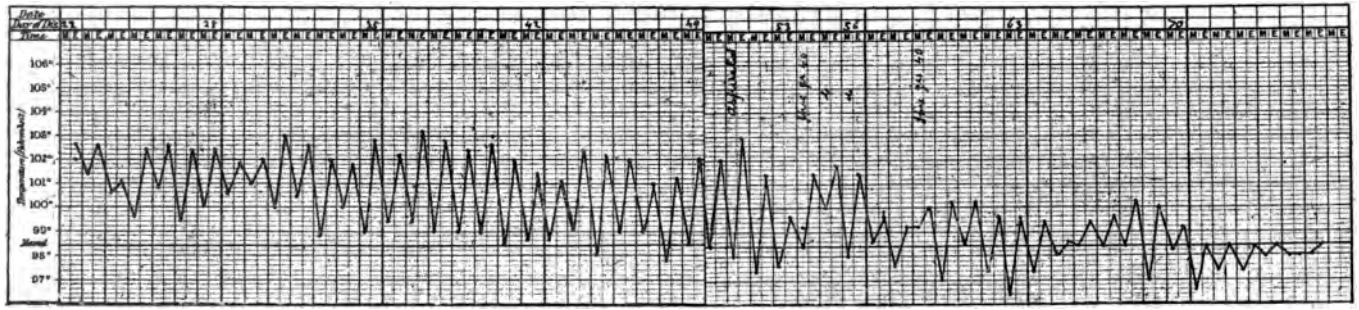
Remarks on Group III.—These cases of hepatitis without dysentery are precisely similar to those of Group II, but the pyrexia and other symptoms rapidly ceased under the ipecacuanha treatment within from one to six days, although they had previously been present for from thirteen to thirty-seven days, and in No. 10 aspiration had been done on account of a suspicion of liver abscess being present. In short, the effect of this treatment was precisely the same as in the first three cases in which symptoms of dysentery as well as hepatitis had been evident.

IV. Cases without Dysentery or Liver Symptoms treated with Ipecacuanha.

Cases 12 to 15 all showed leucocytosis without symptoms of either dysentery or acute hepatitis being present, fever, for which no definite cause could be found, being the condition for which they were admitted. As there was no obvious cause for the leucocytosis I suspected that latent amœbic dysentery and the insidious form of hepatitis which sometimes precedes actual abscess formation might be present. I therefore suggested a trial of the ipecacuanha treatment, which had proved so efficient in the more acute forms of amœbic hepatitis, although there was neither pain nor tenderness of this organ present when they were first admitted, in spite of slight enlargement of the liver in three of the four patients. The results were most satisfactory.

Case 12.—A seaman, aged 40, with alcoholic history, whose pyrexia ceased after six days without ipecacuanha, but who was subsequently treated with it on account of the presence of leucocytosis in order to try and prevent a recurrence of the liver trouble, as the fall of temperature

CHART VII (Case 13).



European, male, aged 28. Fever without any symptoms of dysentery or hepatitis for fifty-three days. Leucocytosis found and liver aspirated with negative result. Fever ceased in a few days under ipecacuanha treatment.

might have been temporary improvement only. This case must be acknowledged to be a doubtful one.

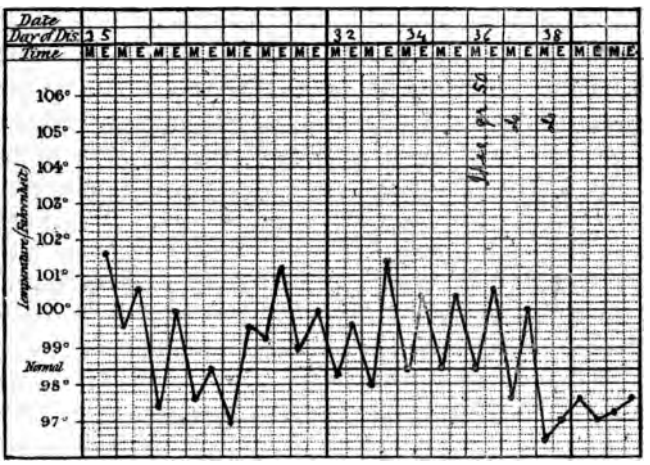
CASE 13.—Male, aged 28, admitted for fever of three weeks' duration, which persisted under quinine treatment, although no cause could be found for it. Bowels normal, both sides of the diaphragm moving well, as seen by the X-rays. Liver slightly enlarged, but not tender, although becoming larger. Leucocytosis was found by me both one week and three weeks after admission, so the liver was explored for abscess with a negative result. Three days later, or fifty-three days after the fever commenced, the ipecacuanha treatment was commenced, and on the fourth day the temperature was normal, but slight low fever up to 100° F. recurred for several days more, after which convalescence set in, as shown in Chart VII.

This was a most remarkable case, as the nature and cause of the fever was quite a puzzle until the presence of leucocytosis lead to a suspicion that latent amœbic dysentery and hepatitis might be at the bottom of it, while the rapid success of the ipecacuanha treatment in such a persistent fever appears to me to lend some support to the correctness of this view of the case.

CASE 14.—Male, aged 24, admitted for irregular intermittent fever, not yielding to quinine, and with no obvious cause. As over 20,750 leucocytes were found the ipecacuanha treatment was again adopted with the happiest results, the fever finally ceasing two days later, and convalescence being quickly established, as in the preceding case (Chart VIII).

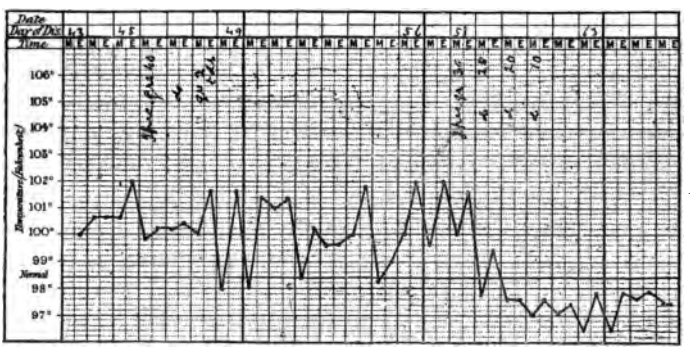
CASE 15.—Male, aged 32, admitted for fever with no ascertainable cause, and resistent to quinine. Bowels constipated; diaphragm moving well. A well-marked leucocytosis was found, so 40 grains of ipecacuanha were given on two consecutive days followed by 2 grains twice a day, but with apparently very little effect on the fever.

CHART VIII (Case 14).



European, male, aged 24. Fever without dysentery or hepatitis. Leucocytosis found. Fever ceased in two days under ipecacuanha treatment.

CHART IX (Case 15).



European, male, aged 32. Fever without dysentery or hepatitis. Leucocytosis found, and fever ceased under ipecacuanha treatment.

Hypodermic quinine was then tried without result, and on the sixteenth day after admission 30 grains of ipecacuanha was again administered, and repeated in doses diminished by 5 grains each day, which treatment was followed by a final cessation of the pyrexia in two days (Chart IX). In this case the fever had lasted for forty-five days before the first trial of ipecacuanha, so that it is, perhaps, not surprising that two large doses failed to stop it, and it had to be repeated again a few days later. The ultimate result was as satisfactory as in the previous cases, so that I do not think the repeated apparently good results of this treatment can be simply a coincidence.

Remarks on Group IV.—The remarkable feature of these last four cases is that there were no bowel or liver symptoms to make one suspect the presence of amœbic hepatitis, until an otherwise unexplainable leucocytosis was found on examining the blood. Yet on administering large doses of ipecacuanha in three of the cases after the fever had resisted quinine and other treatment for thirty-five, forty-five, and fifty-three days respectively, the pyrexia and other symptoms subsided in much the same way as in the previous groups showing definite symptoms of hepatitis with or without dysentery. Moreover, judging from a prolonged experience of similar cases before this treatment was so commonly used in the Calcutta European Hospital, this class of patients do not rarely drift on into liver abscess formation if their true nature is not discovered and the ipecacuanha method vigorously used, while I know of nothing but the presence of leucocytosis which will allow of their nature being suspected in this early stage when they are so readily amenable to medical treatment. I may add that several very similar cases seen in consultation have furnished equally satisfactory results under ipecacuanha to those dealt with above.

The Type of the Leucocytosis in Amœbic Hepatitis.

In the paper already referred to I pointed out that the leucocytosis in amœbic abscess is somewhat peculiar in that the proportion of the polynuclears is commonly comparatively little, if at all, increased, as it is in ordinary suppurative conditions. A reference to the table will show that a similar type is present as a general rule in the pre-suppurative stages of amœbic hepatitis, which is a point in favour of the view that these cases are also secondary to by an amœbic dysentery, usually of a latent nature. Thus, out of thirteen cases in which the differential leucocyte count was done, in none were as many as 90 per cent. of polynuclears present, while in only three were over 80 per cent. found. I have several times found this type a point of diagnostic value in favour of this affection rather than some other suppurative or acute inflammatory condition.

The Importance of always trying the Ipecacuanha Treatment before Operating.

Another striking point in this series is the fact that in no less than one third of them an unsuccessful exploratory operation was undertaken before the disease was rapidly cured by the ipecacuanha treatment, this having been done in several of them at my own suggestion before I had fully realised how often a marked leucocytosis is present in the readily curable early pre-suppurative stage of amœbic hepatitis. In view of the results of that treatment now brought forward I am strongly of the opinion that all cases of this disease should be treated with large and repeated doses of ipecacuanha, whenever there is any doubt remaining as to whether suppuration has already taken place or not, before any exploration of the liver is carried out, for this rule will certainly prevent some unnecessary surgical measures. Moreover, repeated

puncturing of the liver with large aspirating cannulas is by no means without danger, as shown by the number of recorded and still more of unrecorded cases of fatal hæmorrhage following them, while it is especially in these cases of very acute congestion of the organ without actual suppuration that such deplorable deaths have occurred.

The Pathology of the Pre-suppurative Stage of Amœbic Hepatitis.

The explanation of the series of cases just described remains to be considered, and I can best make it clear by beginning with the most straightforward cases and proceeding to the less definite ones, much in the manner in which my present views have gradually become evolved as fresh facts accumulated.

The very favourable position for the study of liver abscess in the tropics which I have long enjoyed, enables me to record in 1902 (3), that I had found living amœba in thirty-five consecutive cases of liver abscess in which scrapings of the wall were examined within less than twelve days of the cavity being opened, while the great majority of them were otherwise sterile when first incised. Further, I showed that, when both the clinical history and post-mortem records of cases were available, dysentery had been noted in 90 per cent. of the cases, always, in my experience, of the amœbic type. In 18 per cent. of the cases the clinical history of dysentery was negative, but nevertheless dysenteric ulcers were found in the large bowel after death, the disease having been present in a latent form ; a sequence of events which has since then repeatedly come within my knowledge. I therefore hold that the amœba dysenterica is constantly present in early amœbic abscess of the liver, although it may very occasionally die out in chronic cases, and that the liver affection is secondary to amœbic dysentery, although this may frequently be present in a latent form and give rise to no

typical clinical symptoms, owing to the ulcers being limited to the cæcum and ascending colon (4).

Now, if the above statements are well founded, then it follows that in the pre-suppurative stages of amœbic hepatitis, in the absence of any clinical history or actual symptoms of dysentery, there must be a latent form of that disease present giving rise to irritation of the liver on account of these protozoal organisms reaching the organ mainly by the portal circulation, and if this source of irritation can be removed the hepatitis should subside. Now it is in just those tropical climates where liver abscess and amœbic dysentery occur that ipecacuanha is looked on as a specific in many cases of dysentery, while I have been informed by several medical men with experience of dysentery in countries where amœbic abscess of the liver is not seen, that this drug is useless in the dysenteries of bacterial origin which they there had to deal with. Personally I look on ipecacuanha as invaluable in the treatment of amœbic dysentery, in fact as a specific against that disease, and in Lower Bengal, where amœbic liver abscess is so common, I regard this drug as second only in importance to quinine itself. If this is so it is easy to understand how large doses of ipecacuanha (not less than 20 to 40 grains once or twice a day some twenty minutes after a dose of tincture of opium) may rapidly abort an early pre-suppurative amœbic hepatitis by curing the latent amœbic dysentery that produces it, although I find no recommendation of the drug in some of the standard works on tropical medicine in acute hepatitis, except when symptoms of dysentery are actually present. The cases narrated are, I venture to think, sufficiently striking to warrant the general extension of the ipecacuanha treatment as a routine method in cases of hepatitis in countries where amœbic abscess of the liver occurs, in the absence of actual history or symptoms of dysentery, and that this treatment should always be given a trial before operative measures are undertaken in all

cases in which any doubt remains as to whether actual suppuration has yet occurred.

Lastly, we also know that amœbic abscess of the liver may arise sometimes in a most insidious manner, there being no very definite indication of hepatitis for a long time in spite of persistent fever. In such cases we may also have a latent amœbic dysentery as the exciting cause of the chronic fever, so that it is but one step further to treat these also with large doses of ipecacuanha as soon as the nature of the case can be determined, while I have shown that they may often at least be suspected from the presence of the type of leucocytosis already mentioned. The rapid cessation of the previously persistent intermittent fever without symptoms of hepatitis under such treatment in some of the cases narrated is the best evidence of the correctness of this argument. I am sanguine enough to hope that when these methods of early diagnosis and prompt ipecacuanha treatment of the pre-suppurative stages of amœbic hepatitis become generally known and practised, much will be done to lessen the occurrence of tropical abscess of the liver, with its very high mortality and prolonged suffering, especially in the case of Europeans, who come early under observation for the fever which so constantly precedes, often for several weeks, actual breaking down of the liver substance.

References.

(1) 'Brit. Med. Journ.,' November 11th, 1905.
(2) 'Brit. Med. Journ.,' June 16th, 1906.
(3) 'Brit. Med. Journ.,' September 20th, 1902.
(4) 'Brit. Med. Journ.,' June 6th,1903.

DISCUSSION.

Sir PATRICK MANSON thought that the statement of Professor Rogers as to the value of ipecacuanha in relation to amœbic hepatitis was of great value. There were two schools in regard to the treatment of dysentery: those who believed in the administration of the aperient sulphates, and those who advocated large doses of ipecacuanha. The term dysentery should be regarded, not as indicating a disease, but as indicating a group of symptoms. There were many kinds of dysentery—amœbic, bacillary, and others—all of which demanded different kinds of treatment. Ipecacuanha, he held, was a specific for amœbic dysentery, but of no value in the bacillary variety for which the sulphates were probably useful. Much depended on the way ipecacuanha was given in amœbic dysentery. It had fallen into disrepute, because it had not been given in sufficient quantity or over long enough periods. His own practice was to give it in large doses for a week, and in smaller doses for at least a month longer, and on these lines he had met with much success in the amœbic form. He had never used it definitely for the purpose of curing amœbic hepatitis, but by its accidental use he had had some successes. In future he should make use of it in hepatitis and threatened liver abscesses.

Sir THOMAS LAUDER BRUNTON.—From a therapeutical point of view Professor Rogers's communication is most interesting, and it tends to remove one more remedy from the empirical to the rational class. For a long time quinine was used as an empirical remedy, but was put into the class of rational remedies when it became known that the disease depended upon a certain parasite to which the drug acted as a poison. Dr. Rogers's observations appear to show that the success of ipecacuanha in dysentery is due to a similar cause. The reason he has given for its use will no doubt lead to a more prolonged trial than has hitherto been given to ipecacuanha in dysentery. When it was administered without any definite idea as to its mode of action, its use was frequently discontinued after three or four doses had been given without any apparent good result. I may perhaps mention a small point in regard to its administration which I found out in my own case. Thirty grains of ipecacuanha, even when opium had been previously administered, generally caused considerable nausea. If I yielded to the desire to vomit and sat up, the whole contents of the stomach were evacuated, but if I kept my head down and put an ordinary dinner plate close to my lips, so that anything expelled by the stomach could simply

be passed out without raising the head, I was able to retain most of the ipecacuanha instead of ejecting it.

Dr. JOHN ANDERSON, C.I.E.—Professor Rogers's paper has been to me equally interesting and instructive. The idea that the suppuration stage of a hepatitis, that is to be diagnosed by a certain type or degree of leucocytosis, can be arrested or prevented by repeated large doses of ipecacuanha is extremely interesting and valuable. It is so new that I presume no one has any experience of it except Professor Rogers himself; but he has shown us some of the temperature charts of his cases. The use of ipecacuanha in amœbic dysentery we are all familiar with, and must have experienced very satisfactory results from its use; but now that Professor Rogers has given us a means of diagnosing the disease, while even in a latent stage, and preventing the common complication of hepatic abscess, the value of the remedy is vastly increased. There is, of course, one great difficulty in the use of large doses of ipecacuanha, viz. its emetic action, and the very distressing and depressing effect produced thereby. I may perhaps mention one method of using it that I do not think is very generally known, and that in my experience has been preferable to giving a dose of opium antecedent to the drug: I refer to the use of chloral hydrate given *with* the ipecacuanha in 10, 15, or even 20 grain doses. This, of course, is only a matter of detail; but as I have found it successful in relieving or preventing distress to the patient it is, I think, worthy of note.

Mr. JAMES CANTLIE thought it necessary to remember that the temperature was often caused to fall, and all the symptoms disappear, in cases of hepatitis by putting in a needle alone, even when nothing but blood was withdrawn. A dose of ipecacuanha often caused the passing of a solid stool loaded with bile, suggesting a direct stimulating effect on the liver. In almost all the cases in the paper an exploratory needle had been introduced, and it was after this that the temperature fell. Almost all the cases of chronic dysentery met with in this country were of the amœbic form, and for this ipecacuanha was a specific.

Dr. ROGERS, in reply, said, I desire, in the first place, to thank those who have spoken for their opinions. The endorsement by Sir Patrick Manson of my view that the ipecacuanha treatment is too much neglected in the tropics at the present time, and that it should be extended to cases of hepatitis without symptoms of dysentery is most important, and will go far to ensure the more general adoption of this method. I am also in entire agreement with his advocacy of the use, this drug being given a more prolonged trial in these cases, as the lesions in amœbic dysentery extend deeply into the sub-mucous coat of the large bowel, so that it cannot be expected that the disease will be permanently cured by one or two doses of the drug; a view which

is borne out by the frequency with which these cases relapse. The valuable suggestions of Sir Lauder Brunton and Dr. Anderson regarding the administration of ipecacuanha will be of material help in extending this somewhat unpleasant therapeutic measure. With regard to Mr. Cantlie's remark that puncture of the liver is sometimes followed by relief of the symptoms of acute hepatitis, and may therefore be advised in the place of the ipecacuanha treatment in some cases, I would point out that in no one of the five cases narrated, in which exploratory puncture was done, did any material benefit or permanent fall of temperature shortly follow this surgical measure. In view, therefore, of the lamentable deaths which sometimes follow this operation —one observer in Bombay alone having recorded six fatal cases— I am most strongly of the opinion that puncture of the liver should never be performed in cases of acute hepatitis, unless there is absolutely clear evidence of abscess formation, without the ipecacuanha treatment being first resorted to.

A COMMON SPORADIC SEVEN-DAY FEVER OF INDIAN PORTS SIMULATING DENGUE

BY

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In order to ascertain what proportion of fevers in Europeans in Calcutta are malarial, and if any other short fevers commonly occur there, I have examined blood-slides taken on admission before quinine was given, and kept notes and charts of every fever case in the large European hospital for two complete years, 1350 consecutive cases being thus worked out. Commencing in the middle of September I found that almost all the short fevers for the first seven months—that is up to the end of April, showed malarial parasites in the blood, although even the malarial cases had become very few since the end of January. In May, and still more from June to September, fevers of short duration began to increase once more, but now malarial parasites were conspicuous by their absence, and it soon became clear that the type and symptoms of the fevers then seen were quite different from those of the malarial series, and more closely resembled in their general characters influenza and dengue. During the following year of 1906 a very similar but shorter prevalence was again noted, while an examination of the hospital

records for several previous years showed that this fever always appeared at about the same time in greater or smaller numbers year by year, while its seasonal incidence was quite distinct from that of true malarial cases. I published a short account of this newly separated fever in the 'Indian Medical Gazette' of November, 1905 (1), and dealt with its seasonal incidence and differentiation from malaria in the same journal in March, 1906 (2), since which I have had much further experience of the disease and studied both its distribution in India and the literature of allied affections, and so am anxious to bring it up for discussion by this Society.

Clinical Description of Seven-Day Fever.

Onset.—The onset of the fever is almost invariably quite sudden, and frequently commences with chilliness, and not rarely with a rigor, which, in a few exceptional cases, may recur, but not with the regularity of a true malarial fever. I have known a hospital patient convalescent from some other disease to be suddenly attacked with seven-day fever while out for a drive, his temperature being found elevated on his return. The onset is thus more sudden than is usual in typhoid fever.

Appearance on admission.—I shall show that about half the cases of this fever seen in Calcutta were admitted during the terminal rise about the fourth or fifth day of the fever, but in those who come in an early stage, or are attacked while in hospital, the following points may be noted: The face was usually flushed, and the conjunctiva presented a vivid red appearance, while the general expression was often dull and listless, and highly suggestive of early typhoid, and in several cases the addition of some abdominal pain and a few suspicious spots gave rise to such a strong suspicion of that disease that a Widal test was performed with a negative result, which was soon confirmed by the early cessation of the fever. Moreover, I have frequently seen such typhoid-like cases in consultation,

and I regard their occurrence as a point of great importance in separating this sporadic seven-day fever from epidemic dengue, which it closely simulates in some points, for I can find no description of typhoid-like cases in dengue, although they could not well have been overlooked in that disease if the two fevers are one and the same.

Headache.—Severe headache, almost invariably frontal, is one of the most constant symptoms accompanying the onset of the fever, often together with pain in the back of the eyes, so that when asked to point out exactly where the pain is felt, the patient places his fore-finger and thumb one on each side just behind the external orbital processes of the frontal bone. The headache in malaria is less frequent, and more variable in position than that of seven-day fever.

Pains in the back and limbs.—In addition to headache, pains in the back, and, only slightly less frequently, in the limbs as well, are almost constantly present, while occasionally they are described as occurring in the joints, but without any local swelling or tenderness. They generally come on suddenly with the initial fever, and may be sufficiently severe to disturb the night's sleep, but according to several medical men with a personal experience of both seven-day fever and dengue (who expressed their opinions at a discussion on the subject at the recently instituted Medical Section of the Asiatic Society of Bengal), the pains in the sporadic fever are nothing like as severe and intractable as those of true epidemic dengue. They more closely resemble those of influenza, and are more constant and severe than the aching pains of malarial fevers.

Alimentary system ; tongue.—Another very characteristic feature is the appearance of the tongue, the dorsum being furred, often markedly so, while the edges are raw and red, and a "strawberry" appearance may be seen. This condition is in marked contrast to the uniform, slight furring usually met with in malarial fevers, but, on the

other hand, it resembles that of influenza and dengue, from which the diagnosis may be equally difficult.

Sickness.—This symptom was not frequently recorded—much less so than in malaria—so its absence is a point in the differential diagnosis against the latter disease.

Bowels.—There were no constant features regarding the bowels, which were usually regular, while constipation and slight looseness were about equally frequently met with in the remainder. Pain in or slight distension of the abdomen was also occasionally noted.

The liver.—Only very rarely was the liver slightly enlarged, and no symptoms referable to derangement of this organ were met with.

The spleen.—This organ was very rarely enlarged, and then only to a slight degree, so that increase in the size of this organ is in favour of malarial as against seven-day fever.

Respiratory system.—Symptoms referable to the lungs were conspicious by their absence, very slight bronchitis being only very rarely seen, while in no case did pneumonia or other acute inflammatory complications make their appearance. Coryza was equally rare, so much so that I failed to find a case showing this symptom from which to try to isolate the influenza bacillus.

This absence of respiratory symptoms is most important in differentiating this disease from influenza, which the general distribution of the disease and its pains caused it to resemble so closely that in my preliminary account I described it provisionally as "a peculiar type of influenza-like fever." Since then I have analysed all the cases of influenza admitted to the European Hospital, Calcutta, in the epidemic year 1892, and found that throat and lung symptoms were almost invariably present, while both the seasonal incidence and temperature curve were entirely distinct from those of seven-day fever, which is, therefore, certainly not influenza. The absence of respiratory symptoms is, then, a very important point of differentiation of seven-day fever from influenza.

Circulatory system.—The heart does not appear to suffer in seven-day fever in the way it so commonly does after influenza, and once the pyrexia has ceased convalescence is fairly rapid.

The pulse, however, presents a very important feature, namely a most constant and characteristic slowness relatively to the temperature, just as is so often seen in typhoid fever. At the very beginning it may be fairly rapid, and rise to over 100 beats per minute, but once the patient has been placed at rest in bed it rarely rises above that point. During the terminal high rise of the temperature it scarcely ever exceeds 100, being more commonly about 80 to 90 only, while after the temperature falls finally to normal it may be 60 or less. The great practical importance of this feature is that it nearly always allows of the difficult cases only admitted during the terminal rise of temperature being differentiated from true malaria, with which they have hitherto been almost invariably confused, for in the latter condition when the pyrexia rises to 103° or 104° F. the pulse is nearly invariably over 100 per minute, and usually up to 110 or more.

Cutaneous system.—Rashes are occasionally seen in this disease, but were only noted in a small proportion even of those patients who were admitted to hospital in the early stages of the fever. In three cases out of twenty-six consecutive early cases a few small rose spots were detected on the abdomen, while in three more a definite rash appeared on the arms and legs, being best seen on the forearms as a mottled or measly-looking eruption. A more extensive mottled appearance may also rarely occur, and such cases may be very difficult to distinguish from measles, and have, indeed, been admitted into the infectious block as cases of that disease. These rashes are very inconstant, and in this respect differ greatly from the text-book descriptions of the secondary eruption of true epidemic dengue.

The Temperature Curve.

I now come to the course of the fever, which can best be illustrated by the temperature charts thrown on the screen. These vary very much in accordance with the stage of the fever at which the patient comes under observation, so that it was only through careful watching of a long series of cases that I was able to recognise that the different types seen were but variations of a single disease, and so to separate it from "abortive typhoid" and "simple continued fever," so-called, on the one hand, and from malaria on the other, these being the terms under which the cases have hitherto been almost invariably returned in the different parts of India where this fever occurs. Nevertheless, the fever has a most characteristic temperature curve, best described by the term "saddle-back," which it will be well to show first, and then to return to the variations from this typical course.

The typical saddle-back temperature curve.—Chart 1 shows the characteristic temperature curve from beginning to end. The temperature, which was taken every four hours, rose rapidly to over 105° F., and was very little reduced by repeated spongings (as shown by the dotted lines in the chart). It gradually declined during the next two days to about 100° F., at which point it remained steadily for three days before the characteristic terminal rise carried it up again to 103° F., and was succeeded by the final fall occupying twenty-four hours. The pulse was markedly accelerated during the unusually high first rise of the temperature, but during the terminal one it is noteworthy that it was not found to reach over eighty-two beats a minute, with a pyrexia of 103° F.

Chart 2 is a two-hour curve showing how continued the fever was between 100° and 102° F. before the typical terminal rise. The pulse in this case was never recorded as above 100, and never reached even 80 during the terminal rise, in spite of the temperature attaining to over 103° F.—a most characteristic feature of this fever. In

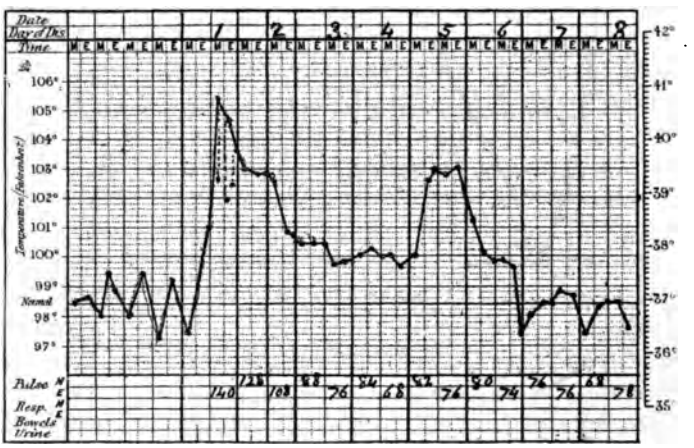
fact, up to the sixth day the pulse and temperature curve might have been those of typhoid, except that in my experience such a saddle-back remission to as low a point as 100° F. is rare in the early stage of typhoid once a higher point has been reached.

Chart 3 again shows the same saddle-back type, but with a still more marked remission to reach 99° F., and a final rise once more to 103° F., accompanied by a pulse never exceeding 100 per minute, except immediately after admission.

One step more to a complete remission of the temperature to the normal point, and we arrive at Chart 4, in which the pyrexia ceased for two complete days, and yet the terminal rise to 103° F. occurred before the final fall to normal. Such a complete remission as this is exceptional, for in five sixths of my cases patients admitted within the first few days the temperature never fell below 99° F. during the usual remission, while in considerably fewer still did it actually reach the normal line, a point in which this seven-day fever differs most essentially from the three-day pyrexia ending by crisis of true epidemic dengue.

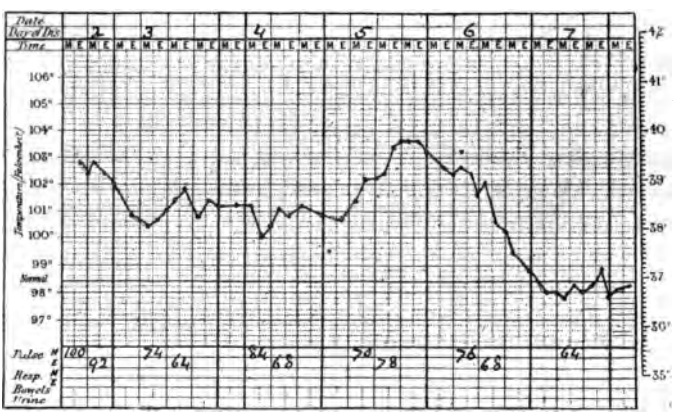
Continued typhoid-like group.—Although the saddle-back temperature curve is by far the most characteristic and frequently met with type in patients admitted in the early days of the fever, still there may be considerable variations from this form, the most important of which is the continued type simulating the early stages of typhoid, for the latter disease by no means very rarely begins fairly abruptly, and without the classical step-like rise in tropical India. Chart 5 is one of the most marked examples of this form I have met with, although I have a number of charts showing an equally continued fever, but usually at a slightly lower level. These continued cases frequently give rise to needless fear of typhoid, but the symptoms already described will, as a rule, allow of a correct diagnosis being arrived at after some experience of the disease. In several private cases in which I was asked to examine the blood for typhoid, I

CHART 1.



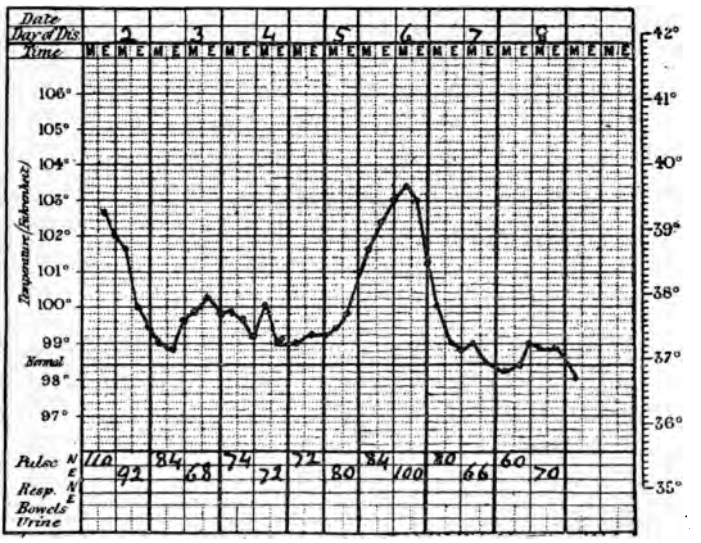
Patient attacked while in hospital, showing complete saddle-back type of fever.

CHART 2.



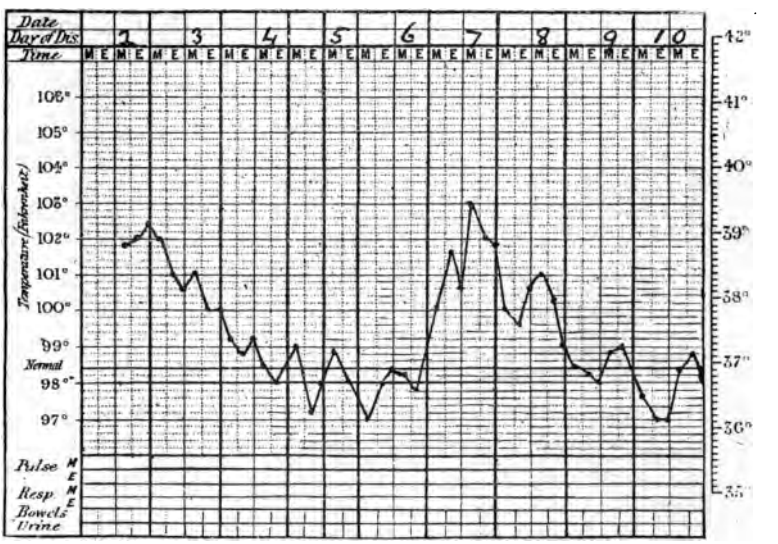
Five-hour chart showing continued type of fever and slow pulse.

CHART 3.



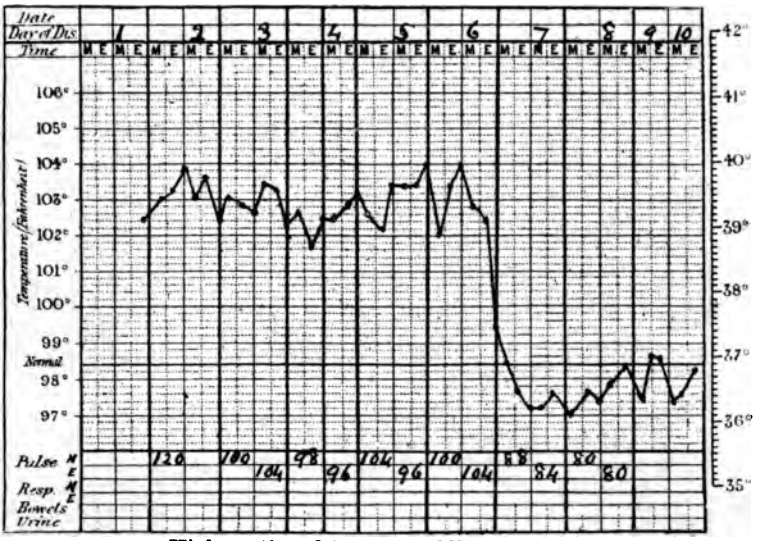
Deep saddle-back remission.

CHART 4.



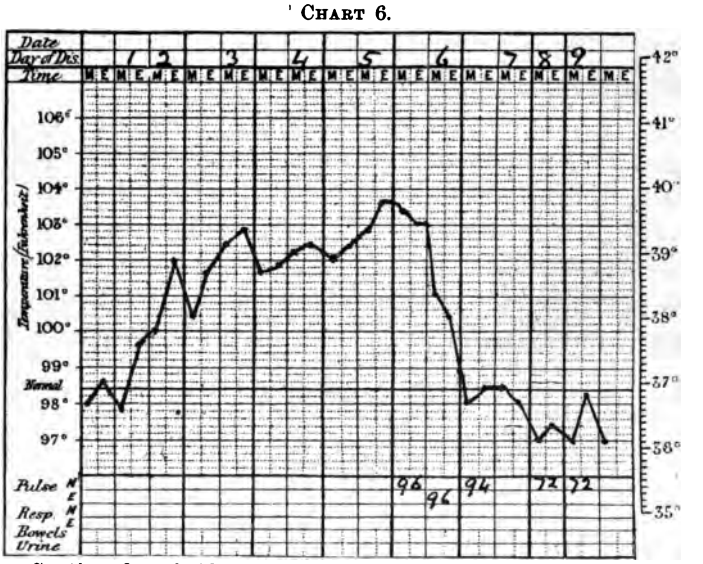
Exceptional remission to normal before the terminal rise, the remission being by lysis.

CHART 5.



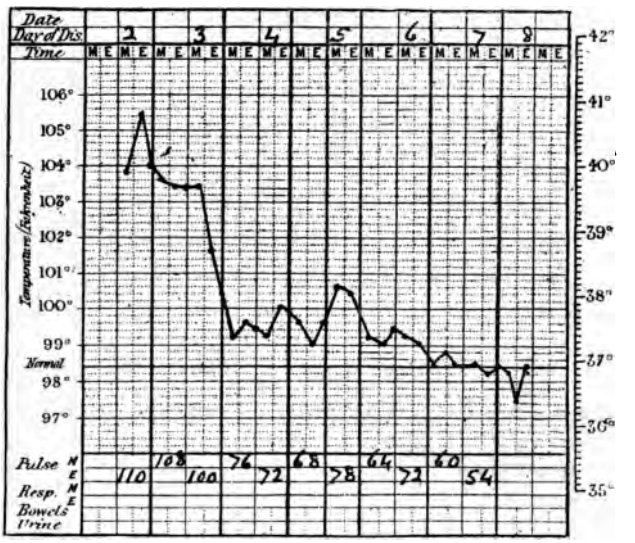
High continued type resembling typhoid.

CHART 6.



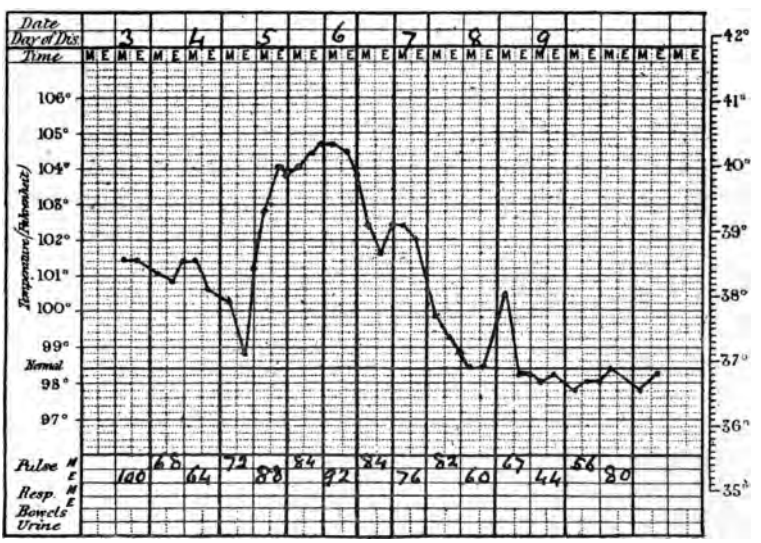
Continued typhoid-like type with highest temperature during terminal rise.

CHART 7.



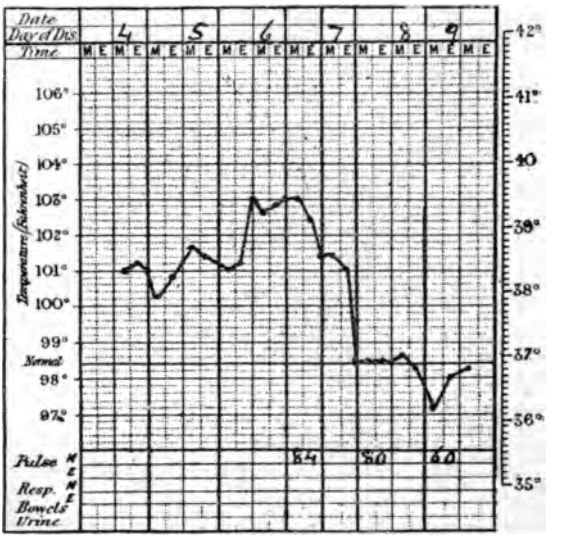
Exceptional case with no terminal rise.

CHART 8.



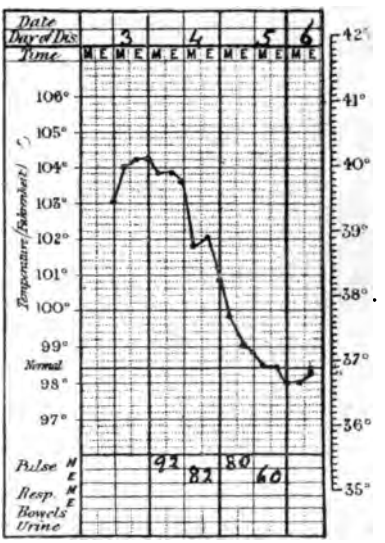
Prolonged high terminal rise with slow pulse.

CHART 9.



Case admitted on fourth day showing terminal rise.

CHART 10.



Short case admitted during terminal rise.

have been able to recognise the new fever clinically, and to correctly assure the friends that the temperature would fall on the sixth or seventh day.

Chart 6 is that of another patient, attacked while in hospital, in which the high initial rise is less abrupt than usual, so that the terminal one shows the highest point of the pyrexia, but with a slow pulse—a less uncommon type which may also give rise to a suspicion of typhoid for several days.

Chart 7 illustrates the opposite condition, also quite exceptional, in which the terminal rise is completely absent, although the temperature did not finally reach the normal until the sixth day.

Terminal cases.—I have already mentioned that just about half these patients only come to hospital during the terminal rise of temperature, and this is easy to understand when we remember that in a large number of the cases the pyrexia falls to about 100, or under, on the second or third day, accompanied by a remission of the severe headache and pains in the back and limbs, so that the patient thinks he is rapidly getting over his trouble. It is only when he is rudely awakened from this happy frame of mind by the second rise of temperature that he comes into hospital for treatment, where his fever rapidly ceases while taking the inevitable quinine, so that both he and his doctor usually have no suspicion that he has suffered from anything but an attack of malaria.

Chart 8 is that of a patient admitted on the third day, in which the temperature fell to almost normal on the following day, after which a well-marked terminal rise took place, during which the temperature remained persistently at about 104° F. for over twenty-four hours in spite of frequent spongings, which had only a very ephemeral effect on the pyrexia. The pulse was also never recorded as rising over 92 during this high temperature, while a very slight and short recrudescence of fever occurred just after the cessation of the secondary curve, as is rarely the case.

Chart 9 is that of a man admitted on the fourth day with a low continued type of fever succeeded by the characteristic terminal rise with a slow pulse.

Chart 10 is that of an unusually short case admitted at about the beginning of the terminal rise, the fever only lasting two days under quinine, but with the slow pulse of seven-day fever and not the rapid one of malaria. In this case, as in every one on which this paper is based, a blood-film, taken before any quinine had been administered in hospital, was examined for malarial parasites, with a negative result.

These last three charts will serve to illustrate the very frequent cases admitted during the terminal rise of pyrexia, many of them only coming to hospital on the fifth or sixth day of the disease, but a few hours before the final fall. In such only the history of the patient, the absence of malarial parasites, and above all, the slow pulse during high fever, aided by their occurring in the regular season for this disease, will allow of a correct diagnosis being arrived at, and the common error of returning them as malaria avoided.

The Duration of the Fever.

On account of the patients coming to hospital in any stage of the disease, the actual duration of the fever after admission varies widely from one to eight, or rarely over eight days. The total duration of the fever in just about three-quarters of them was either six or seven days, so that the temperature either fell to normal on, or finally remained normal during the seventh day in this proportion, and hence the name of "seven-day fever," which I have proposed for the disease. A slightly larger number of cases end on the sixth than on the seventh day, but it appeared to be better to take the latter day for the name of the affection so as not to lead to its termination being expected at an earlier date than actually occurs in almost half the total cases. The following table shows the

number of cases which terminated on different days of the fever, the cases denoted "typical" being those admitted sufficiently early to show the characteristic saddle-back or complete terminal rises of temperature, while the "terminal" cases are those admitted near the end of the disease, and so especially liable to be returned as malarial.

TABLE OF THE DURATION OF THE FEVER.

	— 3 days.	3 days.	4 days.	5 days.	6 days.	6 or 7 days.	7 days.	8 days.	+ 8 days.	Total.
Typical cases ...	0	0	3	5	39	(76)	37	8	8	100
Terminal cases ...	0	4	5	10	40	(69)	29	10	8	106
Total cases ...	0	4	8	15	79	(145)	66	18	16	206

It will be seen from this table that in no case did the fever last less than three days, while in only four, or 2 per cent., did it end on the third day, all these being terminal cases in which the history of the duration of the fever before admission may have been inaccurately given. Further, only eight cases, or 4 per cent., ended on the fourth day, so that in the remaining 94 per cent. the fever lasted five or more days; and in 86 per cent. it lasted six or more days—a point of the utmost importance in separating out this disease from true epidemic dengue with three or less days' fever, according to the great majority of experienced writers on that disease. As I was examining the blood of every fever case in the hospital it is impossible that I could have overlooked an appreciable number of short cases of this fever, for exceedingly few such cases without malarial parasites in their blood occurred during the months the seven-day fever was prevalent.

Convalescence.—Once the temperature is normal and the patient up and on full diet convalescence is very rapid after seven-day fever, as was pointed out by Dr. J. G. Murray, I.M.S., at the recent debate in Calcutta already mentioned, this officer having had a very large experience

of the disease in his wards at the European Hospital. He also stated that he had never seen a case of three or less days' duration, nor the very severe pains and joint complications of true dengue, while the pains did not return during the terminal rise of seven-day fever as in dengue. He regarded the slow pulse of seven-day fever as a characteristic point of difference from the latter disease.

Another important feature of the convalescence of seven-day fever is the absence of the chronic joint pains which are often so prolonged and distressing after true dengue, producing the crippling of the patient from which the name of the affection is said to be derived, but which I have never seen in the Calcutta sporadic seven-day fever.

Relapses.—I have not yet met with a case of seven-day fever which has relapsed during the same year as the primary attack, although the same person very rarely suffers from the disease again in a subsequent year if he remains in Calcutta, usually in a milder form with a more marked and prolonged remission of the fever, as was well illustrated by the charts of two attacks experienced and recorded by my friend, Captain J. W. D. Megaw, I.M.S. (3), in a paper in which he ably advocates the view that the seven-day fever is but a sporadic form of dengue. In the latter disease, however, even repeated relapses during a single season are very common in Indian outbreaks.

The blood changes.—The blood was examined by me in every case for malarial parasites, with invariably negative results, while the protozoan parasite described by Graham (4), of Beyrouth, in dengue, was never seen. A differential leucocyte count was also done in over seventy cases during the first year's prevalence of the fever, but with disappointing results. Without going into full details here it may be said that the total leucocytes were commonly reduced in numbers, and often markedly so, but 3000 to 4000 only being not infrequently met with, while leucocytosis was never seen. The differential leucocyte count furnished

very variable results, a large mononuclear increase, quite as marked as in malaria being by no means rare, while a lymphocyte increase with a normal proportion of large mononuclears, as in typhoid, was also met with, so that this test is of no value in differentiating seven-day fever from those diseases with which it is most frequently confounded.

The Cultivation of a Bacillus from the Blood of Seven-day Fever Cases.

Being struck with the resemblance between the more continued type of the seven-day fever, with its slow pulse, to typhoid and para-typhoid fevers, I made numerous attempts to cultivate a bacillus from the vein blood. An organism was thus obtained in pure culture in six cases during the two seasons' work, which appears to present constant features differing from those of the bacilli of typhoid and para-typhoid infections. The following are its principal characteristics:

In shape and size it resembles those of the coli group, and like them is actively motile, flagella, in comparatively small numbers, having been demonstrated in some of them by my assistant, Dr. G. C. Chatterjee, to whom I am indebted for much help in testing the cultural characters of the organism. It is decolourised by Gram's method of staining, grows in broth with the production of a diffuse haziness; forms a thin film on gelatine at 70° F. without liquefying the medium, and an appearance resembling that produced by the coli group on the surface of an agar tube. In a stab culture of glucose agar it does not grow very readily, except in the upper part of the streak and on the surface, and does not produce any gas-formation. On potato it forms an invisible growth like the typhoid bacillus. In litmus milk no clotting is produced, and only slight acidity results after a few days. In dextrose, levulose, glucose, and maltose broths there is neither acid- or gas-formation, but with mannite slight acid-formation takes place after several days.

The organism thus appears to be related to the great coli group, but differs from the organisms of typhoid and para-typhoid fevers. In addition to the cases in which it was isolated from the blood, a number of other cases yielded negative results, so that repeated examinations are necessary in order to obtain it, but this is, perhaps, not surprising in such a mild and short fever as the seven-day one is. Clumping was obtained with the organisms when mixed with the blood of patients suffering from seven-day fever, up to dilutions of 1 in 20 and 1 in 40, although not sufficiently constant to furnish a reliable diagnostic measure, the fever apparently being too short in duration to yield much agglutinin. The presence of this organism repeatedly in the blood of seven-day fever patients points to its being probably the cause of the disease, but my observations require confirmation before this relationship can be positively asserted, while it is also desirable that similar methods should be employed in true dengue, which somewhat resembles seven-day fever.

Treatment.—Quinine is useless, and tends to aggravate the headache, while I know of no drug which influences the course of the fever, although salicylate of soda is useful in modifying the pains, and belladonna is worth trying if they are unusually severe.

Mortality.—No fatal case has been met with.

Race and sex incidence.—The most striking fact in the incidence of the disease is that for the first month or two of its prevalence each year practically every patient is a sailor or someone connected with shipping on the river Hooghley. Later in the season patients are admitted from other sections of the community all over Calcutta, but it especially affects new comers rather than those who have lived for some years in the town. For this reason comparatively few cases were seen in females, and extremely few in children, almost all the women and children admitted to this hospital belonging to the classes who are born and bred in India.

Among the native classes admitted to the Medical

College Hospital cases are also occasionally seen, but this fever is many times rarer in the indigenous population than among Europeans, especially if the proportions of the two living in Calcutta are taken into consideration—another point in which it is at total variance with epidemic dengue.

The Seasonal Incidence of Seven-Day Fever compared with that of Malaria.

The monthly incidence of seven-day fever and malaria respectively during the two years that I microscoped the blood of every case is shown in the accompanying diagram. In 1904 the seven-day fever was prevalent from June to August, disappearing again in September, which is the month during which malarial cases rapidly increased from a previous low level. The former remained practically absent from September, 1904, to the following April. In 1905 the seven-day fever was unusually prevalent, beginning to increase as early as May and being very numerous throughout from June to September, but rapidly declining during October and November, and disappearing once more from December to the following April. Owing to a late rainy season the usual autumnal increase of the malaria did not appear until November and December, when the seven-day cases had markedly decreased. In 1906 the new fever cases were less numerous than in 1905, but they began to increase in May, reaching the highest point in July and declining again to very few in September, the fall, as usual, taking place just before the main rise in the malarial curve.

If, however, a curve of the incidence of both fevers together is worked out (as shown in a paper on malaria in Calcutta in the 'Indian Medical Gazette' of March, 1906) (3), then a single curve is obtained with two maxima, the first due to the seven-day and the second to malarial fevers. It is not therefore surprising that both these fevers

have been so long confused together and regarded as malarial in nature. The late Dr. A. Crombie, I.M.S., may have been thinking of the seven-day fever now described when he stated that, in his belief, 75 per cent. of the fevers in Calcutta returned as malaria did not belong to that category.

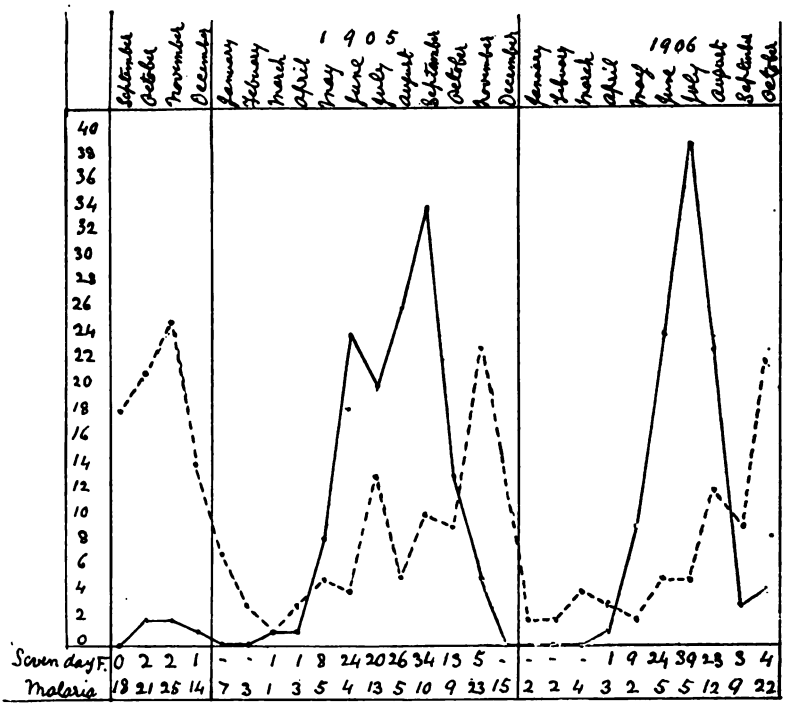


Diagram of monthly prevalence of seven-day and malarial fevers. Continued line = seven-day fever. Dotted line = malarial fever.

Distribution in India.—An examination of the records of all the fever cases for two or three years in the large hospitals of Bombay, Madras, and Lahore, showed this seven-day fever to occur at the two first places only, but I have not found any cases in Lahore or in the United Provinces.

It is thus a common fever with a definite seasonal distribution in the large seaports of India, among which Rangoon should also be included, for Captain Windsor, I.M.S., has reported to me that it is commonly seen there at about the same season that Calcutta is attacked. It is too early to say that it does not occur inland, but it seems pretty certain that it is at least comparatively rarely seen far from the coast-line.

The Differential Diagnosis of Seven-day Fever.

From malaria.—Briefly, the diagnosis of seven-day fever from malaria, with which it has been so long confused, can usually be made clinically by attention to the points described in my paper (3) on the latter disease. The chief of these are, the totally different temperature curve in the cases admitted early, the more severe frontal headache, the red edge to the tongue, the rash when present, and pains all over, the absence of enlargement of the spleen and of malarial parasites in the blood, and, especially in the terminal cases, by the slow pulse-rate accompanying high fever.

From influenza.—In Calcutta I find that influenza cases occur mostly from January to March, and disappear in the hot weather before the seven-day fever season commences, and do not appear again till late in the Autumn, when the other disease is practically at an end. Further, the temperature curve is usually an irregular, intermittent one, and never shows the typical saddle-back type of the seven-day fever, while the latter is scarcely ever complicated with the inflammatory signs in the lungs and throat which were nearly constant in the Calcutta influenza of 1892.

From dengue.—A much more difficult question is its separation from dengue, for the seven-day fever resembles the latter in its sudden onset, the severe headache and pains, the characters of the tongue, the rash, when present, and, to some extent, in its seasonal incidence and its dis-

tribution; so much so that I find from an extensive examination of the literature and of fevers in India, that the typical seven-day fever has been described as dengue by Edward Goodeve, I.M.S., in Calcutta in 1853 (5), and by Staff-Surgeon Bassett-Smith in Bombay in 1897 (6), in addition to a recent outbreak at the Mount in Madras.

On the other hand, the fever now being described differs most markedly from the classical accounts of dengue epidemics, as well as from the two great Indian outbreaks of 1824 (7 to 10) and 1872 (11 to 13), so much so, indeed, as to make me think that it is quite distinct from dengue.

Table of the Differences between Dengue and Seven-day Fever.

	Dengue.	Seven day fever.
Prevalence... ...	At long intervals in epidemic form attacking large proportion of residents.	Annually in sporadic form.
Distribution ...	Specially attacks coast towns, but spreads far inland.	Only known near the coast, so far.
Race incidence ...	Europeans and natives equally attacked.	Very common in Europeans, comparatively rare in natives.
Seasonal incidence	Mostly in hot months, but may prevail in cold season (1872).	Prevails in hot and rainy seasons only.
Relapses	Very common in same year as first attack.	Rare, and not in same year as first attack.
Pains	Very severe and breakbone in character.	Moderately severe, as in influenza, and not of breakbone character.
Joint symptoms ...	Very common and characteristic.	Absent, or only present as slight pain.
Convalescence ...	Very tedious, lasting one to three months, with persistent joint-pains.	Rapid. No after joint-pains.
Fever	Lasts two or three days, falling to normal with crisis. Occasional very short secondary rise. Markedly remittent.	Five to eight or more days, with typical saddle-back remission to 100° to 99° F. only. Otherwise continued in type.
Pulse	Rapid.	Slow, especially in terminal rise.

The main points of difference are shown in the accompanying table.

In a recent discussion on this subject at the Medical Section of the Asiatic Society in Calcutta my friend Captain J. W. D. Megaw suggested that the seven-day fever is but a sporadic form of dengue, but Lieut.-Colonel G. F. A. Harris, I.M.S., Major D. M. Moir, I.M.S., and Dr. A. Caddy, who had each seen both dengue and seven-day fever, were all positive that the two diseases are quite distinct. Further, Major W. G. Pridmore (14) and Captains Cornwall and Cato, I.M.S., who had all recently seen epidemic dengue in Burmah, Madras, and Singapore respectively, are also of the same opinion, which I share. As, however, I have never seen epidemic dengue, I hesitate to pronounce a decided opinion on the subject, and have therefore brought it up for discussion to-night, and hope to get valuable enlightenment.

One thing at least appears to be quite certain—namely, that the seven-day fever has existed as one of the commonest fevers of Europeans in Calcutta year by year for a long period. Yet it has passed quite unrecognised by many medical men with experience of true dengue, so if it be but a sporadic form of that disease the descriptions of dengue require to be completely re-written. It appears to me to be more likely that it is a new fever, not previously differentiated, than that every writer on dengue has overlooked the most characteristic temperature curve of the disease which I have illustrated in the charts shown this evening.

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DISCUSSION

Dr. JOHN ANDERSON, C.I.E.: Professor Rogers' description of this "seven-day fever" reminds me of what is called in India a heat, sun, or ardent fever that is frequently met with in the Punjab, or the north-west provinces of India in the hot, dry weather. It is by no means uncommon, and chiefly attacks recent arrivals. Those who are most exposed to the direct heat of the sun, and those who predispose themselves by indulging rather freely in alcohol (even though not to actual excess) are the chief sufferers. In many points it resembles closely the fever that has just been described to us, particularly in the fact impressed upon us by Professor Rogers, of the high temperature accompanied by a low pulse, but I cannot say that the temperature curve in the cases I remember resembled that of the charts shown on the screen to-night. As to treatment, I obtained most success by using bromides, cold sponging, douching, and restricting the patient to a simple and non-stimulating diet with the use of mild aperients.

Sir PATRICK MANSON said that the diagnosis of the different fevers in tropical countries was a difficult task. It was now possible for everyone to recognise malaria, kala-azar, trypanosomiasis, etc., but he believed there were in addition in the tropics a crowd of undifferentiated fevers, of which he believed that described in the paper was one. He thought it quite distinct from dengue, there being in that disease a distinct rash frequently followed by a late desquamation, and the pains were an outstanding feature. He thought the fever described in the paper was probably the fever previously described by Crombie as simple continued fever, or fever of acclimatisation. The saddle-back temperature curve, or secondary fever, was seen also in yellow fever. The three-day fever of Chitral described by Captain McCarrison, which was accompanied by much depression, seemed similar to the present fever. It affected Europeans and Gurkhas, but not Hindoos, who were, presumably, immune. He hoped Professor Rogers would continue his work on these fevers. He believed the Indian fevers were, for the most part, protozoal and not bacillary.

Dr. ROGERS: I am much indebted for the opinions which have just been expressed. In the course of my investigations I have met with the class of cases termed "ardent fever" by Dr. Anderson, which occur, especially in the very hot season, in the Punjab and United Provinces, together with heat-stroke cases. Their temperature curves are distinct from the saddle-back one of seven-day fever, and I believe that these "ardent fevers," which are few in number, are, like heat-stroke, due to excessive tem-

peratures, and not a specific fever. Sir Patrick Manson's opinion that the fever I have described is a definite specific one not hitherto differentiated and absolutely distinct from dengue is of the greatest importance on account of his personal experience of the latter disease. As he remarks, it has commonly been returned as "simple continued fever," as described by Dr. Crombie, but I found by an analysis of all the fever cases at the Calcutta Hospital before my present investigations that the curves of the monthly prevalence of the cases returned as malaria and as "simple continued fever" respectively were practically identical, showing that the two diseases had not been accurately diagnosed, many of the terminal seven-day cases being classed as malarial, and many truly malarial cases as simple continued fever. I have not had any personal experience of the "three-day fever" of Chitral described by Captain McCarrison, except that I examined blood-slides of cases sent me from Chitral by Captain R. P. Wilson, I.M.S., several years ago, and found no malarial parasites in any of them, so am inclined to think it is a distinct fever, but different from the seven-day one I have now described. I think the separation out of the seven-day fever clears up the short fevers seen in Calcutta, but the longer fevers require much more work.

THE DIAGNOSIS OF ADDISON'S DISEASE

BY

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RECENT progress in the treatment of diseases has accentuated the importance of early recognition of the site of the affected tissues and the nature of the morbid process attacking the organ.

It is easy to understand that the earlier the case is seen the more difficult the diagnosis, and the less likely will the inspection of the tongue, along with the laying on of hands, lead to the correct solution of a difficult problem. If diseases which cure themselves, along with a few others, such as syphilis, malaria, and myxœdema, be excluded, the general rule holds that advanced condition of organic disease can be but slightly ameliorated by medical treatment, and the quack alone claims to cure.

Addison's disease must be defined as a condition which gives rise to a definite series of signs and symptoms, and in the majority of cases marked lesions of the medulla of the suprarenal gland are found at the autopsy. In some apparently typical cases these glands prove to be healthy, not only upon macroscopic and microscopic examination,

but also when the potency of their extract is tested physiologically.

In these cases frequently disease of the solar plexus can be demonstrated, and the explanation of the observation may lie in the fact that the internal secretion of the medulla of the suprarenal gland acts by stimulating the sympathetic system, for it is quite comprehensible that the effects of destruction of the sympathetic system or of the producer of the activator of that system will lead to identical trains of symptoms, and therefore the two conditions will resemble one another very closely. Along with the sympathetic nerves in the abdomen in some animals there are a series of cells which have been termed chromaffine cells, because they yield a yellow colour on treatment with chromic acid in a similar manner to the cells of the medulla of the suprarenal gland. The intensity of the colour developed is said to bear a definite direct relation to the activity of the extract prepared from the tissue, and that the effect following the injection of the extract cannot be distinguished from that produced by extract of the medulla of suprarenal gland.

It is the assumption of the duties of the suprarenal gland by these chromaffine cells, in the same way as the parathyroid glands take over the function of the thyroid gland after its extirpation, I venture to suggest as the explanation of the cases in which the signs of suprarenal inadequacy have been absent, in spite of the adrenal glands having been completely destroyed by disease.

The cortex of the suprarenal gland is developed from quite a different series of cells to that of the medulla—namely, those which ultimately give rise to the genito-urinary apparatus, and does not manufacture a hormone for the sympathetic system, but possibly one which controls the development of the reproductive system.

The pathognomonic signs and symptoms of Addison's disease are asthenia, pigmentation, vomiting, and attacks of fainting. When all the symptoms are well marked, diagnosis is not difficult, but by the time these are evident

the chances of any treatment being applied successfully is at least remote. Possibly some day the methods of transplantation which have been devised and used with such success upon animals by Guthrie and Carrel will be applied to man. If this be the case early diagnosis will still be essential, for it is found that grafting can be successfully accomplished in animals when tissue similar to the graft is already in the animal, but if this normal tissue be removed before the graft is made the transplanted tissue is absorbed and cannot take over the function of the extirpated organ.

Pigmentation.

Pigmentation is the most obvious sign of the disease, although far from the most pathognomonic, for there are cases of Addison's disease which do not develop pigmentation, whilst development of pigment may occur in many other conditions. The pigmentation in Addison's disease is usually but an increase in depth of tint of those parts of the body which are normally dark, along with patches of pigment upon mucous surfaces.

The parts subjected to light or irritation, such as the face and hands and the axillæ and groins, are often the first to show definite darkening. Where the shoulder-straps or braces cross the shoulders or the garter encircles the leg, pigmented bands may be seen, which, if they do not yield to repeated applications of soap and water, are suggestive of the disease. It must be borne in mind that these bands are often present in brunettes, but rapidly disappear when confined to bed and the source of irritation removed. The scalp and nails usually escape, although the latter may be affected in advanced cases which have run a chronic course. Often brown spots may be seen distributed irregularly over the limbs and trunk. Scars are usually pigmented, but may escape with only a dark halo.

Pigmentation of the mucous membrane was at one time

considered of exceptional diagnostic value, but I have seen similar pigmentation in patients who have not had, nor developed, any signs of Addison's disease.

The conditions, which must not be confused with the pigmentation due to suprarenal insufficiency, are numerous:

(1) The pigmentation accompanying pregnancy.

(2) The increased pigment occurring in chronic tuberculosis.

(3) Vagabond's disease, in which the skin becomes dark from continued irritation.

(4) Von Recklinghausen's disease.

(5) Hæmochromatosis.

(6) Exophthalmic goitre.

(7) Pigmentation due to the prolonged ingestion of arsenic.

(8) Argyria.

(9) The darkening of the exposed parts in men exposed to heat, such as occurs among stokers.

(10) The pigmentation arising amongst workers in some chemical factories, especially those engaged upon anthracene compounds.

(11) Melanism accompanying melanotic sarcoma.

(12) Freckles; similar in some respects to Kaposi's disease.

There are other conditions which are accompanied by considerable increase in pigment, but the above are the commoner, and, in the majority of cases, may be distinguished from melanasma suprarenale by careful observation; the main points will be dealt with in order.

The pigmentation accompanying pregnancy is usually of but recent date, but may, of course, be accompanied by vomiting and a languor which makes definite exclusion of Addison's disease (by the usual methods) far from easy. The increase of pigment occurring in advanced chronic tuberculosis may be due, in a certain number of cases, to the adrenal glands becoming affected, and in those it may be impossible to distinguish it from the ordinary typical disease, but in the majority, without suprarenal destruction, the pigmentation is much more uniform.

Vagabond's disease usually permits of ocular demonstration of the cause of the irritation, and on removal of the cause the pigmentation gradually fades.

Von Recklinghausen's disease presents very varying aspects: occasionally the pigmentation is marked, whilst the other skin alterations and the neurofibromata can only be detected upon minute examination.

Hæmochromatosis is a very rare disease, and in the only case which has come under my observation the pigmentation simulated that represented in skin atlases as the pigmentation in argyria. The upper part of the man's body was a much darker shade than the lower, the legs appeared as if they had had a thin layer of black lead applied, and as one travelled upwards the thickness of the layer appeared to be greater. The man's face was the colour of a full-blooded negro. The mucous membranes were of a dark violet. As a rule the pigmentation in Addison's disease is of a much browner colour.

Exophthalmic goitre is usually associated with increased sweating, and this is said to cause pigmentation through irritation. The nature of the pigmentation is very similar, but the other symptoms permit of the differential diagnosis.

Pigmentation due to arsenic, in my experience, simulates most closely that of suprarenal disease, and cannot be distinguished from it merely by inspection. There are a multitude of methods of differentiating, but with those we shall deal later.

Argyria is very rare at the present time, and according to report the colour is similar to that of hæmochromatosis, some authorities stating that the classic plates of argyria have in reality been taken from a patient suffering from hæmochromatosis.

The darkening of the skin due to heat does not, as a rule, present much difficulty, for usually the lower part of the body is normal in colour, since stokers are in the habit of wearing trousers whilst at work.

Melanasma, due to melanotic sarcoma, is usually

uniform, and in the majority of cases, the primary lesion can be found. When this is not possible the differential diag. osis is often difficult.

Kaposi's disease in its typical form leads to the development of epitheliomata, but there seem to be a number of varieties of different malignancy. I have seen such a close crop of freckles that the face seemed of a dark brown colour, as an advanced case of Addison's disease, whilst the pigmentation in the mouth resembled that of melanotic sarcoma, being intensely black and extremely patchy.

Asthenia.

The commonest early symptom is a lack of desire to do work—either mental or physical—but this condition, unfortunately, is not confined to the disease under discussion.

The rapid fatiguing of the muscles is occasionally sufficiently well-marked to be pathognomonic of the disease, whilst in the less pronounced cases graphic record may assist.

Langlois has laid great stress upon the value of the ergograph in the diagnosis of suprarenal inadequacy.

It was found that the power of the muscle to contract two or three times was not far below normal, but the power diminished very rapidly. For instance, a patient suffering from advanced suprarenal inadequacy might raise 5 klg. through 20 cm. three or four times, and then the distance would decrease to 15 cm., then to 13 cm., and gradually fall, so that the fifteenth contraction might be so small as not to be visible. This form of ergographic tracing contrasts strongly with that obtained in phthisis and other wasting diseases; in them the initial contraction is weak, but the diminution in the succeeding efforts is but slight, and the general form of the tracing is similar to that obtained in health, but of course on a different level.

Very great care must be taken in using this method, especially when applying it to female patients, for not infrequently they require considerable verbal stimulus to make them call forth their maximum power, and it is far from a rare thing to obtain typical myasthenic curves due entirely to laziness and not to muscular weakness.

When an ergograph has not been obtainable I have made the patient squeeze my hand repeatedly, at intervals of two seconds; in normal cases the force diminishes but slowly, whilst in well-marked cases of Addison's disease it is very rapid.

It is not at all uncommon, however, to find that the muscular power is only diminished at times, and for quite long periods the patient's muscles act normally. On one occasion, after a man had squeezed my right hand some dozen times, I asked him to squeeze my left, and this he did with so much vigour that I felt the effects of his efforts the next day, although he then appeared on the post-mortem table and presented adrenal glands destroyed by tuberculous invasion.

The adverse criticism of one method does not enhance another, but nevertheless this fact is not sufficient cause for suppression of experience, and on the whole, in my hands, the ergograph method of diagnosing Addison's disease has proved disappointing.

Vascular System.

Since Addison's disease is due to interference with the sympathetic system, and it is through this that the vascular mechanism is controlled, one would expect a very considerable alteration in blood-pressure, and one's expectations are fully realised.

Often it is possible to diagnose the condition from the pulse alone, for it is usually weak and of extremely low pressure. The alteration of this pressure under treatment has assisted me in diagnosis, but for that purpose the estimation must be made with considerable accuracy.

It has been stated that attempts to estimate blood-pressure with the finger accurately are as futile as those to determine temperature with the hand. Even the most experienced physician may be absolutely wrong in his estimation of the blood-pressure in an artery when he uses his fingers alone, however well trained they may be. This is not difficult to understand when one remembers that the pressure required to occlude a vessel not only depends upon the pressure in that vessel, but also varies directly as the square of the radius of the vessel. A small radial artery with a high internal pressure will be occluded with but slight pressure, and this was well brought home to me in a case of hemiplegia in which there was universal accord that the condition was due to cerebral thrombosis, because of the apparently low blood-pressure. On measuring the pressure with an instrument it was found to be 195 mm. Hg., or more than half as much again as the normal.

The best method of measuring blood-pressure is with the Hill and Barnard sphygmometer, preferably one with a mercury or simple air manometer. It is of no little interest to see how the tendency of the Englishman to enhance the value of the foreigner's work has led to the instrument being termed the Riva-Rocci, although that observer published his work in a comparatively obscure Italian journal a few months before Hill and Barnard perfected their instrument.

In estimating the blood-pressure the patient should be set at ease, physically and mentally. An extensible air-tight bag of not less than four inches in breadth should be applied to the upper arm so as to encircle it completely, and be supported externally by an inelastic cover. Air is then pumped into the bag, which is connected with a manometer, and the pressure noted when the pulse just ceases to be palpable at the wrist. The patient is then told to take a few deep breaths, and usually, if the pressure has been only just sufficient to occlude the artery, an occasional throb may be felt in the radial artery. The

pressure is then raised another millimetre or two, and at this pressure the pulse is obliterated even on deep respiration. The reading of the manometer is recorded as the systolic pressure in the brachial artery. Although fully recognising the importance of the diastolic pressure when working at hæmodynamic problems, for the special diagnostic purpose in view I did not consider it essential. It is wise to make the patient assume a recumbent position before taking the observation in order that the heart, bag, and wrist may be in the same horizontal plane. Speed in work is always valuable so long as it is not indulged in at the expense of accuracy, and in the above estimations it is essential, for if the pressure is maintained in the bag for any considerable period the forearm becomes hyperæmic and very uncomfortable, and even a purpuric eruption may follow, due to rupture of small vessels.

In Addison's disease the systolic pressure is usually low, less than 100 mm. Hg. In one case I found it to be 67 mm. Hg.

In 1898, I carried out a series of experiments with the object of determining whether extract of suprarenal might be used as a hæmostatic in hæmatemesis, and found that even large quantities administered by the mouth did not lead to any recognisable rise in blood-pressure in normal individuals.

It has been my rule to take the blood-pressure on at least three occasions in all cases suspected of suprarenal inadequacy, and if the blood-pressure has proved to be low, suprarenal extract has been administered by the mouth. In many cases this has been followed by a very distinct rise in the pressure. These cases have proved to be suffering from Addison's disease.

It was found that in those cases which were pigmented from other causes the blood-pressure was not raised on administration of the drug. The results have been sufficiently constant to permit me to base diagnosis upon the following method:

The blood-pressure is estimated on three occasions, and

the heart is examined to determine whether there is any valvular lesion. If the pressure be low, and there be no valvular lesion, a reliable extract of suprarenal gland is administered three times a day in doses of 3 gr. for three days. The blood-pressure is then estimated again, and if there be a rise of more than 10 per cent., the probability that the patient is suffering from adrenal insufficiency approaches a certainty.

It is scarcely necessary for me to mention that no other drugs must be taken for at least three days before the first observation is made. Not infrequently a patient with a low pressure does not react to suprarenal gland, but does to strychnine; this, if of any value, is additional evidence against the probability of the disease being Addison's.

Blood.

It is very doubtful whether any alteration in the condition of the blood occurs in Addison's disease. The hæmoglobin often remains the normal quantity and the number of red cells is not diminished. In a few cases, but it is doubtful whether these are uncomplicated, an increase in the number of white cells has been recorded, and, upon differential count, the number of lymphocytes has been found to be above the normal, and the polynuclear cells diminished. It has been asserted that the eosinophil cells are present in larger numbers than normal, but this is not infrequently the case after an inoculation of tuberculin, and does not permit of any conclusion being drawn, for it may arise from auto-inoculation.

Having arrived at the conclusion that the suprarenal glands are diseased, the next question which must be decided is the nature of the morbid process which is taking place in them. Undoubtedly the commonest cause of destruction is invasion of the tubercle bacillus, although in the small series which I have had the opportunity of observing I think at least one has been due to some other

condition. Not infrequently this infection of the suprarenal gland takes place without any macroscopic evidence of tuberculous disease in any other part of the body, and therefore examination of the lungs often fails to assist in deciding the nature of the invasion of the adrenals.

Comparatively recently the opsonic index of the serum to the tubercle bacillus has been used to permit diagnosis. In health it has been shown that the variation of the index is limited from ·8 to 1·2, whilst in tuberculosis it may be between these limits, but is often above and below, and if the blood of the patient suffering from tuberculosis be examined from time to time the index will be found to vary considerably. It has been asserted that if the index be high the opsonin is not destroyed at 60° C. in cases who are suffering from tuberculosis, whilst the opsonin in non-tuberculous cases cannot withstand this temperature. I find upon inquiry that this method has yielded very different results in the hands of different investigators, all of whom, in my opinion, were equally skilled.

Probably the most reliable method is by noting the opsonic index, and then determining, by repeated examination of the blood, the duration of the negative phase which follows an injection of a five hundredth of a milligram of tuberculin. In the majority of normal individuals the negative phase is of short duration—twenty-four hours or less—whilst in the hosts of the tubercle bacillus it is prolonged, and may even be for a period of three weeks.

I admit that the number of cases in which the opsonic index has been estimated is too small to permit of any conclusion, and this is primarily because Addison's disease is rare, even in a large hospital, and since this method has been in use the number of such cases admitted has been small.

There is no reason to believe that the treatment with tuberculin would be less efficient in preventing the progress of the disease than in other cases where the invasion is but local, such as in tuberculous glands of the neck,

but so far this is purely speculative, and I have not had opportunity of seeing it carried out in any case.

A few minor signs are of value in the diagnosis of the disease, but to which system they should be ascribed is not clear.

It is not uncommon for advanced cases to complain of flashes of light, which seem to be of very short duration and do not assume the appearance which is described in teichopsia.

Vomiting is often a distressing symptom which cannot be controlled by any of the usual methods. Occasionally it is attributed to suprarenal gland, which is usually administered in this disease, but cessation of the drug does not cause relief of the symptoms.

Constipation is very often difficult to deal with because asthenia attacks the plain muscles as well as the striped muscle, and the usual stimulants of that tissue apparently do not act.

Protocols of a few Cases.

Jane B—, aged 37. Ill about three years. Has been gradually getting darker during that time. Repeated vomiting during last five months. Nails pigmented; lines of palms dark. Pigment in the mucous membrane of mouth and lips, many brown spots distributed over trunk and limbs. Scalp apparently normal. Myasthenic action not at all well marked. Pulse, low tension, 72 mm. Hg., 88 per minute. On administration of 8 grains of suprarenal powder, three times a day, the blood-pressure rose to 84 mm. Hg., but fell again to 68 on ceasing the drug. This fall of blood-pressure occurred more than three weeks before death, and therefore could not have been due to a terminal paralysis of the vaso-motor centre. The opsonic index for tubercle bacillus was found to be (by Dr. Bulloch) ·52. The autopsy revealed complete destruction of the suprarenal glands by tuberculous disease.

Thomas B—, aged 21. Has become darker during the last eleven months and is now so far pigmented as to have been a source of interest to the other inmates of Rowton House. He suffers from attacks of giddiness, and fainting, vomiting, and diarrhœa. Patches of pigmentation may be seen in the mouth, and a scar from an operation is of deeper colour than the surrounding skin. The blood-pressure was found to be 96 mm. Hg., which rose to 124 mm. Hg. whilst under the influence of suprarenal extract. On one occasion the blood-pressure was found to have fallen; and on making inquiry I learnt that the drug had been omitted for twenty-four hours. On re-administering the extract the blood-pressure returned to 124, a pressure which was maintained during the patient's stay at the hospital. In this case the opsonic index tubercle bacillus was found to be normal. This is of considerable interest, because the patient apparently improved in every way whilst under treatment, and it seems likely that the disease was not due to tuberculous invasion of the gland, but possibly to hæmorrhage or some other temporary disease.

George C—, aged 40. I record this case to show that occasionally complete destruction of the adrenal bodies may occur without any of the typical signs arising. Ill eighteen months. Noticed that his skin was getting yellow, had several bilious attacks, one twelve months ago, another ten months ago. Was quite well between these attacks. Upon examination was found to have a dilated stomach, but a normal quantity of hydrochloric acid in the gastric contents. His condition did not suggest suprarenal inadequacy, but upon death from acute pericarditis it was found that both his suprarenals were caseous.

Isaac F—. Ailing three months, languor, discomfort in the stomach, skin becoming darker. Pigmentation of the face and hands, axillæ and upper parts of the thighs, and also of the buccal mucous membrane. In this case

the blood-pressure was raised from 97 to 108, but the evidence of Addison's disease was not definitely forthcoming.

Beatrice B—, aged 24. Illness dates from about two years; the patient had fits, apparently of an epileptic nature, following a fright. In these fits she struggled and bit her tongue, but did not relax the sphincters. She has been losing weight and getting weaker, and her friends assert that she is becoming darker. On admission, there is considerable pigment in the axillæ and the upper part of the thighs, neck, and other skin-folds. Garter marks and shoulder-strap marks are absent. There is no pigmentation in the mouth nor of the lines of the palms. The scalp is pale. Persistent headaches and vomiting were the chief signs, along with occasional fainting. Examination of the discs showed them to be normal. The pulse was of small volume, the blood-pressure 108 mm. Hg., regular, 84 per minute. Administration of suprarenal gland by the mouth did not increase the blood-pressure. The patient died suddenly, and a tumour in the right frontal lobe was found. The pigmentation in this case was in all probability due to the long-continued ingestion of arsenic. The fits were of an epileptic nature, and bromide along with arsenic had been taken for slightly under two years.

Agnes A—, aged 12. During the last year has gradually become darker, and complains of languor. Her appearance suggested a typical case of Addison's disease, and for that reason I was asked to see her. On inquiry one found that two years previously she had suffered from epileptic attacks, and that she had been prescribed bromide and arsenic. Her pulse was of very low tension and small volume. On being measured it was 87 mm. Hg. Three grains of suprarenal extract were given three times daily for a week, and on measuring the blood-pressure again it was found to be 87 mm. Hg. The administration

of strychnine increased the blood-pressure to 94. On ceasing to give arsenic the pigmentation gradually diminished, and the patient returned to her normal colour.

DISCUSSION

Dr. ROLLESTON expressed his sense of the value of the paper, with the conclusions of which he agreed. The diagnosis of Addison's disease, though particularly easy when the disease was advanced, was often a very difficult problem at an early stage. The existence of debility and some pigmentation in dark-haired individuals, more often, perhaps, in young women, sometimes raised the suspicion of Addison's disease, and in these cases the estimation of the arterial blood-pressure was a valuable means of coming to a decision, as a pressure above normal ruled out Addison's disease. He quite agreed that in cases of Addison's disease the administration of suprarenal gland extract was followed by a rise in the blood-pressure. The further question arose as to the application of this test to cases with suprarenal inadequacy, but not sufficiently marked to be called Addison's disease; cases, in fact, which bear the same relation to Addison's disease that cases of benign hypothyroidism or fruste myxœdema do to myxœdema. Boinet had described this group under the name of Addisonism. The existence of cases of this kind could seldom be established, but possibly a rise of blood-pressure after suprarenal medication would make it possible to recognise them. A low blood-pressure was very characteristic of Addison's disease; in one case it was 65 mm. of mercury within twelve hours of the patient's death, and in two other cases the radial pulse had been imperceptible within thirty-six hours of death. With regard to pigmentation, patches of pigment in the oral mucous membrane in Addison's disease were often determined by some irritation, especially that of a bad tooth, and the absence of these patches was of no value against the diagnosis of Addison's disease; pigmentation of the mucosa of the vagina was doubtful. Mistakes sometimes occurred in connection with Graves' disease, fruste cases without any exophthalmos being regarded as Addison's disease. The localised pigment spots on which Greenhow laid stress as almost pathognomic of Addison's disease might be fallacious; thus, a man, now alive and vigorous, was definitely diagnosed on these grounds by the late Dr. Greenhow as Addison's disease some thirty years ago.

Dr. F. PARKES WEBER: To the various clinical manifestations of early Addison's disease mentioned by Dr. Grünbaum, in his

practical and interesting paper, I would add another one, namely, *prolonged convalescence or a condition of unusual asthenia following ordinary attacks of infectious diseases.* In December, 1899, I saw a young man, aged 24, suffering from a pyrexial attack, which was regarded as influenza. The illness was accompanied by some pleurisy at the left base, and was followed by remarkable debility. There were several medical consultations about him, and he gradually apparently recovered. Unfortunately, no exact estimations of the blood-pressure were made. Later on, in 1901, when shooting in India, he was attacked by malaria—apparently not a particularly severe attack—and was sent up to the hill-station of Darjeeling. There his condition was one of great asthenia without fever, and he had syncopal attacks, in one of which he died. Some cutaneous pigmentation was observed in India, though none had been noted during the illness in England. A most careful post-mortem examination was made, and in a report sent over to a life insurance office in England it was stated that the suprarenal glands contained yellow, caseous material and calcareous plates, and were matted together across the aorta. It seems to me that suprarenal disease (latent Addison's disease) may have been the real explanation of the asthenic condition connected with the illness in England. If in all cases of tedious convalescence and unusual asthenia following ordinary attacks of infectious diseases, the possibility of the presence of an early or latent stage of Addison's disease were to be thought of, the methods of diagnosis (especially the accurate estimation of the blood-pressure and the effect of treatment by suprarenal extract) discussed by Dr. Grünbaum might be applied, and thus now and then Addison's disease might be detected at a very early stage indeed.

Dr. TOOTH: I have listened with great interest and profit to Dr. Grünbaum's paper, and I only regret that more are not present to take part in the discussion. The facts recorded are so incontrovertible that there seems to be little room for criticism. Dr. Grünbaum has alluded to a long list of conditions in which pigmentation of the skin exists as a factor, and I should like to ask him whether he has come across cases in which a pigmentation resembling that of Addison's disease has occurred in the course of prolonged treatment by the bromides. I can recall one or two in a somewhat long experience among epileptics in which such a symptom has occurred. In one I recollect liquor arsenicalis had been given with the bromide, but in only two-minim doses—so small a quantity that one can hardly think it was sufficient to account for the colouration, moreover the pigmentation continued in spite of the discontinuance of the arsenic until I lost sight of the case. The action of bromides on the skin is so varied that this may possibly be one of the many symptoms produced by it.

Dr. WILLIAM EWART: Impressions gathered from a comparatively limited clinical and pathological experience have led me to doubt whether the expression "Addison's disease" is sufficiently definite for our present practical requirements. So great are the differences to be noted between the cases to which it has been applied that the question arises whether separate affections may not have been included under that one heading which seems to have hitherto almost monopolised the pathology of the suprarenal body. Analogy and inference might justify us in assuming that the suprarenal is not restricted to one affection only, but that, like any other organ, it may be liable to suffer in various directions. At any rate, even if we do not go beyond the classical descriptions and the common stock of clinical histories we cannot fail to be struck with the remarkable difference between cases as regards the degree of pigmentation. It may in the future be possible to trace some essential distinction corresponding to the outward contrast between the two familiar types which may be defined as "the pigmentary type" and "the asthenic." Both these types are probably apt to occur in attenuated degrees; but only the pigmentary variety would be likely to obtain recognition in its clinical attenuations. I believe that I have seen instances of the latter, and that their symptoms may be capable of considerable chronicity and of apparent, if not of genuine, recovery, or at the least of considerable intervals of quiescence. Further information on these points would be of value.

Dr. F. E. BATTEN said it would be of interest to know if Dr. Grünbaum had examined the electrical reaction of the muscles in cases of Addison's disease.

Dr. GRÜNBAUM (in reply): I have compared the rapid fatigue observed in that disease with that seen in myasthenia gravis, but one of the characteristic features of the myasthenic reaction is the ready exhaustibility of the muscle to electrical stimulation. It was whilst under Dr. Rolleston's tuition that I first became infected with an interest in the suprarenal gland, and most of the remarks in the paper are the result of his teaching. He has drawn our attention to "Addisonism," a condition to which I shall apply the test suggested as soon as opportunity shall arise. Mr. MacAlister asked me whether I had observed pigmentation following the administration of bromides without arsenic. I have not. It is easy to understand that a drug which is to some extent excreted through the skin might lead to irritation and pigmentation in specially susceptible subjects. I have, however, been able to observe the diminution of arsenic pigmentation even whilst bromide has been continued. Dr. Parkes Weber suggests that asthenia during convalescence from an acute disease may foreshadow Addison's disease. I shall remember this, and investigate, in the way I have mentioned earlier this evening

any patients who present this condition. Dr. Ewart makes a suggestion, with which I am absolutely at one, that many diseases are included in the class which goes by the name of Addison's disease. The pigmentation varies to a great extent, but, roughly, the more chronic the disease the greater the pigmentation. I have reason to believe that the distribution of the pigment may be different in the two conditions which lead to similar signs and symptoms, viz., destruction of the suprarenal glands, irritation, or destruction of the solar plexus, but, of this, evidence is insufficient. Dr. Batten tells us that in all probability the myasthenia of Addison's disease and of myasthenia gravis might be differentiated by using electrical stimulation of the muscles and recording movements with an ergograph. I shall avail myself of the suggestion as soon as suitable cases come under my observation. I have to thank you, sir, for the kind words which you have applied to my paper and to the members of the Society for the way in which they have received the paper and made valued additions to it.

THE OPERATIVE TREATMENT OF NON-MALIGNANT ULCER OF THE STOMACH AND ITS CHIEF COMPLICATIONS, WITH INDICATIONS, LIMITATIONS, AND ULTIMATE RESULTS.

A DISCUSSION

OPENED BY W. HALE WHITE, M.D., AND A. W. MAYO ROBSON, F.R.C.S.

OTHER SPEAKERS

APPENDIX: STATISTICAL REPORTS

THE OPERATIVE TREATMENT OF NON-MALIGNANT ULCER OF THE STOMACH AND ITS CHIEF COMPLICATIONS, WITH INDICATIONS, LIMITATIONS, AND ULTIMATE RESULTS

A DISCUSSION

Tuesday, November 13th, 1906

OPENING SPEECH (MEDICAL)

BY

W. HALE WHITE, M.D.

Mr. PRESIDENT,—Inasmuch as the opener of a debate has to cover a wide field in a short time you will, I am sure, excuse me if I only lightly touch the various points of the subject.

Often the duty of a physician is to advise the patient whether or not to see a surgeon, therefore he has to consider what are the cases which surgery can benefit. About perforation there is no doubt. Whatever the medical man's engagements may be everything must give way to hastening operation; every hour's delay increases the chances of death by one or two per cent. On the other hand, if operated upon early, the patient will probably survive, and have good health for the rest of his or her life.

The diagnosis is not usually difficult, and, even when a mistake has been made, it is commonly found that the

condition existing is one—*e. g.* perforation of the appendix or acute strangulation—for which the abdomen should be opened. Sometimes acute hæmorrhagic pancreatitis is revealed, and although there is no evidence that laparotomy benefits this, yet the separation of it from perforated gastric ulcer is so difficult that the patient should usually be given the benefit of the doubt, and the abdomen should be opened. A year ago, however, I refrained from asking a surgeon to see a woman who was thought to have a perforated gastric ulcer, my reason being that after a few hours' very acute illness the patient improved for 24 hours, then became very ill again and died. It turned out that we were correct in thinking the case was not one of perforated gastric ulcer, for it was one of acute pancreatitis.

Some cases of hysteria require great care, for they may closely simulate perforation of a gastric ulcer.

With regard to the operation itself, it would be of interest to learn from surgeons whether or not it is better to do a short-circuiting operation at the same time as the operation for perforation is performed. Judging from our results at Guy's Hospital, it appears that patients who survive an operation for perforated gastric ulcer do so well that a gastro-jejunostomy is quite unnecessary.

Next to perforation severe hæmorrhage is the most acute symptom of gastric ulcer, and I think there is no doubt that no patient should be operated upon during an attack of hæmorrhage, but, as we shall see in a moment, if operation is done it should be between the attacks. The mortality from sudden severe hæmorrhage is small; it may be very difficult to secure the bleeding vessel even when the stomach is opened. Patients who are profoundly anæmic from severe loss of blood bear operations badly, and many persons bleeding furiously from what is thought to be a gastric ulcer have not any ulcer.

If the bleeding from an ulcer is so severe that in spite of all medical treatment the patient dies, I think we ought not to regret having neglected to open the stomach, for the patient would have died in any case, and his chance

of recovery would have been lessened by surgical interference.

MacNevin and Herrick's figures are of interest in connection with this subject, for of 55 cases of undoubted gastric ulcer shown at a post-mortem examination, which died from either perforation or hæmorrhage, 25 died from hæmorrhage. Of these, 19 were males and 6 were females; of the 30 dying from peritonitis, 8 were males and 22 were females; and of the 25 of both sexes dying from hæmorrhage, all except one female were 30 years of age or over.

The patients who are thought, because of their hæmatemesis, to have a gastric ulcer, and yet no ulcer exists, form a considerable group which I have described elsewhere.[1] A few generations ago physicians thought that these cases existed, but they wrongly ascribed the bleeding to vicarious menstruation. When this view was shown to be untenable physicians equally wrongly thought that these cases were examples of gastric ulcer. I have collected twenty-nine cases in which either a post-mortem examination or a careful inspection of the interior of the stomach during life showed that although there had been severe hæmatemesis yet no ulcer was present, nor was any commonly recognised cause—*e. g.* cirrhosis—found to explain the bleeding, and I have suggested that the condition might be known as gastrostaxis. Only two of my patients were males, so it is rarer in men than is gastric ulcer; further, it is much less often met with after the age of forty years than is gastric ulcer. Inasmuch as it does not lead to perforation or to adhesions and dilatation of the stomach and the bleeding usually stops, the mortality of gastrostaxis is very low, and its rarity after forty shows that it tends to get well. Further, when the stomach is opened during life while bleeding is going on the blood is often seen to be oozing from many points, so that it is surgically very difficult to

[1] "On Gastrostaxis, or the Oozing of Blood from the Mucous Membrane of the Stomach," 'The Lancet,' November 3rd, 1906, p. 1189.

stop the hæmorrhage, and even if this is done the patients often later get further attacks of hæmorrhage. For all these reasons the patient should not be subjected to operation if she is thought to be suffering from gastrostaxis; indeed, several patients have died after operation, and I have collected twenty-four cases operated upon with eight deaths, or a mortality of 27·5 per cent.; therefore, as the disease is rarely fatal among those not operated upon, we must conclude that several of these eight cases would not have died if they had not been subjected to operation. I cannot possibly in these few minutes discuss the causation of gastrostaxis or the diagnosis of it from ulcer; my only concern now is to point out that examples of it are not suitable for operation.

Passing on to the chronic effects of gastric ulcer everyone will admit that a subphrenic abscess should be opened, but it is, I believe, a rare complication of gastric ulcer, and a physician may be attached to a large hospital and yet meet with very few cases. The common chronic effects of gastric ulcer for which operation is desirable are adhesions, repeated hæmorrhage, and dilatation.

Adhesions are no doubt a very common result of gastric ulcer, and their symptoms are very perplexing. I am not sure, but I think that they most often give rise to symptoms when they connect the stomach with the bowel, and this is what we should expect; for the stomach is constantly in close contact at the upper part of its pyloric end with the liver and at its back with the pancreas, so that actual adhesions in these situations with the pancreas or liver would hardly be likely to give rise to many symptoms, and the upper curvature and posterior surface of the stomach are the parts of it which alter their positions least, but its greater curvature descends when the organ fills, and is liable to form adhesions with the bowels, and then either a movement of the stomach or one of the bowels will drag on the

adhesions and so cause pain. The pain is often paroxysmal, the patient goes days or weeks without any pain, and then he has an attack; why there should be such a long interval between the attacks I do not know. The pain caused by adhesions is often very severe, and if frequent the patient is quite unfit for work. He commonly complains that he does not know what brings it on, and often it bears no relation to food. The ulcer may be quite healed, the patient has no indigestion, his tongue may be clean; indeed, he often suffers from nothing but the pain. In a bad case it may be constant, but even then there are acute paroxysms of greater pain, and the severity and frequency of these may be so great that his general health is undermined, and he may lose flesh. Such cases—especially if there is no history of gastric ulcer—may be wrongly called abdominal neuralgia or hysteria. Sometimes there is distinct tenderness over the adhesions, and sometimes an ill-defined obscure mass may be felt, giving such a feeling as we should expect to be caused by a mass of adhesions. This pain continues for years, indeed so long that it cannot be due to malignant disease.

Careful examination of the patient and his history enables us to put aside floating kidney, biliary or renal colic, and so we are often able to come to a correct diagnosis, which is, of course, much easier if definite symptoms of a gastric ulcer or severe indigestion are, or have been, present, but the patient may lose flesh quite apart from these. If a careful and repeated consideration leads to the opinion that adhesions are present, and if the trouble they cause is sufficient to mar the patient's life, an operation should be advised, for we know of no drug that will get rid of adhesions; but we must not promise certain success, for, in a long-standing case, especially with a woman, so much neurotic trouble is added to the original disease, and so deeply stamped on the patient's individuality that the removal of the adhesions—gastrolysis as the operation has been called—

does not lead to the expected relief of all the symptoms, just as the stitching of a floating kidney does not always by any means relieve all the patient's trouble. It is notorious that diseases of several abdominal organs—*e. g.* kidney, stomach, and female generative organs—are particularly liable to be associated with neurosis. I remember a woman in whom the removal of adhesions led to complete relief of her pain so long as she remained in the hospital; when she returned to sympathetic friends the pain returned, when she returned to the hospital again the pain disappeared. Still, in many cases, especially in men, the relief afforded by removal of adhesions may be complete.

I call to mind the case of a man in whom a long adhesion was found connecting the stomach with the bowel. The paroxysms of pain were so frequent that work was impossible. He never had any pain after removal of the adhesion. He gained weight rapidly, and was able to work hard.

We must always remember that the adhesions may re-form, and it will be interesting to hear from surgeons the best way of preventing this, but nevertheless there is no doubt that often complete relief follows their removal, and in the cases in which they are so extensive that it appears certain they will re-form, gastro-jejunostomy will often relieve the patient. It goes without saying that they may be so extensive that they cannot be removed, but the most extensive adhesions take place at the back of the abdomen, and in this situation their removal is often undesirable and impossible, for the stomach is so ulcerated that the structures on the back of the abdomen are exposed; here, too, gastro-jejunostomy may be of service.

From time to time we meet with cases in which repeated attacks of severe hæmorrhage compel the patient so frequently to give up his or her work that operation should be performed. Not long ago I saw a man who had had a very severe hæmorrhage necessitating a long stay in

bed in September and another serious attack the following November. From this he did not pick up rapidly, and remained so anæmic as to suggest that lesser bleeding continued from time to time, although there was no hæmatemesis. Everyone must have had experience of such cases, and I think that, when an attempt to confer substantial improvement by medical means has failed, the patient should be asked to see a surgeon with a view of performing a gastro-jejunostomy, and if this is done the risk of further hæmorrhage is greatly diminished.

As has been already mentioned, the practical difficulty is to know which are the cases of gastric oozing—or gastrostaxis—and which are the cases of genuine ulcer, a very important point, however, for gastrostaxis is hardly ever fatal if no operation is performed. On the other hand, if an operation is done the patient not infrequently dies or the bleeding continues.

Many cases of gastric ulcer in which the hæmorrhage may or may not be severe suffer from serious indigestion as a result of their ulcer, they have much pain, are thin, and often quite unfit for work; indeed, when we see the ulcers post mortem we can well understand why this should be so. Here again, if repeated medical treatment has failed, the question of operation should be put before the patient. I know of several instances in which such patients have been converted by gastro-jejunostomy from pale, thin, weak, miserable dyspeptics unable to work, to strong, healthy members of society, able to lead a hard-working life.

I need only mention for the sake of completeness that, when a gastric ulcer cicatrises so as to form an hour-glass stomach, and severe symptoms are due to this, surgery should interfere.

In passing I may perhaps allude to the fact that many patients are kept on rectal feeding far too long. All rectal feeding is starvation diet, and therefore should never be long continued; an ulcer cannot be expected to heal if the sufferer from it is starving. Then, subcutaneous

feeding is not often used, though it may be. Sterilised olive oil may be injected subcutaneously night and morning. I have done this daily for a month. Half an ounce or more may be injected each time. Inasmuch as a person at rest in bed hardly requires more than at the outside 2000 calories a day, we see that roughly about an eighth of the necessary energy may be given under the skin, and, as fats are the very kind of food which are extremely badly absorbed from the rectum, they are a very important aid to rectal feeding if it is decided that this must be continued for some time. But usually it need not be, for the chief reason why patients with a gastric ulcer are restricted in their diet is a fear of perforation. But there is a large group of cases in which adhesions render perforation almost impossible, and then perforation is rare in patients who have recently brought up blood. Moynihan gives the histories of twenty-one cases of perforated gastric ulcer, and in not a single one is recent hæmatemesis mentioned. Therefore, if there has been recent hæmorrhage the risk that food by the mouth will lead to perforation is remote, and it may be given carefully in small quantities a few days after the cessation of the hæmorrhage.

Next we have to consider dilatation of the stomach. This is one of the most important evidences of ulcer, but for it to be important evidence it must be considerable; on the other hand, I do not think that in this country we often see great dilatation of the stomach unless due to growth or ulcer; hence, in cases in which growth can be excluded, extreme chronic gastric dilatation is almost proof that there is an ulcer which, by its cicatrisation or the formation of adhesions, has led to gastric dilatation.

Whether or not a dilated stomach should be operated upon depends upon the degree of dilatation; many of the lesser degrees, if the stomach is washed out once in the twenty-four hours, will disappear even when thought to be due to an ulcer, but it may be that in some of these cases the diagnosis is at fault. As, however, with lavage

and appropriate food they may get better the treatment should always be tried. If it succeeds, well and good; if it does not, and the dilatation is slight, the case must be most carefully reconsidered, for often the slighter degrees of dilatation are not due to pyloric obstruction, and unless dilatation is due to this operation can do no good. When, as is frequently the case with patients who come into the hospital, the dilatation is very great—the last patient I had under me used to vomit more than two quarts at a time—then operation may be advised even without going through a course of washing out, for such extreme chronic dilatation must be due to pyloric obstruction, and will not benefit for long by lavage. The improvement that sometimes follows gastro-jejunostomy in such a case is very great, indeed, after a time the patient feels and appears quite well. As tetany is one of the results of gastric dilatation any operation for tetany is the same as an operation for gastric dilatation.

There are some general propositions which apply to operation for any of the results of gastric ulcer, and which must be borne in mind. No operation is quite free from risk; even in the 1905 edition (American translation) of von Bergmann's *System of Surgery* it is stated that the mortality in Mikulicz's clinic among the last forty patients operated upon for benign affections of the stomach is 7·5 per cent., but, on the other hand, the mortality is decreasing. Paterson (1906) puts it down at 3 per cent., but, judging by the results obtained at Guy's, it has not yet reached nearly so low a figure. Anyhow, the patient must always be asked whether he prefers to take the risk or remain as he is. We can hardly ever say to him—unless perforation has occurred —if you remain as you are your ulcer will almost certainly kill you. Next, we must remember that some patients suffering from chronic ulcer are not relieved by operation; it may be that adhesions cannot be divided, or that even when a gastro-jejunostomy has been done pain and even bleeding may continue. Unfortunately, there is no means

by which we can tell beforehand whether or not the patient will be relieved, but great relief follows operation sufficiently frequently for us to be able to say to a patient, for whom medical treatment has failed, that there is a very considerable prospect that he will be much better after an operation.

In a few cases a peptic ulcer in the jejunum follows a gastro-jejunostomy, but these cases are so few that they need hardly influence us much in the advice we give, especially as it will be impossible during life to say whether or not symptoms are due to the persistence of those of ulcer or the formation of a peptic ulcer in the jejunum; and, further, if Paterson's suggestion is correct that a jejunal peptic ulcer is due to hyperchlorhydria, which is itself due to an insufficient anastomotic opening, jejunal peptic ulcer ought to disappear with improved methods of operating, which will also probably get rid of the regurgitant vomiting which occasionally follows.

I think the evidence is conclusive that the metabolism of the body is not appreciably altered by a gastro-jejunostomy, and even if there is a trifling diminution in the absorption of proteid this may be a blessing in disguise, for it seems that most of us habitually take more proteid than we require. I have been at considerable pains to look through the literature of the question as to whether a growth is particularly liable to develop upon an ulcer, and also I have looked at many specimens. I have not been able to convince myself that any such liability is serious, and, at most, it is so slight that it need not weigh with us in considering operation. The greatest difficulty in deciding whether to operate lies in the uncertainty of diagnosis. No statistics from operations are of value unless the surgeon at the time of operation has seen the ulcer, or a considerable mass of adhesions originating in the stomach, or great dilatation of that organ.

Numbers of young adults who would twenty years ago have been said to have an ulcer have not got one; they are examples of gastric oozing, or gastrostaxis, as it may

be called, but, as this condition is commonest in young subjects and in women, it follows that we are most likely to be correct in our diagnosis of ulcer, and, therefore, most likely to do good by operation if the patient is a man and over thirty years of age. Even if there is an ulcer and the patient is a woman probably the likelihood of improvement is less than if the patient is a man, for in women many neuroses that operation may not benefit are liable to be added to the symptoms of organic disease.

We should, I would suggest, be very chary of operation —apart from that for perforation or abscess—without definite evidence of bleeding, because if there has been none it is quite likely that the case is one of neurotic dyspepsia, for this often cannot with certainty be diagnosed from gastric ulcer, and all experience shows that operation upon the stomach for gastric symptoms not due to ulcer or growth does no good, and even makes the patient worse.

I have not said more about the ultimate results, for my colleague, Dr. H. S. French, has kindly undertaken to collect the results from Guy's Hospital, and he will give them to you; but, speaking generally, the results of operation for chronic gastric ulcer in which medical treatment has completely failed to relieve are—if the diagnosis is correct—so frequently good that every patient ought to have put before him the reasons for and against operation. The statistics from Guy's Hospital put the question in a severely unfavourable light, for the patients were mostly those whose health was undermined by long illness, a large proportion were over forty, and many were operated on early in the history of these operations, in which operators are gaining skill daily.

THE OPERATIVE TREATMENT OF NON-MALIGNANT ULCER OF THE STOMACH AND ITS CHIEF COMPLICATIONS, WITH INDICATIONS, LIMITATIONS, AND ULTIMATE RESULTS

A DISCUSSION

Tuesday, November 13th, 1906

OPENING SPEECH (SURGICAL)

BY

A. W. MAYO ROBSON, D.Sc., F.R.C.S.

MR. PRESIDENT,—I thank you for the honour that you and the Council of the Society have done me in asking me to open this discussion.

An experience of nearly 600 operations on the stomach for various diseases, together with observations on a large number of cases in which no operation was performed, has naturally given me much material for study, on which I have, rightly or wrongly, based certain definite opinions; and the only difficulty I now find is how to condense into a short paper the points most appropriate for a useful discussion. For it is manifestly impossible in one, or even in two or three, meetings to fully consider and discuss the indications, limitations, and ultimate results of operations for all the non-malignant diseases that may call for surgical treatment; as this would involve a consideration of acute and chronic ulcers, with their complications—perforation, hæmorrhage, hour-glass contraction, cicatricial stenosis and dilatation of the stomach, perigastritis with

adhesions, perigastric abscess, inflammatory tumours, fistula, etc.—as well as the subjects of gastroptosis, acute and atonic dilatation, gastric tetany, pyloric and cardiac spasm, congenital hypertrophic stenosis, and other important diseases.

I propose therefore to limit my remarks to the question of the indications, limitations, and ultimate results of operation in ulcer of the stomach and two or three of its chief complications.

I feel sure that gastric ulcer is a much more serious disease than it is ordinarily thought to be, and that the profession has generally considered it more amenable to medical treatment than the facts really warrant.

The accuracy of the observations of Leube, who states that one half or three fourths of all cases of ulcer will be cured by four or five weeks of treatment, but if not cured in that time they will not be cured by medical treatment alone, has been more than borne out by the careful investigations undertaken by Dr. Bulstrode at the instigation of Mr. Mansell Moullin in 500 cases of ulcer occurring in the London Hospital from 1897 to 1902. He showed that 18 per cent. died while under medical treatment, and these cases did not include any of those patients admitted suffering from the serious complications of ulcer, such as pyloric stenosis, hour-glass contraction, gastric dilatation, etc.

Of the 82 per cent. discharged as cured, Dr. Bulstrode calculated that in at least two fifths relapse would occur, for out of the 500 cases no less than 211 had suffered and been under treatment for ulcer from one to four or more times previously.

The more recent observations made by Mr. Paterson and Dr. Rhodes at the London Temperance Hospital on 158 consecutive cases under the care of Dr. Soltau Fenwick and Dr. Parkinson are still more striking, showing that the proportion of real cures in cases of gastric ulcer, even after prolonged treatment by diet and rest in hospital, is under 25 per cent.

Private patients, amid more favourable surroundings, who can rest longer and are more willing to bear restrictions of diet over a lengthened period, suffer less from relapses, though probably not less from the dangers of the serious complications, hæmorrhage and perforation, occurring in acute ulcer.

We may thus on ample evidence accept the fact that at least one third of all cases of ulcer of the stomach treated medically ultimately succumb to the disease or to one of its many complications, and that one half or two thirds of the cases that recover relapse. How does surgical treatment compare with this?

As surgeons we only see the worst cases that have failed to yield to medical treatment, or cases suffering from relapsing or chronic ulcer, or from one or other of the serious complications; yet, arguing from my own experience alone in over 400 operations of various kinds, such as pylorectomy, excision of the ulcerated area, gastro-enterostomy, ligature of bleeding gastric vessels, pyloroplasty, gastrolysis, etc., undertaken for ulcer or its complications (excluding perforative peritonitis), usually in-patients at the time very seriously ill, the total mortality has been only a little over 3 per cent.; but, as I shall show later, the actual risk of posterior gastro-enterostomy in my private practice, the most frequent operation in this class of cases, has only been 1·7 per cent, and the patients completely relieved have been over 90 per cent.

From the point of view of treatment ulcers may be conveniently divided into two classes—acute and chronic.

In *acute ulcer* the treatment should at first be medical, and, above all things, it should be thorough, but, if after a month or six weeks in bed on milk diet followed by a further similar period of from two to three months in which only soft food, chiefly milk, is taken—the patient is not free from digestive troubles, or if, after a period of freedom from symptoms, relapse occurs, the question of surgical treatment should be seriously considered.

In *chronic*, or relapsing ulcer, if medical treatment has not had a fair trial, a rigid course of diet and rest, as in acute ulcer, should be tried; but in case of non-relief or of relapse after temporary freedom from symptoms, surgical treatment ought to be urged.

Limitations.—Before surgical treatment is recommended, it must be ascertained as far as possible that the symptoms clearly and definitely point to ulcer, and to this end a chemical, as well as a clinical, investigation should be made, since operation is, as a rule, contra-indicated in purely functional cases, and, when carried out, not only gives no satisfaction, but tends to bring discredit on operative treatment.

Surgical treatment may be direct or indirect. By *direct* treatment excision of the ulcer or of the ulcer-bearing area is referred to; by *indirect* treatment is meant gastro-enterostomy or one or other of the modifications of pyloroplasty, operations the aim of which is to relieve obstruction and to secure physiological rest to the stomach.

The *direct* operation has been practised since 1881, when Rydygier excised a large ulcer from the posterior wall of the stomach, but his results, and the experience of other surgeons appear to show that the direct operation is more dangerous than gastro-enterostomy. Moreover, gastric ulcers are frequently multiple, and the excision of one ulcer will not cure the remaining ones; hence, as might be expected in practice, excision of ulcer has been frequently followed by relapse. My own experience has extended to 9 of these direct operations, and the fact that all the patients recovered shows that the dangers with modern technique need not deter us from excision if it be the better operation; but of the 9 cases, while 6 are well from three to eight years later, 2 have relapsed, and 1 cannot be traced. Other surgeons, including Mr. Mansell Moullin, Mr. Keetley, and Mr. Sinclair White, have also recorded cases that have relapsed and ultimately proved unsatisfactory.

In future, should I deem it wise to excise the ulcer or

ulcer-bearing area in any exceptional case, as, for instance, in indurated, bleeding, or a directly circumscribed ulcer, I should also, as I did in my last cases, perform at the same time a posterior gastro-enterostomy, in order to secure physiological rest for the cure of any other ulcers that might be present.

The operation of excising the ulcer-bearing area with the performance of an independent gastro-enterostomy will probably in the future be more frequently performed for, as I tried to show in my Bradshaw Lecture at the Royal College of Surgeons, no less than 59·3 per cent. of cases of cancer of the stomach on which I had operated gave a history of chronic ulcer.

Dr. Graham states that a pre-cancerous history of ulcer was obtained in 36 per cent. of cases of cancer operated on in the Rochester clinic, and a clear evidence of cancer development on ulcer in 30 per cent. of the last 40 partial gastrectomies. This affords the most potent argument for the radical operation.

The indirect operations are pyloroplasty and gastro-jejunostomy.

Pyloroplasty, invented by Heinecke in 1886, and improved by Mikulicz in 1887, although excellent in principle and safe and simple in practice, has been followed in a large proportion of cases by relapse. Although the symptoms may be relieved for months or possibly years, the tying up of the pylorus by adhesions or the subsequent contraction of the cicatrix ultimately in about a quarter of all the cases in which operation has been done has led to relapse and the necessity of a further operation. I have personally performed 28 pyloroplasties with immediate success in all except 1, that died a fortnight later from perforation; but of the 27 that recovered and remained well for various periods, in only 16 have the results been perfect, the patients being well from four to eleven years later; 8 have required a subsequent operation; 1 was quite well for nine months, relapsed and died of

acute tetany some time later; and 2 are said to have developed cancer after six years and eighteen months respectively of good health.

Dr. W. J. Mayo, out of 21 cases, had 7 that required a secondary operation, and Mr. Rutherford Morrison had four relapses out of 28 cases.

The modification of pyloroplasty invented by Dr. Finney and improved by Dr. Gould, of Boston, which gives a very wide opening from the stomach into the duodenum, is still on its trial; it is a severe operation when the pylorus and duodenum have to be extensively freed from adhesions. Out of 58 cases operated on by Dr. W. J. and C. Mayo the mortality was 7 per cent., and of 112 cases collected by Dr. Finney it was 9 per cent.

I have no personal experience of Finney's operation, as I prefer to perform gastro-enterostomy, but Dr. Munro, of Boston, says that the operation has not given him good results and he has abandoned it, and Dr. W. J. Mayo thinks it only available in certain selected cases.

Gastro-enterostomy, first performed by Wolfler at the suggestion of Nicoladini in 1881, in a case of obstruction of the pylorus, due to cancer, was first employed for the treatment of ulcer by Doyen in 1893, and in the same year by Talma, who independently came to the conclusion that spasmodic stenosis of the pylorus was the chief factor in maintaining the hyperacidity and unrest which prevent the healing of gastric ulcers.

It acts by affording a free outlet from the stomach, thus overcoming stasis, relieving hyperchlorhydria, securing rest, and preventing the tension induced by spasm of the pylorus.

The after-results of the operation in ulcer of the stomach and duodenum are usually really remarkable. The pain vanishes, food can be readily taken and retained, vomiting ceases, in a little time the anæmia improves, and as a rule the patient rapidly puts on weight.

The ill effects said to follow the operation can probably all be avoided by correct technique. I well remember in

the early cases of gastro-enterostomy the fear that was entertained of regurgitant vomiting—the so-called vicious circle—a complication that I have not experienced in my work since adopting the posterior operation without a loop; neither have I seen a case of closure of the anastomotic opening nor any of the forms of internal hernia, either through a loop or through a slit in the mesocolon, after any of my operations.

Death from asthenia, which at one time was to be feared after stomach operations, when starvation for some days was adopted in already enfeebled patients, is no longer a danger, as feeding can be begun immediately after operation.

The danger of primary hæmorrhage is entirely avoided by continuous sutures embracing the margins of the opening between the two viscera, though it is not always possible to avoid the dangers of secondary hæmorrhage from pre-existing ulcers.

The only complication that perhaps is unavoidable is extremely rare—peptic ulcer of the jejunum, which apparently arises from an excessively acid gastric juice passing directly into the jejunum, just as one not infrequently occurs in the duodenum, leading to duodenal ulcer. The fact that I have not had a case of peptic jejunal ulcer in over 200 posterior gastro-enterostomies shows that this danger is a very remote one.

I have operated on 3 cases of jejunal ulcer, although in only 1 of the 3 did it occur after I had myself operated, and in that case the original operation performed three years and four months previously had been an anterior gastro-enterostomy with a long jejunal loop; of the other 2 cases in which the original operation was performed by friends or colleagues, 1 was after an anterior and 1 after a posterior operation.

I think the condition will in the future be avoided by making the opening sufficiently large so that there can be no possibility of stasis, and by taking pains to carefully diet and treat the patient medically for some time after

gastro-enterostomy so as to thoroughly cure the hyperchlorhydria.

It has been clearly proved that this complication occurs more frequently after the anterior operation, where there has been a long jejunal loop, but whether occurring after the anterior or posterior operation it is very serious, as perforation is very liable to occur.

There is no time to enter into minute details of the technique of gastro-jejunostomy, but I cannot help giving expression to my feeling that many details described as essential by various operators are by no means necessary to success. For instance, the oblique incision of the stomach-wall is quite unnecessary, and I have never adopted it, the only essential so far as the incision is concerned being that it should be sufficiently large, not less than two inches, and that it should be close to the lower border of the stomach. Nor is excision of the mucous membrane necessary or to my mind desirable, as all that is essential is that the mucous membrane of the jejunum and stomach should be brought into contact by the marginal continuous suture which must for hæmostatic purposes take up all the coats. The use of the clamps, though not essential, has, I believe, contributed largely to the diminished mortality of the modern operation.

Though I personally prefer the posterior operation with the so-called no loop, which has given such satisfactory results, yet, as proved by the permanent success of many of the earlier cases, the anterior operation is quite capable also of yielding good results, but I think at a greater risk of complications from the necessarily long jejunal loop. Roux's operation and short-circuiting of the loop are, in my opinion, unnecessary procedures except occasionally when the anterior operation is alone feasible.

Statistics.

The statistics of gastro-enterostomy for ulcer cannot be accurately estimated by merely collecting a large series of

cases from the records of all the general hospitals, in which perhaps only a few operations are performed by each member of the staff and with very varying results; but a true estimate may be obtained by a consideration of the results of individual operators who have accurately recorded and followed up the after-progress of their cases.

Nor do I think it desirable, in estimating the mortality of gastro-enterostomy in simple ulcer, to put the cases of ulcer complicated by hæmorrhage or hour-glass contraction into separate lists, as it may open the way to misconception of the actual results if the simple uncomplicated cases are placed in a separate category.

The following statistics include the results of gastro-enterostomy for ulcer and its complications, hæmorrhage and hour-glass contraction (excluding perforative peritonitis), following the plan employed in the 'Report' of St. Mary's Hospital, Rochester, Minnesota.

Dr. W. J. Mayo reported ('Annals of Surgery,' November, 1905) having performed 307 gastro-jejunostomies for *non-malignant* disease, with a mortality of 6 per cent., but in the last 81 operations of the series there had been only 1 death; and in the 'Report' for 1905 of St. Mary's Hospital, Rochester, I notice that out of 109 gastro-jejunostomies for ulcer, or the results of ulcer of the stomach and duodenum, only 1 patient died. In a supplement to that paper, published in the 'Annals of Surgery,' April, 1906, Dr. Mayo reports 136 posterior gastro-enterostomies, with but 1 death.

At the British Medical Association meeting at Leicester, July, 1905, Mr. Moynihan gave the result of his experience up to date. The cases included 171 gastro-jejunostomies for chronic ulcer, with 2 deaths; 27 cases in which hæmorrhage was a prominent symptom, with 4 deaths; and 8 cases of hour-glass stomach, in which gastro-jejunostomy had been performed, with 3 deaths, thus making 206 cases of ulcer, with its complications of hæmorrhage and hour-glass contraction, in which gastro-jejunostomy had been performed, with 9 deaths, giving a mortality of

4·3 per cent. The after-results of operations for chronic ulcer uncomplicated by acute perforation or severe hæmorrhage yielded 82 per cent. cured and 14 per cent. relieved. In a communication to this Society, read May 22nd, 1906, Mr. Moynihan reports 249 cases, with 9 deaths, equal to 3·6 per cent. mortality.

Hartmann reports his death rate as 6 per cent. and his final result as 90 per cent. of cures.

Von Eiselberg gives his final results as 90 per cent. well two years after operation.

Rotgans of Amsterdam gives his immediate risk as 5 per cent. and a permanent cure in 80 per cent. of cases.

The following is my own personal experience; it includes every instance in which I have performed the operation of posterior gastro-enterostomy, either in hospital or in private practice. There have been 210 cases, of which 202 recovered from operation, thus giving a mortality of 3·8 per cent.

As the after-histories are almost impossible to obtain with any degree of accuracy from the class of patients seen in hospital, I have based my observations on my private cases in which the after-histories have been kindly furnished by the medical colleagues with whom I was associated.

Of the 112 posterior gastro-enterostomies that I have performed in my private practice for ulcer or its complications, including hæmorrhage and hour-glass contraction, 110 have recovered, thus giving an operative mortality of 1·7 per cent.

Out of the 110 recoveries 96 are in good health now, at periods varying from within a year up to five years after operation. Two were alive and well three years subsequent to operation, but no reply has been received as to their present condition. Two were alive and well some time after operation, but no word has been received of them later. One lived in good health for two and a half years, and when travelling in Italy died of acute pneumonia. One remained in perfect health for three years,

but has lately had some vomiting. One lived in good health for three and a half years after operation, and then developed cancer in the abdomen. One lived two and a quarter years and died of cancer of the stomach. One had perfect health for a year, when a tumour at the pylorus developed, but no further operation was permitted and she died two months later. One had improved health for two and a half years, when she had hæmatemesis and died shortly after an attack. One was well for two years, when she had recurrence of vomiting; a further operation was performed, but the old gastro-enterostomy opening was found to be perfectly patent, and though the patient is living, her condition is not satisfactory. One has never been well since operation two years ago, and now has developed a tumour in the colon. One has since died of pernicious anæmia. One has been relieved, but still suffers at times from pain and flatulence after food.

Thus it will be seen that 101 out of 110 posterior gastro-enterostomies for ulcer, or its complications, performed in private practice, apparently resulted in cure: 7 were relieved, 2 were not materially improved, and 2 died.

Excellent results have also been obtained by Ochsner, Murphy, Deaver, Kocher, Munro, Brewer, Littlewood, Monprofit, and others.

The only complications of ulcer of which I propose to discuss the operative treatment are perforation, hæmorrhage, hour-glass contraction, dilatation due to pyloric stenosis, tetany, and perigastritis.

Perforation.

This complication, which is estimated by various authors to occur in from 12 to 28 per cent. of all cases of gastric ulcer, is fatal, unless surgically treated, in 95 per cent. There can, therefore, be no room for any difference of opinion as to the wisdom of operation in this serious accident. As shown by the statistics drawn from a very large series of cases, every hour's delay adds to the danger; hence it is of the utmost importance that an

early diagnosis and immediate operation should be insisted on.

In cases operated on within twelve hours of rupture the mortality has been 25 to 28 per cent.; in from twelve to twenty-four hours 63 per cent.; in from twenty-four to thirty-six hours 86 per cent.; and after thirty-six hours the mortality has been so great—95 to 100 per cent.—that purely medical treatment would seem to give an equal chance of recovery.

The results of operation in late cases will probably always be unsatisfactory, but I believe that the cases seen within twelve hours of rupture should give much better results than those yet attained; my feeling is that the mortality should be brought down to from 5 to 10 per cent.; in fact, in a continuous series of 11 cases operated on within a few hours of rupture and reported last year by Dr. T. Sinclair Kirk there was no mortality, showing that such a forecast is possible.

My experience is that excision of the ulcer is not necessary to success, and that a folding in of the edges of the rupture, with the careful application of a continuous serous suture, and, if possible, an omental graft laid over it, gives the best results. I prefer to wash out the abdomen with a hot, normal saline solution, and to drain above the pubes with the patient well propped up in bed; but, as shown by a series of cases operated on by Mr. Littlewood in which lavage was not adopted, washing out is not always necessary to success.

My rule is that where the effusion is general lavage should be adopted, but where it is local a mere wiping out of the soiled area is only necessary.

The important point is the actual drainage to the peritoneum; for if it has not lost its polish it is capable of absorbing any amount of effusion, but if the peritoneum has lost its polish it must be assisted by artificial drainage.

Seeing that in one third of all the fatal cases other ulcers have been found, that in 20 per cent. of cases of ruptured ulcer the perforations are multiple (Finney), and that in

a large proportion of cases recovering from operation the patients have subsequently had other gastric symptoms or even a second perforation, the question of the desirability of gastro-enterostomy at the time of operating for the perforation is well worthy of consideration.

Of 15 cases traced by Mr. Crisp English, 4 suffered subsequently from gastric trouble; and of 35 traced by Mr. Paterson, 1 died from perforation within two years, 2 required subsequent gastro-enterostomy, 9 had definite symptoms of gastric ulcer, and 5 had gastric symptoms.

In Mr. Moynihan's cases 7 out of 24 required an immediate or subsequent gastro-enterostomy.

The cases submitted at the same time to gastro-enterostomy are too few to argue from, but of those that recovered cure has been complete and permanent in all up to the present.

The chief argument against gastro-enterostomy is, of course, the prolongation of the time of operation in cases already very seriously ill from the perforation, but in cases not too ill to bear it the following arguments are decidedly in its favour:

(1) Other ulcers present at the time of perforation will probably be cured by the operation, and in case of ulcer at the pylorus the effects of cicatricial contraction will be averted.

(2) If a second ulcer is on the point of perforation such perforation will probably be prevented, as tension and pressure on the stomach walls will be avoided.

(3) After gastro-enterostomy more secure healing of the sutured ulcer is likely to occur and there will be less likelihood of the stitches giving way.

(4) It diminishes the risk of hæmatemesis occurring after operation.

(5) It enables saline aperients to be given shortly after operation, and so secures more efficient drainage of the peritoneal cavity.

(6) It permits earlier feeding than would otherwise be the case had no gastro-enterostomy been done.

On all these accounts therefore it is important that where the patient's condition will permit of it the question of the performance of a gastro-enterostomy should be seriously considered at the same time that the perforating gastric ulcer is closed.

Hæmorrhage.

The complication of hæmorrhage occurs in a greater or less degree in from 50 to 80 per cent. of all cases of gastric ulcer, and according to various authors is fatal in from 3 to 11 per cent.

From the point of view of treatment it seems convenient to classify the cases under two divisions:

(1) The acute, always alarming, and sometimes, though rarely, fatal attacks, that occur without any warning and without any premonitory symptoms pointing to ulcer, attacks that when occurring in young anæmic women usually cease spontaneously or under treatment, and that do not tend to recur.

(2) The attacks associated with or preceded by definite symptoms of ulcer, which may be (*a*) acute, ending rapidly in death, (*b*) acute, temporarily subsiding and recurring in a few hours or a few days, or after longer periods, and (*c*) the chronic hæmorrhages, often slight in amount, but frequently recurring and leading to anæmia.

Treatment.

From the fact that medical and general treatment is successful in arresting acute hæmatemesis in from 93 to 97 per cent. of all cases, and that it is difficult in the present state of our knowledge to say at first that the bleeding is not occurring from capillaries or small arterioles, it necessarily follows that medical treatment should always have a fair trial in every case of acute hæmatemesis. The very fact of medical treatment being so often successful in cases of apparently alarming hæmate-

mesis goes to show that capillary oozing or bleeding from arterioles, as in the first division, accounts for many cases of gastric hæmorrhage. But while thoroughly believing this, we must also not close our eyes to the experience we have in general surgery of bleeding from medium-sized arteries, such as the radial or ulnar, which we know would rapidly bleed a patient to death, if only perforated on one side and surrounded by warm compresses, a condition that practically applies in all cases of hæmatemesis where the larger vessels are eroded. If, therefore, medical treatment and rest properly carried out are not successful in arresting the bleeding, or if after being arrested it recurs, we should be suspicious that a large vessel is perforated, and if a surgeon has not been previously asked to see the case, I would say emphatically that a surgical consultation ought to be held with a view to considering the question of operation and arrest of bleeding by direct treatment if the patient is in a fit condition to bear it.

Where there have been distinct signs of gastric ulcer preceding the hæmorrhage, and where a sudden hæmatemesis has occurred with great loss of blood, accompanied by an attack of syncope, a large vessel will usually be found to be the source of the bleeding. In such hæmorrhages not speedily yielding to medical and general means, or recurring, surgical treatment will probably be advisable, though there can be no absolute rule formulated that will apply to every case, and each must be considered on its merits. The present condition of the patient, the previous history, the surroundings, the possibility of skilled surgery and of good nursing, and other circumstances, will all help in the decision.

Although both surgical and medical treatment in cases of fulminating hæmorrhage have so far yielded disappointing results, in the acute cases not immediately fatal, where repeated bleedings occur and the interval between the first seizure and death varies from a few days to two or three weeks, medical treatment will have been

fully tried and failed, and there can be no question as to the advisability of surgical procedures being adopted.

At present, with the exception of Dieulafoy, who advocates operation during the first bleeding if as much as half a litre of blood is lost, all other surgeons who have written on the subject agree that general means ought to be relied on during and after a first attack, as in from 93 to 97 per cent. of cases such treatment succeeds, and until our means of diagnosis as to the size of vessel injured is rendered more reliable we must advise assent to this rule; but after a second bleeding I have no hesitation in advising surgical treatment as soon as the condition of the patient will permit operation to be done, for experience tells us that further hæmorrhages are almost certain to occur unless preventive measures be adopted.

The surgical treatment of hæmatemesis may be direct or indirect.

By the *direct* method is meant—

(*a*) Excision of the ulcer or of the ulcer-bearing area.

(*b*) Arrest of hæmorrhage by ligature of the bleeding vessels, by cauterisation of the ulcer, or by ligature of the mucous membrane *en masse*.

By the *indirect* method, gastro-enterostomy is meant.

Any operation for hæmatemesis must, as a rule, be at first exploratory, and when the condition of the stomach is made out the question of direct or indirect treatment can be decided on.

In all the early cases of operation for hæmorrhage from the stomach the direct method was adopted, as it had not then been realised that by securing physiological and physical rest to the stomach by a well-executed gastro-enterostomy not only could bleeding as a rule be arrested, but the condition of ulceration giving rise to it could be cured.

Mr. Mansell Moullin still advocates the direct method by ligature *en masse* of the bleeding area, or by excision of the ulcer, and he urges in support of his view the fact that there have been cases recorded in which hæmorrhage

has persisted, or recurred, after gastro-enterostomy, but we must not lose sight of the fact that several cases have been recorded in which hæmatemesis has also occurred after local treatment of the bleeding ulcer.

My only experience of recurrence of hæmorrhage after short-circuiting has been in two cases of tumour of the pylorus, probably malignant supervening on ulcer. In one a fatal hæmatemesis occurred one month after what appeared to be a very successful operation, when the patient was apparently well. The other occurred in a man, aged 75, operated on for pyloric stenosis with persistent vomiting. He had had profuse hæmatemesis two weeks previously; after the operation the bleeding recurred in forty-eight hours, and proved fatal. In neither of these cases could excision of the ulcerated area have been performed with a probability of success, as extensive adhesions with enlarged glands prevented the pyloric tumour being brought forward even for inspection. Had it been practicable I should have performed wide excision of the tumour in both of these cases.

An extensive personal experience, and the consideration of a large number of cases operated on by others whose work I have had the opportunity of seeing or studying, has convinced me that the operation of gastro-enterostomy is usually an efficient means of treating hæmorrhage from the stomach, and it is only under exceptional circumstances that I should now think it worth while to open the stomach and treat the ulcer directly; but even should I deem direct treatment of the bleeding point necessary, I should also think it equally desirable to perform a gastro-jejunostomy in order to secure rest to the stomach and to the ulcer, so as at the same time to stop the bleeding and to cure the condition giving rise to it.

Gastro-jejunostomy possesses the advantage that it is applicable to both acute and chronic hæmorrhage, that it avoids the necessity of a prolonged search through a gastrotomy opening, that it is quickly performed, and, not

least, that it involves little shock and has a very small mortality.

A collection of a large number of cases of the direct treatment of the bleeding point not only shows a very heavy mortality, but also want of relief to the condition giving rise to the bleeding in a large proportion of cases, unless at the same time gastro-enterostomy had been performed.

Statistics of Operations for Hæmatemesis from Ulcer.

I have operated in 4 cases of acute hæmatemesis during the continuance of the bleeding. All the patients recovered and remain well. In 2 of these cases numerous bleeding points were ligatured *en masse* and gastro-enterostomy was done; in 2 gastro-enterostomy was alone performed.

I have also operated between the attacks on 22 patients suffering from hæmatemesis due to ulcer; in all a gastro-enterostomy was performed, with recovery from operation in 21. In one case death occurred at the end of a week from exhaustion. To put the matter succinctly I would say:

(1) In a case of acute hæmatemesis from ulcer or erosion when the onset of bleeding is sudden and the previous history of ulcer is absent, medical and general means should be very thoroughly tried, and if the bleeding ceases as the result of rest and general treatment there will be no cause to interfere surgically, though treatment should be carried out thoroughly and persistently until the ulcer has healed.

(2) In case of recurrence of bleeding, or if the bleeding persists despite treatment, surgical measures are called for.

(3) In case of bleeding from a chronic ulcer, whether the bleeding be slight or severe, surgical treatment is demanded, not only for the arrest of hæmorrhage, but also for the curative treatment of the ulcer itself.

(4) As the risk of operation during the quiescent interval

is less than if undertaken while the bleeding is continuing, it is desirable if possible to secure arrest of the hæmorrhage even in chronic ulcer before undertaking operation; but if the hæmorrhage is persisting the surgeon is not justified in waiting until the patient is reduced to such a condition that it is too late to operate.

Pyloric Stenosis and Obstructive Dilatation.

The very process of cure in pyloric ulcer can only result in stenosis, which, if moderate in extent, is overcome by hypertrophy of the gastric muscle. While this compensation is maintained symptoms may be slight or even absent, but sooner or later compensation fails and dilatation occurs, leading to stagnation of the stomach contents, with fermentation and the generation of acrid acids and offensive gases, giving rise to gastritis. Relief is obtained for a time by vomiting, but as the stenosis increases less and less food passes into the intestine, and death from starvation is the inevitable result should not tetany or perforation hasten the end.

In perhaps no other variety of so-called chronic indigestion are the patients so miserable, and as the disease may drag on for months or even years the sum total of suffering probably exceeds that from almost any other form of disease.

Treatment.—In the early stages of obstructive dilatation, when the symptoms are slight, relief will doubtless have been given by semi-liquid diet and lavage of the stomach; but as soon as the symptoms are pronounced it is a mere waste of time to persevere with the use of drugs, massage, electricity, or even lavage, except in those rare cases where the stenosis is due either to syphilitic ulcer or gumma, which should speedily respond to specific treatment.

Surgical treatment is alone of avail in order to remove the cause of the stenosis or to create a new channel by

which the stomach contents may pass onwards into the intestines.

It may sometimes be possible to remove the cause of the stenosis by division of peritoneal bands or adhesions or the removal of a tumour obstructing the pylorus, but in the majority of cases it will be necessary either to enlarge the contracted pyloric orifice or to perform a gastro-enterostomy.

The operations available are: (1) pylorodiosis, or forcible dilatation of the pylorus, (2) pyloroplasty, (3) Finney's operation, (4) Kocher's gastro-duodenostomy, (5) pylorectomy, (6) gastrolysis, (7) gastro-jejunostomy.

It is not necessary to describe or dilate on the various operations that may be performed in case of dilatation of the stomach due to simple pyloric stenosis, as the greater number of experienced surgeons are agreed that gastro-jejunostomy is the procedure that should be followed in such cases, not only because it can be done with very little risk, but also because the after-results of the operation in these cases are extremely satisfactory. The risk, as I have shown, is under 3 per cent., and the cases permanently relieved or cured are 90 per cent.

Although pylorectomy or partial gastrectomy is a much more severe operation than gastro-enterostomy, yet there is a certain class of cases of pyloric stenosis associated with tumour in which it is difficult to say whether the tumour of the pylorus is simple or malignant.

If the obstruction of the pylorus is associated with a tumour due to inflammatory disease, in all probability it will be so adherent to the under surface of the liver or to the pancreas that pylorectomy will be extremely difficult and hazardous. In such cases it will probably be deemed necessary to rest content with gastro-enterostomy in the hope that the rest induced by the operation will cause a subsidence of the tumour. I have found this to apply in many such cases in which at the time there was a question of malignant disease, but in which after gastro-enterostomy complete and permanent recovery

followed. If, however, under these circumstances the pylorus should be free and the disease limited, it may be quite justifiable to perform pylorectomy in case of doubt.

As pylorectomy, even in cancer, can be performed with a mortality of 15 per cent., or less, the risk of operation, should the disease prove to be simple, should not exceed half that amount.

My views on pyloroplasty and its modifications I have already given.

The operation of pylorodiosis, though recommended and performed by Loreta in some cases of cicatricial contraction of the pylorus, is not a procedure to be recommended, as though it has given good results in some cases of obstruction due to simple spasm of the pylorus, the procedure is attended with much more risk than either gastro-enterostomy or pyloroplasty, and the only form of obstruction in which I should consider Loreta's operation at all justifiable is in congenital hypertrophic stenosis where spasm is taking a share in the obstruction.

Gastric Tetany.

To make a distinction between the severe and fatal form of gastric tetany and the tetanoid spasms associated with gastric dilatation is purely arbitrary, and it seems more rational to include all cases of tetany and tetanoid spasms dependent on stomach diseases under the term "gastric tetany."

Morover, to conclude that gastric tetany is almost necessarily fatal, as is insisted on in certain medical works, is to my mind a great mistake, as an extensive experience of the disease, both of the mild and severe varieties, has convinced me that under efficient surgical treatment hardly any case is hopeless.

The treatment of gastric tetany is essentially surgical, as I had the privilege of first pointing out in a paper in the 'Lancet,' November 26th, 1898, when several cases were given as examples of successful surgical treatment.

Since that time my experience has increased, and out of a large number of cases in which I have operated I can state definitely that in no single case has drainage of the stomach by gastro-enterostomy failed to give relief. Moreover, at the same time that relief to the tetany is secured a cure of the disease producing it is accomplished, as in almost all cases there is a grave mechanical obstacle to the onward passage of the food. It is this obstacle which causes dilatation and hypertrophy, to be later followed by stasis and fermentation of the contents of the stomach. To relieve this obstruction and to do away with the stagnation of the stomach contents surgical measures are necessary. In simple cases pyloroplasty or gastro-enterostomy will be the methods of choice, in malignant cases a partial gastrectomy or gastro-enterostomy, as circumstances dictate. In hour-glass stomach gastro-gastrostomy combined with gastro-enterostomy performed in the distal pouch will, as a rule, be the suitable operation.

Hour-Glass Stomach.

Hour-glass stomach owes its origin to definite organic disease—chronic gastric ulcer, cancer, or perigastritis—but the greater number of cases are caused by the cicatricial contraction due to ulcer. Doubtless very rarely the condition may be congenital, just as congenital narrowing may be found at the pylorus, in the intestine, or in the rectum. As yet, however, in an experience of considerably over 1000 operations on the upper abdomen, in which the stomach could be seen and examined, I have not met with a single instance in which I could say that the case was one of congenital hour-glass contraction.

For chronic gastric ulcer to be allowed to pursue its course until this extreme deformity, hour-glass stomach, occurs, can only be described as a disgrace to modern medicine, for the disease giving rise to the conditions has

definite signs and symptoms, and is one attended by considerable suffering. In nearly all my cases the patients had been ill for years, some of them for ten, twelve, or sixteen years, whereas by a timely operation they might have been cured before the development of the deformity.

Surgical treatment is alone of service in this disease, and the conditions to be aimed at are to overcome the obstruction and to secure physiological rest for the healing of the ulcer which is by far the most frequent cause of this deformity. Out of 26 cases operated on by myself and 22 reported by my friend Mr. Moynihan 41 were the result of ulcer.

The operations available are: (1) gastroplasty, (2) gastro-enterostomy, (3) gastrolysis, (4) gastro-gastrostomy, (5) excision of the ulcerated area or partial gastrectomy, (6) divulsion of the stricture, and (7) jejunostomy.

It would occupy too much time to consider the indications for the different operations mentioned, but seeing that the disease is usually associated with active ulceration in the centre of the stomach, no operation will be likely to be permanently effective that does not provide for efficient drainage of both pouches, in order to secure healing of the ulcers; hence a double operation is often necessary, and a gastro-enterostomy into one or both pouches is generally advisable.

Of 7 cases in which I performed posterior gastro-enterostomy for hour-glass contraction, all recovered, and 5 are well at the present time, three to six years later; 1 died of cancer of the sigmoid flexure of the colon four years and 1 of cancer of the stomach a year after operation.

Of 10 cases in which I performed simple gastroplasty, all the patients recovered, and 6 are known to be in good health at the present time from three to six years later; 1 was well a year after, but cannot be traced; 1 required gastro-enterostomy two years later for ulcer of the pylorus; 1 was well for four years, had recurrence of ulceration and died of hæmatemesis, and 1 writes to say that after five

years she has had some gastric pain but has not needed medical attendance.

Conclusions.—Since gastroplasty has been followed by 25 per cent. and gastro-gastrostomy by 30 per cent. of relapses, while gastro-enterostomy alone has given much better results and the combination of the two former operations along with gastro-enterostomy has been uniformly successful, it seems quite clear that whatever procedure is adopted for making a communication between the two stomach cavities, a gastro-enterostomy ought to form part of the operation in order that the ulceration giving rise to the disease may be cured.

Perigastritis

leading to disabling adhesions is so commonly associated with ulcer that I must mention it among the principal complications.

When the adhesions tie up the pylorus so as to produce a kink, the normal peristalsis of the stomach may be interfered with, leading to dilatation and stasis so extreme that stenosis of the pylorus may be suspected. If slight, the adhesions may be separated and the right free border of the omentum may be interposed between the pylorus and its abnormal attachments so as to prevent close adhesions re-forming, but if the adhesions are extensive a gastro-enterostomy will be found to be the more satisfactory operation and to give excellent results. If adhesions form on the anterior wall of the stomach, fixing it to the abdominal parietes, normal peristalsis will be interfered with, and the patient's sufferings after meals or on exertion may be considerable; the condition may give rise to irregular dilatation or to hour-glass deformity and to stasis of the stomach contents.

The adhesions must be detached, but are apt to re-form if at all extensive unless the raw surfaces can be covered by sliding the omentum or grafting the peritoneum over them.

Simple gastrolysis or detaching adhesions is practically unattended by risk, and in many cases I have seen excellent results by this simple operation, but in several cases relief has been followed by relapse necessitating a short-circuiting operation. Posterior gastro-enterostomy should under such circumstances be performed where feasible, but in case of adhesions being very extensive and involving the posterior wall, an anterior Roux's operation will probably be found to be the best procedure to adopt.

In conclusion, I should like it to be understood that I do not advise operation in ordinary acute gastric ulcer, the medical treatment of which should, I think, be much more careful and more prolonged than was formerly considered necessary.

Nor do I advise operation in chronic ulcer before medical treatment has had a fair trial; but I think it is unfair to our patients to advise a continuance of general treatment for chronic or relapsing ulcer until serious complications supervene, when by timely surgical treatment many of them can be prevented.

In perforating ulcer, in chronic or relapsing hæmorrhages, in pyloric obstruction, or other mechanical causes leading to dilatation, medical treatment is as inadequate and useless as is operative treatment in gastric neuroses and other functional diseases, and to continue it beyond the period in which it can benefit is as unscientific and unwise as it is to operate and expect good results in improper cases.

THE PRESIDENT.

The very interesting communications from Dr. Hale White and Mr. Mayo Robson, to which we have had the privilege of listening, evidently furnish a very large amount of material for discussion. And the importance of the subject has, as you would suppose, led a great number of our Fellows to wish to take part in the discussion. I would therefore suggest that although it will clearly be impossible for us to conclude the matter this evening, yet it will be convenient if those who are good enough to speak on the matter will endeavour to confine their remarks within moderate limits, in fact, perhaps, to ten minutes, so that at any rate we may be able to have the pleasure of listening to some of those who are present here this evening from a distance.

Sir DYCE DUCKWORTH.

Dr. Hale White has done well to bring forward this subject at a time when the procedures of modern surgery in regard to abdominal operations compel the serious attention of the physician, and lead him to take a larger view of the possibilities which may be in store for certain of his patients.

With respect to gastric hæmorrhage in young women, I am in full agreement with the author as to the undesirability of any operative interference in the majority of cases. It is certainly as rare for such hæmorrhage to prove fatal as it is for ordinary hæmoptysis, always excluding the rupture of a pulmonary arterial aneurysm. Under ordinary treatment such bleeding ceases. The source of it is more commonly from an erosion with many oozing points or fissure in the gastric mucosa. I am aware that successful operations have been conducted for this condition, and Mr. Butlin effected it in one case for me. In another case one of my colleagues entirely failed to find the oozing points, although the stomach was everted and carefully studied. Death followed, and the bleeding surface was found very near the cardiac end, quite beyond any reach during life.

There can be no doubt that gastro-enterostomy has proved a great boon for many rebellious gastric ailments, including chronic ulcer, dilatation, and adhesion to various adjacent structures, and no less for obstinate forms of dyspepsia which yield to no form of medication or diet. I agree with the author that minor degrees of dilatation may often be adequately met by washing out and appropriate diet, and as many patients refuse surgical aid under these circumstances, we have to be content with

medical measures. Dr. Hale White may hardly be correct in considering rectal feeding as "always a starvation diet."

Dr. Sharkey in his recent Bradshaw Lecture has given good reasons for believing that this is not so, and that we have still something to learn in conducting this method. I agree that great care is necessary in deciding on any abdominal operation, and especially because in women too often we have to deal with forms of dyspepsia which closely resemble those of chronic ulcer. I also regard it as a duty of the physician to carry on the recognised treatment of gastric ulcer for a much longer period than is commonly practised. Absolute rest in bed, and appropriate feeding—truly nutritious—for many weeks should be the rule, and these weeks may safely sometimes run into months. We must be careful in the highest interest of the patient neither to hurry him into the hands of the surgeon, nor to imperil his best chance of relief by prolonging ineffectual treatment till he becomes an unsatisfactory subject for an operation.

Mr. Moynihan.

Sir,—I have presented to the Society a printed contribution which sets forth the total number of cases upon which I had operated up to September, 1906. I do not propose to inflict the reading of that paper upon the Society, but I shall be glad to refer very briefly to certain of the more important points which seem to me to be brought out by my series of cases.

First, with regard to the perforating cases. I hope there will be a good deal of discussion with regard to them, particularly upon two points. And first of all with regard to the diagnosis, especially in an early stage, of perforation. I am convinced that what one might call the text-book description of a case of perforating gastric ulcer is entirely inapplicable to the case as we ought to see it within the first two or three hours after perforation has occurred. In the recent cases which I have seen—and I say it very deliberately—there has been no suspicion of shock; the pulse-rate in the last three cases I have seen has been under eighty, and there has been no abdominal distension, there has been no abdominal rigidity, but there have been signs and symptoms which make the diagnosis perfectly easy. In the first place, there has been a sudden onset and continuance of absolutely intolerable pain. The patient complains very, very seriously of excessive pain, generally in the lower part of the chest, pain of such a character as to prevent him or her from breathing at all deeply, and that restriction of abdominal movements in respiration seems to me to be a very important sign in cases of perforation early on. In addition, there is always an excessive tenderness of the skin, there is a tenderness long before there is anything like abdominal rigidity, so that when one puts one's hand out to examine the patient's abdomen the hand is nearly

always met by the hand of the patient endeavouring to keep one away. When putting the hand on the skin there is excessive sensitiveness, and one which, moreover, is localised to a degree which will enable one, certainly in the majority of cases, not only to diagnose the existence of perforation, but to recognise the part of the stomach in which it has occurred. That is a matter which is also of very considerable importance from the purely surgical point of view, because in the last three cases that I have operated upon I have made an incision deliberately to left or right, according as I had diagnosed the position of the ulcer, and in the three cases, which are not many, but enough for my argument, the ulcer was in the exact position which I had predicted before the operation began. And it is not enough to predict verbally; I have made a point in my recent cases of drawing a picture and marking the place where it is likely to be found. With regard to the performance of gastro-enterostomy at the time I cannot see eye to eye with Mr. Mayo Robson in this particular. I have performed gastro-enterostomy at the time in six cases instantly—that is, as soon as the perforation was closed I have performed gastro-enterostomy, in my series of twenty-seven cases. I think the indication for gastro-enterostomy in perforating ulcer should be a positive one. It is not enough to say that because a patient has perforating gastric or duodenal ulcer, therefore, in addition to the closure of the perforation, gastro-enterostomy should be performed. I think gastro-enterostomy should only be performed, first of all, when the closure of the ulcer has produced such a condition in the stomach as will impede the digestion and progress of the food, when there will be a permanent obstruction from the closure and the subsequent cicatricial contraction of the ulcer, or, again, when there is a reasonable suspicion of the presence of a second ulcer, and especially if that ulcer be nearer to the pylorus than the one which has been closed. In those two circumstances only, I think, is gastro-enterostomy justified or compulsory.

With regard to the cases of hæmorrhage, these seem to me to be the most important of those which are down for discussion to-night. I entirely agree with Sir Dyce Duckworth with regard to the infrequency of death from hæmorrhage in gastric ulcer. Everyone who has had much experience of gastric ulcer will agree with that. At the same time I am bound to say that, when I am called, as I not infrequently am, to operate on cases where hæmorrhage has occurred, I am met frequently with the statement of the doctor, "The last case I had died." The last medical man said, "I have had two cases this year which died of hæmorrhage." Therefore, I am inclined to think that what one might call the academic opinion with regard to death from gastric ulcer is not an absolutely correct one, because many of these cases of hæmorrhage are of such an intense character that

the patients die before they reach hospital. What I want to endeavour myself to find out from this discussion is exactly the kind of case in which operation is indicated. For myself, I have formulated a rough-and-ready rule that, if a patient is bleeding in increasing quantity at decreasing intervals to such an extent as to threaten life in the opinion of those who are called upon to decide the issues, surgical treatment is called for. But if one adopts that the results are not entirely satisfactory. In my experience I have had to operate thirty-three times, and I have operated with six deaths, but I am bound to say that, on looking back on these cases, I think they would legitimately come into the category which Mr. Mansell Moullin described, of cases where death from hæmorrhage would be 90 per cent.; therefore I am accustomed to look upon thirty-three cases with six deaths as rather thirty-three cases with twenty-seven recoveries.

With regard to the grouping of these cases, Mr. Mayo Robson cavilled, in a good-natured way, at the grouping, and he quoted Dr. Mayo, of Rochester—an intimate personal friend of my own—as having rather a different classification. I am very glad to be able to tell him that in the last two papers Dr. Mayo has written he has adopted the classification which I have adopted in this paper, and it seems to me that such a subdivision is absolutely necessary. One cannot possibly put side by side a patient who has bled so that the hæmoglobin is only 18 per cent. and who has almost bled to death, with the patient who has merely a dilated stomach due to pyloric obstruction, and say, "If you do a gastro-enterostomy upon the first and upon the second also, these two are to be classified, not from the disease which necessitated the performance of an operation, but are to be classified according to the operation which is performed upon them." That seems to me to be an untenable position, and I am glad to see that Dr. Mayo, in his last two papers, has adopted a classification which I believe I was the first to suggest in connection with this subject.

With regard to the third division, into chronic gastric ulcer, I agree entirely with, in fact, I am entitled to say that I took my stand in this matter from, Mr. Mayo Robson, to whom I owe whatever enthusiasm I possess on the surgery of gastric diseases. And I feel that the operation of choice, in the vast majority of cases, is gastro-enterostomy. Pyloroplasty, as far as my work is concerned, is a thing of the past. Finney's operation I have done twice, but the results were not so satisfactory as I think they would have been if gastro-enterostomy had been performed. Therefore, I think that gastro-enterostomy is the thing by which we have to stand. But there is one thing which I do in addition to gastro-enterostomy to which Mr. Mayo Robson has not referred. In chronic ulcer and

in bleeding ulcer the direct method of treatment by excision of the ulcer is certainly not necessary, for exactly the same result can be achieved, and at very much less cost of time and trouble, by infolding the ulcer. Every ulcer which bleeds ought to be infolded; there is no need to excise it. Infold it as if perforated. It produces precisely the same result, as can be verified by post mortem, and it produces it at less sacrifice to the patient. I feel, with regard to the treatment of chronic gastric ulcer, that gastro-enterostomy is the operation of choice. One is accustomed to feel that it is not now a question as to what method of gastro-enterostomy shall be done; that has been settled. Posterior gastro-enterostomy without a loop is emphatically the operation of choice. What is the subject for discussion and discovery now is the kind of case upon which gastro-enterostomy ought to be done. And with increasing knowledge, side by side with careful analyses and careful examination of the patient, we have constructed the pathology which we possess, which enables an accurate diagnosis to be made in the majority of cases of duodenal ulcer, which used to be a matter of great difficulty. That which was said to be an extreme rarity is now found to be an extremely common disease. I have operated upon 100 up to to-day. One can diagnose duodenal ulcer in nine out of ten cases without examination of the patient at all. The history given by the patient is so characteristic that it might have been learned off by him from the book. Therefore, what one wants to know in regard to treatment of chronic gastric ulcer in the future is, not what technique is to be adopted in the treatment, but rather, what is the kind of case for which surgical treatment is absolutely necessary.

Again, I side with Mr. Mayo Robson and against Dr. Hale White, with regard to the frequency of the implantation of malignant disease upon chronic ulcer. In a paper I read before the Clinical Society last winter, I gave an analysis of my cases of malignant disease of the stomach, and in 60 per cent. of my cases there was a positive and unmistakable history of previous gastric ulcer. And it is curious to see that in all surgical cliniques where gastric work is done to any extent, the same result has been arrived it. In two out of three cases of carcinoma of the stomach, a clear history of chronic gastric ulcer is given.

With regard to hour-glass stomach, I have nothing to add to what Mr. Mayo Robson has said: it is one of the latest manifestations of merely gastric ulcer, and it ought never to be attained. I am content with submitting my results in twenty-seven cases of gastric ulcer, and I thank you for the opportunity of speaking.

[*Tuesday, November 27th*, 1906.]

Dr. NORMAN MOORE.

Sir, I have to congratulate the Society on this discussion, so full and so very useful, and, in view of the recent Hunterian Lectures at the College of Surgeons, which we have all read with great interest—I am sure I have—extremely opportune. Of course, the sole question with regard to ulcer of the stomach, non-malignant ulceration of the stomach, whether it is considered by a physician or by a surgeon—and I only say this because of one or two observations which were made at the last meeting—is, Is this the best method of treatment? I entirely repudiate the idea that a physician is in any way anxious to avoid treatment by operation. He is neither anxious to avoid it nor to seek it: the sole question is, Is it the best proceeding? And I think that there is a certain misfortune in considering the subject too much from the point of view of operation, that rather a better way of considering it would be to try and arrive at a conclusion as to the different species of ulcer. Such a distinction must precede the determination of treatment. When you look at the cases of ulcer of the stomach which are seen in the medical wards of a general hospital there are very striking differences between the cases that occur at one period of life and those which occur at another. I am speaking, of course, exclusively of cases of non-malignant ulcer. I took the cases at St. Bartholomew's Hospital during the ten years immediately preceding and including the year 1905. In those years, of patients under the age of thirty—I am speaking only of the medical wards—323 cases of ulcer of the stomach were admitted, and there were 6 deaths, less than 2 per cent. Over the age of thirty there were 188 patients admitted, and there were 23 deaths, nearly six times as many deaths in proportion as in the younger people. Then when you look at the question of sex there is again a very striking point about the statistics. Of the 323 patients under thirty 308 were women; of the 188 patients over thirty 117 were women. So that, although the proportion of men is much larger in the cases of gastric ulcer in what I might call the second half of life, the proportion of women is enormously larger than that of men in either group. Surely that is a condition for which there must be some definite pathological explanation. And we might go on from these statistics. I should myself be inclined to classify the cases—I am only speaking of my own limited experience of the cases I have had the opportunity of seeing, either in St. Bartholomew's Hospital or outside—into the following groups: First of all, there is one form of certainly non-malignant ulceration of the stomach, the clinical aspect of which we are all familiar with, occurring in a pale—I do not say anæmic, because the precise characters of the blood in these cases still remains to be investigated—in a pale young

woman. That kind of ulceration runs a certain definite course, has certain possibilities of treatment, and so on, and is easily recognised as a clinical entity. Then there is a second group, of which, of course, no one has seen many cases, but of which there are a few to be observed, and that is the ulceration of the stomach which follows the taking of a corrosive poison. That is a form of ulceration of the stomach which deserves special consideration and, in my opinion, needs to be treated in a particular way. The third group is an excessively rare one. I cannot say that I have seen more than three perfectly certain cases of it post mortem, and that is, tuberculous ulceration of the stomach. And a fourth group includes most of those, perhaps all, occurring in the second half of life, and is associated with vascular degeneration, often with very widespread vascular degeneration. I do not say that those are the only four species of this genus. I daresay there are a great many more, but those are four which I think most people must feel that they can distinctly recognise as clinical entities. The tuberculous one is so exceptional that it is perhaps only worth mentioning on an occasion like this when we are trying to look at every aspect of the subject. Now, I want to turn to the morbid anatomy before considering the question of treatment. I gather from the writings of gentlemen who have performed numbers of operations on these cases, in some of which ulcer has been found, in others of which ulcer has been suspected but has not been found, that they very often come across adhesions in the region of the pylorus. Now, my experience of post mortems suggests to me that such adhesions are rather rare. In considering the peritoneal cavity, if you exclude adhesions due to tuberculosis, adhesions due to new growth, and adhesions due to general peritonitis, whether tuberculous or acute; if you exclude all those, adhesion of any part of the gastric wall is, in my opinion, rather a rare occurrence. For instance, it is a great deal less common than adhesion of the spleen; that is often to be found. Nothing has been known about it during life, and in making the post mortem some adhesions in the neighbourhood of the spleen are found. Now, as far as my experience goes, such adhesions of the gastric wall, with the wide exceptions I have mentioned, are uncommon. Out of 2038 post mortems made by myself, in only two did I find adhesions to the gastric wall, excluding those of tuberculosis, new growth, general peritonitis, in which ulcer of the stomach or duodenum was not present. Of those two, one of them was an adhesion to a mass of calcified and, I have no doubt, tuberculous glands—because there was tuberculosis elsewhere in the body, though not elsewhere in the abdomen—and the other was associated with multiple thrombosis, including the whole of the vena cava. In seventeen examples, again from post mortems made by myself, in which there were adhesions of

stomach or duodenum, all were adhesions near the site of gastric or duodenal ulcers except the two just mentioned. The adhesions in these seventeen cases seem worth noting, whether they were associated with gastric ulcer or not; I mention them in relation to what my own observations seem to show, that adhesions to the gastric wall are rather uncommon, except in the presence of ulcer. In one case there was adhesion to the pancreas, in the second to the pancreas, in the third to the colon, the fourth to the liver and pancreas, the fifth to the pancreas, the sixth and seventh to the liver, two more to the pancreas, one (a duodenal ulcer) to the pancreas and to part of the peritoneum outside the pancreas; the others all to the pancreas except one, in which the adhesion was to a piece of omentum. There is one other point, also in relation to the morbid anatomy, that I should like to allude to. It is with regard to hæmorrhage. When you find blood in the stomach post mortem, what is the commonest explanation? The commonest explanation of blood in the stomach post mortem is that there has been a hæmorrhage from the lung. I am surprised when I look carefully through the notes—I only looked at the notes of post mortems made by myself—to find how much commoner a cause of blood in the stomach post-mortem the lung was than any other. I think it is quite worth while bearing that in mind, as notwithstanding the skill with which the examination of the lungs is invariably practised at present, some cases of supposed gastric hæmorrhage may not improbably be cases in which the lung is the true origin of the blood. It is, I say, worth bearing that post-mortem fact in mind in relation to diagnosis. Then, next to hæmorrhage from the lung, the commonest cause of blood in the stomach post mortem is cirrhosis of the liver. And the third cause is gastric ulcer. I am aware, of course, of the series of cases which Dr. Hale White has mentioned and has written about, but other causes than cirrhosis and gastric ulcer are, so far as my experience goes, rarely demonstrable. I am not speaking now of hæmorrhage into the mucous membrane merely—that, of course, occurs in a great variety of other cases—but of the actual presence of blood in the cavity of the stomach. Those are the three commonest conditions under which it is found post mortem in my partial experience. Now, returning to the groups of gastric ulcer which I have mentioned. To begin with, let us take the condition in the pale young woman. My opinion is that in those cases operation may be considered as an extremely exceptional mode of treatment; they generally get quite well if fed by the rectum, and if asked, how long, I should say I always feed such cases ten days by the rectum. I will not say I have never observed any bad effect from that. I think there are a few cases which do not bear it, but they are extremely rare. In the majority of such cases, in which you are quite certain from the

symptoms that there is a gastric ulcer, the patient recovers after being fed for ten days by the rectum. Something was said, I think by Mr. Mayo Robson in his remarks, which contained such a large number of valuable facts that it is not easy to recall them all, about the great importance of care in relation to diet in such cases, and that perhaps sufficient care was not given to it. Of course it is right that there should be great care; but I think there is another part of the system which it is important to pay attention to, and that is the circulation. These are nearly always people whose hearts are acting very imperfectly, who have those loud murmurs, whatever their cause, known as hæmic murmurs. And in my opinion, after all danger of further acute symptoms from the ulceration has disappeared, those patients ought for many weeks, and perhaps months, to be given as much rest as possible, and their hearts carefully regulated by such drugs as strophanthus, digitalis, and strychnia. And I think I have observed that the careful observation of the circulation in those cases is of at least equal importance with the observation of their digestion. The observation of their digestion includes a point which is often overlooked, and that is ascertaining that the patient's teeth are in such a condition as to enable her to properly masticate her food. The omission of that last consideration, I think, is more often the cause of the recurrence of gastric lesion in these people than any particular arrangement about the variety of food which they take. I consider both are important, but unless the teeth are attended to the effect of any other treatment may be nullified. And there is, of course, one class of case in which there can be no doubt whatever that operation ought to be immediately performed. It is one of the enormous benefits which surgery has conferred upon our generation that it can, if operation is performed sufficiently soon, save the life of a patient in whom gastric ulcer has perforated. And I think it is our duty, and everyone's duty, to urge upon the whole profession the importance of making up the mind in each case as to whether perforation has taken place or not. I am not going to intrude any remarks on so trite a subject to this Society. I agree with what Mr. Moynihan said, that with sufficient care one need be very rarely mistaken about the occurrence or not of perforation. As regards ulcers from poisoning, they last so long—the cases which I can remember lasted very nearly a year or over a year, and after that time still did not heal, but led to great wasting and to the patient's death—I cannot help believing that in those cases, and certainly in all such cases, gastro-jejunostomy is indicated, and that most of them could be so saved. There are generally several ulcers in such cases. With regard to the group of cases occurring in the second half of life, I feel that in those, if the case is treated early enough, before the ulcer has lasted too long, operation might be

avoided in a great many cases. In these cases, also, attention to the state of the teeth as well as to the diet must be given. Where the ulcer is probably very large, or where no improvement is perceptible, I think seizing the proper week or proper period for the operation becomes almost the sole point in the treatment. I cannot agree with those who think that existing evidence proves that prolonged gastric irritation is of itself a cause of malignant disease of the stomach, but I have seen two cases in which malignant disease of the stomach occurring early in life gave rise to symptoms closely resembling those of simple ulcer.

Mr. Herbert J. Paterson.

Sir, I must first of all thank Dr. Norman Moore for his kindly reference to my Hunterian Lectures, and must confess that it has been a source of considerable satisfaction to me to find that some at least of the opinions which I expressed in those lectures have been corroborated by one who has had such an unique experience of gastric surgery as Mr. Mayo Robson. I was particularly interested in what he had to say on the subject of peptic jejunal ulcer, for in my lectures[1] I suggested that peptic jejunal ulcer is due to hyperchlorhydria, the result of insufficient size or inefficient working of the anastomotic opening. I am glad to see that Mr. Mayo Robson now adopts that view. If it be correct—and I think there is good evidence to support it—it is of considerable importance, because it follows that peptic jejunal ulcer is a preventible complication, and may be avoided in the future by making the anastomotic opening of sufficient size. If that be so, then the complication of peptic jejunal ulcer follows the *circulus vitiosus* into that category of complications which need no longer disturb the slumbers of the surgeon who performs gastro-jejunostomy. With regard to the distinction between acute and chronic ulcers, the clinical difference is of such importance that I think we ought to remember that this distinction was first pointed out by Dr. Soltau Fenwick. It is rare indeed that an acute ulcer, apart from its complications of perforation or hæmorrhage, calls for surgical interference. But with chronic gastric ulcer the case is very different. Some time back I made an investigation into the after-history of a series of 147 patients who were in the wards of the London Temperance Hospital suffering from gastric ulcer, and the results are given in the pamphlet which has been handed round. I may mention that although nineteen of these patients (out of seventy-two who were traced) are classified as cured, the use of the term is only relative, and not absolute, for several of them had suffered from gastric symptoms between the time they left the hospital and the time at which the inquiry

[1] 'Gastric Surgery,' p. 20.

was made. Indeed, only seven of the nineteen patients classified as cured had remained absolutely free from gastric symptoms after they left the hospital. So one was led to conclude, from a study of those cases, that probably not more than 25 per cent. of the subjects of gastric ulcer, so far, at any rate, as hospital work is concerned, are really cured. From a consideration of those and other cases it is quite clear that the time usually allowed for the treatment of chronic or acute gastric ulcer is not nearly sufficient; and I formulated and gave in my lectures the following indications for surgical treatment: If after six weeks' complete rest on a milk diet, a further period of six weeks on a milk diet with comparative rest, followed by three months' careful dieting, the patient is not free from definite symptoms, or if after apparent cure the patient has a relapse, operation is probably in the best interests of the patient.[1] The question has been raised as to excising the ulcer-bearing area, to prevent the future development of carcinoma. I think that perhaps this is carrying anticipatory surgery too far. Are we justified in regarding a clinical history as proper and scientific evidence of the existence of a previous gastric ulcer? Even granting that gastric ulcer predisposes to cancer, we must remember that we have considerable clinical evidence that a gastric ulcer will heal after the performance of gastro-jejunostomy. Have we any evidence that a cancer will develop in a soundly healed scar? And if so, with what frequency? I think that before we submit our patients to the added risks of excision we require further proof on this subject than has yet been given. As regards the subject of gastric hæmorrhage, I think one important point which requires discussion is whether we are justified in operating during the progress of gastric hæmorrhage. Operation during hæmorrhage, as far as I can make out, from an examination of all the cases I could find, as well as from hospital statistics, is attended with a mortality of at least 66 or 70 per cent., while probably of all gastric ulcers which bleed the mortality from hæmorrhage is not greater than about 5 per cent. It appears therefore that of those patients who have gastric ulcers which cause recognisable hæmorrhage one in twenty dies, whereas of those submitted to operation two out of every three die. In other words, if a patient is operated upon for gastric hæmorrhage during the attack, the chances are three to one that the result will be fatal; whereas if nothing is done, the chances are twenty to one in favour of his recovery. And even as regards those cases which are operated on, I think we are entitled to ask, in the words of Sir James Paget, "What would have happened if this operation had not been done?" For it must have been within the experience of all of us to have seen cases operated on in which recovery ensued, not because but in spite of operation.

[1] The 'Lancet,' vol. i, 1906, p. 500.

I think, therefore, from a consideration of the facts as we have them at present, that it is open to grave doubt whether it is ever expedient to operate during the progress of gastric hæmorrhage. There is one distinction as to treatment in regard to acute and chronic hæmorrhage upon which stress has not already been laid. As is well known, hæmorrhage from an acute ulcer tends to cease spontaneously, and not to recur. Occasionally, however, hæmorrhage from an acute gastric ulcer does recur. If, in spite of absolute rest, the hæmorrhage starts anew, it is clear that Nature has failed, and if the second hæmorrhage has been a severe one surgery must step in. But—and this is the point I wish to emphasise—in such cases we must be quite sure that *absolute* rest has been tried and has failed. I lay stress on this because in several of the cases I have seen, and in several recorded cases, the same seems to have been true: the second attack of hæmorrhage from an acute ulcer has been apparently reactionary or recurrent, due to the mechanical displacement of the clot owing to the too early stimulation or injudicious movement of the patient. In these circumstances we may fairly give Nature one more chance before deciding on operation. In hæmorrhage from chronic gastric ulcer, on the other hand, it is not, as a rule, mechanical displacement but septic disintegration that starts the hæmorrhage afresh, and so operation is necessary in most cases in order to prevent the possible serious effect of another large loss of blood. Although, as Mr. Mayo Robson has said, we cannot lay down hard and fast rules for the treatment of these gastric hæmorrhage cases, yet we must each of us have some general working rules, so I venture to give to you those which I have drawn up for my own guidance. And I give them, not in any boastful spirit, but because I think, in a discussion such as this, that we shall do much good to each other if we give our individual opinions, so that we may compare them, and see in what points they agree and in what they differ. In hæmorrhage from acute ulcer give a thorough trial to absolute rest in bed, hot water injections by the rectum, and avoidance of food by the mouth. If a second profuse hæmorrhage occurs, perform gastro-jejunostomy, provided it is clear that this second hæmorrhage is not brought on by want of absolute rest, by too early stimulation, or by too early administration of food. In hæmorrhage from a chronic gastric ulcer perform gastro-jejunostomy after one severe hæmorrhage or after several slight attacks of hæmorrhage if the loss of blood is causing serious anæmia. And I think that the indication for operation which Mr. Moynihan gave us at the last meeting is very valuable—operate if the hæmorrhage is increasing in quantity and recurring at decreasing intervals. With regard to perforation, we shall all agree with the advisability of early operation. While, however, it may be true that every hour of delay adds to the danger, I am not sure

that this statement is borne out by statistics. Mr. Mayo Robson quoted statistics compiled by Keen and Tinker, in which the mortality of the cases operated on within twelve hours was only 28 per cent., whereas of the cases operated on in from twelve to twenty-four hours the mortality was 63 per cent. I do not think that these cases justify the conclusions which have been drawn from them. They are not consecutive, but isolated published cases, and therefore comprise an undue proportion of recoveries. In two series of consecutive cases which I collected and analysed for my lectures the mortality of those patients operated on within twelve hours was 47 per cent., and of those operated on in from twelve to twenty-four hours 53 per cent. So that in this series of consecutive cases there is a difference of only 6 per cent. between the cases operated on within twelve hours and those operated on within twenty-four hours, as compared with the 35 per cent. difference in Keen and Tinker's cases, drawn from isolated sources. The analysis of my series of collected cases indicates that other factors besides early operation may have an important bearing on the result, and of these probably the amount and nature of the extravasation, the presence of other ulcers, and the rapidity and completeness with which the operation is performed, are the most important. I need hardly say that I am not urging that early operation is inadvisable. My point is this, that if we expect the mortality to be diminished greatly, solely because we are operating upon our patients at an earlier period after perforation, we shall be disappointed. In 1898 Dr. Keen prophesied that the time was approaching when operations for perforation would be performed within twelve hours, and the mortality would be reduced to 10 per cent. Although operations are usually performed much earlier now than then, we have not yet reached that happy time, for in the year 1904 the mortality of the cases operated on in twelve of the London hospitals was still 48 per cent. Proclaiming the necessity for early operation will not command success; we must do more—deserve it. It is to improvements in technique that we must look if we are markedly to diminish the mortality which attends operation. And I believe that when we regard gastro-jejunostomy as part of the routine treatment of perforated gastric ulcer we shall attain much better results than we have done in the past. In my Hunterian lectures[1] I gave fully my reasons for adopting this procedure, and I shall not now do more than refer to one or two points. Previous writers have suggested that gastro-jejunostomy should be performed at the time of operation in order to improve the after-condition of the patients who recover. This argument is supported by an investigation which I made into the after-history of patients upon whom simple suture of a perforation had been performed, for I

[1] 'Gastric Surgery,' p. 82.

found that 23 per cent. of the patients relapsed within a year or two of operation, in one instance with a fatal result. A perusal of the very valuable and interesting collection of cases from St. Thomas's Hospital tabulated by Dr. Herbert P. Hawkins and Mr. A. R. Nitch reveals the striking fact that 23 per cent. of the patients sought relief at the hospital for gastric trouble after their operation. We may therefore safely assume that had an attempt been made to trace the other patients, the proportion of relapses would be much higher than 23 per cent. There appears, therefore, from the after-histories of patients operated on for perforation, some justification for the prophylactic performance of gastro-jejunostomy. But some more urgent reason is required. Our concern is the immediate saving of life: the ultimate result is of secondary consideration. My contention is, however, that the routine performance of gastro-jejunostomy would diminish the immediate mortality from operation, and the following are some of my reasons. In my series of 112 consecutive cases I found that 11 per cent. of the patients died shortly after operation either from perforation of a second ulcer, from hæmatemesis, or from leakage along the line of suture. And I find in the series with which Dr. Hawkins has furnished us that 12 per cent. died from similar causes. I think we may fairly regard these causes of death as preventable; and there is a strong presumption that had gastro-jejunostomy been performed in these cases the patients would have recovered. I do not claim a certainty, only a possibility. But I submit that a procedure which holds out even a possibility of reducing the mortality from perforation by 11 per cent. deserves a very thorough and extended trial at the hands of surgeons. But there is, I think, an even more weighty reason than any I have already advanced. There can be little doubt that the most effective means of draining the peritoneal cavity is through the intestines and through the intestinal lymphatics. So in order to encourage that drainage our aim should be to establish peristaltic movements as soon as possible. After gastro-jejunostomy purgatives can be given at once with absolute safety, and from my own experience I am convinced that they act much more certainly and more quickly after a gastro-jejunostomy has been performed. Mr. Mayo Robson, I see, goes so far as to say that gastro-jejunostomy should be performed when the patient's condition permits of it. I would go further and say, the worse the patient the more necessary it is to perform gastro-jejunostomy. Most of the patients who die after suture of a perforated gastric ulcer die, not from shock, but from peritonitis or chest complications, and these are best combated by establishing early drainage of the peritoneal cavity through the intestines, by the early administration of purgatives. The chief objection to gastro-jejunostomy is of course, that it prolongs the operation. I think Mr. Mayo

Robson will agree with me that the performance of the short curcuit need not take more than six or eight minutes. And I think it will be conceded—I am convinced of it from the experience I had as anæsthetist for two years at St. Bartholomew's Hospital—that a patient who is too ill to bear prolongation of the operation for six or eight minutes has, under any circumstances, little chance of recovery. But I would go further and say that gastro-jejunostomy need not prolong the operation at all. Most authorities are agreed that a thorough search should be made for a second perforation. If gastro-jejunostomy has been performed, I think a second perforation may be left to take care of itself. Gastro-jejunostomy is probably of advantage in every case, while search for a second perforation will, in four cases out of five, prolong the operation to no useful purpose. If, then, instead of spending time in searching for a second perforation, the time so saved be utilised for effecting the short circuit, the duration of the operation need not be extended at all. Again, the more we trust to intestinal drainage, the less attention we need pay to the toilet of the peritoneum; and so the time saved from mopping and irrigating the peritoneum may be put against the time which is consumed in performing gastro-jejunostomy. In my last five cases I have performed gastro-jejunostomy, and dispensed altogether both with irrigating and mopping out the peritoneum, and four of those five patients recovered. I quite admit, sir, this is too small a number from which to draw conclusions, but I submit that they warrant a more extended trial of this method at the hands of other surgeons. There are just a few more words I have to say, sir, if you will allow me, as regards the results of gastro-jejunostomy. The mortality from gastro-jejunostomy is now so low in skilled hands that the exact percentage is mainly a matter of academic interest. What we want now is accumulated evidence as to the remote condition of these patients. I have attempted to gain some information on this subject by collecting a number of cases, to which I need not refer, as they are given in the table which has been handed round. I might just point out that in about 82 per cent. of the patients relief has been complete, and in about 10 per cent. relief has been almost complete. There can be little doubt that at the present time gastro-jejunostomy is in danger of becoming too fashionable. In an admirable address on "Self Restraint in Surgery," which Dr. Basil Hall published in the 'British Medical Journal,' [1] two weeks ago, he tells us of a physician who said that patients could be divided into two classes, those who were willing to have gastro-jejunostomy performed and those who were not, and he asks the pertinent question with regard to the published statistics of the past four years, "Is it reasonable to suppose that all those operations were

[1] Basil Hall, 'Brit. Med. Journ.,' vol. ii, 1906, p. 1355.

really necessary?" and he instances four cases in which gastro-jejunostomy was performed, one a case of uræmic vomiting, another a case of tabes with gastric crises, another a case of carcinoma of the colon, and another a case of phthisis with troublesome vomiting. Dr. Hall asks, "Can anyone doubt that such catastrophes as these are the result of hasty and indiscriminate resort to operation, occurring as they did in the practice of able and experienced surgeons?" May we not hope, sir, that this discussion, by defining more clearly the uses and limitations of gastro-jejunostomy, will stem this gastro-jejunostomic tide, and so prevent the occurrence of such surgical calamities as these? Dr. Norman Moore's remarks as to the necessity for investigating the condition of the teeth in cases of gastric trouble are very much to the point. Some years back I saw a patient for whom gastro-jejunostomy had been advised, who was really suffering from indigestion the result of insufficient masticatory power due to defective and carious teeth. An artificial denture was prescribed; this was duly obtained, and nothing more has been heard of the gastric ulcer from which she was supposed to be suffering. Judging by the cases which we see from time to time, I think we cannot repeat too often that gastro-jejunostomy is not a panacea for all gastric ills, and that its performance in vague disorders of the stomach, unaccompanied by definite clinical or chemical signs, cannot but bring discredit on a valuable and, in suitable cases, a legitimate and brilliantly successful operation.

Dr. H. P. Hawkins.

The total mortality of 556 cases of gastric ulcer (including its consequences) at St. Thomas's Hospital is 13·3 per cent. The question is not only how to reduce this mortality rate, but also how to shorten the long periods of continuous or intermittent illness and disability, which often amount to several years. A consideration of this series of cases shows—(1) that the acute recently-formed ulcer is seldom a cause of death; (2) that nearly the whole of the mortality and certainly the whole of the long invalidism occur in connection with the chronic thick-walled ulcer. Any improvement in treatment must be directed more particularly to the prevention of the chronic ulcer.

From this series of cases the following idea can be gained of the average history of patients suffering from gastric ulcer. If 100 patients are considered, they are found to come under treatment for the first time in one of two forms: (*a*) a small proportion, between 2 and 3 per cent., are admitted suffering from general peritonitis due to perforation of an ulcer with little or no history of preceding gastric symptoms. They go direct to the surgeon; (*b*) the remainder, about 97 per cent., having a

history of gastric symptoms for a few weeks or months, pass through the routine course of medical treatment. Of these a very small proportion die, certainly not more than 2 per cent., and generally less.

The remaining 95 patients are discharged after their course of medical treatment. They are generally still anæmic, but they are able to take fish and light food without pain or sickness. They have not, however, been tested as regards their capacity for ordinary life and work on a full diet. Of these I think it may be reckoned with certainty—(1) that not more than two-thirds remain well; (2) that about 10 of them are re-admitted with a recurrence of symptoms after a few months or a year (incomplete healing or development of a fresh ulcer); (3) that about 15 of them come under treatment subsequently, on account of perforation of a chronic ulcer (generally by leakage from an adherent ulcer) or on account of perigastric adhesions, pyloric stenosis, or hour-glass constriction. It is in this group (roughly 10 per cent. relapsing ulcer and 15 per cent. perforating ulcer and various sequelæ) that the bulk of the deaths from gastric ulcer occurs.

Prevention of chronic ulcer.—The question is how to lessen the numbers in this group. As regards the general plan of the medical treatment which they undergo in the first instance, I do not think that any improvement can be made. The essentials of the medical treatment are much the same all over the world, though details vary. I believe the milk, egg, and sugar plan of Lenhartz has much in its favour. I agree with Dr. Hale White as to the disadvantage of unduly prolonging a period of rectal feeding. Such feeding is in many cases necessary at the onset of symptoms, but it is obviously not the diet on which we can expect rapid healing of an ulcer to take place, and it should be discontinued as soon as possible. If the plan of medical treatment is good, its duration is quite insufficient. There are difficulties, however, in its extension when we are dealing with patients who have to work for a living, but there is a fair probability that the numbers of patients who enter into the chronic-ulcer-group might be reduced if the duration of medical treatment and dieting could be extended to a minimum of six months.

As regards surgical treatment, as a measure aimed at preventing the chronic ulcer, gastro-enterostomy is to be recommended. The question of gastro-enterostomy, as a routine matter in all cases which either do not make a good recovery after medical treatment or, having made a good recovery, quickly relapse, turns entirely on the mortality-rate which is to be expected from the operation itself, and on the efficiency of the anastomotic opening. There are thus two sides to the question. In the first place we have the balancing of the risks to life. It

is among these chronic-ulcer cases (roughly, 25 in every 100 patients) that the chief mortality of gastric ulcer arises. I think it may be accepted that at least 9 of the 25 ultimately die from perforation or from sequelæ, and the remainder are crippled for some years. I believe that, though the mortality-rate from the operation of gastro-enterostomy itself must be reckoned with, it is so low (and is likely to become lower) that on the score of risk to life the balance is greatly in favour of the operation. Our figures at St. Thomas's Hospital are too small to bear a conclusion. In 47 instances of gastro-enterostomy there were 11 deaths. Of these, I think, 8 must be credited to disease, and 3 to the operation. But these cases are not wholly to the point, for they were mostly instances of cicatricial effects of chronic ulcer, in other words instances of the very conditions which the operation as here recommended is intended to prevent, and it must be remembered that in the cases under consideration the operation would be undertaken, as it were, in a quiescent interval, as a preventive and not a curative measure, under the best conditions, and at a selected time.

In the second place there is the question how far gastro-enterostomy may be considered as truly preventive of subsequent trouble, and as a really scientific method of treatment. In this respect I think the figures from St. Thomas's Hospital are satisfactory, the more so, in fact, because, as is stated above, they are not strictly applicable, being designed mainly to relieve sequelæ rather than to forestall them. Of 33 cases of gastro-enterostomy I think 17 may be described as giving a good result after one to four years, 5 derived slight benefit, 2 no benefit, and 9 were discharged apparently well, but the operation was too recent to justify a verdict. Certainly, in two points, the comparative rest to the stomach afforded by its early emptying, and the shortening of the period of high acidity, the operation has everything to commend it, and I believe that this advantage comes equally into play, both as regards the speedy healing of a chronic ulcer, and as regards the prevention of fresh ulceration. The occurrence of pneumonia after the operation and the development of peptic ulcer in the jejunum receive illustration in our series of cases.

Hæmatemesis.—As regards the question of hæmorrhage, I should prefer to depend on medical treatment during the actual bleeding, being sceptical as to the utility of gastrotomy and a search for the bleeding point, and being influenced by the fact which is shown in two of our cases, and in another case within my knowledge, that there is not necessarily any bleeding point which can be successfully dealt with. The mortality from hæmorrhage is very low in our series of cases, less than 1 per cent. of all cases. But I think that genuine hæmorrhage, with

a preceding history indicating an ulcer of long standing, necessitates gastro-enterostomy in a quiescent interval.

Perforation.—Perforation with general peritonitis must occur in a certain number. It may he actually the first sign of the formation of an ulcer, and I do not doubt that in these cases the ulcer is actually only of a few hours' standing. But in the majority of cases perforation occurs in connection with a chronic and generally an adherent ulcer, a class of case which one may hope that an earlier preventive gastro-enterostomy will make of less frequent occurrence. The mortality from perforation can be kept down to a low level by prompt operation. In our series the average time elapsing between perforation and operation was 25 hours in fatal cases and 16 hours in those that recovered. In one successful case 60 hours elapsed between perforation and operation. The mortality from this cause in our series is 8·5 per cent. of all cases, and it accounts for more than half the deaths. Of 67 cases of general peritonitis from perforation submitted to operation the mortality is at the rate of 55 per cent., but in the last five years this has been reduced to 43 per cent. (23 deaths, 26 recoveries).

Perforation with local and often concealed peritoneal infection is even more disastrous than a general peritonitis, owing to the delay which commonly occurs before such cases come under surgical treatment. Of our 15 cases 11 died, the mortality being at the rate of 73·3 per cent. In this condition we meet with subphrenic abscess, perforation of diaphragm, empyema, gangrene of lung, pylephlebitis, hepatic abscess, and septicæmia. It may be hoped that the number of cases in this group will be reduced by a readier and earlier recourse to gastro-enterostomy.

Sequelæ.—The late cicatricial effects of gastric ulcer, adhesions, pyloric stenosis, and constriction, can only be dealt with surgically as soon as they are recognised. The diagnosis of adhesions is exceedingly difficult. One sees patients who have heard the word adhesions vaguely applied to their state, and who thereupon clamour for operation. Many of them not only have no adhesions, but have never had an ulcer.

The whole subject of the accurate recognition of this class, and the appreciation of the amount of pain which can be experienced by the owner of a hyperæsthetic though otherwise healthy stomach, is one of the great difficulties of the borderland between medicine and surgery. Finally, it should be mentioned that, during the period of ten years from which our series of cases has been drawn, there were admitted, one woman with tabes and gastric crises having an incision scar above the umbilicus contracted under other hands, and two men with tabetic crises, both labelled gastric ulcer, one of them always having slight hæmatemesis at the height of his vomiting.

Dr. S. H. Habershon.

The question of the indications for surgical interference in cases of simple gastric ulcer is one of the utmost importance for the physician. Dr. Hawkins has stated that the medical treatment adopted by the physician has reached finality. Some years ago I wrote an article in the 'St. Bartholomew's Hospital Reports' on the prognosis of gastric ulcer, and in my conclusion I found that errors in diet were the main causes disposing to a recurrence. I believe that solid meat diet should not be given for at least a year after the occurrence of the original symptoms, and that one reason for previous return of symptoms is that solid food and butchers' meat is given at too early a period. In acute cases, except in the case of perforation, it is very rare for any question of operation to arise.

It is in the *chronic cases* that urgent symptoms occur which cannot be relieved by ordinary medical treatment, and in which the advice and skill of the surgeon are called for.

The symptoms which are severe enough to call for one of the various surgical procedures may be classed under five heads.

1. *Excessive vomiting and pain*, due to the inclusion of a branch of the pneumogastric nerve in the cicatrix of a gastric ulcer.

Several cases have been recorded by my father in his work on 'Diseases of the Abdomen' (1888), in which this was the cause either of uncontrollable vomiting or of intense suffering and pain.

One word must be said as to a difficulty of diagnosis that sometimes occurs. The condition may be simulated in hysterical young women when the incessant vomiting is a neurosis, and the pain is described in such exaggerated terms as to be entirely misleading to the practitioner.

In such cases the general appearance and nutrition of the patient or the careful arrangement of the hair in preparation for the doctor's visit may lead to the suspicion that the vomiting is really under the patient's control and the pain fictitious. With regard to pain, I am accustomed to make use of a physical means of diagnosis which is not generally known, and very rarely adopted. If in such cases pressure is applied to the epigastrium with one hand great tenderness is complained of, and the patient may even cry out on account of the intense hyperæsthesia. Now, if with the fingers of the other hand firm pressure is applied to the carotid artery on either side of the neck (but, in my opinion, better on the left side), so as to almost obliterate the circulation, it will be found that the hyperæsthesia disappears instantly on epigastric pressure in all neurotic cases. I have satisfied myself that it is not necessary

to press upon the pneumogastric nerve, but that carotid pressure is sufficient.

2. *Recurrent hæmorrhage*, especially when a large vessel is the source of bleeding.

This rarely occurs except in chronic cases, when the fibrous nature of the scar prevents closure of the ulcerated vessel.

3. *Gastric dilatation, and symptoms* occurring from the interference with the muscular movements of the stomach by adhesions of the peritoneal surface of the scar to surrounding organs.

In this category may also be included those disastrous deformities of the stomach due to contraction of the cicatrix (the so-called hour-glass stomach) which (I agree with Mr. Mayo Robson in believing) ought never now to occur.

4. The *remote sequelæ* of the *perforation* of a chronic gastric ulcer with the formation of a localised or subdiaphragmatic abscess. The need for operation when the condition has once been diagnosed is too obvious to call for much discussion.

5. *Pyloric spasm.*

In another place I have described a group of symptoms that are associated with a simple chronic ulcer in the neighbourhood of the pyloric orifice. They are so definite and characteristic that, in a classification of symptoms based on the position of the ulcer, I should unhesitatingly divide them into two types:

a. The symptoms due to a pyloric ulcer, or to an ulcer on one side or other of the pylorus, but in its immediate neighbourhood.

b. The symptoms dependent on the occurrence of a gastric ulcer elsewhere.

The former type owes its peculiarity to the fact that pyloric spasm is set up.

In the early stages of an ulcer in this position (or, in other words, when the ulceration is recent) the well-known combination of pain immediately after the ingestion of food with vomiting causing relief to pain is occasionally a part of the history of the patient's malady, and with or without hæmatemesis.

When the ulcer has become chronic the patient is liable to recurring attacks of irritability without fresh ulceration necessarily taking place. The outcome is that every few weeks or months attacks of distressing symptoms recur.

Each attack may confine the patient to bed for some weeks, and may subside under appropriate treatment. In others the patient is seldom free from discomfort, though there are usually exacerbations. Emaciation from malnutrition is often extreme, and the life may be rendered burdensome by the constant suffering. Sooner or later the operation of gastro-enterostomy becomes a necessity.

The group of symptoms to which I refer has only obtained recognition in recent years.

They are as follows:

(*a*) Pain or a feeling of increasing weight or uneasiness or discomfort occurs from two to four hours after a meal, and is more common towards the close of the day.

(*b*) Pain is *always relieved by food,* and the patient often seeks relief by taking a cup of coffee or bread and milk, etc.

(*c*) Vomiting or retching may or may not be present, but occurs at a considerable period after the ingestion of food. Pyrosis with excessive hyperchlorhydria often replaces the vomiting. When stenosis or obstruction of the pylorus takes place from contraction or hypertrophy the symptoms simulate those of permanent pyloric obstruction, when quarts may be vomited at the close of the day, while only a few pints of fluid have been taken.

Hæmatemesis and coffee-ground vomit are not common.

(*d*) *A variable dilatation of stomach* is a special feature. During the earlier hours of the day the stomach may be of entirely normal size; a few hours after a meal the stomach becomes extremely dilated, with the presence of succussion splash, excessive flatulence, and the sensation of great weight and oppression at the epigastrium.

The results of a posterior gastro-enterostomy in such cases are so striking that I have no hesitation in saying that they afford a most brilliant picture of the results of modern surgical methods.

I always recommend the operation in cases presenting the symptoms I have narrated as soon as they are definitely established.

In a few cases immediate relief is afforded by operation, only to be followed a few weeks later by a recurrence of the above chain of events.

I wish to say a few words as to the cause of a return of vomiting and pain, and as to the remedy.

It will be remembered that when an ulcer occurs in the vicinity of the pylorus (either on its gastric or duodenal side) a spasm of the sphincter is set up at the time when food is intermittently expelled through the orifice into the intestine.

When a short circuit is established, and the food escapes by the new opening made, the pylorus is at rest, and the movements of the pyloric end of the stomach are probably less vigorous than normal. If by the character of the operation a loop of intestine is left between the pylorus and the new opening, it is obvious that stagnation of the contents is possible, and with a relaxed pylorus regurgitation into the stomach is a probable explanation of the recurrence of pain and vomiting.

In three cases brought to me last year, and in which I considered that a pyloric ulcer was present and recommended

operation, there was a recurrence of symptoms though a posterior gastro-jejunostomy was performed.

Mr. Bidwell suggested a remedy which in two cases relieved the patients, and the third only produced partial relief. Another short circuiting oporation was performed, by which the duodenum was connected with the nearest loop of intestine. In these cases the recurrent vomiting was of a bilious nature, but there is no evidence to prove that a regurgitation of bile or pancreatic juice into the stomach acts in any way as an irritant. The operation performed eliminated any question of stasis in the duodenum, and merely transferred the intestinal process of digestion to a portion of intestine a little further removed from the pylorus.

For a recurrence of symptoms I should always recommend this second operation of entero-duodenostomy.

In the last few years I have advised operation in some forty-three cases of pyloric ulceration, and in every case a posterior gastro-enterostomy was performed. Four cases proved to be malignant, and in each of these death occurred either immediately (in two cases from the shock of operation) or in a few weeks after operation. The remaining cases recovered with the exception of four cases of relapse, three of which I have referred to.

Mr. C. Mansell Moullin.

The results of the present method of treatment, so far as cases sufficiently severe to be admitted into hospital are concerned, are very far from satisfactory. Four years ago, at my suggestion, Dr. Bulstrode collected and analysed the statistics of all the cases of gastric ulcer admitted into the London Hospital from January, 1897, to August, 1902—a period before operations for gastric ulcer, except for perforation, were at all common. They were just 500 in number—402 women and 98 men. Of these, 48 (nearly 10 per cent.) died from perforation and peritonitis, 13 (2½ per cent.) from hæmatemesis, and 28 (5½ per cent.) from other causes. The total number of deaths was 89, or approximately 18 per cent. This is, of course, only the direct mortality, and does not include the deaths due to the more remote sequelæ. As all the cases of simple gastrostaxis were included in the number, and this disease has, so we are assured, a very low rate of mortality, it follows that the direct mortality of cases of true gastric ulcer, treated as it is at present, must be a great deal higher than even 18 per cent.

The proportion of cases cured is equally unsatisfactory, for by cure I mean real cure, not merely discharge from hospital with the most pressing symptoms relieved for a time. But the estimation of this is almost impossible. Dr. Bulstrode, however, did the next best thing for me. He found by careful

inquiry that, of the 500 cases, no less than 214 had suffered in the same way previously, and had been relieved by treatment, some of them many times. In other words, 42 per cent. had relapsed.

These figures do not include cases admitted for what may be termed the late complications of gastric ulcer. They do not take into account cases of pyloric stenosis, hour-glass contraction, perigastric adhesions, subphrenic abscess, or any of those other disorders which result from persistent ulceration. Nor do they take into account the effects of prolonged starvation and anæmia; nor cases in which carcinoma has developed at the site of an old ulcer. There is a growing tendency to believe that whatever may be the proportion of cases of gastric ulcer that end in carcinoma, carcinoma of the stomach nearly always develops at the site of an old ulcer.

The results therefore of applying the same method of treatment to all cases indiscriminately, so far as hospital cases are concerned, are, briefly, a mortality of at least 18 per cent. (perhaps much higher), 42 per cent. of relapses, a large number of grave complications setting in later, and serious risk that carcinoma may follow. What is required is a more accurate diagnosis so that cases can be selected and classified.

All recent and acute cases should have one thorough and prolonged course of medical treatment, unless there is either perforation, actual or threatened, or hæmorrhage endangering life. The former of these exceptions is admitted by all. The latter is not; but I maintain that if a patient is obviously dying from hæmorrhage, and has reached that stage when it is practically certain that a little further loss will prove fatal, it is our duty to try and arrest that hæmorrhage at all cost. I admit there may be merely general oozing and no ulceration or definite bleeding-point; but in every case in which I have opened the stomach the bleeding has been easily arrested, and I have seen two cases and know of a third in which the hæmorrhage was allowed to continue until it proved fatal. It is no argument that in several cases in which an operation has been performed there have been subsequent attacks of bleeding. The operation stops the hæmorrhage at the time, which is all it is asked to do. It does not cure the condition which gives rise to the hæmorrhage, and does not pretend to do so.

All recurrent cases, that is to say, cases in which the patient is perfectly well, with no suspicion of indigestion in the intervals between the attacks, should be treated in the same way.

All cases of chronic ulcer of the stomach in which there has been one thorough trial of ordinary measures without relief, or with only temporary relief, should have an exploratory operation performed. If the ulcer has not healed in one thorough prolonged trial, it is not likely to heal in a second. If it has

healed and left a scar which more or less disables the stomach by its contraction, nothing but operation can do any good.

The ideal operation is excision; but this is rarely practicable. Posterior gastro-enterostomy, with a lateral anastomosis lower down in the loop, is a highly successful operation, both so far as immediate and permanent results are concerned. The mortality is barely 2 per cent., and would be lower still if cases in which it is done as a last resource, after everything else has been tried and failed, are left upon one side, as they should be. What is known as a "vicious circle" never occurs now, and, though I cannot say the same of peptic ulcer, I have been so fortunate as never to meet with one. But the remedy is a purely mechanical one, and its success is proportionate to the mechanical nature of the patient's trouble. Where the vomiting, etc., are dependent upon other than mechanical causes it is of little or no use, and, though it gives some relief, it will not cure atonic dilatation, such, for instance, as is often associated with enteroptosis. What is wanted is an earlier diagnosis, and at present there is only one means at our disposal by which this can be effected—exploratory laparotomy. It is no use waiting for a diagnosis until the condition is incurable—which is what we are doing now.

[*Tuesday, December* 11*th*, 1906.]

Mr. Gilbert Barling (Birmingham).

Allow me, in the first instance, to thank you for inviting me to take part in this interesting discussion. I am glad to think that surgical intervention in the treatment of ulcer of the stomach and its complications occupies a more favourable position in the estimation of the medical profession than it did a very few years ago. This I attribute to two factors: they are the greatly diminished risk now attached to surgical procedures in this particular field, the result of increased experience, and the recognition that gastric ulcer is responsible for a greater mortality and for more grave illness than was formerly attributed to it. In his address delivered at the opening of the discussion Mr. Mayo Robson so thoroughly and so fairly traversed the whole field from the surgical point of view as to leave little further to be said, certainly very little in the way of criticism. However, before referring particularly to the material I wish to bring before the meeting from my own experience, I would like to say a word as to the wisdom or as to the necessity of short-circuiting from stomach to jejunum when operating for perforation of the former organ. Dr. Hale White also raises this point. I would suggest that the patients who die after operation for perforation rarely die from want of the anastomosis. They die because they are sent to the surgeon too late; they die of sepsis which has passed beyond the surgeon's control.

In the early hours succeeding perforation the work of Dr. Hewetson, and cultivations taken from my own cases, show how slight is the degree of infection, but every two or three hours which elapse before surgical intervention intensify rapidly the bacterial growth which finds such favourable conditions. The intense pain usually following perforation appears to me to be due simply to the scalding action of the acid stomach fluid which sets up a rapid secretion of fluid from the irritated peritoneal surface, and in this septic organisms, though few in number at first, develop most rapidly. Operation within from three to six hours after perforation would probably reduce the mortality to 10 per cent. or less. Unfortunately, at present we are doomed to operate in the majority of cases far later than this, and whilst some of them have local conditions which would benefit by short circuiting, most of them, as we see them now, would have their chance of recovery depreciated by an extra twenty minutes' exposure on the operating table with the prolonged anæsthesia, which latter appears to me especially baneful in septic peritonitis from whatever cause. Twice I have found it necessary to perform subsequent anastomosis in patients who have recovered from perforation, but had the anastomosis been made at the time of the perforation my belief is that the chance of recovery for these two patients would have been gravely compromised, as both were late cases when operated on for the emergency. The most successful results with which I am acquainted are those of Dr. Sinclair Kirk, of Belfast, who last year published a list of eleven successive cases operated upon for perforation of stomach or duodenum, all recovering. None of these patients were submitted to gastro-jejunostomy at the time, though three were subsequently, but the most striking fact in them is the short time which elapsed between perforation and operation; eight of them were operated upon in less than six hours, one in seven hours, one in ten hours, and the other in twenty hours. These figures confirm the point I most wish to emphasise, that the greatest factor in success is a brief interval between the calamity and surgical intervention. One other point in the management of perforation well worthy of attention is the necessity for absolute recumbency after the accident has occurred. I am persuaded that diffusion of extravasated material by the action of gravity plays a considerable part in the widespread sepsis which we not infrequently see. A word as to cancer of the stomach following upon simple ulcer. Whilst prepared to acknowledge that this does occur, I have seen the sequence but rarely, certainly in nothing like 30 to 40 per cent. of the patients with cancer of the stomach who have come under my observation, and I shall be interested to hear from other speakers whether this grave indictment of ulcer as a common provocation of cancer of the stomach is sustained by their observation. To illustrate the methods I personally adopt in the treatment of gastric ulcer

and its sequelæ I propose to bring before you, as being fresh in my memory, my work of the last twelve months, during which time, in hospital and in private practice, I have operated upon twenty-nine patients, excluding those with malignant disease and with congenital hypertrophic stenosis. The final condition of some of these it is too early to speak of, and I am not for the moment concerned with final results. I merely want to show here: (1) the safety with which these operations may be performed, (2) the conditions which appear to me to require operation, (3) the methods adopted, and (4) to mention one or two failures. (1) With regard to the safety of the operations it is satisfactory to state that all the patients recovered, and that in no case was there any trouble from regurgitation of bile into the stomach. One patient only gave me anxiety. In this case a band of omentum constricted the colon, causing symptoms of incomplete obstruction. The abdomen was reopened, the colon released, and a satisfactory recovery followed. (2) As to the conditions requiring operation; in three patients severe hour-glass constriction was found, which in two cases was relieved by gastroplasty. The third of these patients suffered from repeated hæmatemesis, and at the operation it was found that active ulceration and widespread adhesions were associated with the hour-glass contraction. Posterior operation was impossible, so an anterior anastomosis was performed with a second junction between the two limbs of the jejunal loop. Seventeen patients suffered from stenosis at or about the pylorus, in some of whom active ulceration co-existed. In all of these symptoms had existed for years, causing much misery and disability for work. The only matter for regret is that most of them were not submitted to surgical treatment much earlier, by which means only was permanent relief to be hoped for. Three patients were the subjects of active ulceration complicated by adhesions resulting from old perforation, which clearly had nearly cost the lives of all three. Six patients were the subjects of uncomplicated ulcer, which lesion was found to exist in every case. In these cases, again, there was a long history indicating that they were not likely to get material relief from further medical treatment. All these patients suffering from stenosis or active ulceration were submitted to posterior gastro-jejunostomy. Formerly I practised anterior gastro-jejunostomy, but have, I think, finally discarded it as a routine procedure on account of the feeling of anxiety I always had as to a vicious circle with its persistent vomiting, distress, and occasionally danger. I employ it now only when the anatomical conditions present render the posterior method unsuitable, and then I supplement the junction between stomach and jejunum by a lateral junction of the two limbs of the jejunum. The junction of stomach and intestine through an opening in the transverse mesocolon by two rows of suture, both viscera being controlled by clamps to prevent

leaking of contents, and, temporarily, to control bleeding, is a method, I believe, unsurpassed for obtaining a safe and effective union. The portion of jejunum selected for approximation should be as close to the duodeno-jejunal junction as can be managed. In these details I follow what I may speak of as the teaching of the Leeds school in gastric surgery, to which I should like to acknowledge my indebtedness. I have been in the habit of excising some of the redundant mucous membrane of the stomach and jejunum, but without any strong conviction that this is an additional safeguard to the patient. Apparatus such as button or bobbin I employ only when some exigency arises which seems to call for such assistance, as, for instance, when only such a small surface of the stomach can be exposed as gives insufficient room for safe suturing, but I always have a feeling then of regret and anxiety that I should be glad to dispense with. Whilst it is too early to speak of final results in these twenty-nine patients, from previous experience I have no doubt that the very large majority will be restored to health and capacity for work. One of the patients with hour-glass constriction still complains of discomfort at times, though she is relieved of her former severe pain and vomiting, and she is able to discharge her household duties, which she was formerly quite unable to do. One patient with pyloric stenosis, though she no longer gets the pain which formerly followed the ingestion of food, complains of pain in the region of the pylorus when she is up and about; the same area is quite tender on palpation, but I confess I cannot explain this partial failure where no active ulceration was discovered at the operation. The patient is the subject of renal mobility and is neurasthenic. One complete failure I have to acknowledge. A male, aged 30, with an evidently dilated stomach and extreme mobility of the right kidney, complained of pain in the stomach after meals, which was not constant but came on periodically and was followed by profuse vomiting. I advised and carried out fixation of the right kidney, thinking that the drag of this organ kinked and thus obstructed the pylorus, as in a case operated upon some time previously in which undoubtedly the kidney was responsible for the symptoms complained of. The kidney fixation in this man, however, failed in its object, and I referred the patient to my colleague, Dr. Saundby, who has paid much attention to gastric troubles and whose assistance I have often sought. He found that the stomach was a good deal dilated and that gastric stasis existed, as shown by a test meal. With Dr. Saundby's advice and concurrence I performed posterior gastro-jejunostomy in July last. The pylorus was narrowed but no ulceration was detected. A good recovery followed the operation but the periodic vomiting remains unrelieved. It may be suggested that the attacks were of the nature of gastric crises such as are seen in locomotor ataxy, but there was not the least

evidence of any disease of the central nervous system. Failures such as I have just described are exceptional and do not shake my opinion that the results attained after operation for gastric ulcer and its complications are generally brilliant. The relief of the patient's local trouble, however, does not alter his general tendencies, which remain what they were. If previous to operation he was the subject of gastric catarrh from exposure or from food which irritated his mucous surface, he is not given an immunity from these things by operation. The nervous, overworked dyspeptic with poor digestive juices returning to his work and nervous strain is likely still to find that his digestive power is not that of a healthy ploughman; despite the benefit conferred upon him by operation he must still recognise that for him there are limitations which he must not transgress. One of the subjects in gastric surgery which I suppose gives rise to most difficulty in our minds is what cases of hæmatemesis should be submitted to surgical treatment, and what treatment should be adopted—the direct, by excision of the ulcer or local treatment by cautery or similar means, or the indirect, by gastro-jejunostomy. Of the former I have no experience; all the cases of hæmatemesis I have treated, not a very considerable number, have been submitted to indirect treatment, and three of them have died, and for that reason remain somewhat prominently in my mind. One, a young woman, had, with only a brief history of stomach trouble, vomited blood for several days in considerable quantities; the amount altogether was very large and the patient was thoroughly exsanguine. I felt impelled to operate, believing that the patient was the subject of gastric ulcer. The bleeding practically ceased after short-circuiting, but the patient died on the fourth day from pneumonia, and at the autopsy no ulcer could be discovered but simply a swollen, soft, and turgid condition of the stomach mucous membrane. The second death occurred in a very feeble, emaciated man, aged 58, with long-standing gastric ulcer. The loss of small quantities of blood had been very frequently repeated and the patient had been in bed for weeks on rectal feeding. Subsequent to operation a little blood was vomited, and the patient died on the third day of asthenia. At the autopsy, in addition to an ulcer close to the pylorus, there were several excoriated patches in the stomach and a few ounces of blood which the pathologist considered had oozed from the excoriated patches rather than from the original ulcer. The third death occurred in an alcoholic man, aged 46, who, before admission to hospital, had suffered one enormous hæmorrhage which caused him to faint; this followed long-existing symptoms indicating ulcer. After operation several small quantities of blood were vomited, and death occurred from pneumonia and a small empyema on the sixth day, the last possibly due to infection from the base of an adherent ulcer on

the lesser curvature and posterior surface. Across this ulcer ran a large artery with an opening of some size in it, the orifice being imperfectly closed by a soft clot. I have often turned over in my mind these three fatalities to consider what guidance they would give me for future emergencies. My first conclusion is that I shall much hesitate to operate in future in cases of women whose history begins acutely with hæmorrhage and with no former symptoms of gastric ulcer. My conclusions as to the two ulcer cases are different. The first man was far too feeble to allow of any extensive search for bleeding points such as would have been necessary to stop his loss of blood; the only lesson of the case is that it is a discredit to medicine if patients are allowed to fall *in extremis* before surgery is resorted to; this man ought to have been submitted to operation some years earlier. With regard to the third case, I regret that I did not open the stomach and seek the bleeding vessel, as there was, in the one profuse hæmorrhage following long-persisting symptoms of ulcer, an indication that some considerable vessel was involved. I pass on now to the question of operation, where there is reason to suspect mechanical obstruction either at the pylorus or in the body of the stomach. The diagnosis may be easy, as the symptoms are sometimes absolutely pathognomonic; the dilated organ is easily percussed and palpated; the profuse vomiting with old retained food remnants and the presence of peristaltic waves in the stomach, which can be seen, form a classical picture. Such picture is only produced, however, after prolonged misery and should not be waited for. Early diagnosis can be attained by distending the stomach with an effervescent and by a test meal withdrawn after from nine to twelve hours to show if stasis exists or not. Dilatation alone does not call for operation, as it may exist in merely atonic stomachs, which I do not think are benefited by anastomosis. When with dilatation we have evidence of stasis then I would always press for short-circuiting in patients otherwise fit for operation. When to operate in ulcer without stenosis is a much more difficult problem, dependent upon the uncertainty of diagnosis of ulcer. To illustrate this difficulty I may briefly mention four cases in my recent experience. (1) A girl, aged 19, with what seemed to be a typical history of gastric ulcer, the symptoms of two and a half years' duration including three attacks of hæmatemesis, yet at the operation no ulcer could be found and the only explanation of the patient's illness lay in an adherent gall-bladder containing calculi and pus. (2) A male, aged 31, diagnosed as suffering from gall-stones, who was really the subject of a pyloric ulcer, which it was evident had at some time given rise to peritonitis, probably from a little leaking, and yet this patient had never at any time vomited. (3) A woman, aged 35, who was suspected of gall-stones with catarrhal cholecystitis. The opera-

tion was practically exploratory in the first instance and no gall-stones were discovered, but a stenosed pylorus from contraction of a healed ulcer, and yet at no time in her history had this patient vomited. (4) A male, aged 35, under observation by several experienced observers who regarded him as a neurotic dyspeptic; pain was the chief symptom, vomiting was rare, hæmatemesis was absent. After an unusual bout of pain a mass was felt in the region of the pylorus which prompted immediate operation, when a pyloric ulcer was discovered which had given rise to a circumscribed leaking from the stomach. Recovery followed operation, but the risk run in waiting for the diagnosis had been great. The difficulty was the greater because the patient was and is distinctly neurotic. Difficulties in diagnosis such as I have just quoted were not my own difficulties only but were shared by others whose opinions were worthy of respect, and they make me mistrust statistics such as are often quoted to show an infinitesimal death rate from gastric ulcer. Another reason for doubting lies in the disproportion between men and women which these figures seem to establish. I find that at the General Hospital, Birmingham, the patients requiring operation for gastric ulcer and its results are almost evenly divided between the sexes, and in my own twenty-nine cases for the past twelve months, to which I have already referred, eighteen were men and eleven women. This suggests either that many men with gastric ulcer are not so classified or that they suffer more severely from the disease than do women. I am no advocate for indiscriminate short-circuiting in dyspeptics, in those who are not the subjects of ulcer or mechanical obstruction resulting from it. Short-circuiting in these is, I believe, likely to be a dismal failure; but with the uncertainty of diagnosis in ulcer a reasonable use of exploratory operation will, I think, avoid exposing some patients to misery and to danger whilst we are waiting for more certain indications of the nature of their trouble.

Dr. E. I. Spriggs.

There is a general agreement that conditions arise in connection with gastric ulcer, mainly of a mechanical nature, which can only be efficiently treated by surgical procedures. A claim has also been put forward to include among these hæmorrhage, if severe or if recurrent, and those cases which, after ordinary medical treatment, relapse. Such a claim, to be established, must be supported by the experience of those who have observed the results of both medical and surgical treatment, and it must also be supported by statistics. Statistics, to be of value, should express, not only the immediate result of treatment as regards the mortality and the proportion of patients discharged well, but must give accurate details of the after-history of these patients. Furthermore, in

the comparison of one set of figures with another it must be clearly recognised that the average duration of treatment must be stated, that hospital patients cannot be compared with private ones, and still less is it fair to compare the ultimate results of treatment of the poorest classes with the immediate results of the treatment of patients who are in better circumstances. Some months ago, having become interested in a special form of treatment, to which I shall shortly refer, I had occasion to look up some of the figures. The statistics collected by Dr. Bulstrode, at the instance of Mr. Mansell Moullin, have been used as a basis in this discussion by some speakers. These are not supported by any other large series which I have found in the literature. A direct mortality of 18 per cent. during treatment is contrary to personal experience—it is equal to saying that the mortality of gastric ulcer is greater than that of many enteric fever epidemics. In the St. Thomas's Hospital statistics, presented by Dr. Hawkins and Mr. Nitch, the mortality was 13·3 per cent., and of the cases treated medically 2 per cent. The mortality in the Liverpool Royal Infirmary from 1896 to 1905 was 4·8 per cent. (Abram, 'Lancet,' November 3rd, 1906, p. 1217), and in Dr. Norman Moore's series 5·7 per cent. In the Boston City Hospital the mortality from gastric ulcer, taken a considerable time after the patients had left the hospital, was 7·5 per cent.; in Leube's series of 556 cases, many of whom, however, were not of the hospital class, only 2·2 per cent. died, and in 135 cases under the Lenhartz medical treatment quoted by Haberman ('Lancet,' July 7th, 1906, p. 25; *v.* also 'Münch. med. Woch.,' 1904, p. 1, vol. li) three died under treatment—a mortality of 2·2 per cent. The statement that one third of the patients succumb eventually to this disease is also unsupported by other figures. In the Boston series the proportion of the patients in the last twenty years who succumbed to gastric ulcer or its complications was, as stated above, 7·8 per cent. The unfavourable nature of the London Hospital results is perhaps due to the extreme poverty of the district from which the patients are drawn. The patients were probably most of them unwisely fed before and after their stay in hospital. It is clearly unjust to operate upon better-class patients with gastric ulcer because ignorance and poverty are rife in Whitechapel. The third statement founded on the London Hospital statistics is that two fifths of the cases under medical treatment relapse—that is, suffer two or more attacks. This is not so open to controversy; nearly all figures show a large proportion of second attacks, though not so large as this. In a proportion of these cases the doubt arises as to whether the treatment had really resulted in the healing of the ulcer, especially as we know that gastric ulcers have been found after death which had given rise to no symptoms during life, though in many, occurring in the ex-

perience of us all, little doubt can be harboured of complete cure—for instance, in the case of a young woman, at present under my care, who was free from dyspepsia in any form, and actively working for an interval of five years between the first and second attacks.

The figures referring to the medical treatment of gastric ulcer therefore show that the disease is very liable to relapse, and the argument for some other method, such as the surgical, may justly be advanced. We must therefore inquire what surgical figures have been gathered, with details of the cases, and in a similar class of patients, which will inform us as to the liability to relapse after gastro-jejunostomy. In the series of excellent results put before us by the distinguished surgeons in this discussion one cannot fail to recognise the technical skill which has been displayed. But in a matter of such paramount importance it is essential that detailed accounts of cases should be presented; this has been recognised in the institution of new methods by other pioneers; for instance, the first sixty cases treated by the Lenhartz method were published in the 'Münchener medicinische Wochenschrift,' a few lines to each case, so that all may judge of their severity and circumstances. Without this evidence the objection may be urged that a large number of these operations were unnecessary. Also, to be convincing, such a history should be accompanied by a note as to the present condition of the patient. Further, in those cases in which medical treatment is said to have failed, it would be of importance to know of what kind was the medical treatment, and how carefully it was applied, and what the average duration of treatment was, or whether whatever treatment had been provided previous to a surgical consultation was regarded as efficient medical treatment. A medical consultation might have prevented, at all events, a proportion of the operations which have been done through errors of diagnosis. A series answering to these requirements has been published by Mr. Moynihan in the 'Lancet' embracing cases of duodenal and of gastric and duodenal ulcers, in a good number of which there could be no doubt that surgical treatment was advisable. Of the cases as to which there was a definite report of the present condition of the patient I think not more than three refer to a period of more than two years. Ricard (Soc. de Chirurg., 1904) records 100 cases of gastro-enterostomy, 4 for gastric ulcer, and says the operation is useless except for complications; criticisms have also been offered by Mr. Barling ('Brit. Med. Journ.,' May 1st, 1904).

The results obtained by operation for hæmorrhage, as Mr. Paterson has pointed out, compare unfavourably with those from skilled medical treatment. It does not follow that because a patient dies of hæmorrhage he could not have been saved by any form of medical treatment, or that he could have been saved by

operation. In all the series of cases treated by physicians who have given special attention to the disease the deaths from hæmorrhage are exceedingly rare—in Leube's series 6 out of 556, in the Lenhartz series 2 out of 135, in the series from St. Thomas's Hospital less than 1 per cent. The surgical results, on the other hand, are not so favourable. Mr. Paterson has already referred to a mortality of 66—70 per cent.,; in Mr. Moynihan's 6 out of 33 died; in Quénu's series (Soc. de Chirurg., 1904) in 32 gastro-enterostomies for bleeding ulcer, in 9 the bleeding returned and 8 died. Krogius ('Schmidt's Jahrbuch,' 289, 1906, s. 101) records 6 cases in which after gastro-enterostomy or pyloroplasty bleeding or perforation followed. Barling ('Brit. Med. Journ.,' May 1st, 1904) also concludes that the cases for operation in hæmorrhage must be selected, and that fatal bleeding may not be obviated by operation.

I think, therefore, that a comparison of some of the series of foreign statistics with our own shows that, although the modern surgical procedures which are available may be safely relied upon to reduce the mortality from this disease in those complicated and chronic cases about which so much has been said, occurring for the most part in older people, as far as present experience goes, no evidence has yet been brought forward to show that operation is beneficial in the majority of cases. If it can be shown that symptoms do not recur after gastro-enterostomy, a great advance will be made. But in many cases they do, as probably every out-patient physician can testify. Such patients do not go back to the surgeon, they seek medical advice at the out-patient department of some other hospital. An accumulation of similar information to that afforded by Mr. Paterson will be of inestimable value in years to come. In the meantime we are constantly meeting with cases of recurrence of symptoms after gastro-enterostomy similar to that which occurs after medical treatment. Dr. Habershon has also stated that 10 per cent. of the operation cases reported by him relapsed, 4 out of 39.

The total metabolism of patients who have been subjected to gastro-enterostomy has been investigated in five women; in one case the assimilation of proteid was normal, in the others below normal; the general nutrition was, however, stated to be improved in each case. In a patient whose stomach was excised in three months after the operation, Deganello ('Arch. Ital. de Biol.,' 33, 1) found that digestion and assimilation were normal. We know that digestion is more complete chemically if the action of the peptic is followed by that of the pancreatic juices. We may, therefore, still venture to doubt whether it is desirable to deprive the stomach of the normal mechanism for the discharge

of its contents unless there is good reason to believe that recovery can be attained in no other way.

Experience shows that two conditions frequently accompany gastric ulcers, anæmia and hyperacidity. Hyperacidity is not present in all cases, especially, it is said, in those with little pain, but if gastric juice possessed of even normal acidity is in contact with an ulcer, the healing will be greatly delayed. Mathes (v. Wagner, 'Münch. med. Woch.,' 1904, 1, Bd. li), who artificially produced defects of the gastric mucous membrane, found that they healed rapidly except when weak hydrochloric solution was put in the stomach. With respect to anæmia Quincke and Daettingler, also Silbermann, found that ulcers produced in normal dogs healed quickly, while in dogs rendered anæmic they did not. Now gastro-enterostomy offers an alternative drainage route for the gastric secretions which are prevented in many cases from passing through the pylorus by spasm of its musculature; it also offers, and this I consider a very great advantage, the opportunity of rapidly getting the patient on to a nutritious diet. And this brings us to the very pertinent question asked by Dr. Hawkins—Has our modern medical treatment reached finality? Dr. Hawkins is of opinion that it probably has. It is obvious that medical treatment by saline or nutrient enemata with a graduated milk supply has the great defect that it is not calculated to improve the anæmia. It should be followed by a further rest cure with a liberal diet, but in hospital cases this is often impossible. Rectal feeding, as far as it has hitherto been carried out, is starvation diet. In the Oliver Sharpey Lectures delivered in April this year, at the Royal College of Physicians, I reviewed accurate metabolism experiments which have been carried out on this subject, and concluded from the most recent experiments that about 500 calories a day have been shown to be absorbed, about double their food value having been introduced into the rectum. This is about half the need of a woman of 50 kilogrammes (8 stone) lying in bed. Such a food-supply is, of course, of very great value, but is still sub-nutrition. Dr. Sharkey, in the Bradshaw Lecture, has since reviewed the literature of the subject, and came to a rather more favourable conclusion. I believe such a favourable conclusion can only be reached by ignoring the quantitative results. It is of interest and importance to know what percentage of various foods are absorbed from the rectum, but the only information which is satisfactory is that telling us how much food value was absorbed per day and for how many days it could be kept up. We cannot hope, therefore, to improve the anæmia by rectal feeding, and with respect to the acid of the gastric juice, doubt has of late been thrown upon the view that gastric juice is not secreted into the ulcerated stomach when a nutrient enema is given, resting on the statement of Umber. I have made inquiry of the sisters at St.

George's Hospital, and have found that patients with gastric ulcer receiving nutrient or even saline enemas frequently complain of pain, referred to the stomach, and nausea. It appears probable to me that this is due to an irritation of the ulcer by gastric juice. We know from the work of Pawlow that animals in a state of hunger produce gastric juice on the very thought of food. We should, therefore, expect a patient on a starvation diet to readily secrete it in response to any stimulus. It is likely, therefore, that ordinary medical treatment is not efficient in many cases in preventing the secretion of acid gastric juice into the stomach.

These considerations lead me to the conclusion that the treatment introduced by Professor Lenhartz is worthy of an extended trial in this country, and I venture to mention this method here because the statistics published in reference to it are much more satisfactory than many upon which the argument for more frequent surgical treatment is based. Lenhartz, while keeping his patients absolutely at rest, feeds them at once, even after hæmorrhage, with milk, and raw eggs beaten with sugar, beginning with 100 c.c. ($3\frac{1}{2}$ oz.), iced milk and one iced raw egg per day, and increasing up to 800 c.c. milk ($1\frac{1}{2}$ pints), and six or eight eggs. The milk and eggs are sipped out of a spoon and made to last as long as possible, in order that there may be constantly proteid in the stomach to combine with any HCl secreted, and protect the ulcer from irritation. The quantity of milk is not increased above 800 c.c. *per diem*, and is not drunk out of a glass so that the stomach may never be dilated by a bulk of fluid. This diet is reasonable on theoretical grounds. In the first place milk calls forth less secretory activity of the stomach than any other food. Egg-albumen calls forth no juice in the dog; and though a secretion follows its ingestion in man, it is probably less than that produced by many foods. Fat, which is present in egg-yolk, inhibits the secretion of gastric juice. The proteid of the milk and the albumen will combine, as above mentioned, with the HCl of the gastric juice. Such a diet is, therefore, calculated to call forth but little gastric juice, and to combine with the HCl of that secreted. The patients may be very rapidly supplied with ordinary food and Blaud's pill. Raw mince is supplied from the tenth to the eleventh day, and if well borne is soon followed by boiled rice. It may seem somewhat heroic to put food into the stomach so soon after a hæmorrhage. Theoretically, the fear of dislodging a clot suggests complete rest for that organ as essential, but, on the other hand, the danger of the digestion of the clot by gastric juice indicates that it may be advisable to provide some other material for any gastric juice to act upon. The use of small quantities of mainly proteid food combines with the HCl and

does not stretch the stomach-walls, either by the bulk of ingesta or by the production of gases.

I bring this method forward because, as I have said, of the results which are claimed for it, but I should not venture to do so if I had not had some personal experience of it. During the past three months, however, I have treated a number of cases of gastric ulcer in this way at St. George's Hospital, both in my own beds and in beds kindly lent me by my senior colleagues Dr. Rolleston and Dr. Latham, and although the patients so treated are few in number they are sufficient to convince me that the method is worthy of trial. I have seen enough to show me that the method possesses the enormous advantage that the patients are well fed almost from the beginning, are more satisfied, usually lose their pain sooner, gain weight, and most of them are discharged in a much healthier condition in a shorter time than is the case with the older methods. I have seen no untoward result of any kind, but my own results are as yet so few that I will quote Lenhartz's figures (Habermann, 'Lancet,' July 7th, 1906). In 135 cases were 3 deaths, one from anæmia, 17 per cent. hæmoglobin on admission, one with five ulcers, and one with femoral thrombosis and pulmonary embolism. Wagner followed up a number of these patients and got twenty-five replies. Two patients were still unable to work, and five had occasional pain, the other eighteen being perfectly well. It is clear that this treatment is like gastro-enterostomy in that a sufficient time has not yet elapsed to judge of the after-results. We can only judge of it after it has been tried on a larger number of cases. Nevertheless my own experience leads me to think that the combination of such a rational treatment for ordinary cases, with surgical treatment for such as may prove resistant, should result in a considerable reduction in the frequency of relapse and in the mortality from gastric ulcer.

Mr. D'Arcy Power.

I understand that the object of this discussion is to obtain from each speaker the results of his individual experience in the treatment of non-malignant ulcer of the stomach whether by medical or surgical means. The experience must be personal if it is to be of any value, and it should be presented as far as possible without conscious bias. I have no hesitation, therefore, in giving my own results and the conclusions at which I have arrived.

I performed my forty-first gastro-enterostomy for non-malignant disease of the stomach last week, and of these forty-one patients three died within twenty-four months and one two months afterwards. The rest, so far as I have been able to discover, are still living, some cured and others better for the

treatment. The mortality of gastro-jejunostomy, therefore, in my hands has been 7·3 per cent.

The cases had all been under medical treatment for a long time. Many were suffering from chronic gastritis due to ulceration of the stomach, two had drunk corrosive poisons, and four had suffered from symptoms of duodenal stenosis.

I have also had twelve cases of perforated gastric ulcer with three deaths, and six cases of perforated duodenal ulcer with four deaths.

In performing gastro-jejunostomy I have varied my operations from time to time partly to suit the exigencies of the case, partly as the result of experience, and partly to verify the statements of those who said that their own methods were better than those I was using. On one occasion I used McGraw's ligature, once or twice the anterior method, usually the post-colic operation with a twelve-inch loop; the last operation was post-colic without a loop, in deference to the opinion expressed in the course of this discussion. Except in the case where I used McGraw's ligature, all my anastomoses have been made by direct suture without the use of any mechanical appliance, and I am satisfied if the operation is completed in thirty minutes from the first incision to the last suture. Two of the three patients who died were women reduced to the last extremity by pain, sickness, and starvation. They died of shock because I had under-estimated the depth of their exhaustion.

I am well satisfied with gastro-jejunostomy as an operation, for in none of the three fatal cases was there any leakage through failure of technique, whilst in the patients who recovered there was always some improvement and often a cure. In some of the cases the improvement was immediate as soon as the shock of the operation was over, and this was especially noticeable where the symptoms had been due to duodenal obstruction. In other cases it was remarkably delayed, though it was eventually well maintained.

All the cases of gastro-jejunostomy ran a straightforward course except the following, and in no instance was there any serious vomiting of the nature of a vicious circle.

Leaking Gastric Ulcer; subsequent Gastro-jejunostomy; Vomiting of Ascarides; Recovery.

The first case of gastro-jejunostomy which presented anything unusual was that of a girl aged 20, whose stomach I had sutured seven weeks before on account of a leaking gastric ulcer. The patient made a good and uneventful recovery from the operation, but she soon began to complain of abdominal pain radiating to the small of her back. She was put upon nutrient enemata, and I kept her under observation in St. Bartholomew's Hospital from the 9th to the 30th of June. She suffered from dyspepsia

and vomiting after her food, and as she did not improve in spite of all that could be done for her by variations in diet, I performed a post-colic gastro-jejunostomy with a loop on 30th June. The patient subsequently passed a very bad night, and was constantly sick until she brought up two large specimens of *Ascaris lumbricoides*. The attacks of vomiting then became less violent and gradually ceased, until from July 3rd she began to make satisfactory progress. The wound healed by first intention, and the stitches were removed on July 9th. At 11 a.m. on July 14th she began to complain of pain, and her abdomen was found to be tender but not distended. At 2 p.m. on this day she had an attack of severe epigastric pain; there was considerable abdominal distension, and her liver dulness had markedly diminished. At 4.30 she vomited eighteen ounces, and at 6.15 p.m. four ounces. Her pulse rose from 104 to 148, and, as she was in great pain, I ordered her a hypodermic injection of morphia and opium fomentations. She vomited a further twelve ounces and then passed a good night, the abdominal distension disappeared, and she had no further hindrance to convalescence. She left the hospital on July 23rd. I believe that I should have saved her this attack of pain and discomfort if I had performed the gastro-jejunostomy without a loop, for I have very little doubt that it was due to duodenal stasis.

Gastro-jejunostomy; Recurrent Hæmorrhage.

A deaf and dumb girl, aged 19, was admitted into the Bolingbroke Hospital on 1st October, 1905, for pain and vomiting due to an ulcer of the stomach for which she had been under medical treatment on three occasions previously. She had vomited a considerable quantity of blood three days before I saw her. She was watched for a month in hospital when it was found that she had many attacks of sickness and that she suffered much pain. I performed a post-colic gastro-jejunostomy with a loop on October 28th. The walls of the stomach were congested and thickened. During the operation there was a great deal of bleeding from the vessels in the submucous coat of the stomach and several ligatures had to be applied. The patient vomited repeatedly on the 28th and 29th of October, and at 6.30 p.m. on the latter day, twenty-eight hours after the operation, she vomited a little blood: an hour later she brought up a pint of blood, at 10 p.m. half a pint of blood, and at 11.30 p.m. she brought up another half pint, making a total loss of two pints in five hours. I reopened the abdominal wound at 1 a.m. on October 30th, and found that the anastomosis was satisfactory. The stomach was therefore opened by a linear incision carried just above and parallel to the gastro-jejunostomy so that the inner aspect of the anastomosis could be examined freely. No actual bleeding point was detected, but there was a small patch

of ulcerated mucous membrane covered with blood clot close to the margin of the anastomosis. I passed a curved needle, armed with a silk ligature, beneath it and sutured healthy mucous membrane over it. A ligature was afterwards applied to another piece of mucous membrane which looked as if it might ulcerate, and the stomach was closed with Lembert's sutures. The abdominal wound was subsequently closed.

The patient vomited a little altered blood on October 30th, and on October 31st, as she was still retching, the stomach was washed out, and again on November 5th. She left the hospital on November 27th, and I have seen her since more than once. Her friends tell me that she keeps well and is at work.

Hour-glass Stomach; Gastro-jejunostomy; Recurrence of Symptoms; Second Gastro-jejunostomy; Uncured.

The next troublesome case was that of a domestic servant aged 27, who had suffered for twelve years from pain and vomiting after food. She was treated for three months in a medical ward, and as she did not improve I performed a post-colic gastro-jejunostomy with a loop. I discovered during the operation that she had an hour-glass stomach. The patient left the hospital a month after the operation weighing half a pound more than when she was admitted, and somewhat improved as regards the pain and sickness. The improvement was maintained for six months, and she then began to suffer from her former symptoms. A year later she was readmitted, and she then weighed two pounds more than when she had been discharged, and her stomach only held a pint and a half instead of two pints as it did before the operation. After a test meal six ounces less fluid could be withdrawn from the stomach than was introduced. There was a large increase in the acidity of the gastric juice. A second gastro-jejunostomy was performed in the middle of September, two inches beyond the previous anastomosis, which proved to be patent. The patient made a good recovery from the second operation, but her condition showed no improvement. She remained in hospital until November 1st, when she was admitted into one of the Homes for the Dying near London. She died a fortnight later, but unfortunately I did not hear of her end until it was too late to ask for a post-mortem examination.

Recurrence of Hæmorrhage after Gastro-jejunostomy for an Active Duodenal Ulcer.

The patient was a man aged 31, whose wife said that "he never denied himself anything." He was admitted into St. Bartholomew's Hospital on 18th May, 1904, for pain in the lower part of the abdomen and vomiting. The pain was first

felt six months previously, and it gradually became worse until, on rising one morning nine weeks since, he had felt faint, and "vomited a quart of blood all in lumps." He was at home five weeks, and was then sent to an infirmary for three weeks. On April 19th he retched after food, and on the following day he vomited a pint of blood after food. There was no history of melæna, and he had never suffered from any illness of importance. The patient had a point of maximum tenderness in each iliac fossa, and there was also a tender spot one inch to the right of the umbilicus, which varied somewhat in position at different times of the day. He was kept under medical observation from April 23rd to May 19th, and I saw him at intervals, and diagnosed the presence of an ulcer in the first part of the duodenum close to the pylorus, and probably on the posterior wall. I performed a posterior gastro-jejunostomy with a loop upon this patient on May 19th, and he was discharged from the hospital on June 17th after a perfectly uneventful recovery, saying that he had had neither pain nor sickness since the day after the operation.

The attacks of pain and vomiting recommenced five weeks after the patient left the hospital, and he was readmitted on 10th January, 1906. Whilst he was under observation he had several characteristic attacks of duodenal hæmorrhage. Sudden blanching, with the signs of severe collapse and melæna, or the vomiting of blood some hours later. The attacks bore no relation to his meals, and one of them, occurring at three o'clock in the morning, was so severe as to lead the nurse to send hurriedly for the house physician. They appeared to be somewhat more severe and frequent when he was in bed on a low diet than when he was up and about, taking good but plain food.

In regard to the operation of gastro-jejunostomy, I welcome Mr. Mayo Robson's suggestion that it is unnecessary to pare the mucous membrane at the edges of the gastric and intestinal incisions, because the operation is thereby shortened; but, at the same time, I rather grudge the loss of the histological examination which such self-denial implies, and hitherto I have caused sections to be made of the mucous membrane in many of my gastro-enterostomies.

I have very few remarks to make on the subject of acute perforation of the stomach and duodenum, except to say that the practitioners in the neighbourhood of the large hospitals are now quite alive to the value of immediate diagnosis and operation. The last patient upon whom I operated for a perforated gastric ulcer was taken ill at half-past twelve o'clock, and by three o'clock he had been operated upon and was back in bed. In like manner, my last case of duodenal ulcer perforated acutely at eight o'clock one night, and at twelve o'clock I had operated upon him, although he was six miles away; and duodenal ulcer

is not so easy to recognise as gastric ulcer. It is needless to say that both patients did well.

Personally, I find that the difficult cases are those to which I give the name of "leaking" gastric ulcers, where the patient presents many of the signs of acute perforation in a modified degree, and is just not bad enough to operate upon. In three such cases the patient has recovered without operation, but the last one died, and a post-mortem examination showed a perforation, which was so recent that it was questionable whether the actual perforation had not occurred at the time of death. I know quite well that broadly in all doubtful cases an operation should be performed, but the three cases which recovered have somewhat biassed my judgment.

Mr. G. E. Gask.

(*Unfortunately unable to be present, submitted the following note, which was read by Mr. D'Arcy Power.*)

I have confined my attention to three points. (1) The results after gastro-enterostomy performed for fibrous stenosis of the pylorus. (2) The results after gastro-enterostomy performed for gastric ulcer. (3) The results after laparotomy for suture of perforated gastric ulcer. The results were obtained after personally seeing and examining the patients. The numbers, therefore, are small, owing to the great difficulty in tracing patients to their homes and getting them to come up to London from different parts of England.

Gastro-enterostomy for Fibrous Stenosis of the Pylorus.

Total number of cases collected during a period of three years (operations 1903—1905). Nineteen cases—thirteen male, six female. Of these four died as the result of the operation. Immediate mortality 21·05 per cent. Of the remaining fifteen cases six were seen personally. Of these four were a complete success. One was much improved. One, stomach trouble, was relieved, but much troubled by a renal calculus. I had also from three others letters which seemed reliable, in which it was said the operation had been a success. The remaining cases I have not been able to trace.

Gastro-enterostomy for Gastric Ulcer.

Total number of cases collected during a period of three years. Sixteen cases—five male, eleven female. Of these two died as a result of the operation; immediate mortality 18·1 per cent. Of the remaining fourteen cases five were seen personally. Of these four were very much improved (slight indigestion). One was only slightly improved (vomiting and pain). All these cases were operated on in the year 1905.

Abdominal Section for Suture of Perforated Gastric Ulcer.

Total number of cases collected during a period of nine years (1897—1905). Sixty-nine cases—twenty-eight male, forty-one female. Of these thirty-four died following the operation. Immediate mortality 49·2 per cent. Of the remaining thirty-five cases eleven were seen personally. Of these nine were a complete success. One had been in hospital suffering from gastric ulcer and hæmatemesis (operation 1900). One still suffers from indigestion and occasional slight hæmatemesis (operation in 1900); therefore gastro-enterostomy not needed (Hale White).

Ventral hernia.—In three of these cases the scar bulged unduly, and in one there was a definite ventral hernia. In the others the scar was sound.

Mr. G. H. MAKINS

(who was unfortunately prevented from being present at the Adjourned Meeting, sent the following contribution to the Discussion).

Perforated Gastric Ulcer.

In the report drawn up by Dr. Hawkins and Mr. Nitch, the results of operations for this condition, performed at St. Thomas's Hospital during a period of fifteen years, have been collated from a paper by Mr. Sargent in the 'St. Thomas's Hospital Reports' (vol. xxxiii, p. 477, 1904). From the numbers there quoted it is seen that the total mortality for the whole period amounted to 55 per cent., and for the latter five years to 43 per cent., the improvement being no doubt due to perfection in the method of dealing with the condition.

As details of these cases are before the Society, I do not propose to remark upon them further than to point out that this high post-operative mortality, combined with the fact that the mortality from perforation *per se* was 8·5 per cent. of the 556 cases of gastric ulcer treated within the hospital during the same period, is a strong argument for the more frequent performance of anastomosis at an earlier period, with the view of removing this most serious cause of mortality from the disease.

As to the details of operations for perforated ulcers, my own experience is in favour of inversion of the ulcer, and the limitation as far as possible of the employment of irrigation. Only that portion of the peritoneal cavity actually soiled by escaped stomach contents should be irrigated or, preferably, moist sponged, and the remaining reactionary peritoneal effusion removed by dry sponging or syphonage. A pelvic drain acts for a very short time, and in my experience is of little value, if not objectionable.

I have no experience of the immediate establishment of an

anastomosis in these cases; but in the future I shall, when opportunity allows it, certainly try the method. The fact that a second ulcer existed in no less than 15 of 77 cases of perforated gastric ulcer seems a strong argument in favour of the procedure.

Perigastric Abscess.

In the 'St. Thomas's Report' fifteen such cases are referred to with eleven deaths. I have operated on four such cases (three of which are included in the report, Nos. 78, 79, and 83). Two of the four cases died from exhaustion and septicæmia after operations for the evacuation of pus. In one no attempt was made to deal directly with the gastric condition, and at the post-mortem examination perforated ulcers were found at the posterior aspect of both the stomach and duodenum. In this case the condition of the patient did not encourage me to operate. In the second fatal case the perforation was anterior, and incision of the abscess was followed by so free an escape of the gastric contents that I attempted to close the opening by a plastic operation on the third day; this operation failed, and on the sixth day an anterior gastro-enterostomy was performed, the patient dying twenty-three hours later.

In the remaining two cases a simple incision and evacuation of the pus was followed by recovery, and no further treatment has been attempted. I think that in both it would have been preferable if I had undertaken an anastomosis.

These cases as a class also seem to support strongly the performance of an anastomosis at an earlier date in the disease, both by reason of the difficulty of dealing with them surgically and their fatal nature.

Hæmatemesis.

The St. Thomas's statistics show that death from this cause alone is rare. Dr. Hawkins puts it at 0·7 of the whole 556 cases dealt with. This is an important fact, as this class of complication is the most difficult to deal with in the light of present experience, and has afforded the most indifferent surgical results.

Three classes of case have to be dealt with.

1. Cases of the condition in which general oozing from an intact mucous membrane occurs (gastrostaxis).

2. Cases in which the hæmorrhage originates from an acute erosion.

3. Cases in which the hæmorrhage is from an old extending ulcer.

I have had no operative experience of the first class, and am in full accord with the view expounded by Dr. Hale White that they are best treated expectantly, and not by operation.

In the second class I have operated on one case. Female, aged 25. There was no previous history of any disease except some attacks of bronchitis.

A sudden severe hæmatemesis came on without any previous warning; considerable collapse resulted; during the next twenty-four hours small quantities of blood were spat up, then a second severe hæmorrhage occurred; the same train of events followed in the third twenty-four hours. The patient was now very much collapsed, and on three occasions more than a pint of blood had been vomited at a time, beyond the small quantities between.

It was thought well to explore the stomach. No ulcer could be discovered until the whole mucous surface had been extroverted, when a small erosion of the diameter of a split pea was found at the posterior aspect of the lesser curvature close to the cardiac orifice, at the bottom of which a branch of the coronary artery was spurting.

Hæmorrhage was controlled by a purse-string suture, and the wounds closed.

It was unfortunately necessary on account of the collapsed condition of the patient at the end of the operation to leave her in a very cold room in which the operation had been performed. Nevertheless she progressed well until the third day, when she began to suffer acutely with bronchitis, and on the eve of the sixth day she died from this cause. No post-mortem examination was allowed by the friends, but the after course left no doubt that the hæmostasis had been immediately satisfactory, and no gastric symptoms were noted.

Although this case was an unfortunate one, yet death, in my opinion, cannot be ascribed solely to the fact that the operation was for gastric hæmorrhage, and under similar circumstances I should feel inclined to take the same course in the future.

The difficulty of discriminating cases of acute erosion is obvious, but the fact of rapidly recurring severe hæmorrhage in patients with no previous history of gastric disease affords strong presumptive evidence of the condition, and should lead to the consideration of operation. A comparison of the total mortality in all cases of gastric hæmorrhage, treated by medical means, with the mortality which has followed all operations, is unsatisfactory. The result no doubt goes to show that hæmorrhage *per se* is a rare cause of death, but such series include a number of cases in which, at the present time, no surgeon would wish to operate, while the number subjected to operation include, at any rate, the most unfavourable that have been met with.

As to the third class of case, where hæmorrhage occurs from ulceration of long standing, my own experience is in favour of an expectant attitude. I have had no cause to regret this in the few instances in which I have had to consider the question, and in the two cases quoted in the Report from St. Thomas's Hospital,

the condition found in two cases not operated upon was not one favourable for direct local treatment.

A difficulty which attends an expectant attitude in such cases exists in the disinclination of patients who have recovered from severe attacks of hæmorrhage to agree to the performance of an anastomosis when they are "well."

Other Complications of Gastric Ulcer.

I have little experience of operations for gastric ulcer, except in connection with its sequelæ. I have notes of eighteen cases of which number thirteen are included in the report of Dr. Hawkins and Mr. Nitch.

Of the whole eighteen, eleven were cases in which some degree of pyloric obstruction or gastric dilatation was present.

Of these, ten were subjected to anastomosis, and one to a limited partial gastrectomy made on the erroneous impression that the disease was malignant.

Of these eleven cases one died from collapse an hour after the completion of the operation. The patient, a man aged 36, was in a very exhausted state, and for three weeks before the operation had been subsisting entirely on rectal alimentation. He bore the actual operation fairly well, and no particular anxiety was felt about him when he left the operating theatre, but he collapsed shortly after his return to bed. At the post-mortem examination extensive atheroma of the aorta and coronary arteries was revealed.

Of the remaining ten cases one has since died, probably from perforation. The patient, a man aged 45, who had suffered for many years from gastric dyspepsia, and for a year with signs of pyloric obstruction, had an anterior gastro-jejunostomy performed with very satisfactory immediate results. He returned some months later to the colonies considering himself quite cured, when one morning he was seized with sudden pain in the abdomen, became greatly distended, and died in thirty-three hours, seven months after the performance of the operation. I think it is probable that the perforation was of an old latent gastric ulcer rather than of a recent peptic one, since no symptoms of any kind led up to the final attack.

The remaining nine patients are alive and well, but three of them suffer still with dyspeptic symptoms of a minor degree. The first of the series is now of six years' standing; the others have been spread over the succeeding years.

Hour-glass contraction existed in three cases; of these one was treated by a plastic operation only, one by a plastic operation combined with anterior gastro-jejunostomy, and one by gastro-jejunostomy alone. These have all progressed satisfactorily, but the patient for whom no direct treatment was adopted suffers from signs indicating pyloric obstruction and

temporary lodgment of food in the distal sac. The symptoms, however, have not been sufficiently irksome to make her consent to a second operation which has been proposed.

The last four cases, which come under neither of the above headings, are the most important and the least satisfactory. Two of them are fatalities. One a woman aged 21, with symptoms of gastric ulcer of twelve months' standing, died from suppurative peritonitis due to infection at the operation.

The second fatal case is referred to on page 2 of the Report, a female aged 22. She died from pneumonia on the nineteenth day after the operation, and at the *post-mortem* examination no signs of gastric disease could be discovered, although she had been under competent treatment for gastric ulceration for a period of four years, and no doubt was felt as to the correctness of the diagnosis.

Deaths from pulmonary complications, although rare in the extensive series of cases which have been submitted to the Society by Mr. Mayo Robson and Mr. Moynihan, have occurred with some frequency in the various series of statistics from other sources, and I have lost another patient, besides the one mentioned, where the operation was performed for malignant disease. This cause of death seems worthy of more mention than has been bestowed upon it, especially in so far as it may be influenced by the anæsthetic and the position in which the patient is arranged after the operation.

The third and fourth are both failures. In one the patient has obtained no material relief, still suffering with pain and vomiting, and the other is perhaps worthy of short narration.

The patient, a married woman, aged 29, had suffered for nine years with attacks of gastric pain, vomiting, and hæmatemesis. She was under treatment in three separate hospitals for these symptoms during the four years prior to the anastomosis, and four years previous to the operation her stomach had been negatively explored by gastrotomy by Mr. Walsham.

The patient had had syphilis, and a significant point, perhaps, was a history of an attack of "melancholia" when fourteen years of age.

In December, 1903, she was under treatment for gastric ulcer at St. Thomas's, when to her other symptoms was added the development of occasional attacks of high fever reaching 103° F.

As no signs of anterior perigastric inflammation could be discovered, it was thought that ulceration might be extending into the pancreas, and an operation was determined upon.

An anterior gastro-jejunostomy was performed, with the result of considerable temporary improvement. A very firm broad adhesion between the stomach and the anterior abdominal wall at the site of Mr. Walsham's prior exploration was not interfered with.

Five months later the symptoms recurred in a lesser degree, but were alleviated by abstention from solid food. At the end of eleven months after the operation she returned to the hospital in much the same condition as she was before the operation, except that the fever was not present. Since that period the abdomen has been again explored, and the anterior adhesion released. The anastomosis allowed the finger easily to invaginate the stomach wall into its lumen, and appeared in all respects satisfactory.

The last operation was followed by some superficial suppuration in the abdominal wall, and the gastric symptoms continued without any abatement for many weeks. Since then, now about a year, the patient has been at home suffering with similar symptoms of varying degree, but is certainly in no way improved.

These two cases illustrate the difficulty which may occur in the selection of proper cases for treatment by anastomosis, yet such instances should rather impress the necessity for extreme care in the exclusion of subjects of the neurotic class, than be raised as arguments against the treatment.

A considerable consensus of opinion appears to show that patients suffering from recurrent severe attacks of gastric ulceration, if subjected to anastomosis after a prolonged period of medical treatment, may secure immunity from frequent periods of incapacitation, and many of the dangers which may subsequently prove fatal in this disease.

As to the method of performing anastomosis, except in cases which specially indicate the choice of one or other method as a result of the location of the disease there does not appear to be much importance as to whether the anterior or posterior route is chosen. I have usually done the anterior operation, but more lately have several times employed the posterior no-loop operation. The points of importance in either operation being the fixation of the intestine close enough to the greater curvature, and the provision of a large enough opening (for which latter point I attach much importance to the excision of the mucous membrane, which I have adopted on all lateral anastomoses since reading Mr. Littlewood's remarks some years ago.)

In the performance of the anterior operation I also attach importance to the fixation of the intestine to the stomach wall at least an inch and a half to the left of the anastomosis opening.

I have had no experience of the establishment of "a vicious circle" or of secondary peptic ulceration, and I have included no cases of anastomosis for duodenal disease, the results of which, in my experience, are more uniformly satisfactory than in operations for advanced gastric conditions.

Dr. HALE WHITE (in reply).

Sir, the first duty which devolves upon me is to thank the Society for the kindly way in which they have criticised some statements which I made if they disagreed with them, and it is obvious that as there have been so many speakers it will be impossible to deal with them in detail, even if I disagree with them, which, in the main, I do not. But there seem to be two or three points about which there is considerable difference of opinion. And one is, as to whether ulcer of the stomach is very frequently followed by cancer on its site. Now, cancer of the stomach is a common cause of death. It is the cause of death in 1·5 per cent. of the patients who die in a large general hospital, including among those a number of children, so that in adults it is a common cause of death. And many Fellows of this Society must have been present at many thousands of post-mortems. I think, if it were common for gastric carcinoma to follow upon gastric ulcer, those who are in the habit of frequenting the post-mortem room would be more cognisant of it than they are. Speaking from my own experience, I should say it is an exceedingly rare event to be met with in the post-mortem room. It may be replied that by the time the post-mortem is made you would not be able to recognise that the cancer had begun from the ulcer. I do not think that is a valid argument, for cancer of the stomach kills as quickly as cancer in any other part. And not only is there dyspepsia and indigestion, but there is the cancer itself killing the patient. So we should certainly, sometimes at any rate, see cancer occurring on ulcer. In their very exhaustive examination of a number of cases in a classical paper, Drs. Perry and Shaw were only able to find nine instances in point. And the largest collection of cases of cancer of the stomach in existence is the monograph by Osler and Gray, and so little do they think of the evidence in favour of the fact, that they are strongly inclined to doubt whether any relation exists between cancer and ulcer. And that is the opinion in the largest work on the subject. I myself think there is a connection, and that a certain number of cases of ulcer of the stomach do later on become cancerous, but I do not think it is as large a number as some speakers put forward. And I would ask the Society to bear in mind, in that relationship, that the proportion of cancer of the stomach in men to women is as five to two. Now, even those who think that ulcer of the stomach is more common in men than in women—and most think it is more common in women than in men—have not suggested that ulcer of the stomach is common in men in the proportion of five to two as against women. So there is considerable divergence between the incidence of ulcer of the stomach and cancer of the stomach in the two sexes.

The next point which has given rise to much discussion is the question of hæmorrhage. And there are two sharply-divided camps here evidently. Some surgeons, such as Mr. Mansell Moullin, would, sometimes at any rate, operate during the occurrence of profuse hæmorrhage. Others, such as Mr. Paterson, would not. I must say I am in Mr. Paterson's camp, and I do not think the patient stands the best chance of recovery if operated upon whilst the hæmorrhage is in progress. Physicians are accustomed to see severe hæmorrhage from the lungs, severe hæmorrhage from cirrhosis of the liver, and extraordinarily severe hæmorrhages will get well if the patient is left alone. I would call the attention of the Society to the fact that Dr. Hawkins, speaking from a very large experience at St. Thomas's, mentioned that he had quite recently seen the first fatal case of death from the bleeding of gastric ulcer. For my own part I think that if we are at the bedside, and the patient is bleeding furiously from gastric ulcer, that patient will probably stand a better chance of recovery without surgery than with, even if the patient seems desperately bad. By all means operate after the hæmorrhage, and stop its recurrence. And I think another aspect has to be borne in mind. To some extent, possibly the discussion here will help to mould the opinion of the profession on this subject, and it must be borne in mind that those surgeons who advocate operating during the hæmorrhage are all of them skilled, highly-skilled surgeons, well versed, and accustomed frequently to deal with difficult abdominal operations. And because they are occasionally successful it ought not, I think, to go forth as the right teaching that when a doctor thinks that a patient is in imminent danger of losing his life from gastric hæmorrhage, that that doctor ought then and there to operate. I can only say that if I were the doctor in charge I am sure the patient would stand a better chance if I did not attempt operation.

Another point which, perhaps, does not appeal to the physician is the attitude of the mind at the bedside which enables one to say, "This patient is bleeding; I think she will in all probability die; I must do something." I do not think it follows, because you feel that a patient is going to die that you must necessarily do something. It might very well be that that "something" might make the patient's short life shorter still.

The next point which has given rise to considerable discussion is the performance of gastro-jejunostomy at the time of perforation. That is entirely a surgical matter. Before the question can be settled we must have before us a large number of cases in which it has been done, and in which it has not been done by equally competent men, and we must know the longevity or expectancy of life of each of those two groups of cases, and the mortality at the immediate operation. No one has yet had

experience enough to be able to answer the question in the only way in which it can be answered.

I think one remarkable thing which has come out of this discussion is, what we all were surprised at before, that considering the great importance of recent experimental work showing the importance of the gastric juice, because of the manner in which it leads to the pancreatic secretion, it is a remarkable thing that gastro-jejunostomy cases do so well as they do. Mr. Paterson called my attention to the fact a moment ago, that he has mentioned two cases, living nineteen and seventeen years afterwards, and they are both perfectly well. I think we should have expected it to be otherwise, and I think it would be most valuable if we could have a large collection of cases of gastro-jejunostomy who have lived twenty years, and thus get a knowledge whether the lives of such patients are appreciably shortened.

I was very glad, indeed, to hear what Dr. Spriggs said about the treatment of gastric ulcer by medical means. I have been accustomed for some time to teach that the older method was wrong. I do not see how you can expect the ulcer to heal if you feed your patient *per rectum* for a long while, nor do I see that any harm can follow giving the patient food by the mouth-early, especially such food as was mentioned by Dr. Spriggs, and many of my own patients in whom there is reason to suspect ulcer, take food by the mouth within three or four days of their admission for severe hæmorrhage.

Another point, which has, perhaps, been left out of the discussion, is the importance of diagnosis. So many of the cases are difficult to diagnose, and until we get over that difficulty I do not see how we shall get our statistics quite accurate. I pointed out, in the remarks I made in opening the discussion, that there were a large group of young women who bleed furiously and have no ulcer. I strongly suspect that that group is larger than many of us think, and I agree fully with what Mr. Mansell Moullin said, that if those women are taken away from the group of gastric ulcers, as I believe they ought to be, gastric ulcer is a serious disease. Dr. Hawkins came to the same opinion on medical grounds. I strongly think that in properly selected cases—and they amount to a great many—the right treatment for many and many a person with gastric ulcer is gastro-jejunostomy. The evidence is overwhelming. And in passing I may say that probably the statistics which have been quoted from two large London hospitals are unduly depressing because they include even the earlier cases, which were done when the operation was on its trial, and when the staffs at the hospital were learning to do that operation. They go back too far. Technique has improved so much that the figures are now much better. I fancy Mr. Mansell Moullin thought me a greater pessimist than I am in connection with these cases. The words

I used in connection with it are, "Great relief follows operation sufficiently frequently for us to be able to say to a patient, for whom medical treatment has failed, that there is a very considerable prospect that he will be much better after an operation."

Mr. Mayo Robson (in reply).

I think, sir, the Society is to be congratulated on the excellence of the discussion to which we have all listened with such interest. I have never attended any medical society at which I have heard such good speeches as have been given during this debate. It is very satisfactory to find that there is so much agreement on the main issues before us. First, I think we are all, both physicians and surgeons, agreed that ulcer of the stomach is a much more serious ailment than it used to be considered, and that the treatment of it is not by any means to be carried out in the out-patient departments, but that it involves a prolonged and a careful course of treatment. And I think we all of us agree that a prolonged course of medical treatment ought to be fully tried before any question of surgical treatment arises. I am very much impressed by the arguments of Dr. Hale White and Dr. Hawkins on this point. Dr. Hawkins was very emphatic in saying he believed that every patient suffering from gastric ulcer ought to have a definite six months' treatment by dieting and by rest. If at the end of that time the patient was not relieved, or if in a short time there was a relapse, then the case was a fit one for surgical treatment. And that, I think, is the opinion of Dr. Hale White and of many other physicians, and I believe of all the surgeons present. The medical statistics which have been furnished, it seems to me, show a curiously varying rate of mortality. It was mentioned by Dr. Spriggs, I think, that no foreign statistics of the medical treatment of gastric ulcer had been quoted. I will give him those of Debove and Rémond, quoted by Einhorn; I did not quote them previously because they put the mortality so high. They say that of every 100 gastric ulcers carefully traced and followed to their ultimate conclusion 50 per cent. will die, either from the direct effects of the gastric ulcer, or from the complications which ensue in consequence of the ulcer. That is not quite borne out by the statistics that we have in England. I do not think that any really good attempt has been made to follow up accurately any 100 special cases; to follow them up, I mean, from the time they were treated in hospital or in private, as the case may be, to their ultimate issues in recovery or death. It would be very interesting if this could be carried out, and I

think it might be done by some of the physicians, just as it has been done by some surgeons connected with large hospitals. Certainly it could be done in regard to patients seen privately, but I have no doubt that the private cases would give infinitely better results than the hospital cases, because of the care which private patients can, and do as a rule, take in their diet. Leube's statistics, again, are very impressive; I did not quote them previously, for I preferred to base my conclusions as to medical treatment on those statistics which had been collected and published by Dr. Bulstrode at the suggestion of Mr. Mansell Moullin, and on the others collected and published by Mr. Paterson and Dr. Rhodes at the Temperance Hospital; and certainly those statistics do go very far to prove that medical treatment of gastric ulcers, as at present conducted, is by no means a successful treatment. The proportion of deaths is large, and the proportion of relapses is extremely large, so that Mr. Paterson, in his lectures, said that careful observation on a long series of cases resulted in his finding only 25 per cent. of real cures by medical treatment. The statistics of surgical treatment that have been furnished by various authorities present also the great differences in mortality of from 1 to 40 per cent. If we were to be guided by the statistics which have been furnished us by Dr. French to-night, I think we should all go away and say that gastric patients must no longer be treated surgically. Dr. Hale White has made some excuse for these statistics. I did not intend myself to touch upon them, because I felt they were too astounding to deal with—27 per cent. of deaths and 32 per cent. of failures to relieve. Now, in my opening address I drew attention to a large number of statistics from America, from France, from Germany, and from England, and I showed that those operators who had had the largest experience had brought down their mortality to under 5 per cent., some to even 1 per cent., with permanent relief in about 90 per cent. When we come to see that there is a mortality of 27 per cent. from gastro-jejunostomy in one hospital, and in another of 40 per cent. it is almost incomprehensible. These results are infinitely worse than in any series of cases treated by medical means, and, unless the institutions acknowledging such statistics can bring their rate more into a line with what has been shown to be possible, they had better stop doing gastro-enterostomy altogether, and leave gastric cases to be treated in the medical wards. I have been by this drawn out of what I intended to say, sir, but we cannot allow such remarks as have passed here this evening to go without comment, for it would not be just to the subject we are dealing with. And when I look at the methods of performing these operations—the Murphy button, the anterior operation, and obsolete methods which have been proved to be likely to lead to failure, I am not much sur-

prised. If we want to have success we must advance with the times, and we must not employ obsolete methods and expect to get good results.

We are all fairly well agreed, with regard to most of the complications, that surgical treatment is the only method of treating many of them, but we do not agree, or some of us do not, with regard to the treatment of hæmorrhage, though I think that Dr. Hale White and I agree. But I cannot altogether agree with what Mr. Paterson said. I cannot be sure about quoting the exact words, but I believe he said it could never be justifiable to operate during the course of the hæmorrhage. It happens that I had, some time ago, to operate with a former President of this Society, and, while we were waiting to do the operation on a patient who had duodenal and pyloric ulcer, he vomited a large bowl full of blood. Was I to stop because the patient was bleeding, and was apparently going to die? I thought not, and I immediately performed gastro-enterostomy. That gentleman to-day, nearly three years later, is in perfectly good health. Only a few weeks ago, since this discussion began, I saw an American surgeon who was taken ill in London. He had a severe hæmorrhage from gastric and duodenal ulcer, and I said "Wait until the hæmorrhage is over, and then I must do a gastro-enterostomy for you." He waited a week, and was moved to a nursing home, where I was to operate. During the night preceding the operation he began to vomit blood again, and next morning when I saw him the blood which he had vomited was shown me, and the bleeding was still continuing; was I to wait and accept the risk of death from hæmorrhage? I thought not. I did a gastro-enterostomy, and he has had no further bleeding, and has made an uneventful recovery. These are examples of several cases of the kind which I have had. I think that even Mr. Paterson would agree that those were justifiable cases to operate upon. But we are quite agreed, I think, that we should not operate on cases of hæmatemesis occurring for the first time while the bleeding is continuing. As a rule such an operation would be unjustifiable, but it must be borne in mind that if an operation has to be done, gastro-enterostomy is a comparatively safe operation to do, and it will accomplish all that is necessary. While I think, however, that it is a very rare event for the surgeon to be called on to operate during the course of the hæmorrhage, if it should be necessary to operate, the mortality need not be what Mr. Paterson said he thought it was, from 70 to 80 per cent.; my statistics quoted in my opening address prove it to be very much less.

With regard to the question of perforating gastric ulcer, we are all agreed that time is of the utmost importance. I think that the profession owes a great deal to Mr. Paterson for bringing forward the subject of gastro-enterostomy, or rather for

renewing that subject, for the operation was performed in perforating gastric ulcer years ago. Mr. Paterson is, however, carrying it out systematically in all his cases, and out of five there have been four recoveries. Some of the reasons he has advanced for this treatment I mentioned in my opening address. I cannot agree to go the length which Mr. Paterson does in thinking that it is advisable in all perforations, as I see some speakers have thought I did. What I said at the end of my address was "I think it is important that where the patient's condition will permit of it, the question of the performance of gastro-enterostomy should be considered," etc. I feel that this would be advantageous in most cases where there is a perforation at the pylorus, and where we know subsequent troubles will be certain to develop. Mr. Paterson's results, after he has operated upon a large number of cases, will be extremely interesting, and we shall be glad to hear further about them.

I should have liked to mention several other subjects, but the usual time for the meeting to cease has already passed.

But I must mention one thing about cancer. In my Bradshaw Lecture at the College of Surgeons I mentioned the fact—and, sir, this is a fact, however it may be translated—that in 59 per cent. of the cases of cancer of the stomach on which I had operated there had been a definite history of gastric ulcer for a long period. In the Rochester Clinic, also, in 30 per cent. of the large number of cases operated on for gastric cancer there had been a history of gastric ulcer preceding the operation for cancer. I do not think that the argument used by Dr. Hale White as to gastric ulcer being more frequently found in the female, while gastric cancer oftener occurs in the male subject, proves his point in any way, for it is not contended that acute ulcer, which is more common in women, predisposes to cancer; my argument being that chronic ulcer, which is much more prevalent in the male sex, predisposes to cancer.

The concluding paragraphs of my opening address are, I think, practically agreed on by nearly all the speakers:—"I should like it to be understood that I do not advise operation in ordinary acute gastric ulcer, the medical treatment of which should, I think, be much more careful and more prolonged than was formerly considered necessary.

"Nor do I advise operation in chronic ulcer before medical treatment has had a fair trial, but I think it is unfair to our patients to advise a continuance of general treatment for chronic or relapsing ulcer until serious complications supervene, when by timely surgical treatment many of them can be prevented.

"In perforating ulcer, in chronic or relapsing hæmorrhages, in pyloric obstruction, or other mechanical causes leading to dilatation, medical treatment is as adequate and useless, as is operative treatment in gastric neuroses and other functional diseases, and

to continue it beyond the period in which it can benefit is as unscientific and unwise as it is to operate and expect good results in improper cases."

THE PRESIDENT (Mr. WARRINGTON HAWARD)

(who was unfortunately prevented from being present at the end of the discussion, contributed the following note.)

I think all those who have listened to this discussion will agree that it has called forth a large amount of valuable information on an important subject, as well as expressions of opinion on various points of treatment by those specially qualified to speak on the matter. The frequency with which ulcer of the stomach is met with gives it a great practical interest to both physician and surgeon. One advantage of our being a medical and chirurgical society is that the physician has opportunity of hearing questions discussed from the surgical point of view, while the surgeon may similarly derive instruction from hearing the medical point of view. It is doubtless natural that physicians should have laid stress upon the importance of persevering in carefully devised medical treatment for gastric ulcer; and surgeons should remember that they do not see the cases successfully treated by the physician, inasmuch as the cases which come to them are mostly those in which medical treatment has failed. One point on which all are agreed is, that when the symptoms of perforation are decided an immediate operation is imperative, and that the shorter the time which elapses between perforation and operation the better is the chance of recovery for the patient. Moreover I have observed that even when recovery takes place the longer the time between the occurrence of perforation and the operation for its relief, the greater is the probability of subsequent complications, such as basal empyema and sub-phrenic abscess. It seemed also to be generally agreed that when possible gastro-enterostomy should be performed at the same time that the perforation is closed. It was shown by Dr. Hale White that gastric bleeding in young women often comes from other sources than an ulcer and is generally recovered from under medical treatment. But if repeated attacks of hæmorrhage are undermining the health, and medical treatment has failed, gastro-enterostomy should be performed. It is advisable that operation should, if possible, be performed in a quiescent interval. Some difference of opinion was expressed as to the relative merits of anterior and posterior gastro-enterostomy, the great experience of Mr. Mayo Robson, Mr. Moynihan, and others being in favour of the posterior operation. Evidently the point of primary importance is an

accurate diagnosis. Dr. Norman Moore and others have reminded us of some of the causes of gastric hæmorrhage besides ulceration; and Dr. Hale White pointed out some of the difficulties of diagnosis, and showed that we should be chary of operation without definite evidence of ulcer, inasmuch as experience shows that operations upon the stomach for gastric symptoms not due to ulcer are as a rule unsatisfactory. The statistics furnished by Dr. French, Mr. Gask, Mr. Hastings, Dr. Hawkins, Mr. Keyser, Mr. Moynihan, and Mr. Paterson furnish valuable information, although it must be admitted, as was pointed out, that no such statistics are free from the possibility of error. Dr. Norman Moore expressed the opinion, based on a large pathological experience, that adhesions of the stomach to neighbouring viscera are less common than is generally supposed. Operations for symptoms attributable to gastric adhesions were shown to be attended by a variable amount of success (partly, no doubt, because of the difficulty of preventing fresh adhesions forming). It appears that while slight degrees of dilatation of the stomach are often curable by medical treatment, the severer cases call for gastro-enterostomy, which is usually followed by great benefit. Accurate records of the ultimate result of treatment in cases of gastric ulcer and its complications are doubtless difficult to obtain, but sufficient evidence has been furnished in the course of this discussion to impress upon us the importance of accurate diagnosis, persevering medical treatment, and timely and judiciously selected operation. It will certainly be agreed that we are greatly indebted to Dr. Hale White and Mr. Mayo Robson for their admirable introductory addresses, and I am sure you will wish to accord to them, as well as to those other Fellows who have contributed to the discussion, the best thanks of the Society.

The CHAIRMAN (Dr. BUZZARD, Vice-President).

Gentlemen, this most interesting discussion, which has occupied the attention of the Society for three nights, has now come to an end, and there is only one duty which remains to us. I think that everyone who has either heard or read the reports of the various speeches which have been made, following the introductory addresses by Dr. Hale White and Mr. Robson, must have been struck with the unusual ability which has characterised the remarks of the speakers in this debate. We shall all agree, I think, that whether by reason of the extraordinary interest and complexity of the subject and its great gravity, or on account of the remarkable results which have been shown to have been achieved, thanks to the development of surgical methods, the discussion on this topic will probably prove something of a record one amongst the important debates of interest

which have characterised from time to time the proceedings of this Society. It remains only now for us to give a cordial vote of thanks to Dr. Hale White and Mr. Mayo Robson and the other speakers for their most interesting contributions.

The vote was then carried by acclamation, and the meeting then terminated.

SPECIAL DISCUSSION

ON

THE OPERATIVE TREATMENT OF NON-MALIGNANT ULCER OF THE STOMACH AND ITS CHIEF COMPLICATIONS, WITH INDICATIONS, LIMITATIONS, AND ULTIMATE RESULTS.

REPORT ON GASTRO-JEJUNOSTOMY CASES

Contributed by HERBERT FRENCH, M.D., Assistant Physician to Guy's Hospital.

THE two points which have interested me most are:

I. The immediate results and the subsequent effects of gastro-jejunostomy in non-malignant affections of the stomach.

II. The after-histories of patients who have recovered after operation for perforated gastric or duodenal ulcer.

I. In regard to the first point, I find that up to 1905 only *47* gastro-jejunostomies for non-malignant gastric conditions had been performed in Guy's Hospital. The following are short notes of each of these cases.

A. *Cases of Gastro-jejunostomy for Non-malignant Conditions of the Stomach, in which death resulted within a fortnight of the operation.*[1]

1. Male, aged 28, admitted in April, 1895. The history was indefinite, except that there had been no hæmatemesis, and that for seven weeks there had been repeated vomiting of large quantities of fluid food. The man was very thin. A lump was felt at the pylorus. This was thought to be carcinoma, so laparotomy was performed, and a posterior

[1] My best thanks are due to Dr. G. A. Ticehurst, who lent me part of the manuscript of his M.D.(Cambridge) Thesis, in which he gives the after-histories of many cases. His thesis, which I hope he will publish at length, deals amply with the question of gastro-jejunostomy, both in malignant and in benign cases, and he has collected not only the cases from Guy's Hospital, but also a very large number from the literature. I confine myself to consecutive hospital cases of non-malignant nature.

gastro-jejunostomy by direct suture done. The patient died six days later of exhaustion. At the autopsy there was no peritonitis; the lump was not carcinoma, but a chronic simple ulcer with much matting, and partial stenosis of the pylorus.

2. Male, aged 46, admitted in April, 1897. Two months previously he had swallowed two ounces of spirits of salts; he recovered from this, but soon after symptoms of pyloric obstruction set in. He was repeatedly sick every day. Anterior gastro-jejunostomy was performed, a Murphy's button being used; vomiting was worse after the operation than it was before, and the patient died exhausted on the fifth day.

3. Male, aged 52, admitted in January, 1898. There had been no trouble till twelve months previously, when persistent vomiting and signs of pyloric obstruction with dilated stomach set in. There was never hæmatemesis nor melæna. At the operation fibrous stenosis of the pylorus was found; posterior gastro-jejunostomy, with Halsted's method of suture, was performed. The patient did not gain strength after this, but sank and died on the thirteenth day. At the autopsy all the organs seemed healthy; there was no obvious dilatation of the stomach, no peritonitis, and no real obstruction to the pylorus.

4. Male, aged 42, admitted in September, 1899. He had a fibrous cicatrix of the pylorus. Posterior gastro-jejunostomy was performed, a Murphy's button being used. The patient died of shock within twenty-four hours.

5. Female, aged 51, admitted in September, 1900. She was found to have a cicatrising ulcer of the pylorus. Gastro-jejunostomy was performed, a Murphy's button being used. The patient died on the sixth day of general peritonitis. At the autopsy it was found that no union between stomach and jejunum had taken place; the button was free within the stomach.

6. Male, aged 43, admitted in November, 1900. He was found to have a cicatrising pyloric ulcer. Anterior gastro-jejunostomy by direct suture was performed. The patient died on the ninth day of syncope. At the autopsy it was found that the jejunum had become doubled round a peritoneal band.

7. Male, aged 67, admitted in January, 1902. He was found to have a cicatrising and leaking pyloric ulcer. Anterior gastro-jejunostomy was performed, a Murphy's button being used. There was continual vomiting after the operation, and pneumonia supervened. The patient died on the thirteenth day. At the autopsy the button was found in the duodenum.

8. Male, aged 46, admitted in June, 1902. He was found to be suffering from peri-pyloritis. Anterior gastro-jejunostomy by direct suture was performed. The patient developed broncho-pneumonia, and died on the fifth day.

9. Male, aged 42, admitted in February, 1903. He was found to have a cicatrised pyloric ulcer. Posterior gastro-jejunostomy by suture was performed. Vomiting after the operation was so extreme that a second operation was performed next day, and jejunostomy done. The patient died the day after. At the autopsy there was no peritonitis, and no obvious obstruction to the bowel.

10. Female, aged 25, was admitted in March, 1904, for dilated stomach, and apparent pyloric obstruction. At the operation the trouble was found to be due to a cicatrising duodenal ulcer. Posterior gastro-jejunostomy by means of a Murphy's button was performed. Symptoms of acute peritonitis set in on the second day, and the patient died on the third day after the operation. At the autopsy the gastro-jejunostomy was found to be all right, but the duodenal ulcer had itself perforated and caused the peritonitis.

11. Male, aged 47, was admitted in January, 1905, for traumatic cicatrix of the pylorus. Anterior gastro-jejunostomy was performed by means of a Murphy's button, and entero-enterostomy was done at the same time. The patient died of shock and collapse on the third day.

12. Female, aged 44, was admitted in November, 1902, for excessive and repeated hæmatemesis. Anterior gastro-jejunostomy was performed by means of a Murphy's button. The hæmatemesis persisted after the operation as badly as before; a second operation was performed, gastrotomy done, and search made for the bleeding point. There was general oozing from the mucosa, but no definite single bleeding point. The patient died on the sixth day exhausted and blanched from further hæmorrhage. At the autopsy the intestines were full of blood; the stomach contained no ulcer, nor could any erosions nor other abnormality be seen.

13. Male, aged 18, was admitted in January, 1905, for repeated hæmatemesis and blanching. Anterior gastro-jejunostomy by means of a Murphy's button was performed. When the stomach was opened general oozing from the gastric mucosa was seen, but no ulcer. The patient died on the second day collapsed from further hæmorrhage. At the autopsy the intestines were full of blood; there was no ulcer; on careful searching multiple minute hæmorrhagic erosions were found in the gastric mucosa.

B. *Cases of Gastro-jejunostomy for Non-malignant Conditions of the Stomach in which Recovery from the Operation occurred, but in which no Relief to the Symptoms Resulted.*

14. Male, aged 53, was admitted in October, 1895. He had been quite well till February, 1891, when he first had severe epigastric pain lasting on and off for four months. At that time he had neither hæmatemesis, melæna, nor

vomiting. In December, 1894, the pains recurred; a week later he brought up three pints of vomit containing altered blood. Since then he had had copious vomiting at intervals; as much as sixty-eight ounces were measured at a time. On November 12th, 1895, posterior gastro-jejunostomy by means of a Murphy's button was performed. The diagnosis of cicatrised pyloric ulcer was confirmed. He passed the button *per rectum* on December 6th. The vomiting persisted after the operation, occurring on November 13th, December 2nd, 6th, 12th, 13th, and 23rd in hospital, and at similar intervals since discharge.

15. Male, aged 36, was admitted in March, 1902, for symptoms which suggested perigastric adhesions. He had previously been successfully operated upon for perforated gastric ulcer. Posterior gastro-jejunostomy was performed. It is not stated whether a Murphy's button was used or not. No relief to his symptoms followed the operation.

16. Female, aged 40, was admitted in August, 1903, for the usual symptoms of pyloric obstruction. Gastro-jejunostomy was performed by means of a Murphy's button. It is not stated whether the gastro-jejunostomy was anterior or posterior. The diagnosis of cicatrising ulcer of the pylorus was confirmed at the operation. There was no relief to the symptoms even at the time, and the patient died of exhaustion six months later.

17. Female, aged 45, was admitted in March, 1905, for the usual symptoms of pyloric obstruction. The diagnosis of cicatrising gastric ulcer was confirmed at the operation. Posterior gastro-jejunostomy by means of a Murphy's button was performed. The symptoms persisted after the operation as badly as before.

18. Female, aged 40, was admitted in March, 1905, for the usual symptoms of pyloric obstruction. The diagnosis

of cicatrising gastric ulcer was confirmed at the operation. There were extensive perigastric adhesions, and great dilatation of the stomach. Posterior gastro-jejunostomy by means of a Murphy's button was performed. On discharge the dilatation of the stomach was no less than before the operation, and the symptoms persisted as before.

19. Female, aged 42, was admitted in June, 1902, for the usual symptoms of pyloric obstruction. At the operation the diagnosis of cicatrising gastric ulcer was confirmed. The stomach was moderately dilated. Posterior gastro-jejunostomy by means of a Murphy's button was performed. There was no relief to the symptoms after the operation, and the patient died five months later of exhaustion. At the autopsy the cicatrised ulcer was found to have nearly, but not quite, occluded the pylorus; *the gastro-jejunostomy opening had become completely closed by cicatrisation,* and the Murphy's button was free inside the stomach.

c. *Cases of Gastro-jejunostomy for Non-malignant Conditions of the Stomach in which Recovery from the Operation occurred, with Relief for a short time, but subsequent Recurrence of the former Symptoms as badly as before.*

20. Male, aged 47, was admitted in March, 1900, with all the symptoms of dilated stomach from pyloric obstruction. The trouble began eighteen months previously, when he first suffered from acute epigastric pains, which were worst one hour after meals, but lasted as much as six or eight hours after the last meal of the day, so that he was kept awake at night. Later he vomited copiously two or three times a week. He had had no hæmatemesis, and had noticed no melæna. At the operation a chronic duodenal ulcer was found, and a much dilated stomach. Anterior gastro-jejunostomy by means of a Murphy's

button was performed. There was great relief for a short time, but, within a month of his discharge, the symptoms were all back again. He was re-admitted in September, 1900, but declined further operative measures, and was discharged two days later *in statu quo anteâ*.

21. Male, aged 43, was admitted in March, 1903. Fifteen years previously he began to suffer from acute epigastric pains after food, relieved by vomiting. He had had neither hæmatemesis nor melæna. For one and a half years he was quite unable to work; he attended as an out-patient, and ultimately, by careful dieting and medicinal treatment, became quite well, and for thirteen years had no return of the symptoms, and was able to work hard. In 1902 vomiting and pain recurred; a gastro-jejunostomy was performed, and the diagnosis of pyloric cicatrix confirmed. Slight relief followed, but then the symptoms all recurred, and grew worse and worse. When seen in 1903, five months after the operation, he could not stand up straight on account of the pains in his abdomen, and there was a large ventral hernia of the scar, which bulged extremely on coughing. It was decided that nothing more could be done.

22. Female, aged 36, was admitted in February, 1904, for the symptoms of cicatrising ulcer of the pylorus. The diagnosis was confirmed at operation. Gastro-jejunostomy by means of a Murphy's button was performed. On discharge she seemed relieved, but shortly afterwards the old symptoms returned, and she has been re-admitted several times since for lavage of the stomach, which temporarily relieves her.

23. Female, aged 26, was admitted in February, 1905, for perigastric adhesions after a perforated gastric ulcer. It was also thought that there was some pyloric obstruction. Posterior gastro-jejunostomy was performed by means of a Murphy's button. She experienced great

relief at the time, but three months later all her symptoms had returned as badly as before.

24. Female, aged 33, was admitted in January, 1904, having never passed a week since April, 1900, without vomiting. After food she had severe pain at the umbilicus. The vomit occasionally contained blood. She was operated upon; an old fibrosed gastric ulcer was found, the pylorus not being stenosed. Posterior gastro-jejunostomy by Murphy's button was performed. The patient vomited incessantly for eleven days after the operation, and nearly died. The button was passed *per rectum* on the twenty-fourth day. On discharge she still had severe pains in the abdomen; she was kept awake at night by them, and she was sick as before.

D. *Cases of Gastro-jejunostomy for Non-malignant Conditions of the Stomach, in which great relief resulted, and in which either cure or great improvement is maintained.*

25. Male, aged 30, was admitted in December, 1901. At the operation peritoneal adhesions around the pylorus were found. Posterior gastro-jejunostomy was performed with the aid of a Murphy's button. Relief to the previous symptoms was complete, and the patient reported himself "cured" four and three quarter years later.

26. Male, aged 39, was admitted in January, 1902, for symptoms of pyloric obstruction. At the operation a cicatrised ulcer of the pylorus was found. Posterior gastro-jejunostomy was performed by suture. The patient remained "cured" for three and a quarter years, and then died of acute phthisis.

27. Female, aged 23, was admitted in December, 1902, for symptoms of gastric ulcer. The trouble began in 1898 with epigastric pains after food, beginning imme-

diately after a meal, and lasting two hours. A little later vomiting occurred repeatedly, the amount of vomit being the food just taken. The symptoms came and went. Hæmatemesis occurred once in 1899 and twice in 1902, the last attack being very severe, one month before admission. She was vomiting everything she took, so posterior gastro-jejunostomy by suture was performed. At the operation the diagnosis of pyloric cicatrix from simple ulcer was confirmed. There were many peritoneal adhesions around the pylorus, but the stomach was not dilated. On discharge one month after the operation she was able to take full diet without the least discomfort, and two and a half years later she reports herself "cured."

28. Male, aged 46, admitted in May, 1904, for pyloric obstruction and dilated stomach. The trouble dated from seventeen years previously, since when he had always been liable to acute "indigestion" pains, and occasional vomiting. He had never had hæmatemesis nor melæna. Latterly the pains had been acute and almost continuous even upon milk diet, and he had begun to vomit great quantities of fluid at intervals. He was wasted, thin, and anæmic. At the operation many adhesions were found around the pylorus; the condition of the ulcer is not stated. Posterior gastro-jejunostomy was performed by the aid of a Murphy's button which was passed on the fourteenth day. On discharge he was still very weak, and had some pains even on careful diet, but he was much better. One and a quarter years later he reports himself as "improved, but not free from pains after food."

29. Male, aged 65, was admitted in July, 1904. At the operation a cicatrised pyloric ulcer was found. Anterior gastro-jejunostomy was performed with the aid of a Murphy's button. Relief to his symptoms was immediate, and one year later he reports himself as "cured."

30. Male, aged 64, was admitted in November, 1904, for symptoms of pyloric obstruction. At the operation a chronic ulcer was found; it is not stated whether it was in the duodenum or at the pylorus. The stomach was moderately dilated. The symptoms dated from only two years previously, and consisted mainly in acute pains "in the chest" coming on from two to three hours after food. Vomiting had been slight; neither hæmatemesis nor melæna had occurred. Gastro-jejunostomy was performed by direct suture, and jejuno-jejunostomy was done at the same time. Relief was immediate, and nine months later he reports himself "much improved."

31. Male, aged 40, was admitted in January, 1905, for chronic duodenal ulcer. The diagnosis was confirmed at operation. Posterior gastro-jejunostomy was performed with the aid of a Murphy's button. Relief was immediate, and one year later he reports himself "cured."

32. Male, aged 32, was admitted in June, 1905. He had recovered from a perforated duodenal ulcer two months before, and was now admitted for pains which were thought to be due, in part at least, to adhesions. Gastro-jejunostomy was performed, and he reports six months later that he has remained quite free from the pains.

E. *The following cases complete the list of all the Gastro-jejunostomies that have been performed in Guy's Hospital up to* 1905 *for Non-malignant Conditions; but none of them have been traceable since discharge from the Hospital.*

33. Male, aged 34, was admitted in October, 1900. At the operation a cicatrised pyloric ulcer was found, with many surrounding adhesions. Gastro-jejunostomy, with the aid of a Murphy's button, was followed by great relief up to the time of his discharge.

34. Male, aged 39, had for nine years suffered from very bad epigastric pains at intervals of three or four months. He had had but slight vomiting and no hæmatemesis. Medicinal treatment and dieting had always given relief until three years ago, since when the pains had been frequent and very acute. In 1898 he was operated upon for gall-stones, but none were found. In July, 1899, he was admitted with a view to further operation. He improved so much with rest in bed, that laparotomy was postponed. Immediately on going out the pains returned. In October, 1899, he was readmitted. Partial stenosis of the pylorus from perigastric adhesions was found on operating; the pylorus was digitally dilated, after opening the stomach. Relief followed, but it was temporary only. Pains began to recur six weeks later. In June, 1900, the pains were so bad that gastro-jejunostomy was performed. Relief followed at once, but the patient has been lost sight of since.

35. Male, aged 50, was admitted in May, 1901, for the usual signs of pyloric obstruction. At the operation, a cicatrised pyloric ulcer was found. Posterior gastro-jejunostomy was performed by means of a Murphy's button. The patient recovered, with considerable relief, but has been lost sight of.

36. Male, aged 37, was admitted in May, 1901, for simple pyloric obstruction. Pyloroplasty had been performed fourteen months previously, after he had suffered from repeated abdominal pains and vomiting on and off for ten years. The symptoms rapidly recurred after the pyloroplasty, so posterior gastro-jejunostomy was performed with the aid of a Murphy's button. The patient was "cured" on discharge, but has not been traced since.

37. Male, aged 50, was admitted in December, 1901, for pyloric stenosis. At the operation posterior gastro-

jejunostomy was performed with the aid of a Murphy's button. The symptoms were "much improved" on discharge.

38. Male, aged 39, was admitted in April, 1902, for pyloric stenosis, the result of swallowing corrosive acid. Gastro-jejunostomy was performed with the aid of a Murphy's button. The patient was "cured" on discharge.

39. Male, aged 36, was admitted in September, 1902, for cicatricial stenosis of the pylorus. Posterior gastro-jejunostomy was done with a Murphy's button. He was apparently cured on discharge.

40. Male, aged 32, was admitted in December, 1902, for persistent vomiting. The cause was not found. At the operation it was noted that there was no pyloric stenosis, and no ulcer either in the stomach or in the duodenum. The stomach was moderately dilated. Posterior gastro-jejunostomy with a Murphy's button was performed. Very severe vomiting persisted for four days afterwards, but then ceased, and the patient on discharge was apparently cured.

41. Female, aged 30, was admitted in March, 1903. Two years previously she had been successfully operated upon for perforated gastric ulcer. Since then she had had three bouts of acute epigastric pain, and it was for one of these that she was now re-admitted. The ulcer, which had previously perforated, had been on the anterior surface of the stomach near the cardiac end. There was no pyloric obstruction, and the pains seemed due to the many perigastric adhesions rather than to the ulcer. Posterior gastro-jejunostomy by suture was performed; the patient was discharged free from pains.

42. Female, aged 37, was admitted in November, 1903. She began to suffer from great pain in the front of the chest and from vomiting twelve months previously. Dur-

ing all that time she had vomited almost continuously, retaining even milk with the greatest difficulty. On one occasion only was there hæmatemesis, one pint. At the operation an ulcer was found on the lesser curvature of the stomach, the base being adherent to the liver. The pylorus was free, and there was no gastrectasis. There were many recent perigastric adhesions. Posterior gastro-jejunostomy by suture was performed. On discharge the patient retained simple foods, but required to be most careful with her diet. She has not been traced.

43. Male, aged 59, was admitted in May, 1902, for cicatrising pyloric ulcer, verified by operation. Posterior gastro-jejunostomy was performed; the patient was discharged greatly relieved.

44. Female, aged 49, was admitted in January, 1902, for pyloric obstruction, which was found, at operation, to be due to a calcified retro-peritoneal cyst. Posterior gastro-jejunostomy with Murphy's button was performed. Improvement was great, and the patient was well on discharge.

45. Female, aged 50, was admitted in January, 1905, for symptoms of pyloric obstruction. Operation showed the condition to be a chronic duodenal ulcer. Posterior gastro-jejunostomy, with a Murphy's button, was performed, and on discharge the patient was well.

46. Male, aged 33, was admitted in February, 1905, for pyloric stenosis. A cicatrised pyloric ulcer was found at operation. Anterior gastro-jejunostomy by suture was performed, and the patient was quite well on discharge.

47. Female, aged 27, was admitted in March, 1905, for abdominal pains after recovery from a perforated gastric ulcer. At the operation perigastric adhesions were found; posterior gastro-jejunostomy, with a Murphy's button, was performed, and the patient was discharged better, but not quite free from pains.

In two only was gastro-jejunostomy performed for hæmatemesis, and both these patients died. In the remaining cases the trouble was almost without exception chronic gastrectasis, with the usual symptoms of pyloric obstruction from cicatrisation. I have excluded all cases of malignant disease. I have written to all the patients who recovered, and have the after-histories of thirty-two.

I am afraid the results are very much less favourable than those recorded by others. Thus thirteen, or over 27 per cent., died as the direct result of the operation within a fortnight of its performance. If we exclude the two cases in which hæmatemesis was the cause of operation, the direct mortality is still over 23 per cent. I am surprised at this. I can only give you the figures as I find them. If we analyse the causes of these deaths, we find that simple exhaustion accounted for three, continued vomiting two, shock two, peritonitis from non-union one, peritonitis from perforation of the ulcer one, pneumonia one, hæmatemesis two, kinking of small intestine by a band one. Death occurred within twenty-four hours in two cases, on the second day in two, on the third day in two, on the fifth day in two, on the sixth day in three, on the ninth day in one, on the thirteenth day in two.

I take it that this high mortality depends largely upon the fact that the patients almost all had ulcers or their results, and that their health was therefore much below par at the time of operation. I imagine that the mortality for gastro-jejunostomy performed in a series of perfectly healthy men would be extremely small. I *do* think that my statistics overstate the direct risk of the operation, especially if cases be carefully selected; but I by no means agree with the opinion that the risk of gastro-jejunostomy in cases of cicatrisation of pyloric ulcers is small.

I regret to say that I have seen personal friends of mine die from the operation when, without it, there is every reason to suppose that they would still be alive.

Even when the operation is in itself successful I am

afraid I do not find the subsequent results altogether good. Of the forty-seven consecutive cases I have been able to collect from the records of Guy's Hospital there remain thirty-four when I deduct those who died at once. Of these I have been able to trace nineteen. The remaining fifteen were discharged much relieved, and what happened to them afterwards I do not know. For the sake of argument I am going to assume that they have since remained absolutely well. Of the nineteen cases who recovered and have been traced, six got no relief to their symptoms even for a time, one dying six months after; five, though they obtained relief for a short time, had all their old symptoms back again within a few months; that is to say, the result is known to have been a failure in 32 per cent. of the cases who recovered from the operation. In eight only is the result known to have remained good. In the rest the ultimate result is not known, but the 32 per cent. of failures is based on the presumption that the untraced cases all did well.

The net result of these statistics is, therefore, that *all* the cases died in whom gastro-jejunostomy was performed for hæmorrhage; that over 23 per cent. of the other cases died as the result of the operation; and that, at the very least, 32 per cent. of the cases who recovered got no lasting relief from their operation.

I am bound to admit that these figures are far more serious than I had expected. I think this is no doubt partly due to the cases having been operated upon by various surgeons—all good, but some with less experience of gastro-jejunostomies than others. I think it is also partly due to some of the cases having already suffered from ill-health and malnutrition for a very long time previous to the operation. If one could pick and choose one's cases, and operate only on the most favourable, the results would be far better. There are cases in whom I should have little fear as to the result of the operation. Nevertheless, I do think that, taking the cases all round, the performance of gastro-jejunostomy does carry with it a

very real risk to the patient's life; I do think it is an operation which should not be decided upon with any sort of haste. I have a sort of feeling that it is sometimes done for conditions which do not really require it, and such cases would naturally swell the statistics of apparently good results. When it is done for the purpose of making a new passage for the food onwards from the stomach when the proper path through the pylorus is interfered with by an ulcer, by a scar, or by adhesions, I think one must not give one's patient's friends too bright a picture of the results to be expected, or else in a great many cases there is sure to be disappointment.

(II) In regard to the second point, I find that up to 1905 altogether thirty patients in Guy's Hospital recovered from perforation of a gastric or duodenal ulcer. I have written to them all, have traced eighteen of them, and am able to arrange them into two very clear groups, namely:

(*a*) Those who have been quite free from gastric symptoms since their operation, and have been able to follow their laborious occupations as labourers, domestic servants, and so on without any difficulty at all.

(*b*) Those who have suffered from dragging pains and other symptoms, more or less severe, which render them quite unequal to their fellows in working power.

(*a*) Those who did Well Afterwards.

Case 1.—William P—, aged 43. He was admitted on July 30th, 1898, and was operated upon three hours after the perforation took place. The hole was upon the anterior surface of the stomach, near the cardiac end of the stomach. It was sutured, and the patient went out thirty-two days later. He had had lassitude, gastric pain after food, and nausea for two and a half years previously, but never hæmatemesis. He had seen many consultants. The condition had been repeatedly diagnosed as nervous dyspepsia, for which the patient had twice been sent for long voyages. In October, 1906, he came

to show himself; he was strong and well, actively engaged in business, and had had no gastric symptoms whatever since his operation. The scar was from the ensiform cartilage to the pubes, narrow and strong.

Case 2.—Nelly R—, aged 26. She was admitted on March 8th, 1900, and was operated upon seven hours after the perforation occurred. The hole was upon the anterior surface of the stomach, close to the pylorus. It was sutured; the abdomen was washed out and completely closed. The patient went out forty-three days later. Previous to the perforation she had had "indigestion" for two years on and off, with pains soon after eating, and occasional vomiting, but never hæmatemesis. In October, 1906, she writes: ". . . I am now married and have been two and a half years out of London. I am most pleased to tell you that I have had splendid health, with no return of my old complaint whatever. Not only have I been free from pain, but I am quite able to eat anything in the way of fruit and vegetables, which I could never do before."

Case 3.—M—. He was operated upon for perforated gastric ulcer in 1900. The ulcer was simply sutured. The patient recovered well, and had had no gastric symptoms since. In October, 1906, he was in good health, and busily engaged as a bank clerk.

Case 4.—William S—, aged 17. He was admitted on December 28th, 1901, and was operated upon four and a half hours after the perforation. The latter was in the duodenum. The ulcer was excised and the duodenum sutured. There had been no previous symptoms, the patient stating that he had not been ill in any way since he was a child. After the operation there was much local suppuration, and the wound took a long while to heal. There was still a sinus on discharge ninety-one days after the operation. In October, 1906, he presented

himself for examination. He had returned to his heavy work as a waterside labourer one month after he left the hospital, and had continued at it ever since, without a bad symptom of any kind. When seen, he was a big, strong, healthy workman, with an abdominal scar 10 inches long and 2½ inches wide in the middle line. The scar, though it looked thin and weak, showed no sign of developing into a ventral hernia, though the man wore no belt even at his heavy work.

Case 5.—John R—, aged 25. He was admitted on July 4th, 1903, and was operated upon five hours after the perforation. The latter was in the anterior surface of the stomach, near the pylorus; it was simply sutured. The patient went out twenty-six days after the operation. Previously there had been epigastric pain after food for twelve months, with flatulence but neither vomiting nor hæmatemesis. In October, 1906, he writes that he is actively engaged as a stevedore, and that he is perfectly well.

Case 6.—Annie E—, aged 21. She was admitted on March 12th, 1903, and was operated upon three hours after the perforation. The latter was upon the posterior surface of the stomach, near the pylorus. It was simply sutured, and the patient went out thirty-six days later. There had been no gastric symptoms previously and no melæna. She writes in October, 1906, that "I am very glad to be able to say that I am still continuing in the best of health." She works as a ward maid.

Case 7.—Robert C—, aged 43. He was admitted for perforated gastric ulcer on September 4th, 1904. In October, 1906, he writes that "While thanking you for your letter, I wish to say that I am very well; I have only had an occasional slight pain, which perhaps may not be due to the complaint." He works as a stoker.

Case 8.—Isaac L—, aged 35. He was admitted on March

30th, 1905, over twenty-four hours after perforation. At the operation two perforations were found, both in the stomach; one was a small hole in a big ulcer on the anterior surface near the pylorus; the other was near the cardiac end. Both were simply sutured. The patient went out thirty-five days afterwards. He was known to have had a gastric ulcer for a long time. He had had many severe attacks of hæmatemesis from 1897 onwards, but had always been able to work hard as a labourer between the attacks. In October, 1906, he writes that he is at work, and that "I am glad to say that I am getting on nicely and have had no pain since my operation."

Case 9.—Beatrice L—, aged 28. She was admitted on January 22nd, 1905, twenty-six hours after the perforation. The latter was in the stomach, near the pylorus, on the anterior surface towards the lesser curvature. There were many hindrances to her recovery. She developed pleurisy with broncho-pneumonia soon after the operation; the pleurisy led to effusion, and paracentesis thoracis was performed. After this she developed thrombosis of veins in her left arm and right leg, and an abscess in her right breast, which had to be opened and drained. Notwithstanding this she went out 127 days after admission. In October, 1906, she writes: "I am pleased to be able to tell you I have been in the best of health since my discharge from the hospital." She works as a domestic servant. Previous to her admission she had suffered for three years from epigastric pains, which were worst half an hour after food, and she had had severe hæmatemesis twice.

Case 10.—William G—, aged 30. He was admitted on January 9th, 1905, fifty hours after the onset of symptoms of perforation. He had had "indigestion," but neither vomiting nor hæmatemesis, ever since his return from the South African War eighteen months before. The per-

forated ulcer was on the anterior wall of the stomach, near, but not at, the pylorus. The hole was sutured and the patient left the hospital sixty-one days later. He wrote in October, 1906: "My general health has been good since the operation; very occasionally I still have a little dyspepsia, which I suppose was the cause of my complaint, and which I put down to being compelled to lead an indoor life." He works as an indoor porter.

Case 11.—William E—, aged 26. He was admitted on February 17th, 1905, and was operated upon six hours after the perforation. The latter was on the anterior surface of the stomach, near, but not at, the pylorus. It was simply sutured, and the patient left the hospital twenty-seven days later. Previously he had had neither vomiting nor hæmatemesis, but for three months had complained of "some discomfort" in the epigastrium about half an hour to one hour after meals. In October, 1906, he presented himself for examination; the scar, which was from the epigastric angle to the umbilicus, was strong and narrow; the man had been hard at work as a labourer since his discharge; he was looking as well as possible, and had no bad symptoms of any sort.

Case 12.—Charles P—, aged 48. He was admitted on April 16th, 1904, for the usual symptoms, and was operated upon within a few hours of the onset of perforation. The hole was in the duodenum, one inch beyond the pylorus; it was simply sutured, and the patient went out thirty-nine days afterwards. Previously he had had "indigestion" badly for six years, with intervals of perfect health for four or five months at a time. There had been neither hæmatemesis nor melæna. In October, 1906, he writes: "I beg to state that my health is, and has been, very favourable indeed."

Case 13.—William N—, aged 48. He was admitted on June 7th, 1899, and was operated upon within three hours of

the onset of acute symptoms. The perforation was in the anterior wall of the stomach, near the pylorus. It was sutured. The patient went out fifty days later. He had suffered severely from "dyspepsia," which had started at a definite time seven years before; there had been epigastric pains, which had become worse during the two and a half years preceding the perforation. There had been neither hæmatemesis nor melæna. He remained free from gastric symptoms for four years after the operation, and then he died of apoplexy.

CASE 14.—Caroline F—, aged 22. This patient had twice suffered from, and survived, perforated gastric ulcer. The first time was in March, 1900; she was operated upon within twelve hours of the perforation, the ulcer being excised and the stomach sutured. She remained well until the end of 1904, when she had another sudden perforation, which was sutured. Since then she writes that she has remained in excellent health (October, 1906), working as a domestic servant. It is noteworthy that the second perforation occurred notwithstanding excision of the first ulcer. Previous to her first admission she had for two years had "indigestion pains" after food, with very occasional vomiting, but no hæmatemesis or melæna.

CASE 15.—Robert B—, aged 22. He was admitted on July 16th, 1904, and was operated upon within a very few hours of perforation. The hole was upon the anterior surface of the stomach, some way from the pylorus. It was sutured; the patient went out thirty-two days later. Previous to admission he had had gastric pains for two weeks only; he had hæmatemesis the day after the operation, but never before or since. In October, 1906, he came for examination. He was strong and well, and actively engaged as an engineer; his work took him to all parts of England and Scotland, and he had not been a day away from it since his recovery.

(*b*) PATIENTS WHO SUFFERED FROM MORE OR LESS SEVERE AFTER-EFFECTS.

CASE 14.—Alice B—, aged 36. She was admitted on October 28th, 1901, and was operated upon thirteen hours after the perforation occurred. The ulcer was upon the middle of the anterior surface of the stomach, half-way between the lesser and greater curvatures, and half-way between the cardiac and pyloric orifices. The hole was sutured. The patient went out sixty-four days later. She remained well for a short time, but then began to suffer from severe dragging abdominal pains. In April, 1904, she was operated upon again; numerous adhesions were found; many of these were divided. The pains, however, persisted as before, and still do.

CASE 17.—Sophia G—, aged 21. She was admitted in March, 1894, for the usual symptoms of perforated gastric ulcer. She had had no previous stomach symptoms. She was operated upon within a few hours of being taken ill. The ulcer was excised and the stomach sutured. She suffered from abdominal discomfort and pains in the abdomen during the next eighteen months, and in September, 1895, she was readmitted, with typical symptoms of acute intestinal obstruction. An operation was performed; the obstruction was due to the kinking of a loop of small intestine which was adherent to the abdominal wall at the seat of the old scar. The patient died of pneumonia seven days afterwards.

CASE 18.—Frederick W—, aged 34. He was admitted on February 6th, 1901, and was operated upon eight hours after the symptoms of perforation began. He had previously had "indigestion" for some months, but no vomiting, hæmatemesis, nor melæna. At the operation the ulcer, which had perforated, was found on the anterior surface of the stomach near the cardiac end; it was found

impossible to suture the hole; the stomach was plicated so as to form a trough or channel for drainage from the perforation to the abdominal wound; a local abscess developed, and a sinus persisted for a long time. It was obvious at the time that many adhesions must result and much deformity of the stomach. The man, a big, burly market porter, has been almost incapacitated for work since. He has repeated recurrences of agonising abdominal pains. An operation for division of some of the adhesions was performed in November, 1901, and a gastro-jejunostomy a little later. No relief followed even the latter. The pains are apparently due to dragging upon extensive adhesions, and not to the persistence of

The following is a tabular summary of the eighteen cases:

No.	Sex.	Age.			
1	M.	43	Perforated gastric ulcer	Suture	Quite well 8¼ years after.
2	F.	26	„ „ „	„	„ „ 6½ „ „
3	M.	—	„ „ „	„	„ „ 6 „ „
4	M.	17	„ duodenal „	Excision and suture	„ „ 4¾ „ „
5	M.	25	„ gastric „	Suture	„ „ 3¼ „ „
6	F.	21	„ „ „	„	„ „ 3½ „ „
7	M.	43	„ „ „	„	„ „ 2 „ „
8	M.	35	Two simultaneous perforated gastric ulcers	„	„ „ 1½ „ „
9	F.	28	Perforated gastric ulcer; complications	„	„ „ 1¾ „ „
10	M.	30	Perforated gastric ulcer	„	„ „ 1¾ „ „
11	M.	26	„ „ „	„	„ „ 1¾ „ „
12	M.	48	„ duodenal „	„	„ „ 1½ „ „
13	M.	48	Perforated gastric ulcer; ultimately died of apoplexy	„	„ „ 2¼ „ „
14	F.	22	Perforated gastric ulcer second time	Excision and suture	„ „ 1¾ „ „
15	M.	22	Perforated gastric ulcer	Suture	„ „ 2¼ „ „
16	F.	36	„ „ „	„	Bad adhesions; operation for division of these; has bad pains.
17	F.	21	„ „ „	Excision and suture	Intestinal obstruction by kinking of bowel eighteen months after; death.
18	M.	34	„ „ „	Drained without suture	Bad pains, not relieved by division of adhesions nor by gastro-jejunostomy.

an ulcer nor to any pyloric stenosis or dilatation of the stomach.

It seems needless to give the incomplete notes of the twelve cases that have not been subsequently traced.

Of the eighteen cases of perforated gastric or duodenal ulcer which recovered after operation and which have been subsequently followed up for periods varying from under two years to over eight years, fifteen, or 83 per cent., have done absolutely well, without recurrence of any gastric symptoms, whilst only three, or 17 per cent., have done badly.

These figures are, of course, for a comparatively small number of cases ; but they are consecutive, and not in any way selected ; they are very much more favourable than I had expected to find them.

In not one of the cases was gastro-jejunostomy performed at the time of the suture of the perforated ulcer. I think the argument against there being any indication for gastro-jejunostomy in all cases at the time of suturing the ulcer is strong, seeing that 83 per cent. of the cases have since had no gastric symptoms at all, but have been able to carry on their laborious occupations uninterruptedly ; whilst in the 17 per cent. of cases who had subsequent abdominal symptoms the troubles were due to peritoneal adhesions, which were a direct result of the general peritonitis, and which, it seems to me, would be equally liable to form whether gastro-jejunostomy were done or not.

SPECIAL DISCUSSION

ON

THE OPERATIVE TREATMENT OF NON-MALIGNANT ULCER OF THE STOMACH AND ITS CHIEF COMPLICATIONS, WITH INDICATIONS, LIMITATIONS, AND ULTIMATE RESULTS.

REPORT ON THE IMMEDIATE RESULTS OF OPERATION FOR GASTRIC ULCER AND ITS COMPLICATIONS AT THE MIDDLESEX HOSPITAL.

Contributed by SOMERVILLE HASTINGS, M.B., B.S., F.R.C.S., Surgical Registrar of the Middlesex Hospital.

During the last six years (1900 to 1905 inclusive) 69 operations for gastric ulcer and its complications have been performed at the Middlesex Hospital. In 30 the patients were males, and in 39 females. Fifty-six recovered from the operation and left the hospital relieved, and thirteen died, a mortality of 18 per cent. Excluding the 13 cases of perforated gastric ulcer, the mortality is reduced to 12 per cent.

In the following tables, by means of the symptoms and conditions found at operation, an attempt has been made to classify the cases into those of simple ulcer and its various complications. Where, as occurred in not a few cases, several complications were present, the case has been placed under the heading of the one which appeared to be mainly responsible for the symptoms.

(1) Cases where the Chief Symptoms appeared to be produced by the Ulcer Itself.

Twenty-seven cases seem best placed under this heading, but in one of these exploratory laparotomy was performed, and, no indication of ulceration being seen on the peritoneal surface of the stomach, nothing further was done. In a second, where posterior gastro-enterostomy was performed, no pathological condition was noticed at operation, and the subsequent history of the patient seems to indicate that the symptoms complained of were mainly functional. Ten of the 27 cases were in males, and 17 in females. Twenty-six recovered, and 1 died, a mortality of 3·8 per cent. In 25 cases posterior gastro-enterostomy was performed, with one death from general peritonitis two days after operation. No cause could be found for this condition, for the stitches uniting the viscera had held, and the site of operation looked well. In another case some adhesions were divided and the ulcer invaginated, and another, as mentioned above, was only explored.

(2) Perforated Gastric Ulcer.

There were 13 cases of perforation, 3 in males and 10 in females, and in all cases the perforation was sutured; but in 6 the result was fatal, a mortality of 46 per cent. Three of the fatal cases died from peritonitis or shock within two days of operation, but the other 3 lingered for 43, 53, and 54 days, and were finally carried off by subphrenic abscess.

It is interesting to note that in the six cases of perforated duodenal ulcer, which were operated on during the same period, only one death occurred, a mortality of only 16 per cent.

(3) Pyloric Stenosis or Gastric Dilatation.

There were 20 cases where one or both of the above conditions was present. Thirteen were in males and 7 in females; 16 recovered and 4 died, a mortality of 20 per

cent. In 14 of these cases a posterior gastro-enterostomy was performed, with a single fatal case, an old woman of 65, who died of anorexia and exhaustion 20 days after operation. There was no post-mortem examination. In two cases anterior gastro-enterostomy was performed, with one death from continued vomiting 7 days after operation. At the post-mortem signs of general peritonitis were present. A case of pyloroplasty and another of dilatation of the pylorus after gastrotomy were successful. A case of constricted and adherent pylorus where pylorectomy was performed died a few hours after operation from collapse.

In another case adhesions were numerous and firm, and in dividing these a diverticulum from the stomach was opened. This was sutured, but the patient died from shock a few minutes after the completion of the operation.

(4) Gastric Hæmorrhage from Ulcer.

There were 3 cases where operation was undertaken mainly for severe hæmorrhage; 2 of these were females. In all 3 posterior gastro-enterostomy was performed, and in the one fatal case death occurred from continued hæmorrhage on the day following operation.

(5) Hour-glass Stomach.

In 2 cases this condition was found at operation. Both occurred in females. In one posterior gastro-enterostomy and gastro-gastrostomy was successful. In the other, where posterior gastro-enterostomy only was performed, the patient died the same day from collapse, and post-mortem a definite sub-hepatic space was found communicating with the stomach, evidently the result of a perforated ulcer.

(6) Perigastric Adhesions.

Of the 4 cases 3 were in men, and all did well. In one, after dividing the adhesions the ulcer was invagin-

ated. In another adhesions were broken down, and in the remaining 2 posterior gastro-enterostomy was performed.

Summary of Results of Gastro-enterostomies.

Forty-eight gastro-enterostomies for gastric ulcer and its complications were performed during the six years, in 1900 only 1, in 1905, 16. Forty-three of these cases left the hospital relieved, and 5 died; a mortality of 10 per cent. The anterior operation was only twice performed, and one of these cases died of general peritonitis. Posterior gastro-enterostomy was performed 46 times with 4 deaths, or a mortality of 8·7 per cent. The causes of death were peritonitis in one case, collapse resulting in death a few hours after operation in a second, continued hæmorrhage in a third, and anorexia and exhaustion ending fatally 20 days after operation in a fourth.

In conclusion, I should like to thank the Surgical Staff of the Middlesex Hospital for permission to publish the above statistics of their cases.

SPECIAL DISCUSSION

ON

THE OPERATIVE TREATMENT OF NON-MALIGNANT ULCER OF THE STOMACH AND ITS CHIEF COMPLICATIONS, WITH INDICATIONS, LIMITATIONS, AND ULTIMATE RESULTS.

REPORT ON 556 CASES OF GASTRIC ULCER AND ITS CONSEQUENCES

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Number and Sex of Patients.

A review of 556 cases of gastric ulcer and its sequelæ admitted consecutively into St. Thomas's Hospital, of which 443 were females and 113 males.

Grounds for Diagnosis.

The evidence on which a diagnosis is based in these cases consists of:

(1) The presence of cardinal symptoms, viz. pain produced by food, vomiting giving relief to pain, and local tenderness.

(2) The fact that 405 of these patients had at one time or another hæmatemesis or melæna or both.

(3) The fact that in nearly every case which came to

operation or to a post-mortem an ulcer or the consequence of an ulcer was found. Thus of 74 cases which were submitted to operation and recovered, an ulcer or its consequence was found in 65, while in 9 the existence of an ulcer past or present was neither proved nor disproved. And of 64 cases which were examined post mortem 63 showed an ulcer and 1 showed a minute erosion (death from hæmorrhage).

So that the probability of a correct diagnosis is very high. At the same time, the following three cases show that error is possible. All three cases would pass as gastric ulcer :

(*a*) Female, aged 44. Suffered fifteen years ago from pain after food and vomiting, in an illness which lasted six months. No hæmatemesis. Three years ago a similar illness of shorter duration. No other illness, but for a few weeks before death she had been getting thinner. One evening she fainted ; next day she fainted again and brought up a large quantity of bright red blood ; for the next six days she was frequently sick, vomiting small quantities of red blood, and she passed numerous stools consisting of altered blood. Death on seventh day. Post mortem no breach of surface could be found in stomach or intestine, but there were patches of injected vessels in the stomach.

(*b*) Female, aged 22, had suffered from gastric pain, vomiting, and occasional hæmatemesis for four years. Anterior gastro-enterostomy was performed. Six days later lobar pneumonia supervened, followed by signs of gangrene and excavation of lung and death on nineteenth day after operation. Post mortem careful examination after washing for twenty-four hours showed no sign of disease of stomach or intestine. Suture sound. Abdomen normal. Gangrene of lung and minute recent vegetations on mitral valve.

(*c*) Female, aged 20, had suffered from gastric pain, vomiting, and constipation for two and a half years. Never hæmatemesis. More or less under medical treat-

ment for two years without relief. Posterior gastro-enterostomy was performed. Death five days later. Post mortem—general purulent peritonitis. Suture sound. No sign of disease, past or present, in stomach, intestine, or elsewhere.

Classification.

		Total.
(A)	Simple ulcer—females 354, males 65	419
(B)	Perforated ulcer: (*a*) general infection—females 53, males 24	77
	(*b*) local infection—females 9, males 6	15
(C)	Sequelæ: (*a*) adhesions—females 9, males 6	15
	(*b*) pyloric stenosis — females 16, males 12	28
	(*c*) hour-glass constriction—females	2

Mortality.

Total mortality is 13·3 per cent. (74 deaths in 556 cases).

	Per cent.
Mortality from simple ulcer	0·9
„ from perforated ulcer—general infection	8·5
„ „ local infection	1·9
„ from sequelæ	1·9

(A) Simple Ulcer.

Recovery Rate.

Of 419 cases 5 died, giving a mortality rate of 1·2 per cent. This apparent cure in nearly 99·0 per cent. is fallacious; for (1) it is clear that about 10 per cent. of these cases are re-admitted subsequently on one or more occasions on account of recurrence of symptoms; (2) it is probable that a further number are treated on a subsequent occasion in other institutions or at their homes;

(3) during the period in which these 419 cases were admitted into hospital 75 other patients were admitted, representing the other side of the picture and suffering from perforation of a chronic ulcer or the results of long-standing ulceration such as gastric adhesions and pyloric stenosis, with long histories of gastric disease often amounting to many years. The aim of treatment is to abolish this class of chronic ulcer.

Condition of Patient on Discharge.

Exact state of the stomach in these cases at the time of their discharge from hospital as cured is unknown. As a rough estimate the average stay of a patient in hospital under routine medical treatment is probably not more than four weeks, and it is open to question how many of them are discharged with an ulcer incompletely healed. No doubt a recently formed ulcer in a patient who is under care from the first (as in cases of initial hæmatemesis) may heal quickly. From one case (Case 6) information can be gained as to the rate of healing when partial rest to the stomach is afforded by an efficient gastro-enterostomy.

Analysis of Fatal Cases.

Of 5 deaths, 4 were due to hæmorrhage and 1 to exhaustion.

(1) Case 1 in table, male, aged 25. Gastrotomy for hæmorrhage.

(2) Female, aged 58. Chronic ulcer in hour-glass stomach. Fatal hæmorrhage, not from ulcer, but from several minute erosions.

(3) Female, aged 35. No fully formed ulcer. Fatal hæmorrhage from a small superficial erosion near cardiac end, with small submucous vessel exposed.

(4) Female, aged 43. Gastric symptoms for fifteen

years. Large ulcer, so extensive that its floor was formed in front by liver, and behind by pancreas. Fatal hæmorrhage from gastric artery.

(5) Female. Gastric symptoms for thirty years, chiefly severe pain, never hæmatemesis. Death from exhaustion. Chronic ulcer round lesser curvature on anterior and posterior surfaces, part of its floor being pancreas with thrombosed arteries exposed. No hæmorrhage.

Remarkably low death-rate from hæmorrhage, being less than 1·0 per cent. of 419 cases of simple ulcer, and 0·7 per cent. of 556 cases of simple ulcer, perforated ulcer, and their sequelæ.

Surgery Employed.

(1) In one case exploratory cœliotomy; nothing done.

(2) In two cases gastrotomy—(i) nothing found, nothing done; (ii) for hæmorrhage, fatal, Case 1 in table.

(3) In five cases gastro-enterostomy, Cases 2 to 6. The results in these 5 cases are: slight improvement, very little benefit, in 2 cases; very great benefit in 3 cases. In one of these the ulcer was excised and sutured at the same time as the gastro-enterostomy (Case 5).

(4) In one case previous gastrotomy in another hospital, nothing found.

Table of Operations for Simple Ulcer.

Case.	Sex.	Age.	Previous history.	State on admission.	Operation.	Recovery or death.	After-history.	Anatomy.
1	M.	25	Hæmatemesis and melæna 7 years and 1 year ago. Last 2 months pain and vomiting with frequent hæmatemesis	Extremely pale and emaciated with dilated stomach	Gastrotomy Stomach contained much dark blood. Ulcer found within pylorus, bleeding vessel size of a digital artery in its floor. Difficult to secure artery; suprarenal extract applied; final ligature. Infusion	D.	—	Little early peritonitis about pylorus. Sutures at pyloric end of incision had cut through. The incision had been made through a chronic ulcer. Large chronic ulcer in duodenum producing some stenosis.
2	F.	20	Indigestion 2 years. Hæmatemesis 6 times, severe just before admission	Blanched from hæmorrhage	Anterior gastro-enterostomy with continuous silk suture	R.	No great benefit, vomiting continued but no hæmatemesis. Not traced after 4 months	—
3	F.	29	Pain, hæmatemesis 10 years ago. Two severe hæmorrhages 3 weeks ago	Gastric tenderness	Anterior gastro-enterostomy with continuous silk suture. Copious vomiting at once for 2 days. Wound reopened, jejunum found kinked on distal side of anastomosis. Lateral entero-enterostomy with relief of vomiting	R.	No permanent benefit. Pain, vomiting, and occasional hæmatemesis continued	No dilatation of stomach, no ulcer felt.

Case.	Sex.	Age.	Previous history.	State on admission.	Operation.	Recovery or death.	After-history.	Anatomy.
4	F.	21	Symptoms of gastric ulcer off and on for 5 years. Hæmatemesis recently	Gastric tenderness	Anterior gastro-enterostomy with continuous silk suture	R.	Nine months later much better, still occasional discomfort, but can eat any food, not sick, has gained weight	Ulcer felt near pylorus.
5	M	31	Symptoms of gastric ulcer for 8 years. Hæmatemesis 7 months ago	No physical signs	Posterior gastro-enterostomy with continuous silk suture. Ulcer excised and sutured	R.	One year later can eat any food, but has to be careful, slight discomfort but not sick	—
6	M.	42	Vomiting with frequent hæmatemesis for 6 years. Vomited blood 5 times after admission, last time day before operation	Stomach slightly dilated. Nothing felt. Blanched from hæmorrhage	Posterior "no loop" gastro-enterostomy with continuous silk suture. Pylorus adherent. An ulcer found on anterior wall adherent to liver	R.	No more pain, vomiting, or hæmatemesis after operation, could eat ordinary food. Died 2 months later from general tubercle of lungs	P.M.—Ulcer found at operation now healed; also healed ulcer in pylorus. Anastomosis large and patent. Tubercle of lungs.

(B) Perforated Ulcer.

(*a*) *With General Peritoneal Infection.*

Number, mortality.—Seventy-seven cases of general peritonitis from perforation of gastric ulcer were admitted: females 53, males 24. Of these, 47 died and 30 recovered, giving a mortality rate from this cause of 8·5 per cent. (47 deaths in 556 cases). Of these cases of general peritonitis the mortality rate is 61·0 per cent. (47 deaths in 77 cases).

Operations.—Of these 77 cases operation was performed in 67 (ten patients being moribund on admission), with 37 deaths and 30 recoveries, giving a mortality rate in cases of operation of 55·0 per cent. In the last five years the mortality rate has been considerably reduced, being 43·0 per cent. (26 recoveries and 23 deaths).

Time of operating.—Average time between perforation and operation in fatal cases was twenty-five hours, and in non-fatal cases sixteen. In one fatal case suture was carried out four days after perforation; in another case, also fatal, three hours after perforation. Of cases that recovered the extreme limit of interval between perforation and operation was sixty hours.

Position of Ulcer.

Anterior surface	55
Posterior surface	6
Lesser curvature	5
Anterior and posterior surface . .	3
Within pylorus	1

In 15 of these cases there were two ulcers present, and in 3 of these both ulcers had perforated. In one case there was an ulcer also in the duodenum.

Summary of Table of Operations.

(1) *Methods.*—In nearly all cases the perforated ulcer was invaginated and sutured; in 5 cases it was excised. As a rule the pelvis was cleansed and drained by a supra-

pubic incision. In 6 cases the site of the ulcer was covered with an omental graft. In 2 cases (Cases 13, 19) suture was found to be impossible, in one of these the cause being friability of stomach wall.

(2) *Preliminary exploration.*—In 4 cases the abdomen was opened in the first place by incision below the umbilicus for examination of appendix and tubes. In one of these (Case 31) an inflamed appendix with three concretions were removed, and the perforated ulcer sutured subsequently with recovery. In one case (Case 28) a perforated stomach was thought, after examination, to be sound, and the appendix and right Fallopian tube were removed.

(3) *Gastro-enterostomy* was performed in 4 cases; in 3 cases (Cases 36, 39, and 66) four months, and one month, and one year after suture of the ulcer, and in 2 cases (Cases 70, 71) at the time that the ulcer was sutured.

(4) *Complications.*—In 6 cases (Cases 11, 16, 37, 47, 48, 55) various complications ensued, viz. parotitis, thrombosis, pelvic abscess, right and left subphrenic abscess, and intestinal obstruction by adhesions.

(5) *Perforation of second ulcer.*—In 1 case (Case 25) perforation of a second ulcer probably occurred after suture of the first and was the cause of death, in 2 other cases (Cases 42, 49) of two ulcers perforated, only one was sutured. In 1 case (Case 17) the patient died from perforation of an ulcer one year after perforation and suture of a first ulcer.

(6) *Peptic ulcer of jejunum.*—In Case 39 after gastro-enterostomy a peptic ulcer formed, perforated, and was sutured successfully on two occasions with a year's interval. In Case 56 a perforated peptic ulcer was sutured successfully after gastro-enterostomy two years before in another hospital.

(7) *Pneumonia.*—In 4 cases (Cases 38, 43, 71, 72) pneumonia occurred after operation for perforated ulcer, and in 3 of these it was probably the only cause of death.

(8) *Death under anæsthetic.*—In two cases (Cases 44, 63).

Table of Operations for Perforated Ulcer.

Case.	Sex.	Age.	Previous history.	Operation.	Recovery or death.	After-history.	Anatomy.
7	F.	32	Hæmatemesis 2 years ago; gastric symptoms since	Cœliotomy 3 days after perforation; nothing done	D.	—	Leakage from chronic ulcer adherent to liver; another ulcer present and scars of others.
8	F.	22	Scarcely any previous symptoms; pain for 3 days	Operation within few hours. Suture and omental graft	D.	...	General peritonitis, perforation of recent anterior ulcer; suture sound.
9	F.	24	Not recorded	Operation in 3 hours. Suture and lavage	D.	—	General peritonitis, perforated ulcer on lesser curvature, stomach hour-glass, tubercle of lungs; suture inefficient.
10	F.	17	Gastric symptoms for 2 years; never hæmatemesis	Operation in 26 hours. Edges scraped, ulcer sutured, lavage, wound closed	R.	Re-admitted 1 year later with pain and vomiting. Treated medically	Leakage from ulcer adherent to liver.
11	F.	21	Indigestion some months; never hæmatemesis	Operation in 60 hours. Invaginated and sutured, lavage, drainage, supra-pubic incision and drainage. On 3rd day double parotitis; on 16th day pelvic abscess burst into rectum; on 20th day thrombosis in left leg	R.	—	Perforated ulcer near entrance of œsophagus with much induration around.
12	M.	16	Not recorded	Operation in 48 hours. Scraped and sutured, lavage and supra-pubic drainage	D.	—	General peritonitis, ulcer 3 in. from pylorus, another ulcer present; suture sound.
13	F.	22	Indigestion for 6 months; never hæmatemesis	Operation on 4th day. Suture impossible owing to friability of wall; tube inserted, lavage, and supra-pubic drainage	D.	Died in a few hours	No p. m.; perforated ulcer on anterior wall surrounded by adhesions.
14	F.	28	Pain and vomiting off and on for years	Operation in 24 hours. Suture and dry sponging	D.	—	Perforated chronic ulcer of pylorus and duodenum, general peritonitis; suture sound.

15	F.	28	Pain and vomiting for 5 mths.; never hæmatemesis	Operation in 10 hours. Edge of ulcer freshened, suture, lavage, supra-pubic drainage	D.	—	General peritonitis, ulcer on anterior surface near pylorus; suture sound.
16	F.	23	Indigestion 1 year	Operation in 24 hours. Ulcer excised, drainage of pus between liver and diaphragm. Fortnight later transpleural drainage of perisplenic abscess. Month later re-drainage of this with counter-opening. Week later supra-pubic drainage of pelvic abscess	D.	Died on 40th day	General peritonitis with localised collection of pus; ulcer on anterior surface adherent to liver.
17	F.	23	Symptoms for 8 years; hæmatemesis	Operation in 12 hours. Suture, dry sponging, supra-pubic incision and drainage	R.	Died 1 year later from perforation of a second ulcer. P. m. but no operation	Ulcer on anterior surface 3 in. from pylorus.
18	M.	47	Pain for many years; occasional hæmatemesis	Operation in 12 hours. Suture, lavage, supra-pubic incision and drainage. Infusion	D.	—	General peritonitis, dilated stomach adherent to liver and pancreas, large chronic ulcer on middle of lesser curvature.
19	M.	33	Long history of gastric symptoms; hæmatemesis twice	Operation in 14 hours; infusion beforehand. Suture impossible, drainage, supra-pubic incision and drainage	D.	—	General peritonitis; two ulcers anterior and posterior, latter fixed to pancreas and perforated; cirrhosis of liver.
20	M.	56	No symptoms till last 2 weeks; then pain, hæmatemesis	Operation in 34 hours. Milk in peritoneal cavity. Sequestration of ulcer and suture	D.	—	General peritonitis, ulcer with thick edges on anterior surface near pylorus; suture sound.
21	M.	47	Indigestion for 2 years; never hæmatemesis	Operation in 40 hours. Ulcer excised and sutured, lavage, supra-pubic incision and drainage. Infusion	D.	Died in 6 hours	General peritonitis, crater-ulcer perforated on anterior surface near pylorus.
22	F.	17	Indigestion for some years; twice hæmatemesis	Operation in 13½ hours. Ulcer and indurated area invaginated and sutured, sponging, supra-pubic incision and drainage	R.	Discharged in 5 weeks	Ulcer on anterior surface near cardiac orifice.

Case.	Sex.	Age.	Previous history.	Operation.	Recovery or death.	After-history.	Anatomy.
23	F.	22	Indigestion for seven years; hæmatemesis 6 months ago	Operation in 42 hours. Sutured with difficulty, lavage, and supra-pubic drainage	D.	—	General peritonitis, perforated ulcer on anterior surface of lesser curvature near cardiac end; suture sound.
24	F.	16	Ill for 14 days, pain and vomiting	Operation in 12 hours. Suture, lavage	D.	—	No p. m.; perforated ulcer on anterior wall near cardiac orifice.
25	F.	23	Gastric symptoms for 3 years; melæna	Suture, lavage, wound packed with gauze and a few sutures inserted. Two days later wound sutured under anæsthetic	D.	Lived for 6 days, death from perforation of a second ulcer	General peritonitis; perforated ulcer on anterior surface sutured securely; more recent perforation of another ulcer similarly placed on posterior surface.
26	M.	47	Not recorded	Operation in 24 hours. Suture, lavage, supra-pubic drainage	D.	—	General peritonitis, perforation of large ulcer on anterior surface near pylorus; upper part of suture leaked, one stitch torn out.
27	F.	21	Gastric symptoms for 2 years	Operation in 24 hours. Suture and lavage	D.	—	General peritonitis, ulcer on posterior surface adherent to pancreas; suture sound.
28	F.	19	Indigestion for 4 years, never hæmatemesis	Incision above umbilicus, turbid fluid released; after examination stomach thought to be sound. Second incision below umbilicus, appendix removed and also right Fallopian tube, which contained pus	D.	—	General peritonitis, perforated ulcer adherent to pancreas, another healing ulcer on middle of linear curvature.
29	F.	38	Pain and vomiting for 2 years, never hæmatemesis	Operation in 12 hours. Invagination with silk sutures, lavage, supra-pubic drainage	R.	Discharged in 31 days	Ulcer on anterior surface 3 inches from pylorus
30	F.	17	Indigestion for 5 months, one month ago hæmatemesis	Operation in 7 hours. Incision below umbilicus, appendix found to be normal. Incision above umbilicus, suture of ulcer, lavage	R.	Discharged in 37 days	Ulcer on anterior surface near pylorus

31	F	18	Gastric symptoms for 6 months	Operation probably in 12 hours. Incision below umbilicus, inflamed appendix with 3 concretions removed. Incision above umbilicus, ulcer invaginated with double row of silk sutures, lavage	R.	Discharged in 47 days	—
32	F	23	History of gastric ulcer for 2 years	Operation in 6 hours. Excision of ulcer, suture with fine silk, lavage, gauze drain, suprabubic drainage	D.	Died in 36 hours	General peritonitis, large ulcer on anterior and posterior surfaces of lesser curvature, posterior part had escaped suture.
33	F	18	Indigestion for some years. Sudden pain and vomiting while at a meal	Operation in 16 hours. Exploration below umbilicus; second incision above umbilicus, ulcer invaginated with silk sutures, lavage	D.	Died in 24 hours	Ulcer on anterior surface of lesser curvature near pylorus.
34	F	21	History indefinite: ill for 4 days	Operation probably in 48 hours. Infusion necessary, suture and lavage	D.	Died in a few hours	General peritonitis, ulcer on middle of anterior surface, another ulcer opposite on posterior surface.
35	F	36	Long history of pain and vomiting	Operation in 12 hours. Suture and lavage	D.	Died in 24 hours	General peritonitis, perforated ulcer on anterior surface adherent to liver, healed ulcer on posterior surface, suture sound.
36	M	33	Some previous history of vomiting. Sudden pain while pushing a cart 3 days before admission	Suture of ulcer, lavage	R.	Readmitted 4 months later with dilated stomach; anterior gastro-enterostomy with silk sutures; four years later remains well, can eat any food, never sick, has gained one stone	Perforated ulcer on anterior surface.

Case.	Sex.	Age.	Previous history.	Operation.	Recovery or death.	After-history.	Anatomy.
37	M.	61	Pain and vomiting for 14 days	Operation in 16 hours. Suture, lavage, supra-pubic drainage; fortnight later rise of temperature; at end of fourth week left subphrenic abscess opened and drained	R.	Discharged in 68 days	Perforated ulcer on posterior surface near cardiac end.
38	M.	51	Indigestion for some years, hæmatemesis	Circular hard mass on anterior surface of stomach 3 in. in diameter, with a central perforation, edges pared, invagination, lavage, supra-pubic drainage	D.	Died on 13th day	Suture sound, cause of death was grey hepatisation of upper lobe of right lung.
39	F.	37	Long history of gastric pain, worse last 10 weeks, hæmatemesis	Operation in 7 hours. Invagination, suture, lavage, supra-pubic drainage	R	Discharged on 48th day; re-admitted 1 year later for gastro-enterostomy; re-admitted later with perforation of peptic ulcer of jejunum, suture, recovery; re-admitted year later with perforation of 2nd peptic ulcer, suture, recovery	Ulcer on anterior wall near pyloric end of lesser curvature.
40	F.	22	Gastric symptoms for some years	Operation in 4½ hours. Incision below umbilicus, appendix and tubes normal, incision above umbilicus, ulcer sutured, extro-ventration of viscera, lavage	R.	Discharged on 28th day	Ulcer on lesser curvature.
41	F.	19	Pain for some months, never hæmatemesis	Operation in 26 hours. Invagination, lavage, supra-pubic drainage, infusion	R.	Discharged on 31st day	Ulcer on upper part of cardiac end.

42	F.	28	Gastric symptoms for some years	Operation in 15 hours. Hour-glass stomach found, gastroplasty with sequestration of ulcer, lavage	D.	Died on 4th day	No recent peritonitis, stomach full of blood, gastro-plastic wound firmly closed, perforated ulcer in pyloric part of hour-glass stomach; death from perforation of another ulcer into lesser sac.
43	F.	17	Long history of pain after food	Operation in 14 hours. Suture, lavage, supra-pubic drainage	D.	Died on 6th day	Chronic adherent ulcer just below lesser curvature near cardiac orifice; death from pneumonia.
44	F.	20	Indigestion for some months, never any hæmatemesis	Operation in 18 hours. Perforated ulcer found, nothing done, death under anæsthetic	D.	—	Ulcer on anterior wall near cardiac end
45	M.	35	Pain and vomiting for 2 months	Operation in 4 hours. Suture with omental graft, lavage, supra-pubic drainage	R.	Discharged in 19 days; re-admitted year later with gastric pain, treated medically	Ulcer on anterior wall.
46	M	57	Long history of pain after food	Operation in 24 hours. Suture, lavage, wound closed, wound re-sutured later	R.	—	Ulcer on anterior surface of lesser curvature near pylorus.
47	F	23	Pain and vomiting for some years	Operation in 14 hours. Suture, lavage, supra-pubic drainage, 1 month later left subphrenic abscess drained; 5 days later right subphrenic abscess drained	D.	Died on 39th day	Peritonitis only in upper part of abdomen, perforated ulcer on anterior surface had been sutured but had re-opened, scar of ulcer on posterior wall, pylephlebitis, abscesses in liver and spleen.
48	F	21	Pain and vomiting for 10 days	Incision outer border of right rectus level of umbilicus, localised abscess in right iliac fossa drained	D.	Died on 3rd day	Ulcer on anterior surface, not perforated, but floor of peritoneum only, multilocular suppurative peritonitis, abscess round spleen, round cæcum, and in pelvis.

Case.	Sex.	Age.	Previous history.	Operation.	Recovery or death.	After-history.	Anatomy.
37	M.	61	Pain and vomiting for 14 days	Operation in 16 hours. Suture, lavage, supra-pubic drainage; fortnight later rise of temperature; at end of fourth week left subphrenic abscess opened and drained	R.	Discharged in 68 days	Perforated ulcer on posterior surface near cardiac end.
38	M.	51	Indigestion for some years, hæmatemesis	Circular hard mass on anterior surface of stomach 3 in. in diameter, with a central perforation, edges pared, invagination, lavage, supra-pubic drainage	D.	Died on 13th day	Suture sound, cause of death was grey hepatisation of upper lobe of right lung.
39	F.	37	Long history of gastric pain, worse last 10 weeks, hæmatemesis	Operation in 7 hours. Invagination, suture, lavage, supra-pubic drainage	R	Discharged on 48th day; re-admitted 1 year later for gastro-enterostomy; re-admitted later with perforation of peptic ulcer of jejunum, suture, recovery; re-admitted year later with perforation of 2nd peptic ulcer, suture, recovery	Ulcer on anterior wall near pyloric end of lesser curvature.
40	F	22	Gastric symptoms for some years	Operation in 4½ hours. Incision below umbilicus, appendix and tubes normal, incision above umbilicus, ulcer sutured, extroventration of viscera, lavage	R.	Discharged on 28th day	Ulcer on lesser curvature.
41	F	19	Pain for some months, never hæmatemesis	Operation in 26 hours. Invagination, lavage, supra-pubic drainage, infusion	R.	Discharged on 31st day	Ulcer on upper part of cardiac end.

42	F.	28	Gastric symptoms for some years	Operation in 15 hours. Hour-glass stomach found, gastro-plasty with sequestration of ulcer, lavage	D.	Died on 4th day	No recent peritonitis, stomach full of blood, gastro-plastic wound firmly closed, perforated ulcer in pyloric part of hour-glass stomach; death from perforation of another ulcer into lesser sac.
43	F.	17	Long history of pain after food	Operation in 14 hours. Suture, lavage, supra-pubic drainage	D.	Died on 6th day	Chronic adherent ulcer just below lesser curvature near cardiac orifice; death from pneumonia.
44	F.	20	Indigestion for some months, never any hæmatemesis	Operation in 18 hours, Perforated ulcer found, nothing done, death under anæsthetic	D.	—	Ulcer on anterior wall near cardiac end
45	M.	35	Pain and vomiting for 2 months	Operation in 4 hours. Suture with omental graft, lavage, supra-pubic drainage	R.	Discharged in 19 days; re-admitted year later with gastric pain, treated medically	Ulcer on anterior wall.
46	M.	57	Long history of pain after food	Operation in 24 hours. Suture, lavage, wound closed, wound re-sutured later	R.	—	Ulcer on anterior surface of lesser curvature near pylorus.
47	F.	23	Pain and vomiting for some years	Operation in 14 hours. Suture, lavage, supra-pubic drainage, 1 month later left subphrenic abscess drained; 5 days later right subphrenic abscess drained	D.	Died on 39th day	Peritonitis only in upper part of abdomen, perforated ulcer on anterior surface had been sutured but had re-opened, scar of ulcer on posterior wall, pylephlebitis, abscesses in liver and spleen.
48	F	21	Pain and vomiting for 10 days	Incision outer border of right rectus level of umbilicus, localised abscess in right iliac fossa drained	D.	Died on 3rd day	Ulcer on anterior surface, not perforated, but floor of peritoneum only, multilocular suppurative peritonitis, abscess round spleen, round cæcum, and in pelvis.

Case.	Sex.	Age.	Previous history.	Operation.	Recovery or death.	After-history.	Anatomy.
49	F.	21	Gastric symptoms for 10 weeks	Operation in 60 hours. Incision below umbilicus, appendix found healthy. Upper incision, ulcer sutured, lavage	D.	Death in 1 hour	Slight general peritonitis. Ulcer on anterior surface near cardiac end; another opposite on posterior wall. Both had perforated, only former sutured.
50	F.	19	Indigestion for 6 weeks	Operation in 26 hours. Suture, lavage, closure of wound	D.	Died in a few hours	General peritonitis. Perforated ulcer on posterior wall near pylorus. Suture sound.
51	F.	21	Gastric symptoms for 5 years	Operation in 8 hours. Lower incision, appendix and tubes healthy. Upper incision, suture of ulcer, lavage, gauze drain	R.	Re-admitted year later with pain and vomiting. Treated medically	Ulcer on anterior surface, close to lesser curvature.
52	F.	21	No history of previous illness	Operation in 36 hours. Suture, lavage	R.	—	Ulcer on anterior surface near pylorus.
53	M.	22	Gastric pain for 2 years; never sick	Operation in 7 hours. Suture, lavage, supra-pubic drainage	R.		Ulcer on anterior surface near pylorus.
54	M.	48	Gastric symptoms for 2 years; occasional hæmatemesis	Operation in 12 hours. Suture, lavage, supra-pubic drainage	R.		Ulcer on anterior surface near pylorus
55	M.	27	Gastric symptoms for 6 months, a heavy drinker	Operation in 28 hours. Suture, lavage, supra-pubic drainage	R.	Three weeks later intestinal obstruction by adhesion of small bowel to operation-scar. enterolysis, recovery	Ulcer on anterior surface near pylorus

56	M.	30	Gastric symptoms for 14 years. Occasional hæmatemesis. Two years ago, in another hospital, gastro-enterostomy with Murphy's button. Four months ago intestinal obstruction by the button; operation and removal	Suture of ulcer, lavage, supra-pubic drainage	R.	Still ill at date	Perforation of peptic ulcer in jejunum 1¼ inches from anastomosis.
57	F.	18	Gastric symptoms for 6 months	Operation in 12 hours. Closure of ulcer with purse-string suture and a row of Lembert sutures, lavage, supra-pubic drainage	R.	—	Perforating ulcer on middle of anterior surface.
58	F.	17	Indigestion for 1 year never hæmatemesis	Operation in 13 hours. Suture, lavage, closure of wound	R.	—	Ulcer on anterior surface.
59	F.	21	Pain and vomiting for 1 year; never hæmatemesis	Operation in 6 hours. Suture, lavage, supra-pubic drainage	R.	—	Ulcer on anterior surface near cardiac end.
60	F.	29	Slight history of indigestion	Operation in 20 hours. Closure with purse-string suture, omental graft, lavage, no drainage	R.	—	Ulcer on anterior surface of lesser curvature.
61	F.	25	Gastric symptoms for 2 years	Operation in 5 hours. Excision (in which process another ulcer not perforated was found) suture, lavage, no drainage	R.	—	Perforating ulcer on anterior surface.
62	F.	38	Not recorded	Operation in 10 hours. Suture, lavage, drainage	R.	—	Perforation of chronic ulcer near cardiac orifice.

Case.	Sex.	Age.	Previous history.	Operation.	Recovery or death.	After-history.	Anatomy.
63	F.	26	Not recorded	Operation in 10 hours. Death under anæsthetic during suture	D.	—	Perforated ulcer on anterior surface of lesser curvature near cardiac end.
64	F.	20	Pain; vomiting; occasional hæmatemesis for 6 months	Operation in 13 hours. Suture and omental graft, lavage, supra-pubic drainage	D.	Vomiting; hæmatemesis after operation. Died on 4th day	P.-M. — Slight peritonitis, probably subsiding, stomach hour-glass, perforation of chronic ulcer middle of lesser curvature, suture sound; another recent ulcer opposite to it, from which hæmorrhage.
65	F.	24	Slight indigestion; no vomiting; never hæmatemesis	Operation in 27 hours. Excision, suture, lavage, no drainage	D.	Died on 6th day, mainly from tubercle of lungs	P.-M.—Slight peritonitis subsiding, perforated ulcer on anterior wall near cardiac end; another ulcer opposite with floor of peritoneum only, advanced phthisis with excavation, suture sound.
66	M.	50	Gastric symptoms for 3 years	Operation in 14 hours. Suture and omental graft, lavage and supra-pubic drainage	R.	One month later, anterior gastro-enterostomy, pylorus being narrowed by suture. Twenty months later quite well, can eat any food, no pain or vomiting	Perforated ulcer on anterior surface near pylorus.
67	M.	20	Indigestion for 1 month; not sick	Operation in 22 hours. Suture by double layer of interrupted sutures, lavage, supra-pubic drainage, infusion	D.	Died in a few hours	General peritonitis, perforated ulcer on anterior surface near pylorus, another ulcer opposite to it, suture leaked.

68	F	27	History of gastric ulcer for 4 years; hæmatemesis	Operation in 24 hours. Suture, lavage, Paton's serum, supra-pubic drainage	R.	—	—
69	F	17	Gastric symptoms for 3 months; no hæmatemesis	Operation in 12 hours. Suture, lavage, supra-pubic incision, no drainage	R.	—	Ulcer on anterior surface.
70	M.	16	Pain after food for 3 weeks. Fall from bicycle 24 hours before admission; sick 15 minutes after the accident	Operation in 27 hours. Suture, lavage, supra-pubic drainage; posterior gastro-enterostomy at the same time	R.	—	Small perforation on anterior surface near pylorus, just admitting a probe.
71	M.	45	Gastric symptoms for some months	Operation in 5 hours. Suture, lavage; posterior gastro-enterostomy at the same time	D.	—	Ulcer on anterior wall and one on posterior wall, coalescing; latter adherent to pancreas, suture sound, pneumonia.
72	M.	52	Gastric symptoms for many years; hæmatemesis 5 months before admission	Operation in 8 hours. Suture and omental graft, lavage, drainage, supra-pubic drainage	D.	Death from pneumonia	Ulcer on anterior and posterior wall adherent to liver, suture sound, no peritonitis. Death from pneumonia.
73	M.	25	Gastric symptoms for 3 years	Operation in 6 hours. Suture, lavage, supra-pubic drainage	D.	—	Perforated chronic ulcer with adhesions on anterior wall.

(*b*) *With Local Peritoneal Infection.*

Fifteen cases admitted, 9 females and 6 males. Of these 11 died, giving a mortality rate from this cause of 1·9 per cent. (11 deaths in 556 cases).

Recovery in 4 cases of local perigastric abscess (in 1 case associated with subphrenic abscess). The remainder form a series of nearly hopeless attempts to deal with such conditions as subphrenic abscess, perforation of diaphragm, empyema, gangrene of lung, perforation of ulcer into lung, pylephlebitis, hepatic abscess, septicæmia, pyæmia, and intestinal obstruction by perigastric adhesions.

In 3 cases gastro-enterostomy was performed.

Table of Cases of Local Infection.

Case.	Sex.	Age.	Previous history.	State on admission.	Operation.	Recovery or death.	After-history.	Anatomy.
74	F	24	Pain and vomiting for 8 years, never hæmatemesis, 2 days before admission sudden pain and vomiting	Epigastric tenderness and rigidity, slight pyrexia, not very ill and not sick	Area of induration gradually appeared in middle line above umbilicus, incision on 12th day, 3 oz. of pus	R.	Slow recovery, discharged in 60 days	Perigastric abscess.
75	F	26	Pain, hæmatemesis 1 year ago, now pain and vomiting for a few weeks	Physical signs in upper abdomen and left side of chest	Aspiration of left pleura, pus found, transpleural drainage of left subphrenic abscess, incision above umbilicus, no result, incision below umbilicus, drainage of pelvic abscess	D.	Died on 6th day	P. M.—Chronic ulcer adherent to liver and pancreas, leakage, subphrenic abscess, perforation of diaphragm, empyema. No general peritonitis, but pelvic abscess apparently unconnected with above.
76	F	39	Pain and vomiting for 9 weeks	Dilated stomach	Anterior gastro-enterostomy	D.	Died on 19th day	P. M.—Anastomosis sound, no general peritonitis, but localised abscess between stomach and liver due to perforation of an ulcer, probably subsequent to operation.
77	M	27	Gastric symptoms for 9 months, never hæmatemesis	Dilated stomach	Exploratory cœliotomy, thought to be carcinoma	D.	—	P.M.—Tumour round pylorus consisting of adhesions, stomach dilated and adherent to pancreas, perforation of an ulcer 3 ins. in diameter on anterior and posterior walls near pylorus, subphrenic abscess on left of falciform ligament, no general peritonitis.

Case.	Sex.	Age.	Previous history.	State on admission.	Operation.	Recovery or death.	After-history.	Anatomy.
78	F.	23	Gastric symptoms for 5 years, worse last 5 weeks, 2 weeks ago began to cough with offensive sputum, never hæmatemesis	Large tender mass in epigastrium, clear signs of gas-containing left subphrenic abscess with less certain signs of fluid in pleura	Incision into central mass led into gas-containing cavity with a little pus, aspiration of left pleura, no result, resection of rib and drainage of large left subphrenic abscess	D.	Septicæmia, died on 80th day	P. M.—No general peritonitis, hour-glass stomach, pyloric part reaching to pubes, cardiac part mainly under ribs, small thick-walled abscess-cavity which had two openings, one into cardiac part of stomach, the other into duodenum, small empyema, no perforation of diaphragm, subphrenic abscess healed.
79	F.	28	Pain and vomiting for 7 years, never hæmatemesis, present illness 9 weeks	Tender mass in left hypochondrium, dull on percussion, slight movement with respiration	Incision into mass led into small thick-walled abscess-cavity containing gas, which had an opening into stomach, later attempt made to close gastric fistula and anterior gastro-enterostomy performed	D.	Died on 8th day	P. M.—Anastomosis sound, but had been made with pyloric part of hour-glass stomach, with extreme constriction, ulcer in pyloric part opening into abscess-cavity.
80	F.	21	Gastric symptoms for 5 years, sudden pain and sickness 5 days before admission	Not very ill, signs of fluid in left pleura, abdomen normal	Aspiration of left pleura gave 10 oz. of blood-stained fluid, rigors began, left subphrenic abscess drained, rigors continued	D.	Died on 19th day	P. M.—Ulcer on anterior surface, floor transparent, but contained muscle and was not perforated. Cause of death pylephlebitis and multiple hepatic abscesses.
81	M.	30	Gastric symptoms for 4 months, 5 days ago vomited, bringing up blood	Signs indicating disease of left side of chest	Transpleural drainage of left subphrenic abscess	D.	—	P.M.—Healing ulcer on anterior surface, perforated ulcer on lesser curvature, dense perigastric and peri-

			and offensive sputum					splenic adhesions, large gangrenous cavity in left lung, no perforation of diaphragm seen.
82	F.	21	No previous illness, sudden pain in left side, shoulder to abdomen	Signs of pneumonia, lower lobe left lung; pyrexia, 3 days later rigors with leucocytosis, increase of polymorphonuclear cells, later right hemiplegia with aphasia	No operation	D.	Died in 42 days	P.M.—Stomach adherent to diaphragm, direct perforation of ulcer into lung, small empyema, infective thrombosis of portal and mesenteric veins, abscess in liver, many abscesses in cortex of brain, infarcts in spleen, embolism of left middle cerebral artery, *Bacillus coli* grown from lung, portal and systemic pyæmia.
83	F.	35	Pain after food for 10 years, but seldom sick, no hæmatemesis	Tender swelling left side of epigastrium, not moving with respiration, pyrexia	Incision, release of 1 oz. of yellow odourless pus lying between stomach and abdominal wall, no gas	R.	—	—
84	M.	32	Pain and vomiting for 6 months, never hæmatemesis	Inflammatory mass in right hypochondrium	Incision and drainage of a peri-pyloric abscess, later 13 days anterior gastro-enterostomy, parotitis	R.	Discharged well, cannot be traced	—
85	M.	58	Symptoms for 1 month	Signs obscure, hard mass under left costal margin	No operation	D.	—	P. M.—Chronic ulcer on anterior wall, perforation, small localised abscess, strangulation of small bowel by adhesions.
86	M.	58	Symptoms for 4 months	Signs obscure, moribund	No operation	D.	—	P.-M.—Mass of adhesions between liver and pylorus, with chronic pyloric ulcer opening into them, general arterial disease.

Case.	Sex.	Age.	Previous history.	State on admission.	Operation.	Recovery or death.	After-history.	Anatomy.
87	F.	37	Gastric ulcer at age of 19, recurrence recently; acute symptoms for 13 days	Tender inflammatory swelling in epigastrium with pyrexia	Incision into local perigastric abscess containing gas, 3 weeks later abscess in abdominal wall opened, rigors began, 2 weeks later transpleural drainage of left subphrenic abscess, later glandular abscess in left groin	R.	—	—
88	M.	68	No previous gastric symptoms whatever, week before admission ricked himself while making sudden effort	Abdominal pain and tenderness with pyrexia	No operation	D.	—	P. M.—Stomach adherent to liver and localised abscess in the adhesions, chronic ulcer with peritoneal floor, acute pericarditis.

(c) Sequelæ.

Forty-five cases admitted, of whom 11 died.

Mortality rate from this cause (11 deaths in 556 cases) is 1·9 per cent. Impossible to classify these cases accurately, because the condition is often complex and in many of them doubtless an ulcer is present.

Surgical treatment:

(*a*) *Adhesions.*—In 4 cases gastrolysis with no deaths; in 10 cases gastro-enterostomy with 2 deaths.

(*b*) *Pyloric stenosis.*—In 5 cases pyloroplasty with 3 deaths; in 24 cases gastro-enterostomy with 6 deaths (1 of these due to tetany).

(*c*) *Hour-glass stomach.*—In 1 case gastroplasty; recovery. In 1 case gastro-enterostomy; recovery.

Table of Operations for Sequelæ.

Adhesions	Cases 89—103
Pyloric stenosis	,, 104—131
Hour-glass stomach . .	,, 132, 133

Table of Operations for Sequelæ.

Case.	Sex.	Age.	Previous history.	State on admission.	Operation.	Recovery or death.	After-history.	Anatomy.
89	F.	36	Pain and vomiting for 13 years, hæmatemesis several times	Tumour felt under left rectus	Incision, tumour composed of dense fibrous tissue fixing stomach to abdominal wall, gastrolysis and insertion of omental graft, suppuration of wound	R.	Re-admitted year later with pain and hæmatemesis	—
90	F.	28	Pain, vomiting, and hæmatemesis for some years, in another hospital gastrotomy for hæmorrhage, nothing found	Thin, constant pain and vomiting, occasionally small hæmatemesis	Anterior gastro-enterostomy with continuous silk suture	R.	No improvement, gastrolysis 2 years and 3 years later (adhesions of the gastrotomy - incision), still pain and vomiting	—
91	F.	57	Pain, vomiting, and hæmatemesis for 12 years, at other hospitals—5 years ago pyloroplasty, 2 years ago nephropexy	Dilated stomach: mass felt in region of pylorus	Posterior gastro-enterostomy with silk sutures	R.	Discharged well, 2 years later stomach dilated, vomits almost daily, lives on liquids and soaked bread	Pylorus hard and fixed to scar of abdominal incision (pyloroplasty) by firm adhesions.
92	F.	46	Pain and vomiting for 12 years; often altered blood	No signs except free right kidney	Right nephropexy, no relief, later gastrolysis and suture of ulcer	R.	Re-admitted year later and died (*cf.* Case 114)	Ulcer adherent to liver and liver adherent to abdominal wall; in separating adhesions ulcer perforated and was sutured.

93	F.	29	Gastric symptoms for 10 years	No signs	Anterior gastro-enterostomy with continuous silk suture, gastrolysis	R.	Six months later rather better, but still some pain after food	Scar near pylorus with band from it to abdominal wall, giving slight hour-glass shape.
94	F.	32	Gastric symptoms for 12 years, eight weeks ago hæmatemesis	Dilated stomach	Anterior gastro-enterostomy with continuous silk suture	R.	Discharged well, not traced since	Pyloric adhesions.
95	F.	30	Gastric symptoms for 12 years, never hæmatemesis, lately frequent vomiting and loss of weight	Dilated stomach, visible peristalsis	Posterior gastro-enterostomy with continuous silk suture, condition satisfactory till 8th day, then vomiting requiring rectal feeding for 5 days, then improvement, and patient out of bed 24th day	D.	Three days later abdomen opened for recurrence of pain, stomach surrounded by dense adhesions, in separating which an ulcer perforated, suture and drainage, died 13 days later	F. M.—General peritonitis, large chronic ulcer fixed to pancreas, hour-glass stomach, anastomosis sound but had been made with pyloric chamber of stomach.
96	F.	21	Indigestion for 12 months, occasional hæmatemesis	Tenderness in epigastrium, slight dilatation of stomach	Anterior gastro-enterostomy with continuous silk suture, area of scar-tissue felt through anterior wall, stomach freed from adhesions	D.	General peritonitis came on, exploration on 4th day, pus in abdomen, lavage, drainage, death 2 days later	P. M.—Anastomosis sound, chronic ulcer fixed to pancreas, general peritonitis, *Streptococcus pyogenes* and *Staphylococcus aureus* and *albus* grown from pus.
97	F.	35	Gastric symptoms for 14 years, twice hæmatemesis	Dilated stomach	Posterior gastro-enterostomy with continuous silk suture, in 24 hours after operation vomited 2 pints of blood, no further trouble	R.	Discharged well, too recent for real result to be known	Adhesions round pylorus.
98	M.	55	Hæmatemesis and melæna 3 years ago, pain and vomiting 2 years	Dilated stomach, lower border 2 inches below umbilicus	Gastrolysis, adhesions too dense to allow complete separation	R.	Considerable improvement, final result unknown	Pylorus adherent to liver.

Case.	Sex.	Age.	Previous history.	State on admission.	Operation.	Recovery or death.	After-history.	Anatomy.
99	M.	26	Gastric pain and vomiting for 2 years, no hæmatemesis	Emaciated, dilated stomach	Anterior gastro-enterostomy	R.	Discharged well, died 6 months later from tubercle of lungs	Pylorus bound down by adhesions.
100	M.	34	Pain for 1 year, not sick, never hæmatemesis	Epigastric tenderness and resistance	Separation of adhesions over scar on middle of lesser curvature, scar invaginated, plication over it	R.	Discharged well, not traced since	—
101	M.	54	Pain and vomiting for 4 years; sometimes hæmatemesis	Dilated stomach	Anterior gastro-enterostomy with continuous silk suture	R.	Good recovery, but final result not known	Pylorus bound up in mass of cicatricial tissue.
102	M.	45	Pain, hæmatemesis for 18 months	Dilated stomach, free HCl.	Anterior gastro-enterostomy with continuous silk suture	R.	Three years later quite well, can eat any food, no pain or sickness, gained weight	Hard mass round pylorus thought to be carcinoma; subsequent history shows that it was cicatricial tissue.
103	M.	40	Gastric symptoms for 9 months	Dilated stomach	Gastrolysis	R.	—	Dense adhesions between pylorus and gall-bladder; latter healthy.
104	F.	45	Gastric symptoms for 20 years; 3 months constant vomiting	Emaciated, dilated stomach	Pyloroplasty	R.	Well with stomach of normal size on 58th day	Before operation pylorus just admitted little finger.
105	F.	25	Pain and vomiting for 2 years; hæmatemesis	Dilated stomach	Anterior gastro-enterostomy with continuous silk suture	R.	Three years later quite well, can eat any food, has gained 5 st.	Cicatricial stenosis of pylorus.

106	F.	38	Same patient as Case 62. Then perforation of ulcer and suture; now pain and vomiting	Dilated stomach	Anterior gastro-enterostomy with continuous silk suture	R.	Re-admitted year later with perforation of peptic ulcer of jejunum, suture, recovery	—
107	F.	50	Pain and vomiting for 7 years	Dilated stomach	Pyloroplasty, during which an ulcer was found in pylorus and partly excised	R.	One year later much better; still occasional pain but not sick	—
108	F.	33	Pain for 3 years, recently vomiting, no hæmatemesis	Emaciated, stomach slightly dilated	Pyloroplasty; pylorus thought to be scarred	D.	Died on 6th day	P. M.—No peritonitis; pyloroplasty successful; chronic ulcer near pylorus adherent to pancreas.
109	F.	38	Pain, hæmatemesis for 2 years	Dilated stomach	Anterior gastro-enterostomy with 2 rows of silk sutures	D.	Continued to vomit till death on 10th day; vicious circle	P. M.—No peritonitis, pylorus narrowed by scar, anastomosis sound, no kink of intestine.
110	F.	49	Gastric pain for 2 years, vomiting for 8 months	Dilated stomach	Anterior gastro-enterostomy with continuous silk suture	R.	Discharged well	Pylorus thickened.
111	F.	34	Pain for 7 years, occasionally hæmatemesis	Dilated stomach	Anterior gastro-enterostomy with continuous silk suture	R.	18 months later has gained 2 st., but still liable to pain and occasional vomiting, and rarely eats solid food	Pylorus thick and adherent.
112	F.	30	Pain, vomiting, and occasional hæmatemesis for 16 years; last year has vomited daily	Dilated stomach	Anterior gastro-enterostomy with continuous silk suture	R.	Improved at first but 18 months later still discomfort, and can only take light food; hæmatemesis several times; treated at another hospital by lavage	—

Case.	Sex.	Age.	Previous history.	State on admission.	Operation.	Recovery or death.	After-history.	Anatomy.
113	F.	47	Gastric symptoms for 9 years; twice hæmatemesis; latelyalmost daily vomiting	Emaciated, dilated stomach, visible peristalsis	Anterior gastro-enterostomy with continuous silk suture	D.	Frequent vomiting up to death on 9th day	P. M.—Peritonitis through leakage at left end of anastomosis. Pylorus thick, hard, and white; microscopically scar-tissue.
114	F.	47	Same patient as Case 92. Then gastrolysis,suture of ulcer. Recurrence of pain and vomiting for 6 months	Dilated stomach	Exploration. Transverse colon was drawn up and adherent to anterior surface of stomach; gastro-enterostomy, though anterior, was performed through opening in transverse meso-colon. Copious vomiting followed operation.Three days later entero-enterostomy. Death 3 days later.	R.	—	P. M.—Peritonitis,gas in peritoneal cavity, probably due to leakage at site of old ulcer. Coils of small bowel had passed through opening in transverse meso-colon, but were not strangulated. Anastomosis sound. Water passed from stomach into proximal limb of jejunum and thence by second anastomosis into bowel; but water could not be driven into distal limb of jejunum owing to a kink.
115	F.	28	Gastric symptoms for 10 years. No hæmatemesis	Dilated stomach	Anterior gastro-enterostomy. Later, 10 weeks, entero-enterostomy, because pain and vomiting recurred	R.	Discharged well	Pyloric stenosis.
116	F.	32	Gastric symptoms for 7 years	Dilated stomach	Posterior gastro-enterostomy	R.	Discharged well	Pyloric stenosis.

117	F.	42	Gastric ulcer with hæmatemesis 8 years ago. Now pain and vomiting	Dilated stomach	Posterior gastro-enterostomy	R.	Improved greatly at first, but six months later re-admitted with return of pain and vomiting. Second posterior gastro-enterostomy performed, with complete relief	Pyloric stenosis.
118	F.	30	Gastric symptoms for 9 years	Dilated stomach	Posterior gastro-enterostomy	R.	Discharged well	—
119	F.	40	Gastric ulcer 3 years ago	Dilated stomach	Anterior gastro-enterostomy	R.	Discharged well	—
120	M.	45	Gastric symptoms for 5 years. No hæmatemesis	Dilated stomach	Pyloroplasty	D.	Died on 9th day	P. M.—General peritonitis. Repair of pyloroplastic incision good. But perforation of an ulcer on anterior surface at the time of or subsequent to operation.
121	M.	45	Indigestion for 18 months. Never hæmatemesis	Dilated stomach	Pyloroplasty	D.	Sudden death on 11th day	P.M.—Repair of wound good. Cicatrised ulcer in pylorus. Pyloric orifice admitted 3 fingers. No disease elsewhere. No pulmonary thrombosis. Cause of death unknown.
122	M.	50	Pain and vomiting for 12 years	Dilated stomach	Anterior gastro-enterostomy with continuous silk suture	R.	Four years later quite well, can eat any food, no pain or vomiting	—
123	M.	36	Five attacks of pain and hæmatemesis in 9 years	Emaciated, very ill, dilated stomach	Anterior gastro-enterostomy with continuous silk suture	D.	Died in 1 hour	Large, thick stomach, with three ulcers surrounding pylorus. Death from collapse.

Case.	Sex.	Age.	Previous history.	State on admission.	Operation.	Recovery or death.	After-history.	Anatomy.
124	M.	46	Pain, vomiting, and hæmatemesis for 4 months	Dilated stomach	Posterior gastro-enterostomy with continuous silk suture	D.	Frequent, copious vomiting till death on 7th day.	P. M.—No peritonitis. Large chronic ulcer encircling and narrowing pylorus. Anastomosis sound, but mucous membrane swollen and completely blocking the opening so that water could not be forced through it from stomach.
125	M.	34	Hæmatemesis 10 years ago, indigestion off and on since, recently hæmatemesis	Dilated stomach	Anterior gastro-enterostomy with continuous silk suture	R.	Two years later quite well, eats any food, not sick, occasional flatulence, normal weight	—
126	M.	34	Pain and vomiting for 12 months, with occasional hæmatemesis and melæna	Dilated stomach	Anterior gastro-enterostomy with continuous silk suture	R.	17 months later quite well, can eat anything, gain of weight	—
127	M.	44	Gastric symptoms for 7 years, with occasional hæmatemesis	Dilated stomach	Posterior "no loop" gastro-enterostomy, with continuous silk suture, excision of redundant mucous membrane	R.	17 months later quite well, can eat any food, has gained 45 pounds in weight	—
128	M.	39	Gastric symptoms for 9 years, with hæmatemesis and melæna	Dilated stomach	Anterior gastro-enterostomy with continuous silk suture, some vomiting and melæna after operation	R.	10 months later feels well, eats well, has gained weight, but has vomited 3 times	—

129	M.	37	Gastric symptoms for 20 years, 4 ago had hæmatemesis	Dilated stomach	Anterior gastro-enterostomy with continuous silk suture	R.	10 months later quite well, no pain, "can take food he has not touched for 15 years"	—
130	M.	53	Five months of pain and vomiting, no hæmatemesis	Dilated stomach	Posterior gastro-enterostomy and entero-enterostomy	R.	16 months later still gastric pain, can only eat fish and light food, vomits every morning, losing weight	Diagnosis uncertain.
131	M.	35	Long history of gastric symptoms, said to have had 3 attacks of appendicitis, an abscess in right iliac fossa drained 6 months ago	Large dilated stomach, tetany	Posterior gastro-enterostomy and entero-enterostomy, 4 days later no relief, anterior gastro-enterostomy nearer cardiac end, and second entero-enterostomy	D.	—	P. M.—General peritonitis, all junctions sound, ulcer on posterior wall adherent to pancreas near its head.
132	F.	41	Pain after food for 5 years, frequent vomiting	Large dilated hour-glass stomach with palpable mass in region of pylorus	Anterior gastro-enterostomy with continuous silk suture	R.	Great relief, but 2 years later some pain after food and occasionally sick, has gained weight, cannot eat meat	—
133	F.	23	Indigestion for 4 years, has been in other hospitals	Dilated stomach	Gastroplasty of hour-glass stomach	R.	Often indigestion, but on the whole much better, and 4 years later is able to work hard on a simple diet; with bismuth and X rays it can be seen that the deformity has not returned	Sharp constriction 3 ins. from pylorus

Summary of Results of Gastro-enterostomy.

Of 556 cases gastro-enterostomy was performed in 47, with 11 deaths.

(1) *Ulcer with hæmatemesis.*—Four cases; all recovered.

(2) *Chronic ulcer.*—Three cases; all recovered, but 1 died later from perforation of ulcer during a subsequent gastrolysis.

(3) *Perforation—general peritonitis.*—Two cases, with 1 death due to pneumonia. *Local peritonitis.*—Two cases, with 1 death, anastomosis with pyloric part of hour-glass stomach.

(4) *Adhesions.*—Ten cases, with 2 deaths—1 due to perforation subsequent to the operation, 1 due to post-operative peritonitis.

(5) *Pyloric stenosis.*—Twenty-four cases, with 5 deaths —1 by vicious circle, 1 by blocking of the anastomotic opening, 1 by peritonitis through leakage at anastomosis, 1 by peritonitis through leakage at site of old ulcer after the operation, 1 by immediate collapse.

(6) *Tetany.*—One case; fatal.

(7) *Hour-glass stomach.*—One case; recovered.

One patient was admitted twice with perforation of a peptic ulcer in jejunum; suture; recovery. One patient was admitted with perforation of a peptic ulcer, the original gastro-enterostomy having been performed in another hospital; suture, recovery.

In 36 cases of recovery the final results are:

No improvement	2
Slight benefit	5
Good result or complete cure	17
Not traced	2
Too recent for verdict, but discharged well	9
Death from other disease later	1

Table of Results.

Case.	Condition.	Operation.	Recovery or death.	After-history.	Cause of death.
2	Hæmatemesis	Anterior gastro-enterostomy	R.	Hæmorrhage ceased, but no great benefit	—
3	„	Anterior operation with entero-enterostomy	R.	No great benefit	—
4	„	Anterior operation	R.	Good result	—
5	Chronic ulcer	Posterior operation, ulcer excised and sutured	R.	„	—
6	Hæmatemesis	Posterior "no loop" operation	R.	Immediate result good	Died 2 months later from general tubercle of lungs.
70	Perforation, general peritonitis	Posterior operation at same time as suture of ulcer	R.	—	—
71	Ditto	Ditto	D.	—	Pneumonia.
76	Adhesions	Anterior operation	D.	Died on 19th day	Perforation of ulcer subsequent to operation, localised abscess between stomach and liver, the anastomosis sound.
79	Perforation and local abscess	„ „	D.	Died on 8th day	Anastomosis sound, had been made with pyloric part of hour-glass stomach.
84	Peri-pyloric abscess	„ „	R.	Not traced	—
90	Adhesions	„ „	R.	No improvement, symptoms continue 3 years later	—
91	„	Posterior operation	R.	No improvement 2 years later	—
93	„	Anterior operation, with gastrolysis	R.	Some improvement	—
94	„	Anterior operation	R.	Not traced	—

Case.	Condition.	Operation.	Recovery, or death.	After-history.	Cause of death.
95	Chronic adherent ulcer	Posterior operation	D.	—	On 27th day gastrolysis; perforation of ulcer, general peritonitis. Anastomosis had been made with pyloric part of hour-glass stomach.
96	Adhesions	Anterior operation	D.	—	Post-operative peritonitis. Streptococcus and staphylococcus.
97	„	Posterior operation	R.	Too recent for result to be known	—
99	„	Anterior operation	R.	Discharged well	Died 6 months later from phthisis.
101	„	„ „	R.	Final result unknown	—
102	„	„ „	R.	Good result 3 years later	—
105	Pyloric stenosis	„ „	R.	„ „	—
106	„ „	„ „	R.	Good result, but perforation of peptic ulcer of jejunum later; suture; recovery	—
109	„ „	„ „	D.	—	Vicious circle and vomiting.
110	„ „	„ „	R.	Final result unknown	—
111	„ „	„ „	R.	Good result 18 months	—
112	„ „	„ „	R.	Slight improvement	—
113	„ „	„ „	D.	—	Peritonitis through leakage at anastomosis.
114	„ „	„ „	D.	—	Peritonitis through leakage at site of adherent ulcer after operation.
115	„ „	Anterior operation and entero-enterostomy	R.	Final result unknown	—
116	„ „	Posterior operation	R.	„ „	—
117	„ „	„ „	R.	Improved at first; 6 months later recurrence. Second gastro-enterostomy; good result	—

118	„ „	„ „	R.	Discharged well	—
119	„ „	Anterior operation	R.	„ „	—
122	„ „	„ „	R.	Good result 4 years later	—
123	„ „	„ „	D.	Died in 1 hour	Death from collapse.
124	„ „	Posterior operation	D.	Vomiting till death on 7th day	Anastomotic opening blocked by mucous membrane.
125	„ „	Anterior operation	R.	Good result 2 years later	—
126	„ „	„ „	R.	Good result 17 months later	—
127	„ „	Posterior "no loop" operation	R.	Ditto.	—
128	„ „	Anterior operation	R.	Good result 10 months later	—
129	„ „	„ „	R.	Ditto.	—
130	„ „	Posterior operation and entero-enterostomy	R.	Very little improvement 16 months later	—
131	Pyloric stenosis and tetany	Posterior and anterior operation with double entero-enterostomy	D.	—	General peritonitis, junctions sound.
132	Hour-glass stomach	Anterior operation	R.	Great improvement 2 years later, but pyloric part of stomach does not completely empty itself	—
36	Pyloric stenosis	„ „	R.	Four years later quite well, can eat any food, never sick, gained 1 stone	—
39	Chronic ulcer	„ „	R.	Improvement but for perforation of peptic ulcers	—
66	Pyloric stenosis	„ „	R.	Twenty months later quite well, can eat any food	—

SPECIAL DISCUSSION

ON

THE OPERATIVE TREATMENT OF NON-MALIGNANT ULCER OF THE STOMACH AND ITS CHIEF COMPLICATIONS, WITH INDICATIONS, LIMITATIONS, AND ULTIMATE RESULTS.

REPORT ON CASES TREATED AT ST. GEORGE'S HOSPITAL

By C. R. KEYSER, F.R.C.S., Senior Assistant Surgeon at the Cancer Hospital; late Surgical Registrar at St. George's Hospital.

Gastric Ulcer.

Statistics of cases medically treated. *J. W. Russell,* '*Lancet,*' *vol. I,* 1904.

47 *patients*:

2·1 per cent. died of the disorder and 4·3 per cent. died of intercurrent maladies.

42·6 per cent. have ended in recovery, 27·7 per cent. having had but a single attack, and 14·9 per cent. having recovered after one or more relapses.

44·7 per cent. were suffering from stomach symptoms of more or less severity at time of last report, 14·9 per cent. getting repeated and definite attacks with intervals of immunity, and 29·8 suffering from almost continuous pain.

Statistics of cases surgically treated.

Collected by author from 1898—1906 :

18 cases, 8 deaths.

Direct treatment 9 cases, 3 deaths.

Indirect „ 10 „ 4 „

The Cancer Hospital. Collected by author 1905–6.

6 cases, 0 deaths.

Indirect treatment 6 cases, 0 deaths.

Perforated Gastric Ulcer.

Statistics of operative treatment at St. George's Hospital from 1895—1906.

Collected by author. 51 cases, 17 deaths.

Boston City Hospital and Massachusetts General Hospital, 1898—1903 inclusive. 18 cases, 13 deaths.

Carney Hospital. J. C. Munro. 7 cases, 4 deaths.

1 *case reported by J. Porter Parkinson.* 'Trans. Study of Diseases in Children,' 1901. *Male, aged* $2\frac{2}{12}$.

1 *case reported by R. V. Aitken.* 'B. M. J.,' 1904. *Perforation twice in* 5 *months.*

3 *cases in which appendicitis was also present.*

2 *cases of double perforation of stomach.*

In several of the fatal cases where a post-mortem was performed a second or even a third ulcer was found, showing that gastric ulcers are frequently multiple.

Early operation is desirable, but in many cases where operation was delayed recovery followed, notably in one case where 52 hours elapsed between perforation and operation.

Hæmorrhage from Ulcer.

5 *cases collected by author.*

1. Gastrotomy performed, no cause found. Death. P.M.—4 tiny ulcers found, 1 eroding an artery.

2. Gastrotomy performed, no cause found. Recovery. ? gastric erosion.
3. Post. gastro-jejunostomy, no cause found. Death. ? gastric erosion.
4. No operation. Death. Large chronic ulcer found.
5. No operation. Death. Large ulcer perforating coronary artery.

Boston City Hospital, etc., 1898—1903. 10 cases, 10 deaths.

PYLORIC STENOSIS AND GASTRIC DILATATION.

Cases, surgically treated, collected by author. 27 cases, 4 deaths = 14·8 per cent.

PYLOROPLASTY, 7 cases: 2 cured, 1 died, 4 had recurrence of symptoms necessitating a second operation.

GASTRO-JEJUNOSTOMY. 25 cases: 2 deaths = 8 per cent.

Ant. operation	*Suture.*	4 cases:	0 deaths.
	Bobbin or Button	10 cases:	2 deaths.
Post. operation	*Suture.*	6 cases:	0 deaths.
	Bobbin or Button	2 cases:	0 deaths.
Inferior operation.		1 case:	0 deaths.
Y-operation.		2 cases:	0 deaths.
Pylorectomy.		3 cases:	1 cured, 2 died.

THE CANCER HOSPITAL. Collected by author 1905–6.

Gastro-jejunostomy. Post: operation 8 cases: 0 deaths.

In 1 case the Y-operation was performed, and in 1 other case entero-enterostomy had to be performed, subsequently to the ordinary operation.

BOSTON CITY HOSPITAL.—PYLOROPLASTY. 9 cases: 2 cured, 2 died, 5 symptoms recurred.

Gastro-jejunostomy. 28 cases: 12 cured, 8 died, 8 unrelieved.

Post. Gastro-jejunostomy
- *Suture.* 12 cases: 6 cured, 3 died, 3 unrelieved.
- *McGraw ligature* } 3 cases: 1 cured, 2 unrelieved.

Ant. gastro-jejunostomy
- *Murphy's button.* 6 cases: 1 cured, 4 died, 1 unrelieved.
- *McGraw ligature.* 5 cases: 1 cured, 4 unrelieved.
- *Suture.* 2 cases: 2 cured.

Pylorectomy. 3 cases: 1 cured, 2 died.

Gastro-jejunostomy for Simple Pyloric Stenosis.

Name.	Sex.	Age.	History.	Date of operation.	Condition found.	Result.	Remarks.
T. McQ.	M.	34	8 years chronic indigestion	July 18, 1896	Pyloric stenosis	Died 10 days later	Anterior operation by Senn's plates. Death due to constant vomiting and bronchitis.
A. D.	M.	42	14 years pain and vomiting after food	Oct. 21, 1895	Pyloric stenosis; adherent to liver	Cured	Anterior operation by Senn's plates.
M. U.	F.	38	4 months "lump" noticed in abdomen; vomits 2 to 3 hours after food	March 26, 1896	Pyloric stenosis	„	Anterior operation by Senn's plates. June, 1904.—No pain or vomiting, but has to be careful in diet.
S. W.	M.	48	7 years pain and vomiting after food	Dec. 1, 1896	Pyloric stenosis	„	Anterior operation by Senn's plates. Rapid improvement.
W. McC.	F.	27	5 years pain and vomiting after food	Oct., 1903	Pyloric stenosis	„	Anterior gastro-jejunostomy. Pyloroplasty. Oct, 1900.—Symptoms recurred in 3 months.
A. H.	M.	44	9 months pain and vomiting after food	May 22, 1900	Pyloric stenosis; adherent to liver	„	Anterior operation by Allingham's bobbin. Steady improvement.
C. D.	M.	47	9 years pain after food; vomits occasionally	Dec. 27, 1900	Pyloric stenosis	„	Posterior operation. Perfect health till Aug., 1903, when ? symptoms of cancer of stomach set in.
T. D.	M.	50	17 years vomiting, especially at night	Jan. 31, 1901	Pyloric stenosis	„	Posterior operation.
J. C.	M.	54	Pain and vomiting for "years"	June 25, 1903	Pyloric stenosis	„	Anterior operation by Murphy's button. 5 months later button still retained.

Gastro-jejunostomy for Simple Pyloric Stenosis (continued).

Name.	Sex.	Age.	History.	Date of operation.	Condition found.	Result.	Remarks.
G. B.	M.	33	1 year pain and vomiting after food; 9 years "indigestion"	Nov. 20, 1903	Pyloric stenosis	Cured	Posterior operation by Allingham's bobbin. June, 1904.—"Getting fat."
O. G.	F.	40	4 years pain and vomiting after food	Nov. 14, 1903	Pyloric stenosis	,,	Inferior operation, followed by constant vomiting. 1 week later lateral anastomosis of proximal and distal loops of jejunum. Perfect recovery followed.
G. H.	M.	32	"Indigestion" as long as he can remember	Jan, 11, 1903	Pyloric stenosis	,,	Posterior operation by Robson's bobbin. Uninterrupted recovery.
F. B.	F.	43	9 years symptoms of gastric ulcer	Jan. 13, 1903	Pyloric stenosis; ulcer present	Died Jan. 15	Anterior operation by Murphy's button. ? cause of death; none found P.M.
E. W.	F.	57	"Indigestion all her life"	Jan. 20, 1904	Pyloric stenosis	Cured	Posterior operation. Some vomiting for 1 week.
J. R.	F.	47	18 years pain and vomiting	Feb. 7, 1904	Pyloric stenosis; ulcer present	,,	Roux's Y operation.
J. C.	M.	50	"For years" pain and vomiting	Feb. 18, 1904	Pyloric stenosis; many adhesions	,,	Posterior operation by Robson's bobbin.
R. L.	M.	55	20 years pain and vomiting	March 15, 1904	Pyloric stenosis	,,	Anterior operation. Sept., 1904.—Health excellent.
H. H.	M.	34	3 years pain and vomiting	May 4, 1904	Pyloric stenosis	,,	Roux's Y operation.

A. E.	F.	43	2 years pain and vomiting	April 12, 1904	Pyloric stenosis	„	Anterior operation. Pyloroplasty Jan., 1903. Symptoms recurred in 8 months.
E. D.	F.	49	30 years pain and vomiting	May 17, 1904	Pyloric stenosis	„	Anterior operation.
G. K.	M.	23	5 years pain and vomiting	March 3, 1904	Pyloric stenosis; ? cancer many glands	„	Anterior operation by Robson's bobbin. June, 1904—Still has much pain; no vomiting; is unable to walk.
W. B.	M.	34	1 year pain; vomited once, much blood	Sept. 18, 1904	Pyloric stenosis	„	Anterior operation.
W. R	M.	37	6 years pain and vomiting	Sept. 24, 1904	Pyloric stenosis	„	Posterior operation.
H. G.	M.	43	HCl swallowed 1 month before operation	Oct. 6, 1890	Pyloric stenosis	Died in 2 days	Case reported in 'Clin. Soc. Trans.,' 1891.
E. B.	F	46	HCl swallowed, attempted suicide, Sept. 13, 1900	Oct. 27, 1900	Pyloric stenosis	Died in 42 days	Case reported in 'Med.-Chir. Trans.,' vol. lxxxv. Anterior operation by Murphy's button.
J. P.	M.		Pain and vomiting 8 months	Dec. 8, 1905	Pyloric stenosis	Cured	Posterior operation.
S. T.	F.	38	?	Aug. 14, 1905	Pyloric stenosis	„	Posterior operation. Oct. 27, 1906. —Quite well; can eat anything.

Gastro-jejunostomy for Simple Pyloric Stenosis (continued).

Name.	Sex.	Age.	History.	Date of operation.	Condition found.	Result.	Remarks.
J. P.[1]	M.	—	Pain and vomiting for "years"; no hæmatemesis	Dec. 6, 1905	Pyloric stenosis	Cured	Posterior operation. Nov., 1906.—Some pain and vomiting occasionally.
E. D.[1]	F.	40	Pain and vomiting 1½ years; hæmatemesis many times	March 1, 1905	Pyloric stenosis	„	Posterior operation. Readmitted June 22, 1906, for constant pain and occasional attacks of vomiting. June 27.—Entero-enterostomy performed; jejunum at site of anastomosis kinked, due to axial rotation. Aug. 8, 1906.—Except for one attack of pain and vomiting has been quite well.
A. C.[1]	F.	47	Pain after food; vomiting very slight; tumour present	March 15, 1906	Pyloric stenosis	„	Posterior operation.
F. S.[1]	F.	23	Pain and vomiting 3 years	April 11, 1906	Pyloric stenosis	„	Posterior operation. Nov., 1906.—Some pain.
A. B.[1]	F.	31	7 years pain and vomiting; rarely hæmatemesis	May 17, 1906	Pyloric stenosis and adhesions	„	Posterior operation. June 26, 1906.—Better than she has been for years; no pain.
E. B.[1]	F.	51	Pain right side after food	May 21, 1906	Pyloric stenosis	„	Posterior operation. July 28, 1906.—No pain after food.
E. S.[1]	F.	33	Pain, vomiting, and hæmatemesis 14 years	July 11, 1906	Pyloric stenosis	„	Posterior Y operation. Perfect recovery, Nov., 1906.
E. S.[1]	M.	47	Pain and occasional vomiting 5 years	July 19, 1906	Pyloric stenosis and adhesions	„	Posterior operation. Uninterrupted recovery. Can eat anything.

[1] From the Records of the Cancer Hospital.

In no case did peptic ulcer of the jejunum follow.

Where method of operation is not stated simple suture was employed.

Gastric Ulcer.

Name.	Sex.	Age.	History.	Date of operation.	Condition found.	Result.	Remarks.
A. G.	F.	25	6 months dyspepsia	Jan. 10, 1898	Ulcer on point of perforation	Cured	Ulcer invaginated. Parotitis followed. June, 1904.—Has been absolutely free from pain.
L. S.	F.	66	30 years dyspepsia	Sept. 21, 1900	Ulcer anterior wall, adherent to parietes; excised	Died	Hæmatemesis Oct. 1, continued till death Oct. 2. P.M.—Hour-glass stomach; several more ulcers in stomach, also large ulcer in duodenum.
F. D.	F.	19	2 years dyspepsia; hæmatemesis once	June 30, 1900	Ulcer anterior wall, adherent to parietes; invaginated	Cured	April 2, 1902.—Much improved. Has baby 2 months old.
S. A.	F.	34	8 years pain and vomiting; hæmatemesis once	Nov. 7, 1901	Ulcer anterior wall; excision	,,	
E. D.	F.	38	14 years pain and vomiting, and hæmatemesis	Dec. 3, 1901	Ulcer posterior wall; excision	,,	Symptoms recurred April, 1904. June 28, 1904.—Gastrolysis.
G. A.	M.	31	3 years pain and vomiting	Feb., 1901	Ulcer anterior wall	Died	Anterior gastro-jejunostomy by Murphy's button. Died on 5th day of persistent vomiting.
M. H.	F.	31	Over 1 year pain and vomiting, and hæmatemesis	Sept. 22, 1903	Ulcer posterior wall	,,	Posterior gastro-jejunostomy. Died on 5th day of septic peritonitis.
E. H.	F.	48	6 years pain and vomiting, and hæmatemesis	June 19, 1901	Ulcer anterior wall; many adhesions; excision	Cured	Much pain followed. Posterior gastro-jejunostomy and entero-enterostomy July 7, 1904. Uninterrupted recovery.

Gastric Ulcer (continued).

Name.	Sex.	Age.	History.	Date of operation.	Condition found.	Result.	Remarks.
C. D.	M.	47	9 years pain and vomiting	Dec. 27, 1900	Ulcer upper part of pylorus	Cured	Posterior gastro-jejunostomy. Was well for 2 years, then symptoms of cancer.
E. B.	F.	48	7 years off and on pain, vomiting, and hæmatemesis	Sept. 2, 1902	Ulcer anterior surface; adhesions	„	Gastrolysis.
			Symptoms recurred in 1 year	Aug. 11, 1903	Adhesions	„	Anterior gastro-jejunostomy by Robson's bobbin. June, 1904.—Perfectly well; can eat and do anything.
F. R.	M.	63	6 months pain and vomiting; hæmatemesis twice	Jan. 16, 1904	Large ulcer at pylorus on anterior and posterior surfaces	Died	Anterior gastro-jejunostomy. Death on 5th day of collapse.
J. B.	M.	39	Several years pain and vomiting; hæmatemesis	July 1, 1903	Ulcer anterior wall; excision	„ next day	No P.M.
R. W.	F.	53	20 years pain after food; vomiting slight	Feb. 13, 1904	Ulcer at pylorus; adhesions	Cured	Anterior gastro-jejunostomy.
W. G.	M.	27	15 years dyspepsia off and on	July 25, 1904	Thought to be cancer on anterior wall; excision	Died	Death on following day of collapse. P.M.—Large simple ulcer.
A. G.	F.	42	For years dyspepsia; hæmatemesis 3 times	Jan. 11, 1904	Large ulcer lesser curvature and anterior surface	„	Death on 7th day. Operation done for hæmatemesis, which was very severe.

E. K.	F.	21	Pain and vomiting 17 months; no hæmatemesis	Dec. 10, 1904	—	Cured	Anterior operation. Vomited on and off for 3 weeks after operation.
C. D.	F.	30	Pain and vomiting 18 months	Feb. 1, 1906	? nature of ulcer; many glands	„	Posterior operation. Patient re-admitted March 9, 1906, for pain and vomiting; tumour present; ? cancer. May 18.—Laparotomy; tumour found close to cicatrix of ulcer anastomosis apparently efficacious; abdomen closed. Patient recovered. Nov., 1906.—Improving.
J. C.	M.	36	5 years pain; hæmatemesis several times; severe just before operation	July 20, 1906	—	Died next day	Anterior operation.
F. M.[1]	F.	30	6 years pain and vomiting	March 21, 1906	Ulcer on anterior wall	Cured	Posterior gastro-jejunostomy.
M. T.[1]	F.	22	For "years" pain and vomiting; hæmatemesis once	May 16, 1906	Ulcer present	„	Posterior gastro-jejunostomy. Patient has phthisis.
K. Q.[1]	F.	52	7 years pain and vomiting	July 4, 1906	Ulcer present	„	Posterior gastro-jejunostomy.
G. B.[1]	F.	22	2 years hæmatemesis on and off; also pain after food	Aug. 1, 1906	Ulcer present	Improved	Posterior gastro-jejunostomy. Still in hospital. Has pain and vomiting occasionally.
A. S.[1]	M.	43	10 months pain and vomiting; hæmatemesis once	Aug. 30, 1906	Ulcer present at pylorus	Cured	Posterior gastro-jejunostomy.
A. B.[1]	F.	35	8 months pain and vomiting	Oct. 16, 1906	Ulcer present and pyloric stenosis	Improved	Posterior gastro-jejunostomy. Y operation. Still in hospital. Much improved.

[1] From the Records of the Cancer Hospital.

Sex.	Age.	Previous gastric symptoms.	Hæmatemesis.	Operation.	Date.	Result.	Remarks.
F.	20	Dyspepsia lately	No	Suture of ulcer, irrigation and drainage	Jan., 1895	Died next day	General peritonitis; two other ulcers found.
M.	23	?	No	Suture of ulcer, irrigation and drainage	May, 1896	Recovery	Ulcer on anterior surface.
F.	28	Pain and vomiting	No	Suture of ulcer, irrigation and drainage	Nov., 1900	,,	Patient walked up to hospital.
F.	26	Indigestion	No	Suture of ulcer, drainage	Nov., 1897	,,	Operation 52 hours after perforation; slight dyspepsia since.
F.	30	Pain and vomiting	Once	Suture of ulcer, irrigation only	Mar., 1898	,,	Operation 9½ hours after perforation.
F.	30	Indigestion	No	Suture of ulcer, drainage	Dec., 1897	,,	
F.	23	Indigestion	Once	Suture of ulcer, irrigation and drainage	Dec., 1898	Died on 4th day	Intense peritonitis; pus in pelvis.
F.	24	Indigestion	No	Suture of ulcer, no drainage	Feb., 1899	Recovery	Operation 4½ hours after perforation.
F.	21	Indigestion	No	Suture of ulcer, no drainage	Mar., 1899	,,	Operation 17½ hours after perforation.
F.	24	?	?	Suture of ulcer, irrigation and drainage	Nov., 1900	,,	
M.	$2\frac{2}{12}$	—	—	—	—	Died 60 hours later	Reported by J. P. Parkinson. 'Trans. Study of Diseases in Children,' 1901, perforation on posterior surface.
F	28	Indigestion, 10 years	No	Ulcer on posterior surface, packed with gauze	Mar., 1901	Died 2 hours later	Acute general peritonitis.
F	23	Indigestion	No	Suture of ulcer, irrigation	July, 1901	Recovery	Operation 22 hours after perforation. Left empyema followed.
M.	48	Indigestion, 12 years	No	Suture of ulcer	June, 1901	Died on 16th day	Melæna started on 12th day; ulcer eroded gastro-duodenal artery.
F.	19	?	?	Suture of ulcer, drainage	Oct., 1901	Died on 5th day	Diagnosed first as appendicitis.
F.	23	Indigestion for years	No	Suture of ulcer, irrigation and drainage	June, 1902	Recovery	Left pleurisy developed.

F.	19	?	?	Suture of ulcer, no drainage	Feb., 1902	Recovery	Operation 9½ hours after perforation.
F.	17	Indigestion, 1 year	No	Ulcer on posterior surface, gauze packing	Feb., 1902	„	Patient in good health one year after.
F.	25	Indigestion, for years	No	Suture of ulcer, irrigation and drainage	Nov., 1902	„	Patient in hospital at time of perforation.
M.	52	Indigestion, 2 years	Once	Suture of ulcer	Nov., 1902	Died on 3rd day	Second ulcer found close to first.
F.	30	Indigestion, 1 year	No	Suture of ulcer, drainage	Jan., 1903	Recovery	Operation 6½ hours after perforation; double parotitis developed.
F.	23	Indigestion, 1½ years	3 times	Suture of ulcer, drainage	Jan., 1903	„	Operation 17 hours after perforation; pelvis drained.
F.	18	Indigestion, 2 months	No	Suture of ulcer, irrigation	Jan., 1903	„	Operation 9 hours after perforation.
F	26	Gastric ulcer, 3¼ years	5 times	Suture of ulcer, irrigation	Feb., 1903	„	Operation 37 hours after perforation; left pleurisy developed.
F	29	Gastric ulcer, 8 years	No	Suture of ulcer, irrigation	Jan., 1903	„	Operation 36 hours after perforation; left pleurisy and left parotitis developed.
M.	63	Indigestion, 25 years	No	Suture of ulcer, irrigation	July, 1903	Died on the table	Operation 3 hours after perforation; second ulcer found in stomach.
F	26	None	No	Suture of ulcer, no drainage or irrigation	April, 1903	Recovery	Operation 24 hours after minute perforation; old ulcer found.
F	43	Gastric ulcer, 2 years	Occasionally	Suture of ulcer, drainage	April, 1903	„	Operation 14½ hours after perforation.
F	27	Indigestion, 3 months	No	Suture of ulcer, drainage	Mar., 1904	„	Operation 5½ hours after perforation; occurred during sleep.
F	29	Gastric ulcer for years	No	Suture of ulcer, drainage and irrigation	Feb., 1903	„	Operation 17 hours after perforation; parotitis and pneumonia developed. (Same patient.[1])
				Suture of ulcer	July 18, 1903	„	Operation 25 hours after perforation (Same patient.[1])

[1] Reported by R. V. Aitken, 'Brit. Med. Journ.,' 1904.

Perforated Gastric Ulcer (continued).

Age.	Sex.	Previous gastric symptoms.	Hæmatemesis.	Operation.	Date.	Result.	Remarks.
F.	32	Indigestion, 6 years	Once	Suture of ulcer	Jan., 1904	Recovery	Operation 9 hours after perforation; pelvis drained.
F.	23	Indigestion, 7 years	No	Suture of ulcer, irrigation	Feb., 1904	,,	? Symptoms started 10 days before operation; diagnosis uncertain.
F.	16	Indigestion, several months	No	Suture of ulcer, irrigation	April, 1904	Died on 5th day	General peritonitis; second ulcer found.
M.	42	Indigestion, 3 years	?	Suture of ulcer, irrigation and drainage	April, 1904	Died on 5th day	No post mortem.
F.	24	Indigestion, 2 years	No	Suture of ulcer, irrigation and drainage	Mar., 1904	Recovery	Died one month later from sub-diaphragmatic abscess; no post mortem.
M.	23	None	No	Suture of ulcer, irrigation and drainage	April, 1904	,,	Patient walked to hospital; symptoms indefinite.
F.	23	Indigestion, 1 month	No	Appendix explored	Jan., 1904	Died in 1¼ hours	Perforated gastric ulcer; general peritonitis.
F.	21	None	No	Appendix explored, suture of ulcer in stomach	May, 1904	Died next day	No post mortem.
F.	41	Indigestion, 21 years	No	Suture of ulcer, irrigation	June, 1904	Died on 4th day	Operation 6¾ hours after perforation; no post mortem.
M.	13	—	—	Suture of ulcer, irrigation and drainage	Mar., 1904	Recovery	Reported by W. Watson Cheyne, 'Lancet,' 1904. Appendicitis also present.
F.	7	Reported by Lediard and Sedgwick, 'Lancet,' 1904	—	Unsuccessful attempt to suture ulcer	—	Died	? Appendicitis also present.
F.	17	Indigestion, 1 year	No	Suture of ulcer, irrigation and drainage	—	Recovery	Appendicitis also present. Reported by V. Warren Low, 'Lancet, 1904.
F.	16	Indigestion, 1 year	No	No operation. Death immediate after occurrence of pain	—	—	Double perforation of stomach. Reported by L. Lovell Keays, 'Brit. Med. Journ.,' vol. ii, 1903.
M.	10	—	—	No operation	—	—	Double perforation of stomach. Reported by Barber, 'Brooklyn Med. Journ.,' Dec., 1902.

Perforated Gastric Ulcer (continued).

Sex.	Age.	Previous gastric symptoms.	Hæmatemesis.	Operation.	Date.	Result.	Remarks.
F.	36	Symptoms of ulcer for 19 years	Twice	Suture of ulcer, irrigation and drainage	Dec. 25, 1904	Recovery	Nov., 1906.—Some pain at times.
F.	21	Symptoms of dyspepsia previously	No	Suture of ulcer, irrigation and drainage	Feb. 6, 1905	Died in 6 weeks	P.M. — Pericarditis, pneumonia, thrombosis and sub-phrenic abscess.
F.	21	Pain and vomiting, 3 months	No	Suture of ulcer, irrigation and drainage	Feb. 21, 1905	Recovery	Nov., 1906.—Quite well.
F.	19	6 months dyspepsia	No	Suture of ulcer, irrigation and drainage	May 18, 1905	Died on 15th day	Operation 6½ hours after perforation. P.M.—Septic peritonitis.
F.	26	"Always" pain and vomiting	No	Tissues too rotten to hold sutures. Omental graft. Irrigation and drainage	Sept. 10, 1905	Recovery	Nov., 1906.—Quite well.
F.	24	4 years dyspepsia	No	Could not be sutured owing to position of ulcer. Gauze plug, irrigation and drainage	Sept. 18, 1905	Recovery	Ulcer at cardiac end on posterior surface; convalescence long. Nov., 1906.—Quite free from pain.
F.	23	"Previous dyspepsia"	No	Suture of ulcer, irrigation and drainage	Nov. 6, 1905	Recovery	Operation 3¾ hours after perforation. Nov., 1906.—Quite well.
F.	25	Vague history of dyspepsia	No	Suture of ulcer; drainage	Mar. 24, 1906	Died on 13th day	Operation 16 hours after perforation. P.M.—Septic peritonitis.
M.	40	?	—	Suture of ulcer	April 6, 1906	Died on 5th day	No post mortem.
M.	27	?	—	Suture of ulcer	April 21, 1906	Died next day	No post mortem.
M.	22	Practically no symptoms	No	Drainage	July 4, 1906	Recovery	Operation one week after admission. Nov., 1906.—Quite well.

? Gastric Erosion.

Name.	Sex.	Age.	History.	Date of operation.	Condition found.	Result.	Remarks.
H. S.[1]	F.	59	6 years slight epigastric pain; in last 4 months three attacks of hæmatemesis	June 27, 1906	Gastrotomy; cause of bleeding not discovered; some glands enlarged in omentum, one removed for examination; only inflammatory changes seen	Improved	Pain continued. Patient advised to have gastro-jejunostomy, but refused.

[1] From the Records of the Cancer Hospital.

SPECIAL DISCUSSION

ON

THE OPERATIVE TREATMENT OF NON-MALIGNANT ULCER OF THE STOMACH AND ITS CHIEF COMPLICATIONS, WITH INDICATIONS, LIMITATIONS, AND ULTIMATE RESULTS.

REPORT ON 334 CASES OF OPERATION FOR NON-MALIGNANT DISEASE OF THE STOMACH.

Remarks contributed by B. G. A. MOYNIHAN, M.S., F.R.C.S.

In the following report I have analysed and made brief comment upon all the cases of non-malignant disease of the stomach which I had operated upon up to September, 1906. For convenience of description the cases are arranged in four groups:

	Operations.	Recoveries.	Deaths.
Group 1. Cases of perforating ulcer . . .	27	18	9
„ 2. Cases in which recurrent hæmorrhage was the cause of immediate operation . .	33	27	6
„ 3. Miscellaneous cases, given in detail later	248	246	2
„ 4. Hour-glass stomach	26	22	4
Total . .	334	313	21

GROUP 1.—PERFORATING ULCER OF THE STOMACH OR DUODENUM—27 CASES, 18 RECOVERIES, 9 DEATHS.

The perforation of an ulcer of the stomach is a catastrophe which, in my experience, never comes unannounced. In all patients there has been trouble, of a greater or less degree of severity, from the ulcer. In many of the patients the symptoms have persisted for months, and in some they have been of greater severity in the weeks or days preceding the rupture of the ulcer.

When perforation occurs there is a sudden agonising pain, a pain which almost reaches the limit of human endurance. But there is neither shock nor collapse as a rule. The pulse at the first is not rapid, and the abdominal muscles may not be rigid. It is as well to insist upon these points; for now that the surgeon is asked to see these cases almost immediately after the occurrence of the perforation, delay may be considered necessary if the pulse-rate is not quickened, and if collapse and rigidity of the abdominal muscles are absent. Exquisite tenderness of the skin I have always found. The least stroking of the surface with the finger is resented. Tenderness and pain on pressure, though general, are often present in a greater degree at one part than at another. So constantly is this the case that a careful examination of the abdomen will almost always permit of an accurate localisation of the position of the ulcer which has ruptured. The onset of those symptoms formerly considered as necessary to a diagnosis—collapse, abdominal distension, rapidity and smallness of the pulse—should not be allowed to develop. A diagnosis can be made from the history of ulcer, the sudden onset and the continuance of intolerable pain, restriction of thoracic movements, surface tenderness, and abdominal rigidity or restriction of the normally free abdominal movements.

Early operation is indicated in all cases of perforation.

The incision should be made to left or right according to the position of the ulcer as ascertained by examination. The ulcer should be closed and infolded; excision is not necessary. If the ulcer be near the pylorus (on either side), and if its closure is at all likely to cause an obstruction, or if a second ulcer be seen in the vicinity of the pylorus, then gastro-enterostomy should be performed; in other cases it is not necessary. Drainage is only necessary in the late cases. When necessary it should be conducted through a supra-pubic incision and the sitting posture be insisted upon afterwards.

Record of cases.—I have had under my care 27 cases of perforating ulcer of the stomach or duodenum. In these 27 cases there were 18 recoveries (66·6 per cent.) and 9 deaths (33·3 per cent.). There were 10 cases in which a duodenal ulcer had perforated, 17 in which a gastric ulcer had perforated. In the first 10 cases there were 6 deaths; in the last 17 cases there were 3 deaths.

The ages of the patients varied from 17 to 44. The cases of gastric ulcer were 17 in number; of these 3 were males, aged 29, 23, and 24; 14 females of average $27\frac{1}{12}$. The cases of duodenal ulcer were 10 in number; of these 5 were males, aged 44, 25, 22, 29, 40; 5 females aged 17, 17, 25, 29, 38.

The perforation was found on the anterior surface of the stomach in 15 cases, on the posterior surface in 2 cases. In 2 cases two perforations were found. In 1 both were on the anterior surface, about $1\frac{1}{2}$ inches apart. In the other 2 "kissing" ulcers had perforated.

In 1 case the perforation occurred in the centre of an hour-glass stomach, and gastroplasty had to be performed. In the 10 duodenal cases the perforation was found in the first portion 9 times, in the beginning of the second portion once. In 6 cases in the whole series gastro-enterostomy was performed immediately after the closure of the perforation; in 5 of these cases recovery followed. In 2 cases the subsequent performance of gastro-enterostomy has been necessary owing to the persistence of

symptoms due to the ulcer or to the scar left by it. There were, therefore, 18 cases of recovery from the perforation; in 5 of these gastro-enterostomy had been performed and in 1 gastroplasty for an hour-glass stomach; of the remaining 12, 2 subsequently suffered to such a degree as to necessitate the performance of a second operation—gastro-enterostomy.

The present condition of all the patients in this series is perfectly satisfactory.

Group 2.—Hæmorrhage Cases.

The classification of cases of hæmorrhage from gastric or duodenal ulcer which I proposed in a paper read before the American Surgical Association in 1903 (see 'Brit. Med. Journ.,' vol. i, p. 1363), is that which I still adopt. Hæmorrhage may occur in *acute ulcer* or in *chronic ulcer*. Hæmorrhage from an acute ulcer is characterised by spontaneity, abruptness of onset, the rapid loss of a large quantity of blood, the marked tendency to spontaneous cessation, the infrequency of a repetition of the hæmorrhage in anything but trivial quantity, and the transience of the resulting anæmia.

Hæmorrhage from a chronic ulcer may be divided into four classes, the characteristics of which are briefly:

(1) The hæmorrhage is latent or concealed, is always trivial and often inconspicuous.

(2) The hæmorrhage is intermittent, but in moderate quantity, occurring spontaneously and with apparent caprice at infrequent intervals. The life of the patient is never in jeopardy from loss of blood, though anæmia is a persisting symptom.

(3) The hæmorrhage occurs generally, but not always, after a warning exacerbation of chronic symptoms. It is rapidly repeated, is always abundant, its persistence and excess cause grave peril, and will, if unchecked, be the determining cause of the patient's death.

(4) The hæmorrhage is instant, overwhelming, and lethal.

In acute ulceration, under which term probably many different conditions are included, operation is never necessary. In chronic ulceration hæmorrhage is the cause of urgent operation only in those cases included in Section 3. It is the repetition of the hæmorrhage in increasing quantity and at decreasing intervals that is the cause, the only cause, demanding operation for its instant relief.

When operation is undertaken the surgeon should have in view as his immediate object the securing of the vessel which is bleeding. If the vessel can be found, and if it is accessible, it should be ligatured. But this counsel of perfection cannot always be followed, for there may be more ulcers than one in the stomach, and evidence as to which is the offender is lacking; or no ulcer may be discoverable; or the ulcer when found may be of such size and so indurated that a large tumour is formed in the stomach or duodenum (in one of my cases there was an inflammatory mass as large as a billiard ball), a tumour of such size that its removal would involve a serious and prolonged operation unsuited to the patient's weak condition. In place of the ligature of the bleeding vessel, the excision of the ulcer may be performed; this is certainly preferable. But a far simpler method, one I have adopted on many occasions, is to infold the ulcer with a double row of sutures in exactly the same manner as if the ulcer had perforated. This infolding of ulcers should always be performed when feasible. In all cases of duodenal ulcer, as I have before advised, infolding of the ulcer, or the closure of the duodenum or pylorus on the proximal side of the ulcer is necessary.

In my own cases gastro-enterostomy alone has been performed in many cases where hæmorrhage was the cause of operation. The result has been satisfactory in every instance; there has not been one single recurrence of the bleeding in any case. In my recent cases I have, however, always combined infolding of the ulcer

with gastro-enterostomy, and this I now consider the safest and best procedure.

Record of cases.—In 33 cases I have operated for the relief of severe recurrent hæmorrhage. The bleeding occurred as follows in 31 cases; in 2 cases no ulcer was found.

	Hæmatemesis alone.	Melæna alone.	Hæmatemesis and melæna.
Gastric ulcer	7	0	9
Duodenal ulcer	0	4	7
Gastric and duodenal ulcers	1	0	3

There were 16 females among the 33 cases and 17 males. The distribution of the ulcers among them is shown in the next table:

	Gastric.	Duodenal.	G. and d.	No ulcer found.
Males	6	8	2	1
Females	10	3	2	1

It is evident that, while the sexes appear to be equally liable to hæmorrhage from an ulcer, yet as regards the source of the hæmorrhage in men 50 per cent. of the bleeding ulcers are in the duodenum, while in women the majority, 66·6 per cent., are situated in the stomach. In other words, about 73 per cent. of bleeding duodenal ulcers occur in men, while of the gastric ulcers which bleed 62·5 per cent. are in women.

Mortality.—Six of these cases died (18 per cent.), one in four hours from the operation, at which no ulcer could be found. At the autopsy dilated gastric veins and cirrhosis of the liver were demonstrated. One became suddenly collapsed, and died in a few minutes at the end of a week; the cause of death was not known. One died on the eleventh day from pneumonia, one on the eighteenth day from exhaustion, one in three weeks from gradual exhaustion (in this case the blood contained only 27 per cent. hæmoglobin before operation), one a few hours after operation from shock and exhaustion. The patient was a man, aged 62, who had bled repeatedly and copiously from a large pyloric ulcer.

In no case did hæmorrhage recur after operation. In three of the cases which recovered the estimation of hæmoglobin before operation was less than 30 per cent.

Operative treatment.—Posterior gastro-jejunostomy was performed in every case save one. In this case the ulcer was excised; the patient died. In 21 gastro-jejunostomy was the only treatment adopted; 2 of these died. Gastro-jejunostomy was combined with excision of the ulcer in 2 cases, 1 of which died. Gastro-jejunostomy, with infolding of the ulcer, was done 8 times; 2 patients died. Gastro-jejunostomy, with infolding of the pylorus and of the ulcer, was performed once; the patient recovered.

After-results.—All the patients in this series are quite well, and may be considered "cured." One patient suffers a little from the painless vomiting of bile at infrequent intervals. In all other respects she is quite well, suffers no indigestion, and has gained about 2 stone in weight.

Group 3.—Cases of Chronic Gastric and Duodenal Ulcer, etc.

The problems which are raised by a consideration of the cases included in this section are too complex for anything in the nature of a detailed examination in the brief time at one's disposal now. The great majority of the cases, however, may be summarily dismissed by the simple statement that in cases of pyloric or duodenal obstruction gastro-enterostomy is the most efficient operation in affording relief. If the ulcer be active, it should be infolded or excised; the former by preference as being easier, quicker, and equally effective. If the ulcer area be extensive, multiple ulcers being found in the pyloric region of the stomach with, perhaps, an ulcer or ulcers beyond the pylorus, Rodman's operation—that is, excision of the ulcer-bearing area—may be practised. If an ulcer in the pyloric region has to be excised the wound left after its removal may be enlarged to permit of Finney's operation being performed. My own opinion is very

emphatically in favour of posterior gastro-enterostomy for all cases of simple ulcer, whether single or multiple, involving the pyloric region of the stomach or the duodenum. With gastro-enterostomy the infolding of the ulcer should be combined.

When the ulcer is seated along the lesser curvature of the stomach it is better to excise it; in a few cases it is possible to infold it, but excision is probably safer though certainly more difficult. Gastro-enterostomy must be combined with excision if there is a second ulcer near the pylorus. If the ulcer on the lesser curvature is solitary, gastro-enterostomy is not necessary when the ulcer is excised, and is of very little use when the ulcer, if large, is allowed to remain. I have twice performed gastro-enterostomy alone in such conditions, and though both the patients have improved, they are not quite well. In three cases I have dealt directly with the large ulcer, and in all the result has been perfectly satisfactory.

One of the chief difficulties in the early surgical work upon diseases of the stomach was concerned with the making of an accurate diagnosis. Now, after we have been able in a large number of cases to set side by side the details of the clinical history and the disclosures of the operation table, the difficulties are very considerably lessened. For example, duodenal ulcer in a very great proportion of cases can be diagnosed without any physical examination of the patient. The story he tells in his anamnesis might often have been learnt by heart from a written description, so exactly are the details, even the hours of the onset of pain, recited. But in all cases accurate observations should be made as to the length of stay of food in the stomach; the chemical reactions, the bacterial flora, the digestion leucocytosis should all be investigated. Repeated stasis of food for over twelve hours means organic disease; it is not found apart from organic disease. I place a high value, though not a certain reliance, upon the chemical analysis. In addition, the surface examination of the abdomen by Mackenzie's

method enables one quite frequently to localise the position of an ulcer. From every side help is coming, and we are now able to make an accurate diagnosis of ulcer and of its situation, in the duodenum or stomach, and if in the stomach, of its whereabouts therein, in certainly the great majority of the cases.

Case records.—The operations in this series number 248, and were performed on 239 patients. There were 2 deaths. The following classification of the cases seems the most satisfactory:

	Cases.	Deaths.
(*a*) Gastric ulcer Duodenal ulcer Gastric and duodenal ulcer	206	2
(*b*) Cholelithiasis, with gastric or duodenal ulcer	5	0
(*c*) Cholelithiasis causing obstruction	6	0
(*d*) Gastroptosis; no ulcer found	3	0
(*e*) No ulcer found	6	0
(*f*) Pyloroplasty; in two of these gastro-enterostomy had to be performed later (see *h*)	3	0
(*g*) Secondary operations, the primary operation elsewhere	10	0
(*h*) Secondary operations, on cases under headings (*a*) and (*f*).	9	0

Class A.

In this series are 206 cases, 91 males and 115 females. Gastric ulcer alone was found in 138 cases. Duodenal ulcer alone was found in 40 cases. Gastric and duodenal ulcers combined were found in 28 cases. The proportions occurring in the two sexes are shown thus:

	Males.	Females.
Gastric ulcer alone was found in	42	96
Duodenal ulcer alone was found in	30	10
Gastric and duodenal ulcers together in	19	9

Therefore gastric ulcer alone occurred more than twice as often in women as in men, while duodenal ulcer alone is three times as frequent in men as in women. If all the figures are combined, it is seen that over 70 per cent. of duodenal ulcers are found in men.

Class B.

There were five patients in whom an ulcer was associated with cholelithiasis; three of the ulcers were gastric, two duodenal.

All the cases were treated by gastro-enterostomy. In three the gall-bladder was also emptied and drained; in two the gall-bladder was left alone at the operation, in one of these because the patient's condition would not allow of a further operation, in the other because stones were also present in the common duct. In this latter case cholecystectomy and choledochotomy were subsequently performed successfully.

Class C.

In six cases cholelithiasis or its results were found to be interfering with the proper action of the stomach. Five patients were females, one a male. Of these six cases two had had cholecystotomy performed before and adhesions had caused pyloric narrowing. In one of these a gastro-enterostomy had also been done with the aid of Murphy's button. The stomach symptoms returned after a year, and on reopening the abdomen it was found that the anastomosis was almost closed. A second gastro-enterostomy by suture was therefore performed.

In the remaining four cases posterior gastro-enterostomy and cholecystotomy were performed three times and cholecystectomy and gastro-enterostomy once. All the patients recovered. In all these cases the adhesions which existed between the stomach or duodenum and the bile passages made it impossible to say whether ulceration

in these viscera existed. In all probability ulceration was present, or had been present, in every case.

Class D.

In these cases pyloric stenosis due to ulcer was expected. In none was it found, but in all there was marked prolapse of the stomach, which was remedied by gastropexy (Coffey's method in 1 case, with gastro-enterostomy; Beyea's method in 2 cases). In 1 case the gall-bladder was removed. It contained a stone impacted in the cystic duct; there was a large diverticulum of the gall-bladder.

Class E.

In 6 cases no ulcer was found. In 2 of these the stomach and the first and second parts of the duodenum were much dilated. These were examples of a condition to which Ochsner has recently drawn attention. Two of the patients had dilated stomach; 1 of the patients had suffered from hæmatemesis. In 2 cases the pylorus was said to be "narrowed," but no mention is made of either scar or ulcer.

Class F.

Pyloroplasty, 3 cases, no deaths. In 2 cases the symptoms returned after a period of improvement; gastro-enterostomy was performed in both. The third patient remains perfectly well up to the present—that is, seven years after the operation.

Class G.

Ten secondary operations, the primary operation being performed elsewhere. In some of these cases the original condition which made operation necessary was still plainly

in evidence; in others adhesions rendered its discovery difficult or impossible. As a rule a mechanical defect resulting from the first operation was evidently the cause of the symptoms. The cases may be tabulated thus:

Previous Gastro-jejunostomy (6 cases).

Condition found.	Operation.
Long proximal loop	Roux's operation 2 cases.
" " "	Roux's operation and gastro-enteroplasty 1 case.
" " "	Lateral anastomosis 1 case.
Constriction of distal loop by adhesion.	Enteroplasty 1 case.
Nothing abnormal found	Nothing done 1 case.

Previous Gastroplication.

Pyloric ulcer.	Posterior gastro-enterostomy.

Previous Gastrolysis.

Duodenal ulcer; adhesions.	Posterior gastro-enterostomy.

Previous Exploration.

Pyloric ulcer	Posterior gastro-enterostomy.
Duodenal ulcer; ? fistula to cystic duct.	Posterior gastro-enterostomy.

Class H.

In eight patients whose cases are numbered under headings (*a*) and (*f*) I have had to operate a second time. One of the patients was operated upon a third time, so that in all nine operations were performed. The following shows the cases:

No.	First operation.	Result.	Second operation.
1	Posterior gastro-enterostomy with Laplace forceps	Return of symptoms	Gastro - enterostomy with Murphy's button.
2	Posterior gastro-enterostomy by suture	Regurgitant vomiting	Lateral anastomosis.
3	Ditto	Hernia into lesser sac	Reduced ; lateral anastomosis.
4	Ditto	Return of symptoms	Loop found, lateral anastomosis.
5	Ditto	Ditto	Opening too small, gastro enteroplasty.
6	Ditto	Constant vomiting .	Gastrolysis, no relief.
7	Ditto	Ditto	Roux's operation, relief.
8	Pyloroplasty . .	Return of symptoms	Posterior gastro-enterostomy.
9	Ditto	Ditto	Ditto.

The following is a complete tabulation of the 248 operations :

Gastro-enterostomy	Anterior	Simple suture	1
		Roux's operation	2
	Posterior	Simple suture	211
		Murphy's button	1
		Laplace forceps	3
Pyloroplasty			3
Gastro-duodenostomy, Finney's operation . .			2
Gastropexy			2
Gastro-enterostomy and gastropexy . . .			1
Gastro-enterostomy and gastrostomy . . .			1
Infolding of ulcer alone			1
Excision of ulcer alone			1
Secondary operations, first operation by other surgeons			10
Secondary operations, first operation by myself .			9
			248

In 4 cases in the above list other operations were performed at the same time as the gastro-enterostomy. These were—radical cure of a right inguinal hernia in one

case, ovariotomy for large cyst in one case, hysteropexy in one case, appendicectomy in one case. All these patients recovered.

After-results. — In this complete series there are 4 patients who are "no better." In 1 the condition found was a contracted stomach with extensive perigastritis. There are reasons for considering the condition syphilitic. The last report was that the operation "had not made much difference."

In 2 cases where no ulcer was found at the operation the patients are in almost the same condition as before; the patients are "neurotic," and operative treatment has certainly not helped them in the least. In one of these cases gastro-enterostomy had been performed by another surgeon; the symptoms—hæmatemesis, melæna and pain—were not relieved by the operation. I operated a second time but found nothing abnormal, and I therefore closed the abdomen without doing anything further. The symptoms still persist.

In 1 case a large ulcer on the lesser curvature adherent to the liver was found. This patient has, after temporary marked improvement, relapsed, and is probably to be operated upon again.

In addition, there are 10 cases which must be separately mentioned. In 3 the ulcer was on the lesser curvature, close to the cardia, with adhesions to the under-surface of the left lobe of liver and diaphragm. These patients are all better since the operation; 2 of them have gained 1½ stone, but all of them complain still of occasional pains, sometimes severe, radiating upwards into the chest. In 1 of these pyloric obstruction also was present, due to a second ulcer. In 1 the patient, who gained over 3 stone after the operation, has lost almost 2 stone of this increase, and has to restrict her food almost entirely to semi-solids and fluids.

In 3 cases where no ulcer was found and gastro-enterostomy performed some benefit has resulted, but there has been no pronounced improvement in the patient's health such as we are now accustomed to

expect after gastro-enterostomy. In 2 cases the patients suffer from the occasional painless vomiting of pure bile. In other respects the condition of both patients is quite satisfactory; neither suffers from indigestion or discomfort of any kind after food, both eat well, and both have gained weight.

In 1 case, a case of gastric and duodenal ulcer, where hyperchlorhydria was excessive, the patient improved very little after operation, and after some months suffered a relapse, in which hæmorrhage (melæna) occurred. At a second operation, performed elsewhere, adhesions were found around the jejunum, on the proximal side of the anastomosis, and were taken to indicate the possible existence of a peptic jejunal ulcer. One patient has been almost constantly under treatment since her operation, at which only a few adhesions around the pylorus and about the lesser curvature were found. She is reported to be " rather better " than she was before operation.

The result in all the remaining cases has been good. Thus, of 239 patients operated upon 225 are quite well, 4 are no better, and 10 are to be classed as indifferent or fair results. In the last 151 operations in this series there has been no death.

Gastric tetany.—In this series there were 14 cases of gastric tetany of varying degrees of severity, from the simplest to the most serious. In all the same conditions were present—a high degree of pyloric stenosis, moderate or considerable dilatation of the stomach, hypertrophy of the stomach musculature to a degree which made peristalsis quite obvious, and prolonged stasis of the contents of the stomach. In every case gastro-enterostomy was performed and every patient recovered. In 2 cases slight attacks of tetany were noticed for a few days after the operation, and one patient said that the attack which occurred about eighteen hours after operation was the worst he had ever suffered.

Hour-glass Stomach.

This condition is far more common than is generally

supposed. In my recent cases a diagnosis has in every instance been made before operation by attention to a series of signs which I have described fully elsewhere.

Hour-glass stomach is not infrequently associated with cicatricial stenosis at the pylorus. The result is that two pouches are formed in the stomach, each being obstructed at the outlet. Since the cardiac pouch in the majority of cases is very much larger than the pyloric, the operation of gastro-enterostomy, the cardiac pouch and the jejunum being attached, will afford relief to the condition. But in other cases a double operation may be necessary.

In this series there are 24 patients upon whom were performed twenty-six operations, and one operation not completed, the patient dying under chloroform. The 24 cases included 7 males and 17 females.

In several cases the history is strongly suggestive of a former perforation, while in one case a perforation was the cause of operative measures. Trilobed stomach was seen once.

Mortality.—Four deaths occurred among the 24 cases: one on the fourth day from septicæmia, resulting from a strangulated rectal prolapse; one on the third week from suppression of urine; one on the fifth day from pneumonia; and one died on the operating table from the effects of chloroform before the operation was completed.

Operative treatment.	Primary.	Secondary.
Gastro-jejunostomy alone	7	2
Gastro-jejunostomy and gastro-gastrostomy	3	0
Gastro-jejunostomy and gastroplasty	2	0
Gastro-jejunostomy and dilatation	1	0
Gastroplasty	7	0
Gastro-gastrostomy	2	0
Dilatation	1	0
Entero-anastomosis	0	1
Total	23	3

Of the secondary operations, the entero-anastomosis was performed for regurgitant vomiting after gastro-

jejunostomy; one of the secondary gastro-jejunostomies was required for relapse three years after gastroplasty, the other was performed a year later upon a case whose condition at the first operation would only allow of a gastroplasty.

The hour-glass stomach on which Loreta's operation alone was performed was thought at the time to be due to malignant disease, but the patient made an excellent recovery, and four years later was reported quite well.

[I am much indebted to Mr. H. Upcott for his help in the preparation of the statistics in this report.]

SPECIAL DISCUSSION

ON

THE OPERATIVE TREATMENT OF NON-MALIGNANT ULCER OF THE STOMACH AND ITS CHIEF COMPLICATIONS, WITH INDICATIONS, LIMITATIONS, AND ULTIMATE RESULTS.

STATISTICS OF CASES

Contributed by HERBERT J. PATERSON, M.B., B.C.Cantab., F.R.C.S., Assistant Surgeon to the London Temperance Hospital.

Remote Results of Gastro-jejunostomy.

Two hundred and seventy-eight cases of this operation for simple pyloric stenosis or for gastric ulcer have been collected from surgical literature previous to 1904, and an attempt has been made to ascertain the remote history of each patient. The after-history of 143 patients has been obtained.

The results are shown in the following table:

Unsatisfactory cases:		
Died subsequently	4	
Relapsed	5	
"Fairly" good result:		
Occasional dyspepsia	9	
Some care in diet necessary	7	16
"Cured" of gastric trouble:		
Died from other causes	3	
Alive and well	115	118
		143

So that in about 82 per cent. of the patients relief has been complete, while in about 10 per cent. relief has been almost complete.

Eliminating the cases in which a mechanical appliance has been used or in which a small opening has been made, the proportion of patients completely relieved is about 92 per cent.

In one instance the interval since operation is nineteen and in another eighteen years. The operation on both these patients was performed by Dr. Fritzsche, of Glarus, and they are by far the longest intervals of survival hitherto recorded. In none of the patients included in my statistics is the interval since operation less than two years.

Gastric Ulcer.

With the assistance of Dr. Herbert Rhodes an investigation was made into the after-condition of 147 patients who had been in the London Temperance Hospital under the care of Dr. Soltau Fenwick and Dr. Parkinson. Out of a series of 147 patients 72 have been traced. The results are as follows:

"Not cured":		
Signs of gastric ulcer	40	
Operation subsequently necessary	5	46
Died subsequently from gastric disease	1	
"Cured"		19
"Probably cured"		2
"Probably not cured"		5
		72

Of the 75 untraced patients 12, *i. e.* 14 per cent. had previously been in hospital on one or more occasion for gastric trouble.

Perforated Gastric Ulcer.

Site of Perforation.

One hundred and twelve consecutive cases of perforated gastric ulcer:

Anterior wall.				
Pyloric third.	Middle third.	Cardiac third.	Posterior wall.	Not stated.
32	33	32	12	3

Fifty-four of the patients recovered, of whom 35 have been traced.

On 2 of the patients gastro-jejunostomy was performed. Both these patients have remained in good health. The after-history of the remaining 33 patients is as follows:

Quite well	16
Died subsequently of perforation . .	1
Relapsed and required gastro-jejunostomy	2
Symptoms of gastric ulcer . . .	9
Symptoms of dyspepsia . . .	5

Personal Cases of Perforated Gastric Ulcer.

(*a*) Suture only: 4 cases; 3 deaths.

(*b*) Suture (when possible) with gastro-jejunostomy: 5 cases; 1 death.

The Results of Operation for Hour-Glass Stomach.

A series of 92 operations for hour-glass stomach (non-malignant) has been collected, and an endeavour made to ascertain the after-results. Through the courtesy of the various operators I have been able to ascertain the after-results in 61 of the 76 patients who survived operation. These are as follows:

Summary of Operations for Hour-Glass Stomach.

Gastroplasty only: 43 operations; 4 deaths.

Complete relief	16
Relief, but not perfect	3
Unrelieved	1
Relapsed	9
Relief, but subsequent condition unknown	7
Untraced	3
	39

Gastro-gastrostomy only: 14 operations; 1 death.

Complete relief (1 patient has had hæmatemesis once)	4
Relieved, but died from other cause	1
Relieved, but remote result unknown	2
Unrelieved	1
Relapsed	3
Untraced	2
	13

Gastro-jejunostomy only: 19 operations; 4 deaths.

Completely relieved	10
Relieved, but remote result unknown	3
Unrelieved until second operation	1
Relapsed	1
	15

Gastro-jejunostomy subsequent to gastroplasty: 3 operations.

Complete relief	2
Remote result unknown	1
	3

Gastro-jejunostomy subsequent to gastro-gastrostomy: 1 operation with relief.

Gastroplasty with gastro-jejunostomy: 6 operations; recovery and complete relief in 5; 1 died.

Gastro-gastrostomy with gastro-jejunostomy: 3 operations; recovery and relief in all.

Excision of ulcer: 2 operations; 1 died, 1 recovered and relapsed.

Gastroplasty with pyloroplasty: 1 operation; 1 death.

Pylorodiosis with gastro-jejunostomy: 1 operation; 1 death.

Gastro-duodenostomy: 2 operations; 1 completely relieved, 1 death.

Dilatation with gastro-jejunostomy: 1 operation; recovery, but remote history unknown.

Dilatation: 1 operation; complete relief.

From this table it appears that in at least 25 per cent. of the patients upon whom gastroplasty has been performed either no relief has followed or relapse has occurred subsequently, while at least 30 per cent. of the patients upon whom gastro-gastrostomy has been performed have either obtained no relief or have relapsed. Not one of the patients upon whom gastroplasty or gastro-gastrostomy together with gastro-jejunostomy has been performed have yet shown signs of relapse. I think, therefore, that we may conclude that gastro-jejunostomy must form some part of the method adopted for the treatment of hour-glass stomach.

DR. WALTER BAYLEY AND HIS WORKS, 1529-1592

BY

D'ARCY POWER, F.S.A.

Received December 29th, 1906—Read June 19th, 1907.

THE small book which I hold in my hand long aroused my curiosity. It is entitled 'A Briefe Treatise touching the Preservation of the Eiesight, consisting partly in Good Order of Diet and partly in Use of Medicines.' It bears no author's or printer's name on the title page, but there is a contemporary inscription at the beginning of the preface which runs, "To the righte worshypfull my very good frynde Mr. John Pophame Her Ma[jesty's] attornye-generall" (Fig. 1, p. 421), and the preface is signed at the end, "your very lovinge frynde Walter Bayley" (Fig. 2, p. 421). The date is 1586. The book was bought in Dublin some years ago by Dr. Aquilla Smith, who seems to have paid one shilling for it. He gave it to my father, who in turn has given it to me. I had also long known a small book, published in 1622, under the title 'Banister on the Eye,' which contains "Banister's Breviary of the Eyes," Jacques Guillemeau's "Worthy Treatise of the Eyes," "A Brief Treatise concerning the Preservation of the Eyesight," which is a

reprint of Dr. Bayley's book without the preface, and "A Discourse of the Scorby," translated out of 'Wyer's Observations.' At first I thought that Banister was the author of Bayley's work, and he has indeed been credited with it, but I soon discovered that either Banister or his publisher had merely re-issued it to make a volume of moderate size, dealing chiefly with diseases of the eye. I set to work, therefore, to find out more about Dr. Walter Bayley and his works, with results which, I hope, will prove as new and interesting to the Fellows of the Society as they have been to me.

Dr. Walter Bayley, son of Henry Bayley, of Warmwell, Dorsetshire, Esquire, was born at Portisham, eight miles south-west of Dorchester, in that county, in the year 1529, and was educated at Winchester College. He passed from Winchester to New College, Oxford, in 1548, and was elected a Fellow in 1550, probably on the understanding that he would devote himself to the study of medicine, although he was called upon to take orders. He resigned his fellowship of New College in 1560, perhaps on the occasion of his marriage. He was admitted B.A. on October 24th, 1552, and was licensed to proceed M.A. July 6th, 1556. In April, 1558, he was Junior Proctor of the University—Alan Cope, of Magdalen College, who was afterwards a Canon of St. Peter's, at Rome, being the Senior Proctor. Bayley demanded the degree of Bachelor of Physic and supplicated for leave to practise medicine "ad practicandum in re medicâ per totam Angliam," January 28th, 1558—1559. Both were granted him on February 21st, 1558—1559. In 1561 he succeeded Thomas Francis, M.D., of Christ Church, who was afterwards Provost of Queen's College and Physician to Queen Elizabeth, as Queen's Professor of Medicine in the University. This post he retained until 1582, when he resigned, and his place was taken by his son-in-law, Anthony Aylworth, M.D., of New College (p. 438, note). Bayley graduated M.D. July 26th, 1563, and we read that in his capacity as Regius Professor of Medicine on "August 27th, 1566, Dr.

Walter Baylie and Henry Bayly,[1] M.D., conferred the degree of Med. Bac. on Edward Atslow[2] in a room of Dr. Henry Bayly's, next to the highway leading to the Quaterfax, in the presence of William Standishe, Thomas Owen, and Thomas Collyns, notary public: and the degree of Med. Doct. on Robert Barnes[3] and Richard Slithurst."[4] Three days later, on August 30th, 1566, in virtue of a decree of August 29th, Walter Bayly created Roger Gifford[5] Medicinæ Doctor, in the presence of William Standishe, George Caponhurst, M.A., William Gilbert, Superior Bedell of Arts." These irregular creations were made with the connivance of the University to avoid the Comitia and its attendant expense. The ordinary University fee for the degree of Doctor of Medicine was £7 16*s.* 6*d.*, at a time when money was about fifteen times more valuable than it is at present, but the incepting doctor had also to pay 14*s.* 8*d.* *pro vino*, and was obliged to provide "convivia" for the Vice-Chancellor, the Regius Professor of Medicine, the Proctors, the Registrar, and the Bedell of the faculty. He had furthermore to give "gloves" to the Vice-Chancellor, the Regius Professor, the Proctors, the Registrar, and to all Students of

[1] Henry Bayly, Fellow of New College 1534—1552, from Bradford, Wilts. B.A. July 16th, 1538; M.A. April 18th, 1542; Proctor 1547; B.Med. 1547—1548; D.Med. July 20th, 1563. He devised to New College "Le Blew-Bore" in St. Aldates.

[2] A Fellow of New College 1551—1562, afterwards imprisoned for designing the escape of Mary Queen of Scots; physician to the Duke of Norfolk, and racked twice in the Tower on his account; died 1594. His widow received an annuity from the Earl of Arundel.

[3] Robert Barnes, Fellow of Merton College, 1538; Linacre Lecturer, 1558; died 1604.

[4] Richard Slithurst, Demy of Magdalen College, 1537; Fellow of Brasenose College.

[5] Roger Gifford, of Christchurch, B.A. 1556; Fellow of Merton College 1557; Proctor 1562 and 1563; Fellow of All Souls' College 1563; Junior Linacre Lecturer; Physician to Queen Elizabeth; President of College of Physicians 1581—1584; M.P. for Old Sarum 1585—1586; Precentor of St. David's and Prebend of Llanbedr-pont-Stephen 1592. Died January 27th, 1596-1597; buried in St. Bride's, Fleet Street.

Medicine and Bedells who accompanied him to church at the ceremony of presentation.

On September 5th, 1566, when Queen Elizabeth visited Oxford, Dr. Walter Bayley, with his relative, Dr. Henry Bayley, and Dr. Huicke[1] opposed these two questions in physick: (1) Vita potest prorogari arte medica? (2) Cibi tardae concoctionis præferendi sunt cibis facilioris concoctionis? The official record of the visit states: "This day, being Thursday, were disputations in Physick and Divinity in St. Mary's, the University Church, from two of the clock, or thereabout, untill seaven, before the Queen's Majesty; who gave very attent care unto them, and tarryed till the full end thereof. Dr. Francis[2] was respondent in Physick, and Dr. Masters[3] was Determiner. Dr. Bayle jun. gratias egit Principi, et Acad. nomine et suo, quod Regius Professor in Med. erat, egitque hac ratione. Ars Med. non potest retardare senectutem: Ergo nec mortem. Quod

[1] Robert Huicke, B.A. 1528; Fellow of Merton College, 1530; Doct. Med. from Cambridge 1538; incorporated 1566; Principal of St. Alban Hall; President of College of Physicians 1552—1564; Physician to King Henry VIII and Queen Katherine Parr; Physician Extraordinary to King Edward VI, and Physician to Queen Elizabeth; M.P. for Wootton Bassett 1547—1552. He took part in the Physic Act, kept at Cambridge, in the presence of Queen Elizabeth, August 7th, 1564. The Privy Council reported to Mr. Secretary Petre after an examination of the dispute betwen Dr. Huicke and Elizabeth his wife, on May 11th and 12th, 1546, that "we never in all our liefes harde matier that more pitied us: so much crueltie and circumvencion appered in the man, so little cause minstred by the woman." On November 2nd, 1575, he was licensed to marry Mary Woodcocke, spinster, of the City of London.

[2] Thomas Francis, of Christ Church, B.A. 1540; M.A. 1544; B. and D. Med. 1553—1554; Provost of Queen's College 1561—1563; Regius Professor of Physic 1554—1561; President College of Physicians 1568; Physician to Queen Elizabeth. Died 1574.

[3] Richard Masters, Fellow of All Souls' College 1533; of Christchurch 1547; incorporated at Cambridge 1571; President College of Physicians 1561; Physician to Queen Elizabeth 1559; Prebendary of Friday Thorpe, in the Cathedral of York, 1563; had a grant of the reversion of the site of the late Monastery of Cirencester with lands in fifteen counties, dated January 6th, 1564—1565. Died about 1587 (see *Lancet*, vol. ii, 1889, p. 987).

probavit quoniam solidæ partes non poterant humectari."

Dr. Bayley was collated Prebend of Dulcott in the Church of Wells on August 30th, 1572, and resigned his prebendship in 1579. Thomas Bayley, also from Portisham, Dorset, and a Fellow of New College from 1534 until 1552, was Treasurer of Bath and Wells in 1560, and again in 1564. A search at the Record Office shows that Dr. Walter Bayley was appointed "medicus ordinarius ad vitam" to Queen Elizabeth on December 1st, 1581 (xxiii Eliz.). In 1578 a lease of Stanlake, in the County of Oxford, was granted him by the Queen for twenty-one years. In the following year he was negotiating leases with the crown, in which Corpus, Lincoln, and Magdalen Colleges at Oxford were also interested. On June 28th, 1590, there is an entry of "purchase by Dr. Bailie, one of Her Majesty's Physicians in Ordinary, of lands of the yearly value of £27 8*s*. 0¼*d*., for which he is to pay £715 9*s*. 4½*d*. to Her Majesty."

Bayley was elected a Fellow of the College of Physicians of London about the year 1581. He was named an Elect June 10th, 1584, and Consiliarius 1588. He died March 3rd, 1592, and was buried in the antechapel of New College, Oxford, where, under a brass in the floor representing an old man in a gown with hands erect, he is commemorated in the following lines:

"Gualterus tumulo dormit Bailæus in isto,
Cui Doricastrensis patria fundus erat.
Wicchamicis didicit juvenis quam sumptibus artem,
Grandior hanc lector regius edocuit.
Fama virum evexit, Regina accivit ad Aulam
Jungeret ut Medicis Elizabetha suis:
Haec tria lustra egit longe illustrissimus, amplo
Et celebri, quantum dat medicina loco.
Charus erat multis, dum vita manebat, et idem
Deflendus multis vita ubi fugit erat.
Obiit 30 Martii anno salutis
Humanae MCCCCCLXXXXII ætatis suæ 63.
Posuit Gulihelmus Bailey filius
Amoris et pietatis monumentum."

His posterity, says Anthony à Wood, writing at the end of the next century, "Do live at this day at Ducklington, near to Witney in Oxfordshire, some of whom have been justices of the peace for the said county." The following story is told of one of these descendants:

"At Ducklington, neare Witney, in Oxfordshire, hath been a custom for forty or fifty years not to give the parson gloves at weddings, but, if the persons married shall at the year's end after marriage say that they repent them not of the marriage, then shall they give, and the parson claime, a paire of gloves. In 1682 a paire was given to the parson by a couple that had been married a year. Wherefore Walter Bayly, the parson, sometime Fellow of Magdalen College, did show them to his friends for a rarity, and being persuaded to give them to the archives in Bodley's Library at Oxon., did so, and these verses following (made by Ant. Hodges, rector of Wytham, neere Oxon., 1682) were pin'd upon them:

"Chirothecas connubentes
Anno post non pœnitentes
Has dederunt nuptiales
Quis ostendit mihi tales?

Wedded a year we ne'er repented,
But to the Preist these gloves presented
Let Oxford archives never dare
To show me such another paire."

Works and Bibliography.

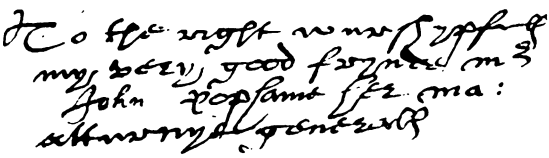
Dr. Walter Bayley published three books—one in 1586, one in 1587, and one in 1588. There remains a fourth in manuscript, which is said to have been in the library of Robert, Earl of Aylesbury. It is entitled 'Explicatio Galeni de potu convalescentium et senum, et præcipue de nostræ Alæ et Biriæ paratione.' I have not been able to find the manuscript.

The three books are 'A Briefe Treatise on the Eye-sight,' printed in 1586; 'A Brief Discourse on the Baths at Newnam Regis,' in 1587; and 'A Short Discourse on

the Three Kinds of Pepper,' in 1588—the year of the great Armada.

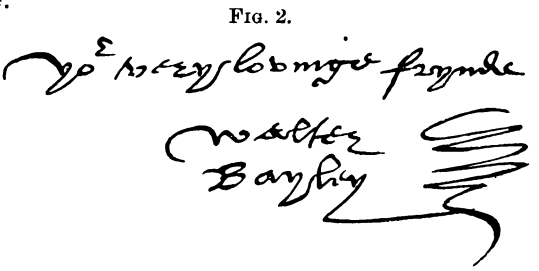
Each book was printed privately, and was issued without any name. They were given away by Dr. Bayley to his friends as New Year's greetings. Each copy, therefore, was tastefully bound, and the offering was made personal by a short autograph inscription at the beginning of the introduction and by the donor's signature at the end of the preface. The private issues of 1586 and 1587 do not seem to have been very numerous, and Dr. Bayley was able to alter his form of address according to the rank of the recipient. But in 1588, as his circle of friends had grown larger and more varied, he made the printer leave spaces in his preface to the discourse on the three kinds of pepper, and these spaces (p. 433) he afterwards filled in with his pen in an appropriate manner. For he seems to have been very methodical, and the courtesy of the time demanded great precision in adapting the presentation formula to the rank of the recipient. The presentations are written thus:

Fig. 1.



in a neat and clear hand, immediately above the preface, and the preface itself is signed with his name in full, and always in this manner, except that he sometimes varied the spelling of the surname—a matter of no importance in his time.

Fig. 2.



The first book was certainly printed by Robert Waldegrave, for in Arber's 'Transcript of the Register of the Stationers' Company' there is the entry—"18 Julii (1586) Robert Wal(de)graue: Receaued of him for printinge *a treatise for the Eiesight.* Entered in full Court . . . vid."

Waldegrave first practised his art in the Strand, without Temple Bar, near Somerset House, in 1578. He then removed to Foster Lane, and got into trouble for printing puritanical books. He retired for a time to Wales, but, being of good family, he finally regained his position, and was appointed printer to King James VI of Scotland, from whom he received a patent.

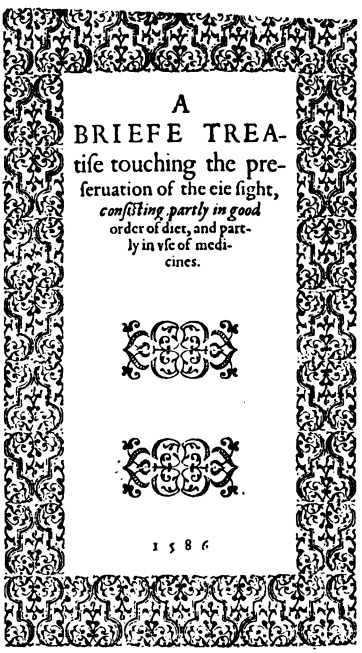
The books are well printed, and the printer's ornaments are sharp and clear. It appears to me that each of the books was reprinted immediately after publication. All the presentation copies that I have seen bear the printer's signature A2 at the bottom of the first page of the preface, and there is no signature on the last page of the preface. The re-issue, which has no autograph inscription, has the printer's signature A iii on the first page of the preface and A iiii on the last page. There is also an ornament on the last page of the preface which is wanting in the original issue, because Dr. Bayley required the space for his autograph. It is evident, therefore, that Bayley was a lover of books who bestowed thought on the format of his little gifts.

(A) (1) 'A Briefe Treatise touching the Preservation of the Eiesight, *consisting partly in Good* Order of Diet, and partly in Use of Medicines,' 1586 [two devices] (Fig. 3, p. 423), 16°, pp. 6 + 23, p. ii, beg. pheasant, *Rasis.* Contents: p. i title, 3–5 preface [a device and an ornamental letter on page 3], 1–23 the Treatise; ornamental letters on pages 1, 17, and 20.

Dr. Bayley introduces his treatise with the following words: "Occasioned thorough certaine speeches had with

some of mine honorable good friends, to write myne opinion of meanes to preserve the sight in good integritie, I have performed the same in this little pamphlet, wherein I have directed my pen rather to leave rules for those, which have not in themselves sufficient knowledge, than to satisfie the learned; who I know can devise much better means and remedies. . . . And now following the

FIG. 3.



laudable custom begun in ancient time; and continued in these our daies of presenting our friends with new yeares gifts, for lacke of the things of greater price, I offer unto you this little pamphlet, as a significance of my good will, wishing that the same may be a token of many good and prosperous yeeres, which God the giver of all goodness, send unto you."

The treatise begins with a consideration of those things upon which the preservation of the eyesight doth consist—air and diet. Dr. Bayley considers that "southerne wyndes doe hurt the sight; so do low rooms, places full of dust and smokie are noysome. Meates are best which are easie to be digested and which do not stay long in the stomache; amongest such a yong henne is greatly commended; so is partridge and pheasant." Amongst medicines he praises most a drink made with *eiebright*, called of the Arabians *adhill*, and in Latin *euphragia*. "This simple may be used in beere, in ale or meade, but 'in countries which for their common drinkes do use wine and water, they alway do mingle thinges for the sight in wine and not in water.'" He recommends as "the simplest order to compound ale or beere to ech mans best liking with eiebright onelie, taking to everie gallon of the drinke a great handfull of the herbe, and binde it togither, or put it in rawe and thin tinsell of silke and so tie the same by a string to the top of the vessell, that the herbe may hang in the midst of the drinke, not too lowe in the grownds, neither too high in the barme, being put into the drinke when it is newly clensed; let all work togither untill the drinke be cleare and ripe to be droonken according to the common use, and then yee may drinke of it at pleasure, in the morning fasting and at meate also if you will, and can well like thereof, and most men may like to drinke it, bicause this herbe doth yeeld no ungratefull taste, but rather with a pleasant sapour doth commend the drinke." Fenill seeds and spices may also be added. "I can witness," he says, "that many by this simple composition of eiebright and fenill seeds, have found great good for their sight, not onlie to continue in good estate, but also that some have found remedie against the dimnes & other impediments growing in their sight. In truth, once I met an old man in Shropshire, called M. Hoorde, about the age of 84 yeres, who had at that time perfit sight and did read smal letteres very wel without spectacles; he

told me, that about the age of 40 yeeres, he finding his sight to decay, he did use eiebright in ale for his drinke, and did also eate the pouder therof in an egge three dayes in a weeke, being so taught of his father, who by the like order continued his sight in good integritie to a very long age. I have heard the same confirmed by many olde men. *Rowland Shorlooke*, an Irish man, physician to Queen *Mary*, did affirme for truth, that a Bishop in Ireland, perceiving his sight to waxe dimme, about his age of fiftie yeres, by the use of eiebright taken in powder in an egge, did live to the age of 80 yeres, with good integritie of sight."

Dr. Bayley gives simple advice in the manner familiar to all conversant with the teaching of the Schola Salernitana, and not differing very much from it. He recognises the value of tinted glasses and the superiority of crystals or pebbles over common glass for spectacles. Amongst local applications he commends "the washing of the eies with the urine of a childe and sometimes to drop the same into the eies," and he advocates the use of "a liquor of the liver of a goate." The last chapter deals with inward medicines, and the conclusion runs: "Where in this little treatise mention is made of distilled waters, I wish the same to be artificially done in stillatories of glasse, that the qualities of the herbes may remain in the distilled waters; and therfore I do not allow of the common manner of distilling in stillatories of leade, by the which the waterie parts onlie are drawne."

The briefe treatise had an extraordinary vogue, and I have seen copies of the following issues:

(2) 'A Briefe Treatise touching the Preservation of the Eiesight, &c.,' sixth edition. At Oxford by Joseph Barnes, printer to the University 1602. This is identical with the edition of 1586, of which there are copies in the British Museum and Bodleian libraries, except for some slight variations in spelling. It is very poorly printed, and although it is called the sixth edition I have found no copies of a third, fourth, or fifth issue. Impr. 24:

1602 (eights) 16°, pp. [6] + 25 + [1] : p. ii, beg. *rected by the.* Pica Roman. Contents: p. (i) title: (3–5) a preface: 1–17, 19–25 the treatise.

Joseph Barnes, the printer, was admitted a bookseller at Oxford, on September 8th, 1573. He was licensed to sell wine from October, 1575 to, at least, October, 1596. He was sole printer to the University from 1585 to 1617; he resigned on February 12th, 1616–1617, and died in 1618. He was buried in St. Mary's church on December 17th, 1618. He lived and printed in a house at the west end of St. Mary's—the University church—where is now St. Mary's Entry. The University lent him £100 on August 15th, 1584, with which to set up a press. The money was to be repaid in six years, but it was still owing in October, 1592. It will be remembered that in 1586 an ordinance of the Star Chamber allowed only two presses outside London, one at Oxford and one at Cambridge, and only one apprentice to each press.

(3) Two Treatises concerning the Preservation of Eiesight, the first written by Doctor Bailey, sometimes of Oxford; the other collected out of those two famous physicians Fernelius and Riolanus, Oxford. Printed by Joseph Barnes for John Barnes, 1616. There are copies of this edition in the British Museum, in the Bodleian, in the Royal College of Surgeons, in the Royal College of Physicians, and in the library of the Royal Medical and Chirurgical Society. Impr. 34: 1616: eights. 12°: pp [8] + 64: p. ii begins *yeeld into*: Pica Roman. Contents: p. (3) title: (5–7) "To the Reader," a preface by I[ohn] B[arnes] 1–24 "A breefe Treatise concerning the preseruation of the eye sight": 25–62 "A Treatise of the principall diseases of the eyes gathered *out of* Fernelius *and* Iohn Riolanus *Doctors of Phisicks.* "Johannes Fernelius and Johannes Riolanus the elder, both French physicians," says Mr. Falconer Madan in 'Early Oxford Press,' p. 105, commenting on this edition, "died in 1558 and 1609 respectively, but neither wrote a special treatise on eyesight. The preface is no doubt by John Barnes, and

alludes to the worth and undeserved obscurity of Bailey's work. The whole book, with the possible exception of the title page, was printed in London, the woodcuts being quite unknown at Oxford. Even the arms of the University on the title page are recut on wood." The preface to the reader says: "It is not unknown to the world with what general applause a certain treatise concerning the preservation of the Eyesight written by Doctor Baylie, sometimes of Oxford hach (*sic*) beene accepted, which by the happy experience of many in the doubtfull cures of that kind stands thoroughly confirmed. Being, therefore, unwilling that a gemme of such worth should lie any longer hidden under the soile of oblivion and withall desirous to give more lustre unto it as well for the ornament of itselfe as for the good of the merchant: I have now at length resolved to set it forth in the world to the publique view and censure of the time, newly and artificially polished with most notable collections out of those very renowned doctors Fernelius and Riolanus written on the same subject. Peruse the book and make use of it. If thou findest benefit by it, thanke God first that hath made his goodness manifest to the world by his singular gifts bestowed on those most excellent men: next to the Authors themselves for theire great paines and studie taken for thy profit: and lastly to mee, for my good will and costes in the publishing of it. Farewell.—I. B."

(4) 'A Treatise of One Hundred and Thirteene Diseases of the Eyes AND Eye-liddes.' The second time published, with some profitable additions of certaine Principles and ex*periments*, by Richard Banister, Mr. *in Chy*rurgery, Oculist and *Practitioner in Physicke*. '*God hath created medicines of the earth and he that is wise will not contemne them.*' [Device crowned Tudor rose.] Imprinted at London by *Felix Kyngston*, for *Thomas Man*, dwelling in Paternoster Row, at the signe of the Talbot, 1622, 16mo. Contents: p. (1) title; 3–7 The Epistle Dedicatory: 8–19 "To the Reader": 20–21 Commendatory ode in Latin:

(22): 23–111 Banister's "Breviary of the Eyes": (112): 113 title, "A Worthy Treatise of the Eyes [by Jacques Guillemeau, 1550–1612, translated by Richard Banister?] containing the knowledge and cure of one hundred and thirteene diseases incident unto them. *The summe of the chapters of euery* section of this booke follow *in the next page.*

Imprinted at London by *Felix Kyngston* for *Thomas Man*, dwelling in Paternoster Row, at the signe of the Talbot, 1622, 16mo.

115–130 the Sum of the Chapters: 131 "The generall heads": (132): 133–134 Preface: 135–367 the treatise: 368–397 A Briefe Treatise concerning the preservation of the Eye *sight*: (398): 399–400 Preface to the Courteous and Carefull Chirurgian: 401–442 A discourse of the Scorby, translated out of Wyer's Observations: 443–477 On the Nature of divers kinds of Cancers or Cankers: (478).

(5) A reprint of John Barnes' edition, dated London, 1626, printed by John Beale for Francis Williams, and are to be sold at his shop at the signe of the Globe in Cornhill, over against the Royal Exchange. This edition, with a separate half-title, is bound up with William Vaughan's 'Directions for Health,' London, 1626. The copy in the British Museum contains the following note in manuscript, but the Mr. Bayly,[1] to whom reference is made, is clearly not Dr. Walter Bayley: "A postscript of a letter of Gilbert, Earl of Shrewsbury, to Sir Michael Hicks, Secretary to the Lord Robert Cecil, Earl of Salisbury, Lord Treasurer, dated May 1st, 1612. 'At Bathe you shall finde a physitian called Mr. Bayly, a man in great practice there and in myne opinion both very learned, discreete and honest. I pray you take notice of him from me as thus recommended unto you, and, as there may be cause, so that for my sake (he being one that I well affect)

[1] Probably Ralph Bayly, of Dorset, who matriculated at New College November 24th, 1581; B.A. April 12th, 1594; Fellow: M.A. March 20th, 1597—1598; licensed to practise medicine July 11th, 1617; B. and D. Med. July 16th, 1617.

you will afford him your favour.' This I have seen in the original in the honorable Mr. West, of Alscot, his MS. collection called 'Scrinia Burleighiana,' vol. 92, No. 94, C. Y. Greene."

(6) A reprint of John Barnes' edition was issued in 4to and dated London, 1633. It is printed by Thomas Harper for John Harrison, and "are to be sold at his shop in Paternoster Row, at the signe of the Unicorn." It has a half-title, and is bound up with Vaughan's 'Directions for Health.' I have seen the copies in the Bodleian and in the British Museum.

(7) 'A Briefe Treatise touching the preservation of the Eyesight, &c.,' by Walter Baley (*sic*), sometimes Fellow of New Colledge in Oxford, Doctor of Physic, Regius Professor in that faculty and Physitian to Queen Elizabeth. Oxford: 1654. Printed by H. Hall, printer to the University, for R. Davis. The preface is addressed "To my very good Cosen Mr. John Bayley of New Colledge," and is subscribed "your Uncle Walter Baley." John Bayly, of Dorset, fil. pleb. matriculated at New Coll. 20 March 1578–9, aged 19; B.A. 1582, M.A. 1586; licensed to practice medicine 1596. His will was proved at Oxford, July 9th, 1602. This edition is printed, I think, from one of the original 1586 presentation copies, as the preface is a copy of the original preface, which had not been reprinted since 1602 in the previous editions. There is a copy in the British Museum.

(8) A re-issue of this edition by the same printer, dated Oxford, 1673. There is a copy in the British Museum.

(B) 'A Briefe Discours of certain Bathes of medicinall waters in the Countie of Warwicke neer unto a village called Newnam Regis, 1587.' Dr. Bayley begins his brief discourse with the following words: "The benefits, no doubt, are great and manifold which almightie God of his large bountie and exceeding goodness of late yeeres hath

plentifully bestowed uppon this little soile of England, since the prosperous reigne of our most gratious Sovereigne, wherein the gospele hath sincerely and freely beene preached: in whose time many and strange events have happened to the great benefit of the countrie. . . . And it is not altogether a vaine conjecture to thinke that God in these daies miraculously revealed wels and springs of medicinall waters never knowen before to work effects strange and marvellous in our sights, thereby to induce all men to forsake such puddle pits which man's devise hath digged and drinke onely of the cleere fountaines of his word, thence only to fetch remedy for our diseased soules. The baths of Bathe and Buckstan for their antiquitie and long proofe in times past, are of great fame and no doubt as of more efficacie than others may justly be most accounted of: nevertheless manie other waters in England of late yeeres discovered have the testimony of experience. . . . Amongst the which the wels in Warwickshire nigh a village called Newnam Regis[1] have most credit, by the use of which great numbers of people have found helpe beyond their expectation."

The discovery of the baths: "It happened not long

[1] I visited Newnham Regis, or King's Newnham as it is now called, last Whitsuntide. It is a small hamlet with a population of about 160 people near Church Lawford, which is situated on the high road between Rugby and Coventry, and about three miles from Rugby. A few enquiries led to the information that the bath still existed at "Rainbow's cottage" on the banks of a small stream nearly a mile away from the hamlet. The cottage is old-brick with a tiled roof. The bath forms an annexe with an entrance from the cottage as well as from the outside. The main bath measures about fifteen feet by six and is five or six feet deep. It is entered from one end by a broad flight of steps, and over it is a beam to which a rope has been fixed for the assistance of cripples taking the water. In another room is a smaller bath which has had a hot water supply. Both the baths are now empty and are falling into decay. All round the cottage are the remains of lime-kilns which are in ruins. The spring is situated a few hundred yards away, and discharges straight into the small river flowing past the cottage. It still yields a perennial supply of water which is clear, hard and tasteless, as in the time of Dr. Walter Bayley.

since in Warwickshire, neer unto a village called Newnam Regis that a certain husbandman occupied in lopping of trees, chaunced to receive a greevous wound in his hand whereupon he repaired presently to a fountain which was not far off: after he had washed and cleansed the wound very well with the water, he presently found all pains of his hurt asswaged and in a short time the wound thoroughly closed and healed, that he needed no further help by the aid of surgerie, which effect he imparted to his neighbours as a strange success: and it so amazed the hearers that after another having a deep wound in his heele with a sieth (scythe) came to the same fountain and recovered and was made hole onely with the lotion of the same water."

Hearing this, Dr. Bayley went to the baths to see how far the rumours were true, and, "after I had a fewe daies observed the success and event of things and seen as much with mine eies as I had before heard with mine eares, I was in truth induced to beleeve that there was in those wels some more secret and hidden qualities than are now found in common waters." He therefore acted in a thoroughly scientific manner worthy of the age in which he lived, for he says: "I tooke certaine quantities of them and distilled them: I have likewise examined them by filtrings and evaporations and have alwaies observed and found in the end . . . . that there remaineth a certaine residence, in color whitish and in taste somewhat salt, which being put upon a glowing hote iron doth foorthwith become cleerer and whiter. And although this be a property of Limestone, Plaister and Alabaster, when they are mingled in both waters, yet I am persuaded that onely the limestone, and neither of the other two, entreth the mixture of these waters. . . . I have also farther probabilities . . . . that there is in the waters som stone juice: for it is ordinarily observed that if there be drowned in them either wood or any other such solid substance it is after a short time covered and overgrown with a hard crust like a stone."

Dr. Bayley thought that lime was the chief ingredient, though the water also contained nitre, alum, and iron in lesser quantities. The waters were useful, therefore, in a variety of maladies, especially "those which are hot and moist in their nature, as in the inflammation of the eyes, in excessive bleeding, in over-much laxitie of the gums being used as a lotion and in the pissing evil." He then gives directions for the use of the wells and for certain things to be observed before drinking the waters. These directions do not differ materially from those enforced at similar cures in the present day. He goes on to say: "Wherefore I do in anywise advise my countrymen which have in purpose to use these wells to forbeare them in wet and rainy seasons: for the rain-water being compounded and mixt with the other maketh them either altogether unwholesome, or at the least less profitable and effectuall than otherwise they would be." The amount to be drunk is from four to six pints a day, fasting, and sippingly; and "the usage and maner of our countrymen in England is utterly condemned, which, whiles they are using the bathes, have no regard to their frugality of diet, but both in their dinner and supper so farre exceed and swarve from the rule, that it is not to be marvelled at there be so few in our countrey which ordinarily receyve any commoditie or health by meanes of the bathes I admonish them therefore to be more sparefull in their dyet, and so shall they gayne health to their bodies and comfort to their purse."

There were two issues of this work, which are distinguished from each other in the same manner as the two issues of "the treatise of the eiesight." I have not been able to ascertain the name of the printer, but it seems probable that they were printed by Waldegrave, though there is no entry of it in the records of the Stationers' Company under the year 1587. In some of the presentation copies Walter Bayly's signature is printed at the foot of the preface, in others it is in autograph.

I have found no trace of any subsequent editions of the treatise on the baths of Newnham Regis.

(c) 'A Short Discourse of the Three Kindes of Peppers in common use and certain Medicines made of the same, tending to the preservation of health,' 1588.

In this book the preface has spaces left blank for the appropriate form of address. The copy in the British Museum is presented to "Ye right honourable my very good Ladye ye Comptesse of Harforde," and the preface is signed "Your honour's alway to commande Walter Bailey." The copy in the Bodleian Library is presented "To the right worshipful Sir Johne Horseley Knight." This copy has bound up with it a panegyric advertisement of Mr. Hugh Morgan, Her Majesty's Apothecary, addressed to Dr. Bailey "by your assured loving friend B. G.," and dated, Alvingham, August 14th, 1587.

Dr. Bayley begins his preface with the following words, those here printed in italics being written into the spaces left blank for them: "As in former yeares at this season, I have alwaies been carefull to discharge my dutie towards *your honour* by offering some present appertaining to the preservation of your healthe: so at this time *right honor*: in token of a good new yeare (God grant you many) I have emboldened myselfe to exhibit to *your honor* in our vulgar toong this short discourse of the three kindes of Peppers, and of those medicines compounded of the same greatly commended by Galen in his book 'de sanitate tuenda.'"

The three medicines are Diatrion pipereon, which, "by consent of all writers hath facultie to warm the stomach." Diaspoliticon, which "keepeth the belly loose," and Diacalaminthe, which "having more subtletie in substance doth penetrate further."

The preface concludes: "Wherefore I remaine to the Almightie a daily orator so to prosper the effects of these

medicines that the same commodities may come unto your *honor* which our authors do attribute unto them, that your yeeres may be enlarged to extreame age. Your honor's always to commande Walter Bailey."

This discourse, which is not very interesting, and is more academic than personal or practical, treats of the spice called "pepper" and of the kinds of pepper, wherein Dr. Bayley says: "The navigations in these latter yeares made by the Portingales into the East Indians and by the Spaniards into the West Indians hath made manifest to us how greatly the old authors, I meane Dioscorides, Galen, Plinie, Avicenna, Serapis and other writers of the former time were deceived in and about the history of pepper. . . . They concluded that these three kinds of pepper were all the fruit of one tree and differed only in that one was not so ripe as the other. But by the navigations of the Portingales and of the Spaniards in those countries in which these pepper trees do grow, it is evident and well knowen that the trees which do yeeld these three spices are divers in kind. . . . And that the plants which do beare white pepper and blacke pepper are not trees of any bigness, but weake shrubs which do clime by other trees as an ivie doth in this our countrie." He goes on to say that although he has never seen any living pepper trees yet: "I have often seene at Poole in Dorsetshire and also in London, the whole clusters of pepper preserved in brine and in salt: these clusters are long and thin and not so thicke together as the cluster of grapes." Dr. Bayley then quotes passages from Dioscorides, Galen, and other pharmacologists upon the methods of choosing pepper and of the uses of the various preparations, but there is nothing original in the rest of the pamphlet.

Arber refers to this book in his 'Transcript of the Register of the Stationers Company under the year 1588,' and says it is uncertain whether it was printed in London or at Oxford. I do not think it is issued from the same press as the two preceding pamphlets. It was not reprinted so far as I have been able to ascertain.

The Friends of Dr. Walter Bayley.

The Countess of Harforde, to whom Dr. Bayley gave a copy of the discourse on "Peppers," which she does not seem to have opened, because it is in the British Museum Library as clean and fresh as when she received it, was the second wife of Sir Edward Seymour, Baron Beauchamp, and Earl of Hertford. She was the sister of Charles, first Earl of Nottingham, and daughter of William Howard, first Baron Howard of Effingham. She died at the age of forty-four, in 1598, and was buried in Westminster Abbey. Edward Seymer, Lord Beauchamp, her step-son, matriculated at Magdalen College, Oxford, in 1576, and about the year 1585 married a daughter of Sir Richard Rogers, of Bryanston, in Dorset. Dr. Bayley may thus have become acquainted with Lady Hertford through her step-son either at Oxford or in Dorsetshire.

The copy of his discourse on the Newnham baths, which he gave to Lady Dacre, is also in the British Museum. It has been well read, and is annotated here and there. Lady Dacre was Ann, sister of Thomas, first Earl of Dorset, and daughter of Sir Richard Sackville. She was married to Gregory, Lord Dacre, who, Camden tells us, was said to be "crack-brained." He died in 1594, and was buried at Chelsea. Dr. Bayley may have attended him professionally.

Sir John Horsley, Knight, was knighted between September 18th and 25th, 1547. He was one of the Knights bannerets and Batchelor knights, made in the camp beside Roxburgh, in Scotland, in the first year of Edward VI's reign, by the hand of the high and mighty Prince Edward, Duke of Somerset, Lieut.-General of all the King's armies by land and sea, and governor of his royal person and Protector of all his realms, dominions, and subjects. I can discover nothing about him, but Dr. Bayley gave him a copy of the short discourse on "Pepper," presenting it to the Right Worshipful Sir

Johne Horseley, Knight, and subscribing himself throughout in more familiar terms than is usual with him.

Sir John Popham was the second son of Edward or Alexander Popham of Huntworth, Somerset. He was a member of Balliol College, Oxford; autumn reader at the Middle Temple 1568; treasurer of 1580; serjeant-at-law 1579; Recorder of Bristol; M.P. for Lyme Regis 1558, and for Bristol 1571 and 1572–1583; Speaker of the House of Commons 1577–1585; Solicitor-General 1579–1581; Attorney-General 1581–1592; Chief Justice of the King's Bench; Knighted 1592; died June 10th, 1607; buried in Wellington Church, Somerset. Dr. Bayley gave him a copy of his treatise on the eyesight, and inscribed it "To the right worshipfull my very goood frynde Mr. John Pophame Her ma: attorney general," signing himself "Your very lovinge frynde Walter Bayley."

The copy of the tract on the baths of Newnham Regis in the Bodleian Library has the dedication obliterated, but the signature remains "Your lovynge frynde Walter Bailey." It is possible that this copy also was given to the Attorney-General as the inscription is about the same length.

I have accidentally come across two references to Sir John Popham whilst reading some speeches of King James I ('Works,' London, 1616, p. 567). The King says in a speech in the Starre Chamber on June 20th, 1616: "Looke to your houses of Correction, and remember that in the Chiefe Justice Popham's time there was not a wandering beggar to bee found in all Somersetshire, being his native countrey." He also says: "Another thing to be cared for is the new buildings here about the City of London; concerning which my Proclamations have gone foorth, and by the chiefe Justice here, and his Predecessor Popham, it hath bene resolved to be a generall nuisans to the whole Kingdome; And this is that, which is like the Spleene in the body, which in measure as it overgrows, the body wastes. For is it possible but the Countrey

must diminish, if London doe so increase and all sorts of people doe come to London? And where doeth this increase appeare? not in the hearte of the Citie but in the suburbes; not giving wealth or profit to the city, but bringing miserie and surcharge both to Citie and Court; causing dearth and scarsitie through the great provision of victuals and fewel, that must be for such a multitude of people; And these buildings serve likewise to harbour the worst sort of people, as Alehouses and Cottages doe. I remember, that before Christmas was Twelve-moneth I made a proclamation for this cause, That all gentelemen of qualitie should depart to their own countreys and houses, to maintaine Hospitalitie amongst their neigbours, which was equivocally taken by some, as that it was meant onely for that Christmas; But my will and meaning was, and here I declare that my meaning was, that it should alwayes continue."

I think that this account of Dr. Walter Bayley and his works shows him to have been a type of what our best physicians were at the most brilliant period of English literary history. A Wykehamist and a West Countryman, he came of a large and influential family whose members for several generations maintained a close connection with the University of Oxford. It is remarkable as showing how closely he maintained his West Country connection to the end of his life that all the presentation copies of his works which I have seen were given to those who were connected with the counties of Dorset and Wiltshire, yet his descendants became an Oxfordshire family. A Fellow of New College, Oxford, in virtue of his education at Winchester, he falsified the gibe that "the scholars of New College are golden, the bachelors silvern, and the Masters leaden." The Patent Rolls and the University records show Dr. Walter Bayley to have been essentially a man of affairs. He amassed and dealt

shrewdly with large sums of money; with his relative Dr. Henry Bailey he was chosen by the University to represent its medical faculty when the Queen visited Oxford, and his treatise on the baths at Newnham prove him to have been possessed by the spirit of the age, so that if he had lived at a later time he would have been a pharmacologist and a scientific physician with a strong leaning towards the chemical side of medicine. I am indebted to the Rev. W. D. Macray, F.S.A., the present rector of Ducklington, for the reference which enabled me to find Dr. Bayley's will. It contains many interesting facts about him, and a copy of it is appended, therefore, to this paper. The will records that Dr. Bayley lived in Salisbury Court, Fleet Street, and that his wife Ann was an invalid, suffering probably from some mental disability. He had two sons and four daughters. The sons were William and Walter. Two of the daughters, Barbara and Marie, were unmarried. One, the wife of Dr. Ailworth,[1] appears to have died before her father made his will, as she is left nothing. The other was Mrs. Covert.[2] Dr. Bayley had two brothers living—Ralph and Jeffrey, and a married sister—Mrs. Alice Evered.

[1] Anthony Ailworth was a Fellow of New College 1563–1582 (from London); B.A. 1566; M.A. 1570; B.Med. 1578; D.Med. 1582; licensed to practise medicine 1582; Regius Professor of Medicine 1582–1597; physician to Queen Elizabeth. He does not appear to have attached himself to the College of Physicians. He died April 18th, 1619, aged 72, and was buried in the antechapel of New College. The inscription on the brass covering him runs:

"Hospes siste, Gradum numerosum perlege funus
Hic Jacet Hippocrates, hic Avicenna jacet.
Ossa Dioscorides sunt hic, sunt ossa Galeni
Et simul Aylworthum contegit iste lapis.
An tot congestos tumulum miraris in unum
At mirare majus, nempe tot unus erat
Qui tamen in vita simplex, ut dicere possis
Quod neque plus uili, nec minus artis erat."

[2] There were two Coverts, or Calverts, at Oxford at the end of the sixteenth century—Alexander, of St. Mary's Hall, born in 1562, afterwards of Gray's Inn; and Robert, of Magdalen Hall, who took his B.A. in 1575.

THE WILL OF DR. WALTER BAYLEY.

EXTRACTED FROM THE PRINCIPAL REGISTRY OF THE PROBATE, DIVORCE, AND ADMIRALTY DIVISION OF THE HIGH COURT OF JUSTICE.

In the Prerogative Court of Canterbury.

IN THE NAME OF GOD AMEN in the fowerthe daie of Januarie in the three and thirtiethe yeere of the raigne of our Soueraigne Ladie Elizabethe by the grace of god Queene of Englande Fraunce and Irelande Defender of the Faithe &c In the yeere of our Lord God accordinge to the Englishe Churche one thousand fiue hundred ninetye I WALTHER BAILY Doctor of Phisicke Phisicon in ordinarie to our said Souereign ladie beinge of whole and pfecte minde and memorie thanckes be given to god doe make and ordaine this my laste will and testamente in manner and forme followinge Firste I doe bequeathe my soule to allmightie god and my bodie to be honestelie buried without anie greate pompe in christiaun buriall at or in the churche of St Marie Colleage in Oxforde called commonlie Newe Colleage if I happenn not to die farr from thence wch I doe leave to the discrecoun of mine exequetor with thadvise of those which I doe put in truste as overseers of this my laste will And my will is that theare be a stone of marble laide vppon my grave my name and the time of my deathe engraived vppon it as shall seeme moste conveniente to mine exequutor and to mine overseers Item I give and bequeathe vnto my sonn William Bailie whome I make my sole Execuutor all my landes tenementes and heriditamentes in Stanlake More alias Northmore Barlie Parke in Ducklingeton in the countie of Oxford and all my lande in the Cittie of Oxforde and the Suburbes of the same And all other my landes tenementes and heriditaments whatsoeuer and whearesoeuer And wheare I haue one patente of dimise of the Chauntrie lande in Stanlake aforesaide made to me by our said Soueraigne the Queenes maiestie that nowe is in revercon of a former patent made theareof by our saide Soveraigne to one Castelman wch patente to Castellman is determined And whear I haue one other patent of dimise of certaine landes in More nowe or late in the occupacon of John Ferberd or his assignes wch twoe patentes of dimise graunted to me as aforesaide in the lawe I did then make ouer in truste to John Bailey of Stranbridge in the countie of Dorcet my neiphewe and to Jeffrie Bailie of the parishe of Winforde in the countie of somersett my brother with a prouiso in thassignmente reservinge full power and aucthoritie

to me and mine heires exequutors administrators and assigns vppon consideracon to revoke and make frustrate the said assignement at mine and theire willes and pleasures as by thindente of assignmente and the prouiso in the same dothe and maie appeare Nowe I giue and bequeathe to my said sonn Willim all the estate titell intereste and claime wch I have or maie have in the said two patentes together wthall aucthoritie right and interest wch I haue or may haue in the saide prouiso Nowe concerninge my wiefe my greateste care for that I doe beste knowe her infirmitie and weakenes to be suche as require the government And that all kindes of government cannott satisfie the same but that shee must have the carefull attendaunce of her deere frendes suche as of nature doe beare ann affection and love towardes her and of suche as also shee dothe naturallie bothe like and love vppon good and deliberate advisemente heere on by me taken I haue with my selfe resolued that my said sonn William Baileie when he shalbe setled in a house is and wilbe the fitteste psonn to take this care vppon him And I doe thearefore ernestelie require him as my speciall truste is in him like a kinde and lovinge childe dilligentelie tattende his aged and sickelie mother as he dothe look to enioie theblessinge to him for soe doinge by God in his holie worde promised And I doe hartelie desier them wch I make ouerseers of this my testamente and moste especiall myne honorable and good cozin Mr John Wolley [1] and Mr Ralffe Sheldon to directe and to take order and aduise my said wiefe wheare and with whome she maie remaine after my deathe vntill my said sonn Will^m^ shall be settled in a house And soe from time to time wheare and with whome it shalbe beste for her to bide and what people shalbe moste conveniente to have aboute her And I doe mosye hartelie praie my saide wiefe for the love wch hath binn betwine vs to referr her selfe to be ruled in this pointe by my said ouerseers especiallie by saide cozin Wolley and Mr Ralffe Sheldon And my will is and by this my testamente I bequeathe vnto Ann my saide wiefe yeerelie of Currante englishe moneie duringe her naturall liefe To be paid by mine exequutor before hande by evenn porcons quarterlie ouer and beside her meate drinke and chamber wch I will that my said sonn shall freelie giue her as longe as she shall continue and abide in house with him And as longe as shee shall like to be in house with anie other of my sonns or doughters

[1] Wolley (Sir) John, Fellow of Merton College 1553; B.A. 1553; M.A. 1557; D.C.L. 1566; of Pirford, Surrey; admitted to Gray's Inn 1592; Latin Secretary to Queen Elizabeth 1568, after the death of Roger Ascham; Canon of Wells 1569; Dean of Carlisle 1578, and Chancellor of the Order of the Garter; a commissioner on the trial of Mary Queen of Scots. M.P. East Looe 1571; Weymouth and Melcombe Regis 1572–1583; Winchester 1584 1587; Dorset 1588 1589; and Surrey 1593. Knighted 1592 and a Privy Councillor. Buried in St. Paul's Cathedral 1595.

or of any other person and personns as shalbe thoughte meete by mine overseers especiallie by my said cozin Wolley and Mr Ralffe Sheldon I will that my said son Will^{m} over and beside the saide somm of shall also yeerelie pay to the pson with whome my saide wiefe shall live and continue twentie marcks of like Englishe moneie for her boorde chamber or after the proporcon and rate of him that shee shall abide in house with suche psonn or psonns The reste for her boorde and maintenaunc to be supplied with her owne porconn of Prouided allwais and my will is that my saide wiefe for and in consideracon of this annuitie of and twentie marcks to be paide in manner and forme aforesaide shall leave yeelde vp resigne and relinquishe all her claime righte titell and demaunde wch shee hathe or maie haue in anie my landes tenementes and hereditamentes or in anie of my goodes and chattells (other then is to her bequeathed and given in this my laste will and testamente) by reason of her thirdes or by reason of anie other claime righte titel or intereste wch if shee my saide wiefe shall refuse to do And shall at anie time after my death claime any intereste porcon or righte in anie of my saide landes tenementes hereditamentes goodes or chattels (other then is to her bequeathed in this my testamente Then my will is that this my legacie for the paimente of and soe for the paimente of twentie marks and soe all other legacies due to her by this my laste will and testamente shall surcease and be meerelie voide to all intentes and construccons Itm my will is that if my said wiefe doe not make claime as aforesaide Shee shall haue the vse and occupacon of soe muche of my beddinge and furniture to the same And the vse and occupacon of soe muche of my linnen and of my plate pewter and brass as shalbe thoughte needefull and conveniente for her by mine overseers or three of them my good cozen Wolley and Mr Ralfe Sheldon beinge twoe of them To be deliuered to my saide wiefe within one quarter of a yeere after my death. Soe that my saide wiefe doe put in suerties to leave the same to mine exequutor or as good after her deathe as well for the plate as for the beddinge pewyer linnen & brasse Item I giue and bequeathe to my sonne Walther Baileie all my lease of Burle ferme in Evershint and Fromquinton in the countie of Dorcet Item I giue to my saide sonn Walther all my lease and leases of or in the mannor of Melsburie or anie parte theareof in the countie of Somersett Item whearas my saide sonn William Baileie is named in one copie nowe in beinge of a copieholde in Nye and Dinghurste in the parishe of Ninchcombe in the said countie of Somersett To enter vppon the same after the deathe of Elizabethe Stone alias Saton mine aunte Wheareof a reuercon is graunted to my said sonn Walther and to others as by the copie in reuercon dothe appeare My will is that when the said Elizabeth Stone my said aunte shall happen to die whereby the said holde shall

then come to my said sonn William by the firste copie nowe in beinge Then my said sonn William shall suffer my saide sonn Walther to occupie the saide holde during my saide sonn Williams naturall liefe he my said sonn Walther paieinge the rente to the Lordes and all other paimentes duties & seruices for the same and theareof dischargeinge my said sonn William his exequutors & administrators Yf my saide sonn Willim shall refuse soe to doe my will is that he my sonn William shall paie to my saide sonn Walther tenn poundes yeerelie and everie yeere as an annuitie of good englishe moneie at twoe vsuall feastes in the yeere by evenn and equal porconns duringe the time that my said sonn William shall occupie the said holde All wch annuitie and profitts of all and singular the premisses bequeathed to my said sonn Walther shalbe emploied as is heereafter expressed viz That wheare my saide sonn Walther is yeat vnder age My will is that the commodities and profitts of the said leases of Burle ferme and Melsbery and also of the copie holde in Eversint and of thother copie holde in Nie and Dingehurste aforesaide if the same shall happen to faull togeather wth thannuitie and paimente as my saide sonn William by this my testamente is to paie to my saide sonn Walther if he my sonn William shall occupie the holde in Ny and Dinghurste himselfe shalbe receaued by mine exequutor (the mine exequutor puttinge in sufficiente assurance to mine ouerseers or to three of them my cozen Wolley or Mr Ralfe Sheldon beinge one of them) That the said profitts and annuitie shalbe emploied as followeth That is to saie that tenn poundes yeerelie theareof shalbe bestowed in and vppon the education and bringing vp in lerninge of my saide sonn Walther vntill my saide sonn Walther shall accomplish the age of twentie and fower yeeres And the residewe to be imploied to the paimente of my debtes And wheare I haue procured to my said sonn Walthr of the Deane and Chapter & the cathedrall churche in Wells one copie holde in possession of certaine overlande in Winchcombe aforesaide late in thoccupacon of George Paine Gentleman or his assignees wch is nowe lett for a yeerelie rente And one other copieholde in Winchcombe aforesaide in reuercon of the saide George Paine and Eyd his sister And one other copie holde in winchcombe aforesaide in reuercon of one John Saunders and Margerett his wiefe as by the copies of the same it dothe appeare My will is that my said sonn Walther shall suffer those wch I doe put in truste or three of them my Cozin Wolley or Mr Ralfe Sheldon beinge one To haue the lettinge of theas and euerie of theas copie holdes when theie or anie of them shall fortune to faull and to suffer mine exequutor to receaue all the profitts of the saide holdes To thintente that the same maie be imploied to the paimente of my debtes vntill the same shalbe paid or vntill my saide sonn Walther shall accomplishe the age of twentie fowr yeeres And then after my debtes paide thoughe

it be before my said sonn shalbe of thage of twentie fower yeeres from thence and after the whole profitts of all the premisses bequeathed and cominge to my saide sonn Walther Shalbe receaued by mine exequutrs To be bestowed by the consente and orderinge of mine overseers or three of them my cozen Wolley or Mr Ralffe Sheldon beinge one To the vse and behoofe of my saide sonn Walther to make a stocke for him vntill he shall accomplishe the age of twentie fower yeeres And after he my sonn Walther to receave and dispose of the premisses dewe to him to his beste likinge And my will is that before my saide sonn William doe receaue anie of the profitts of the premisses due to my saide sonn Walthr he my said sonn Will^{m} shall put in sufficiente assurance for the trewe answeringe of such moneie as he shall receave accordinge to my true meaninge in this my will and testamente Prouided allwaie and my will is that if my said sonn Walther shall not agree and be contented that the profitts of the premisses bequeathed to him in this my laste will or that the profitts and commodities of the copie holdes menconed wch shall in the meane fall to him as is aforesaide shalbe imploied accordinge to this my will in this my testamente Then my will is that he my saide sonn Walther shall loose all his righte wch he hathe or maie haue by this my last will and testamente in the leases bequeathed to him of and in Burle Ferme and Melsburie and that the same shall goe to mine exequutor as if the same had neuer binn bequeathed Item I giue to my saide sonn Walther my third beste feather bed bolster and pillowe and my third beste couerlett my thirde beste paire of blancketts my thirde beste bedsteade and furniture belonginge to the same Item I giue to my said sonn Walther my three haunce siluer cuppes gilte My siluer salte with a couer havinge the crosse keyes in the topp of the couer My greate siluer boull with a couer gilte wch boule I doe vse at the Courte Item I giue to my saide sonn Walther Six siluer spoones the knobbes gil^{t}e Item twoe gilte siluer spoones with letters W.B. in the end of them All theas latter legacies of beddinge and plate my will is shalbe deliuered to my saide sonn Walther when he shall accomplishe the age of twentie fower yeeres In the meane to be in the keepinge and custodie of mine exequutor (he puttinge in sufficiente bond to those wch I make mine ouerseers or to three of them my cozen Wolley or Mr Ralfe Sheldon beinge one trulie to aunswer the same when it shalbe dewe) Prouided allwaies and my will is that if my saide sonn Walther shall happen to die before he doe accomplishe the age of twentie fower yeeres That then all thinges bequeathed and givenn to him by this my laste will shall remaine and be due to my said sonn William and to his childrenn (if he be then livinge) or haue childrenn And if he my said sonn Will^{m} shall happen then to be dead without issue of his bodie Then my will is that all legacies

and thinges bequeathed to my saide sonn Walther shalbe equllie diuided amongste my daughters then livinge Item I giue to my daughter Barbara Twoe hundred pounds to be paide vnto her at the daie of her marriage or when shee shall accomplishe thage of twentie one yeeres So that the same maie by that time be levied as heereafter is appointed by this my testamente or to be paide soe soone as the same shalbe levied in manner as is heereafter expressed Item I giue to my saide daughter Barbara my fowerth beste feather-bed bolster and pilowe my fowerthe beste couerlett and paire of blancketts Item I giue to my daughter Barbara a silver gilte standinge cup with a cover ingraued

Item a siluer gilte tankard with a couer Item one gilte siluer spoone with letters A.S. in the knobb Item a siluer bell salte with a couer wch I doe vse at the Courte And also a siluer salte with a couer called a trencher salte the couer havinge a pepper box in it All wch legacies of beddinge and plate my will is shalbe deliuered to her at the daie of her marriage or when shee shall accomplishe the age of twentie one yeeres Item wheare my daughter Barbara is yet vnder age and hath no yeerelie revennewe to find her selfe My will is that mine Exequutor shall paie to her yeerelie the somm of six pounds at twoe vsuall feastes in the yeere by evenn porcons towarde her maintenaunce vntill twoe hundred marckes of the legacie of twoe hundred poundes shalbe paide vnto her in manner as is heereafter expressed Item I giue to Marie my daughter two hundred poundes To be paid to her at the daie of her marriage or when shee shall accomplishe the age of twentie one yeeres so that the same be by that time levied or soe soone as the same shalbe levied in manner as is heereafter expressed in this my testamente Item I giue to my saide daughter Marie my fiveth beste featherbed bolster and pilloe my fiveth beste couerled and paire of blancketts Item I giue to my saide daughter Marie one siluer salte called a trencher salte And one standinge silver gilte bowle with a couer ingraved Item a standinge cupp with a couer gilte havinge siluer leaves in the foote Item twoe gilte spoones of siluer with great knobbes at thende And the letters M.S. at the one ende And the letters M.B. at the other ende of the steale or hande by the boll of the spoone All wch legacies of beddinge and plate shalbe deliuered to my saide daughter Marie at the daie of her marriage or when shee shall accomplishe the age of twentie one yeeres And my will is that the profitt of a copie holde in Stert in the countie of Wiltes wheare to my daughter Marie is nowe tenente wch holde is in possession and let for six poundes cleere to Richarde Longe shalbe paid yeerelie to my saide daughter Marie by the discretion of mine ouerseers or three of them my cozen Wolley or Mr Ralffe Sheldon beinge one Prouided allwaye and my will is that from and ymeadiately after my deathe by the discrecon of myne ouerseers or three of them my cozen Woolley or Mr Ralffe Sheldon

beinge one of them out of the profittes and commodities of the rectorie and psonage of Asheberie in the countie of Berks theare be yeerelie levied one hundred marks for the paienge to my daughter Barbara her porconn of moneie of twoe hundred pounde bequeathed in this my laste will and soe from yeere to yeere vntill the said somm of CC^{li} shalbe receaued And immediatelie after the said somm of CC^{li} shalbe levied My will is that in like sorte one hundred markes shalbe yeerelie levied out of the profitts of the said psonage of Ashberie for the aunsweringe of my daughter Marie her portion of twoe hundred pounde bequeathed to her in this my laste will vntill the somm of CC^{li} shalbe levied And my will is that as the saide somm or somms of one hundred marckes shalbe levied yeerelie as is aforesaide soe the same from time to time shalbe receaued by mine exequutor to be imploied to the vse and benifitt of my saide daughter Barbara by the discreconn of mine ouerseers or three of them My Cozenn Wolley or Mr. Ralffe Sheldon beinge one of them togeather with mine exequutor And that so soone as twoe hundred markes shall be levied as aforesaide Then and from thence foorthe the legacie of six pounde by the yere bequeathed to my saide daughter Barbara for her mainetenance shall surcease and be meerelie voide In like manner my will is that when anie of the somm or somms of one hundred marckes or anie parte theareof shalbe levied towarde the paimente of my daughter Marie her porconn of twoe hundred poundes The same shalbe receaued by mine exequutor and emploied to the vse and benifitt of my said daughter Marie by the discreconn of mine ouerseers or three of them My cozen Wolley or Mr Ralffe Sheldon beinge one of them Item my will is that the beddinge and plate bequeathed to my saide twoe daughters shall remaine in the handes of mine exequutor vntill the same shalbe due at the times aforesaide Prouided allwaie that my said exequutor doe put in sufficiente bond with suerties to those wch I doe put in trust or three of them my cozen Wolley or Mr Ralffe Sheldon being one of the three for the trewe levinge and paimente of the said somms of monie And for the trewe deliueringe of the saide beddinge and plate accordinge as the same shalbe due accordinge to my meaninge in this my laste will and testamente Prouided also That if anie of my saide twoe daughters shall happen to die before the saide porconn or porcons of moneie and other legacies bequeathed shalbe dewe and paide accordinge to my meaninge in this my testamente Then the portion and porconns of moneie and other thinges bequeathed to her soe dead shall goe and be dewe to the survivor daughter If bothe daughters happen to die before the saide porconn and porconns of moneie and other the premisses bequeathed to my saide twoe daughters shalbe due then my will is that they and euery of theyr porconn and porcons of moneie and other the legacies bequeathed vnto them shall remaine and be due to my

saide twoe sonns equallie to be diuided or to the survivor of them Item I giue to my brother Ralffe Bailie if he be livinge at the time of my death my seconde beste horse mare or colte Item I giue to my brother Jeffrie Bailie if he be livinge at the time of my deathe My thirde beste horse mare or colte And to everie of theire wiues wch shallbe livinge at the time of my deathe a ringe of golde with a deathes head on the foare side and W.B. for my name in the inner side of the value of twentie shillings or thereaboute Item I giue to my sister Alice Evered if shee happen to be livinge at the time of my deathe a like ringe of golde of like value Item I giue to mine aunte Elizabeth Stone alias Eaton in Wells one other like ringe of golde of like value Item I giue to the Wardin and Schollers of St Marie Colleage in Oxforde called New Colleage one greate standinge cup with a cover gilte with a leather case to and for the same wch greate standinge cupp is in my plate cheste at Mr Humphrie Wem's house by the greate conduite in fleete streete and the case is in my gallerie in my house in Sarisburie Courte Item I bequeathe to the saide Wardine and scollars the somm of six pounde thirteene shillinges fower pence in moneie if I be buried theare to amende theire commons To be paide vppon the daie of my buriall Item I giue to the saide Wardine and schollars one of my Galenes Workes in greek of Basils printe bounde in three volumes Item Matthiolus commentaries vppon Dioscorides of the best edicon wch I haue Item my Fuchius herball in folio Item all my bookes of Gesnerus de quadrupedibus de Avibus de aquatilibus de oviparis et reptilibus wch are bound in three bookes or volumes Item definitiones medicæ Gorræi Item Theatrum Galeni in folio Item Brasavolus in aphorismos Hippocratis Item in libros hyppoc. de ratione victus in morbis acutis Soe that the saide Wardine and schollars doe cause all and everie the saide bookes to be placed in the librarie of the saide colleage amongste other phisicke bookes theare And my name to be sett vppon the forrells and coveringes vnder horne as some other bookes in the said librarie are placed and ordred within one yeere after my deathe[1] Item I giue to my sonn in lawe Doctor Ailworthe twentie of my phisicke and philosophie bookes not bequeathed to be chosenn by himselfe And my skeliton of bones in Oxford and a ringe of golde with deathes head and my name to it as is aforesaide of the value of fortie shillinges Item I giue to my daughter Em Covert a silver boull without a couer gilte waienge aboute thirteene ounces Item I giue to my sonn in lawe her husbande if he be livinge at the time of my deathe a ringe of golde with deathes head and my name to it as is aforesaide of the value of fortie shillinges Item I

[1] Prof. Osler has very kindly visited New College. He tells me that the books still remain, but the standing cup has long since gone, though there is a cocoa-nut cup presented by Katherine Bayley.

giue to everie Servaunte wch hathe dwelte with me one yeere at the leaste before my deathe and shalbe in seruice in house with me at my deathe twentie shillinges To be paide within one quarter of a yeere after I am dead All other my landes tenementes heriditamentes goodes and chattells not bequeathed I giue and bequeathe vnto Willim Bailie my sonn whome I make my sole exequutor of this my laste will and testamente And wheare I anie at this pnte in debte and doe owe as appearethe by a shedule annexed to this my laste will the somm of
And I desier the speedie paimente theareof for that a great porconn of my plate liethe in pawn to my good frende Mr Humphrie Wems at and for five shillinges the ounce being more worthe for parte of my saide debtes And I doe paie intereste for the greateste parte of my debtes I will that the lease wch I haue of corpus christi colleage of a ferme in Litle Staughton in the county of Bedforde for wch lease I haue binn offred fowerscore poundes shalbe sould by the discrecon of mine ouerseers or three of them my cozen Wolley or Mr Ralffe Sheldon beinge one Togeather with the consente of mine exequutor And that the stuffe wch I haue in my house wch I haue in Sarisburie Courte viz duringe the naturall liefe of mine honorable good ladie the Ladie Dacre of the South be soulde by the discreconn of mine overseers in manner and forme aforesaide with the consente of mine exequutor And I will that the moneie wch shalbe receaued for the premisses soe solde shalbe receauid by mine exequutor and imploied by mine ouerseers discreconn towarde the paimente of my debtes wch mouere together with the debtes owinge to me and the fines wch are yeat vnpaide for thinges allreadie lett and wch maie be made of thinges to be lett of the Chauntrie lande in Stanlake (as appearethe by a schedule annexed to this my testamente) will make vp the somm of or theareaboute towarde the dischardge of my debtes And then twoe hundred-poundes a yeere out of the profittes and revennues of thinges bequeathed and lefte to my sonn William and to my sonn Walther as is aforesaide will in shorte time aunswer all my debtes Notwithstandinge this mine opinion I leave thorderinge of this matter touching the paienge of my debtes To the discreconn of mine ouerseers or three of them My cozen Wolley or Mr Ralffe Sheldon beinge one of them Togeather with the consente of mine exequutor Praienge mine overseers ernestelie to psuade with mine exequutor in no case to allien or sell my landes nor the lease nor advowson of the rectorie or psonage of Witneie in the Countie of Oxon Nor thadvowson or lease of Ashberie in the countie of Berks And my will is that my saide sonn Willim shall not allien nor sell anie of thease latter menconed premisses without the consent of mine overseers or three of them my cozen Wolley and Mr Ralffe Sheldon beinge twoe of the three And my will is that firste order be takenn by

mine ouerseers and three of them Mr Wolleie or Mr Ralffe Sheldon beinge one for the leavienge of my saide wives porconn to be paid as aforesaide And then next for the paimente of my debtes And after for the paimente of other legacies bequeathed in this my laste will Beside the porcouns of twoe hundred pounde a peece bequeathed to my daughters Barbara and Marie beinge to be levied and paide out of the psonage of Ashburie as is aforesaide Item whearefoure pte of my plate in this my will bequeathed doth lie in pawne with Mr Humphrie Wems as is aforesaide My will is that the same be redeemed so soon as convenientelie maie be And that the moneie for wch the same dothe lie in pawne be firste paid. That then theas my legacies of plate to my sonn and daughters maie be pformed to my sonn and daughters accordinge to my meaninge in this my will freelie without paienge anie thinge for the same And for the better pformance of the premisses I doe make choice of and doe hartelie praie mine honorable good frende and kinseman Mr John Wolley one of her Maiesties moste honorable Priuie Counsell My verie good frend Mr Ralffe Sheldon of Bely in the countie of Worcester Esquier Mr Mr Humphrie Wems of the pishe of St Brides alias Bridgett nighe fleete street in the suburbes of London Grocer Mr Thomas Brigham of Caversham in the countie of Oxon gentleman To take the paines as to see this my will and testamente to be in all thinges pformid in manner and forme as I haue expressed and to doe all thinges requisite for the better pformance of this my will accordinge to my meaninge in the same To wch ende and purpose I doe ordaine and make them mine ouerseers of this my laste will and testamente Reposing in them my speciall truste and confidence for the performance of the same And I doe giue to everie of them to be levied of my goodes and chattells vnbequeathed or lefte to mine exequutor And my will is That my saide ouerseers shall haue full power and auctboritie to dispose of all thinges in this my testamente accordinge to my meaninge in the same And that if anie controuersie shall fortune to arise in or about the same That they and euery of them betweene whom such controuersie shall arrise Shalbe whollie be ruled and ouer ruled and stande to the arbitrumente and iudgmente of mine overseers or three of them My cozen Wolley or Mr Reffe Sheldon beinge one of them And whoe soeuer to whome anie legacie is giveun in this my laste will and testamente when anie suche controuersie shall arrise wch will not abide and stande to the order of my said ouerseers as aforesaide Then he and theie wch shall soe refuse shall vtterlie loose his or theire pte and ptes of the thinge wch he or theie doe strive for as if the same had not binn bequeathed to him or them And the saide pte and ptes shall remaine and be due to mine exequutor as if the same had neuer binn bequeathed

MEMORANDUM that in a certain schedule of paper vnder the hande writinge of the testator aboue named was founde in manner of a codicill or declaracon of the testamente aboue written as followethe:—

Yeerlie to be paide by mine exequutor by this my testamente Anno 1590.

Inprimis to mywieffe during her liefe p annum . L^li
Item to my daughter Barbara vntill her legacie be due and paid yeerelie vj^li
Item to my sonn Walther for his bringing vp in lerninge x^li

Proved with a codicil 25th March 1592.
Fos 63 OB HK.
24 Harrington.

Exhibition of Historical and Antiquarian Objects shown in connection with the reading of Mr. D'Arcy Power's Paper.

Shown by Mr. D'ARCY POWER

Precept for Barbers not to use their Trade on Sundays.— An original manuscript, injured by mice, written on parchment and dated Lambeth, 19th April, 1445. The manuscript is a letter directed to "the venerable and religious Abbot of the Monastery of St. Peter's, Westminster, written by John, Archbishop of Canterbury. The letter states that the Archbishop had received a communication from Eugenius, dated "St. Peter's at Rome, 9th April, 1431, in the first year of our pontificate," "to his venerable brothers, the Archbishop of Canterbury and the Bishop of Winchester as well as to his beloved son the dean of the church of London." The Pope states that he had heard that the Barbers of London kept their shops open and carried on their trade upon Sundays and Feast Days, and he directs them to be warned against so doing under the penalties of the greater excommunication.

Mr. Sidney Young says, in his 'History of the Annals of the Barber Surgeons,' that Thomas Arundell, Archbishop of Canterbury, denounced the barbers for Sunday trading in 1413. His letter is addressed to the Mayor and Aldermen of London, and, after threatening the penalties of the greater excommunication, it goes on to say, "But, dearest children, seeing that so greatly has the malice of men increased in these days, a thing to be deplored, that temporal punishment is held more in dread

than clerical, and that which touches the body or the purse more than that which kills the soul, we do heartily intreat you, and, for the love of God and His law, do require and exhort you that taking counsel thereon, you will enact and ordain a competent penalty in money to be levied for the Chamber of your City, or such other purpose as you shall think best, upon the Barbers within the liberty of your City aforesaid, who shall be transgressors in this respect; that so at least, those whom fear of the anger of God doth not avail to withhold from breach of His law, may be restrained by a scourge inflicted upon their purse, in the way of pecuniary loss." An ordinance was made in consequence of this letter to the effect that no barber, his wife, son, daughter, apprentice or servant should work at such craft on Sundays, within the liberty of the City, either in hair cutting or shaving, on pain of paying 6*s*. 8*d*. for each offence; 5*s*. thereof to go to the new work at the Guildhall and the remainder to the Wardens or Masters of the Barbers within the city. The letter, which is now exhibited, dated twenty-two years later, shows that the ordinance could not have been carried out very effectually.

John, Archbishop of Canterbury, was John Stafford, who succeeded in 1443 Archbishop Chicheley, the founder of All Souls' College, Oxford. He was the son of Sir Humphrey Stafford, called "of the silver hand," Sheriff of Somerset and Dorset, and was educated at Oxford where he became D.C.L. before 1413. In 1419 he became Dean of the Court of Arches; in 1422 Dean of St. Martin's, London, where is now the General Post Office; in 1423 Dean of Wells, and in 1425 Bishop of Bath and Wells. In 1421 Stafford was made Keeper of the Privy Seal; in 1425 he was one of the Lords of Council during the minority of the king, and he accompanied the young king to France in 1430. After his return to England he was made Chancellor in March, 1432, and he is the first who is known to have been called "Lord Chancellor." The novelty of the title is interestingly brought out in the letter shown here where he is content to describe himself simply as "Judex." Stafford was a member of the commission appointed to try the offenders in Jack Cade's rebellion. He died at Maidstone on 25th May, 1452, and was buried in the Martyrdom at Canterbury Cathedral. He was a cautious and experienced official, who did no harm if he did but little good. In 1444 he decreed that in future no fairs or markets should be held in churches or churchyards, or on the Lord's Day or Holy Days, except in the four weeks of harvest.

The Abbot of Westminster, to whom the letter is addressed, was Edmund Kerton, who became a monk of Westminster in 1403, and graduated B.D. from Gloucester Hall (Worcester College), Oxford, and where he was Prior of the Benedictine Scholars in 1423. He visited Rome and preached before Pope

Martin V (1417-31) and in 1437 he attended the Council of Basel where he was cited to appear before Pope Eugenius IV on a charge of heresy. Kerton (whose real name seems to have been Cobbledike) was elected Abbot of Westminster in 1440. He resigned on account of ill-health in 1462 and died in 1466. His tomb in St. Andrew's Chapel, Westminster Abbey, formed part of a screen which he had himself designed. It has long since disappeared.

Pope Eugenius IV was Gabriel Condulmier, a Venetian. His father died young, and, after distributing his fortune of 20,000 ducats to the poor, in a fit of religious enthusiasm he entered the monastery of St. Giorgio d'Alga, in Venice, with his cousin, Antonio Correr. Antonio's uncle was unexpectedly elected Pope Gregory XII, when Condulmier was made Bishop of Siena and Antonio Bishop of Bologna; they were shortly afterwards chosen Cardinals of the United Church. Condulmier was elected Pope at the age of 47 on 3rd March, 1431, and he died, at the age of 67, on 23rd February, 1447. During his pontificate, and chiefly at his instigation, Fra Angelico decorated the Vatican Chapel, and the Pantheon was restored; his gates still adorn the central doorway at St. Peter's. In person he was tall and spare, handsome when young, imposing when old; a martyr to gout, though he drank nothing but water. He slept little, and was reserved and retiring, rarely lifting his eyes from the ground on any public occasion. He was singularly uncultivated, and was in no way distinguished for political capacity, yet he had to manage the affairs of the Church at one of the most dangerous crises of its history.

Four "dissected" plates, mounted on cardboard, representing the male and female body from youth to age allegorically as well as anatomically. The plates are entitled Spring, Summer, Autumn, and Winter. The Autumn plate contains a nativity calculated at Bononia, 4.32 p.m., 22nd May, 1605. Otherwise there is no indication of their source. They are not copies of the Catoptrum Microcosmicum by Johannes Remmelin of Ulm, which were published without his consent in 1613, and they are, I think, earlier, though the Catoptrum is usually said to have been the first "dissected" plates issued.

Pinax Microcosmographicus, by Stephanus Michaelus Sphacerus; a Dutch translation, dated Amsterdam, 1634. These plates were inspired by, even if they are not direct copies of, Remmelin's Catoptrum Microcosmicum.

Shown by Dr. J. F. PAYNE

Medical Diplomas

1. Diploma of Doctor of Medicine conferred by the University of Padua on *Robert Ley*, described as "Præclariss. Nob. Dom. Robertus Ley, Londinensis Anglus, Præclariss. Nob. Dom. Thomæ Ley Filius."

He is said to have answered at his examination in such an "excellent and doctorial manner" that he "not only came up to the great expectations which he had aroused in the minds of all, but very far surpassed them." (This was a common form.) He was admitted with the usual solemnities of the books, the ring, the cap, and the "kiss of peace." Dated 12th January, 1696.

In original morocco binding, with one empty seal-case.

2. Diploma conferred by the University of Caen, in Normandy, upon William Forester, an Englishman described as "Eruditissimus juvenis Gulielmus Foresterus, vir in praxi medicinæ et Chirurgiæ exercitatissimus, idemque in facultate Medicinæ Licentiatus gradu in Academiæ Cantabrigiensi decoratus." The document states that the University of Caen was founded by Henry VI, King of England. With one seal attached. Dated 28th July, 1608.

3. Diploma of Doctor of Medicine, conferred by the College of Physicians of Venice on Marcus Sexterus. The degree was conferred with the books, the ring, the doctor's cap, and the "kiss of peace" as in Universities. Dated 18th September, 1736.

In original decorated binding.

Manuscripts

1. Treatise on Anatomy and Surgery, in English, dated 1392. The writer's name is not given, but he was a surgeon practising in London. It is the only mediæval treatise on anatomy known to the present possessor, which is written in the English language. The language much resembles that of an English translation of Lanfrank's 'Cirurgie,' printed by the early English Text Society in 1894. This book was the original of the printed book called 'Vicary's Anatomy' (see paper by J. F. Payne, on an English Anatomical Treatise of the 14th century, 'Brit. Med. Journal,' 25th January, 1896).

Folio, on paper, double columns, in large Gothic script.

2. Folio MS on vellum, of 15th century; containing—(*a*) An English 'Medicine Book' or collection of receipts. The text is

the same as that of the work published as 'Ein Mittel-Englisches Medizin-Buch,' by F. Heinrich, Halle, 1896, from British Museum MSS. The author speaks of "The good Earl of Hereford, that was a noble surgeon," and since the *Earldom* became extinct in the 14th century, the book was probably written then.

(*b*) Several medical treatises in Latin. One is a treatise 'De Passione Oculorum' (by Zacharias) from which a passage is interpolated into Gilbert's 'Compendium Medicinæ.'

3. Latin MS on vellum, of the 15th century; containing 'Compendium Medicinæ' of Gilbertus Anglicus, imperfect at beginning and end. In old binding; from some monastic library, probably the Abbey of St. Alban's. The text agrees generally with the printed edition.

SHOWN BY DR. F. W. COCK

(*a*) Richard Mead's stop-seconds watch, 1744.

(*b*) A double-cased stop-seconds watch, 1776. Silver gilt. This is very unusual. Copper gilt and pinchbeck common. N.B.—The pulse watch was invented about 1708.

(*c*) Shagreen and silver pocket ointment box carried by surgeons to dress their patients, circa 1780. *Note* that one of the ointments was unguentum hydrargyri, and that the mercury has attacked the silver lining.

(*d*) Silver caustic case. One end for lunar caustic, the other for the oxide of mercury powder. *Note* the "saw" edge. 1791.

(d_1) Canaliculus operation case; early nineteenth century.

(*e*) MS of 15th century on Vellum and Paper, "De Cyrugia."

(e^1) Vigo's Surgery. Only known autograph of Wm. Clowes, Surgeon to Queen Elizabeth, and St. Bartholomew's Hospital, on Title page.

(*f*) and (*g*) Two MS pharmacopœias of the Waylett family, who were apothecaries in the neighbourhood of Rye, Sussex, 1650—1815. The older contains the obstetrical list of the last of the family 1757—1815. The later one should be noted, as the writer quotes from all sources of contemporary medical literature, showing that it was possible even in an out-of-the-way country village for the local doctor to keep abreast of knowledge in the 18th century.

(*h*) Some bills of the same written in Latin.

SHOWN BY MR. L. A. LAWRENCE

1. Transcript (1784) of Lectures on Surgery by Percivall Pott.
2. Works of Taliacotius. Venice, 1597.
3. Myographia Nova, by John Browne. London, 1698. L. P. copy with coloured plates. Red morocco binding.
4. Works of Galen, folio 1562. Stamped vellum binding.
5. Mezzotint portrait. Frater Jacobus de Beaulieu Lithotomus.

DISCUSSION

The PRESIDENT said: I think that the pleasant custom of old times of authors giving their own books as New Year gifts is one to be commended to the notice of the Meeting for adoption. I was struck by the knowledge James I showed of pathology, in noticing that enlargement of the spleen was accompanied with a pale and sallow complexion.

Dr. OSLER said: Walter Bayley did not forget his old college—New College—in his will. The big cup mentioned in it went into the melting-pot in 1643. There is, however, still in existence an interesting cup given by his assistant to the College. There is now no remnant of the funds for the library, and for the support of the high table. I think no Regius professor since had been able to make such a will; those were the palmy days of Regius professors.

Dr. J. F. PAYNE said: I think that all Mr. D'Arcy Power has discovered is entirely new. Doctors of that time probably possessed more real knowledge than is now usually believed, possibly because they did not write large books. Elizabethan medical literature cannot be compared in importance with that of foreign countries; its surgical literature was better. Dr. Bayley probably did not perform operations. He showed remarkable self-effacement by not publishing his name with his books, but writing it in them for his friends.

Mr. D'ARCY POWER said one reason probably why physicians did not write more in Elizabethan times was because they were so much involved in political intrigue, and suffered so much from the consequent racking. Surgeons, on the other hand, were more men of business, and had more time for writing and less physical incapacity.

A CASE OF CERVICAL MYELOPATHIC TRICHOSIS

BY

SIR WILLIAM GOWERS, M.D., F.R.S.

Received March 8th—Read May 28th, 1907

THE occasional occurrence of an abnormal growth of hair across the lumbar region of the back in cases of spina bifida occulta has long been known. Attention was called to it by Virchow,[1] by Fischer,[2] and especially by v. Recklinghausen in his elaborate study of spina bifida published in 1886.[3] The facts were collected and illustrated in a comprehensive lecture by Mr. Bland-Sutton in 1887.[4] In all the cases hitherto described the growth of hair was in the lumbar or lumbo-sacral region, extending on both sides, and was accompanied by obvious defect in the arches of the lumbar spine, and by symptoms in one or both legs of a defective development in the lumbar region of the spinal cord. In one case (that of Fischer) the patient, a girl, had also a tuft of long hair growing from a small spot over the upper cervical spine,

[1] Virchow, 'Zeitschr. f. Ethnologie,' 1875, and 'Berlin. klin. Wochenschr.,' 1884, p. 747.

[2] Fischer, 'Virchow's Archiv,' Bd. cvi, 1886.

[3] v. Recklinghausen, 'Virchow's Archiv,' 1886, and 'Spina Bifida,' Berlin, 1886.

[4] Bland-Sutton, 'Lancet,' 1887, vol. ii, p. 4.

but there was no defect in the bones of this region or symptoms in the arms.[1]

The patient whose case I have to describe to the Society presents a growth of hair in the cervical region only; it is strictly unilateral, being limited by the middle line; it is on the right side and accompanied by paralysis of the right arm and slightly of the right leg; while sensibility to pain alone is lost in a band from the spine to the shoulder—unequivocal evidence that the loss of power is due to syringomyelia on that side. The spine seems quite intact. The case thus presents a simpler problem than those previously recorded. I could wish that my account were fuller, but all know how difficult it is to secure fulness as well as accuracy in cases seen only in private.

Since the cases previously published of what may be called myelopathic trichosis have presented definite indications of defect in the bones, spina bifida occulta, I will add particulars of another case, which I have briefly referred to elsewhere,[2] in which a huge mass of lumbar hair also accompanied definite symptoms of lumbar syringomyelia; defect in the lumbar spine is uncertain.

The first case is that of a man, aged 41, who was sent to me by Dr. C. H. Andrews, of Norwich, on account of paralysis of the right arm, and slightly of the right leg. It had been thought to be of cerebral origin. The arm had never been quite strong, although he was right-handed. The definite loss of power came on gradually three years ago, and slowly increased. For the last six months it has been stationary. I saw him in November, 1906, and in January, 1907.

[1] In Gould and Pyle's 'Curiosities of Medicine,' Philadelphia, 1897, a similar lock of long hair is depicted, growing from a spot to one side of the upper dorsal region. It was supposed to arise from a hairy mole. The source of the figure is not stated. The subject is said to have been exhibited in public.

[2] Lecture, "Dystrophy of Tabes, etc.," 'Brit. Med. Journ.,' June 2nd, 1906.

His state was then as follows: There is no affection of the face, eyes, or any cranial nerve, and all movements of the neck are normal, and so are the neck muscles. The right arm is everywhere weak, but no movements are lost. The grasp of the right hand is 27 lb., of the left 70 lb. The fingers are generally slightly flexed, but can be well extended. There is no wasting of muscles. No change in sensation can be found anywhere below the shoulder. Electric excitability is everywhere the same as in the left arm. Marked rigidity exists, greatest towards the extremity in the muscles of the wrist and fingers. All the arm-jerks are increased to an unusual degree; the increase in the muscle-reflex action is so great that if the slightly-flexed fingers are pressed back suddenly, a persistent clonus in the flexors is produced.

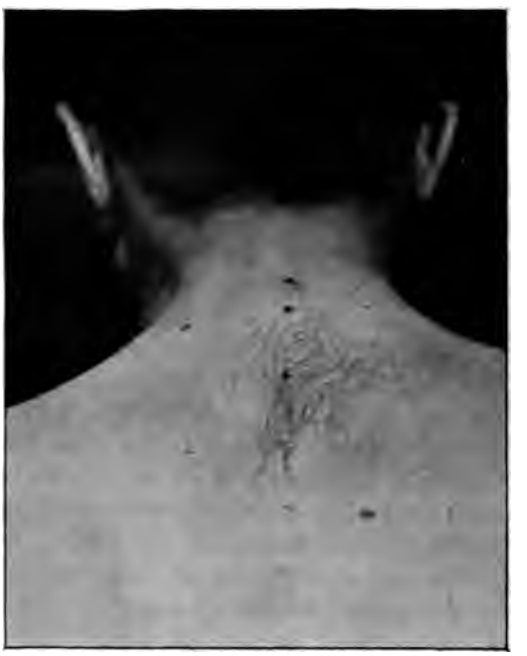
The right leg is a little smaller than the other, and flexion of the right ankle is less strong than of the left, causing him to drag the foot slightly as he walks. He gives a vague history of some skin eruption in this leg at eight years old, to which he attributes the difference in size; the difference in strength is said to be nearly the same as it has always been, but it has been less useful during the last three years. If he walks slowly he can still manage four or five miles. Sensation on the thigh, leg, and foot is perfectly normal. The knee-jerk is much greater on the right side, and well-marked foot-clonus can be obtained, on the right side only. The plantar reflex is flexor on each side.

The lumbar and dorsal regions of the spine are perfectly normal, and no trace of hypertrichosis can be found until the upper dorsal region is reached. The vertebra prominens seems rather broad, and its projection is increased by slight forward recession of the vertebra above, but the condition is merely such as is often met with in men who habitually carry the head a little forward. No imperfection of the cervical arches and spines can be felt.

The remarkable growth of hair occupies a triangular

area to the right of the middle line of the spinal column, along which it extends from the sixth cervical spine to the third dorsal, its vertical extent here being 2¼ in. It is widest—1¾ in.—opposite the first dorsal. To the left of the middle line the skin is quite free from hair, and it appears normal where the hairs grow. These are not

FIG. 1.



Figs. 1 and 2.—Cervical trichosis. From a photograph by Dr. S. A. K. Wilson.

coarse, and are brown in tint, a little lighter than those of the head. Their length is an inch or less, as may be seen in the lateral view. Elsewhere the amount of hair is small.

No wasting of the muscles of the back or shoulder-girdle can be perceived, and the only change in electrical

excitability is a slight diminution in the trapezius opposite and below the spine of the scapula.

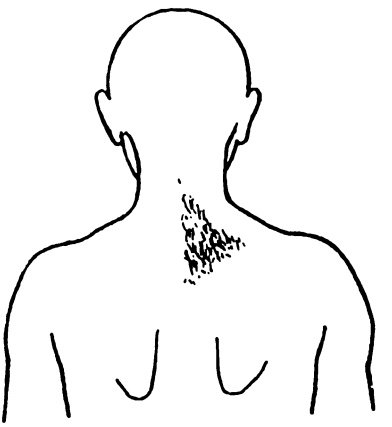
Tactile sensibility is unimpaired in this region, but there is loss of pain in a band from the spine to the shoulder, as shown in the accompanying diagram. It was widest at the spine and extended from about the fifth

FIG. 2.

cervical to the first dorsal level; it has an irregular shape extending directly outwards to the highest part of the shoulder, over which it turns and ends at the outer half of the clavicle, the outer end being somewhat lower, about an inch from the acromion (see Fig. 4). It does not altogether agree with the recognised distribution of the nerve-roots, corresponding with parts of the fourth cervical to the second dorsal, and the roots between. It is probably due to interference with some of their fibres (those for

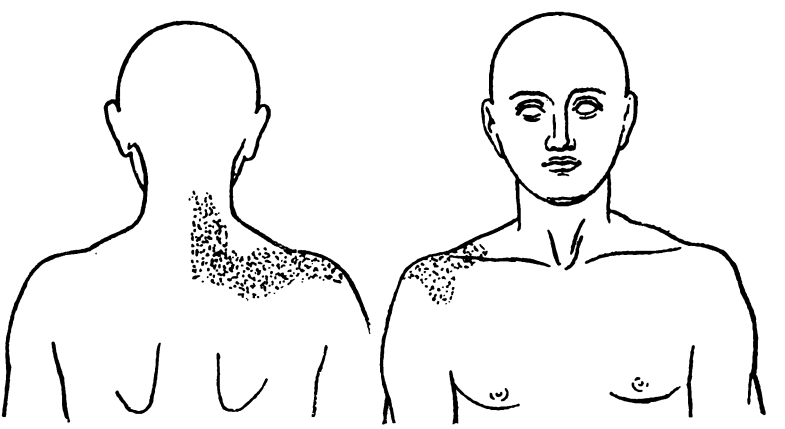
pain) after a rearrangement in the spinal cord. At the edges the loss ceases gradually, except at the middle line

FIG. 3.



Area of trichosis.

FIG. 4.



Area of analgesia.

of the spine, where the transition is sudden. To the left of the middle line sensation is perfectly normal.

Those are the essential features of the case : The motor

paralysis of the arm is clearly due to pressure on the arm-fibres in the lateral column of the cord, just above the grey matter in which they end, since the fibres for the neck muscles have escaped. The motor grey substance had escaped damage, and thus the muscle reflex action, subserving the wrist and other "jerks," had passed into a condition of great excess. The slight degree of similar paralysis in the leg may have been due to the cervical process, or to a slight lumbar syringomyelia, of which, however, there was no other evidence.

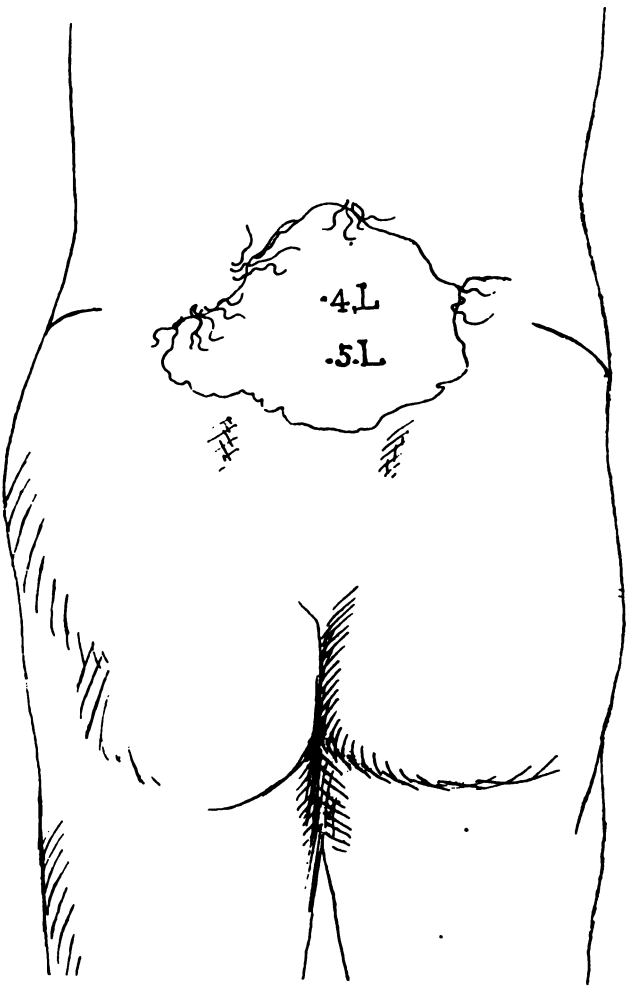
The symptoms of syringomyelia often present a considerable and speedy increase during adult life (and may even seem to begin then), in consequence either of increased distension of the cavity, or of a local growth of the residual gliomatous tissue which is generally found adjacent to the cavity. Either of these processes will account for the damage to the pyramidal tract. The cavity is usually in, or close to, the posterior horn, extending forwards often to the anterior cornu. The extension of the cavity, or the growth, may occur in any part. It is thus quite easy to understand the implication of the pyramidal tract.

The other case of hypertrichosis was in the lumbar region, and its evidence now is a large lumbar scar, with a few hairs on the margin. The growth of hair was destroyed by Mr. Warrington Haward, our President, when the patient was an infant. I recently learned that he is now under the care of Mr. Godlee, to whom I am indebted for the sketch of the scar.

He was a man aged 26. Trophic changes in the left foot began when he was about twelve years of age, and had gone on, although, as far as I could learn, the nature of the case had never been recognised. Obstinate ulceration of the toes was readily produced by pressure. One toe after another had been removed, and when I saw him, in December, 1905, obstinate ulceration had occurred on the extremity, and he was brought to me for my opinion

on the desirability of removing the front part of the foot. The leg had never been quite so strong as the other, and

Fig. 5.



Lumbar scar. From a sketch by Mr. Godlee.

it was assumed (without other reason) that he must have had slight infantile paralysis in early infancy, since all

the muscles were a little smaller than those of the right leg. There was nowhere special wasting, and nowhere diminution of electric excitability. The knee-jerk and ankle-jerk were absent on the left side, although on the right they were normal. Sensation on the left leg was normal to touch, but markedly defective to pain and temperature. The loss extended to the groin in front and the lower part of the gluteal region behind, where it ceased so gradually that the margin was not easy to determine. The degree was much greater below the knee than above it. On the back it seemed normal, and no defect could be found anywhere on the right leg.

Another fact that deserves mention is that at the left ankle there was enlargement of the internal malleolus. The external was normal, but the internal malleolus was sufficiently enlarged to make the circumference here one-third of an inch more than that of the right ankle.

Such a condition of dissociated analgesia, trophic change and enlargement of bone, occurs only in tabes and in imperfect development of the spinal cord,—syringomyelia, spina bifida, and the like. Tabes being, from the history, out of the question, I asked to see the back. I was assured that nothing was to be found there, but I persisted. More to the astonishment of the patient's doctor than of myself, I found the lumbar region occupied by the scar shown in the sketch. It was 5 in. from above down and 6 in. from side to side, and extended equally on each side of the middle line, with a few coarse, dark hairs on the margin at each side. Sensation was quite normal on the skin in the vicinity of the scar and seemed also normal in the cicatrised area. I learned that he was born with an extraordinary growth of hair in this region, which, as I have said, was destroyed when he was eight months old, by Mr. Warrington Haward, who has kindly informed me that it was so abundant and long as to resemble a hairy tail. At first I thought there was a defect of the lower lumbar arches. Mr. Haward, however, was sure that there was not. I had an opportunity of exa-

mining the patient again, and convinced myself that Mr. Haward is right and that there is no defect. I have lately learned that Mr. Godlee is equally sure that the fourth and fifth lumbar spines are absent. I fear it must remain uncertain whether or not there is spina bifida occulta or merely syringomyelia.

The case of cervical trichosis, on one side, certainly presents only syringomyelia, also apparently unilateral. It thus presents the problem of the relation of the peculiar growth of hair to the defect in the spinal cord in a much more simple form than do the cases of spina bifida occulta, the complex pathology of which has been much discussed. Virchow ascribed the hypertrichosis to a process of "irritation," an explanation sufficiently vague to be beyond the range of discussion. It seems to have been accepted by others. The growth cannot be regarded as a trophic effect of the nerve impairment, because there is no correspondence in the area of the two, and also because the trophic effect is a defect of vital nutrition, while the growth of hair must be regarded as an excess of nutritional energy in the epidermis.

The exuberance of the growth of hair at birth in the second case shows clearly, as do others, that it must have begun early in fœtal life. The facts of the cervical case seem to point to its origin during the first weeks of embryonic development. The two structures involved in the deranged development, the posterior part of the spinal cord, and the epidermis over it, both on one side only, arise from one and the same embryonal structure, the neural ridge of the epiblast. The two ridges meet in the middle line, and after uniting, immediately split. The deeper layer completes the neural tube, forming its dorsal portion, from which the posterior part of the spinal cord develops. The superficial layer forms the epidermis of the skin of the back, on each side of the middle line, probably as far as the trichosis extends. The facts of the cervical case I have described may be explained by a

relative disproportion in developmental energy between the two layers of the right neural ridge at the part concerned, an excess in the outer layer, for the epidermis, a deficiency in the inner layer, for the spinal cord. Whether it can be conceived as a mere quantitative difference is doubtful. If it could, it might result from an abnormal position of the division of the epiblast into the two layers, giving too much to the surface and too little to the neural tube. But the split is said to form so soon after the union of the neural ridges that its position must be determined by processes that occur before this union. In the cervical case the difference must have been limited to one neural ridge—the right.

To one other point in the cervical case I may call attention. The growth of hair is in the form of a triangle, which has its longest side at the middle line. It is thus most extensive at the edge of the neural ridge. If it were bilateral and symmetrical the area covered would have been lozenge-shaped. This is strikingly evident in some of the cases of bilateral lumbar trichosis, and an approximation to it may be seen in the scar of the case operated on. It would seem, therefore, that the epidermic developmental energy increases towards the middle line, towards the line of junction of the neural ridges. In one of the cases of spina bifida occulta described by v. Recklinghausen, a depression in the middle of the trichosis passed within the spinal canal, and the hair extended down its sides. Evidently at this spot the neural ridges had failed to unite, and the external epidermic layer of the epiblast had passed down between them. Here, also, the growth of hair within the opening was ascribed to " irritation."

I have purposely avoided referring to the more complex problem presented by spina bifida occulta.

DISCUSSION

The PRESIDENT said: In reference to the case alluded to by Sir William Gowers in his paper as having been under my care, the hair, I may state, was 9 in. long, and might have been taken for a tail; there was, I believe, no defect of the vertebræ, and this Sir William Gowers has confirmed, but he had loss of power in the legs and talipes, with loss of sensation and a great tendency to ulceration of the toes, even leading to exposure of the bone, requiring removal of the toes. The patient, now a young man, is still greatly troubled with ulceration and discomfort of the foot.

Dr. PERNET: I am glad to have had an opportunity of seeing Sir William Gowers' unusual case, for although my knowledge of hypertrichosis and my acquaintance with the literature of the subject are extensive, I cannot recall an instance quite like it. If ever such a case comes my way I shall remember to examine it from the point of view of the dissociation of sensibility (syringomyelia). Acquired hypertrichosis in spinal disease has been observed by Erb and also by Schiefferdecker, but I have not been able to put my hands on their original communications.[1] Hypertrichosis has been observed as a result of local irritation; I have seen such myself, and also in paralysed limbs.[2] In 1902 I saw a case of remarkable growth of hair over the cutaneous distribution of the musculo-spiral nerve, shown by Mr. Clement Lucas at a meeting of the Society for the Study of Disease in Children. The patient was a girl, aged 11, in whom a fracture of the humerus had led to musculo-spiral paralysis, owing to formation of callus about the nerve. Subsequently Mr. Lucas reported that the functions of the limb were completely restored three months later, but the abnormal growth of hair remained when last seen.[3] In that case the hypertrichosis certainly followed the injury to the nerve. As to the growth of hair observed in spina bifida occulta, I have come across it incidentally a good many times in the course of dermatological practice.

Dr. T. GRAINGER STEWART said: I have seen a similar case which was under the care of Dr. Ormerod—a miner, who had wasting and weakness of the right arm, with spasticity of that limb and both the lower limbs. He also had dissociated anæsthesia. The seventh cervical spine was suspiciously prominent,

[1] Cited by Ziegler in his 'Lehrbüch der Allgemeinen Pathologie,' 10th edition, vol. ii, p. 503.

[2] Kaposi-Desnier, 'Maladies de la Peau,' 1891, tome ii, p. 79.

[3] 'Reports of the Society for the Study of Disease in Children,' vol. ii, 1902, pp. 74–76.

and as a baby he had had a growth of hair in that region, with some formation described as a bag of water. It was believed at the time that the seventh cervical spine was bifid. The scar was in the middle line rather more to the right than the left and was about $1\frac{1}{2}$ in. broad. The case had been shown by Dr. Ormerod before the Clinical Society.

Sir WILLIAM GOWERS, in reply, said: The case alluded to by Dr. Grainger Stewart is of great interest, but I doubt whether a bag connected with the spinal canal could have failed to prevent closure of the vertebral arches.

LIGATURE OF THE RENAL VESSELS BY THE TRANSPERITONEAL METHOD FOR THE CURE OF PERSISTENT URINARY RENAL FISTULA,

WITH NOTES OF ONE SUCCESSFUL CASE

BY

MAJOR M. P. HOLT, R.A.M.C.

Received May 14th—Read May 28th, 1907

(1) *Choice of procedure:* (*a*) *Nephrectomy.*—In the absence of well-known contra-indications, nephrectomy is commonly acknowledged to be a legitimate surgical procedure in certain of these cases. But where there is extensive matting of the kidney and perinephritic tissues to surrounding structures, nephrectomy may be extremely difficult or even impossible. The severity of the operation, the risk to the patient from hæmorrhage, shock, and damage to important organs and serous cavities, justify the authoritative admission that the maintenance of good drainage is then the sole resource in the vague hope that at some future and quite uncertain date healing may occur.

(*b*) *Subcapsular nephrectomy;* (*c*) *morcellement.*—In other cases subcapsular nephrectomy, piecemeal nephrectomy, or morcellement may be tried, but these operations

are often followed by prolonged suppuration or by permanent fistula owing to a portion of renal tissue having been left behind, as it frequently is.

(2) *Disabilities attendant on a persistent renal sinus.*—Taking the case of a young, and otherwise robust man, who has to earn his living, and who would find the expenses entailed by the purchase of voluminous and frequently to be changed dressings, or of any special apparatus, quite beyond his resources, or taking, on the other hand, the case of a weakly patient, in whom the drain of prolonged suppuration would appreciably curtail life, the surgeon may well hesitate before coming to a decision as to which of these methods to adopt.

(3) *Dangers and difficulties of nephrectomy.*—In not a few cases death from hæmorrhage alone has been the immediate result of operation.

Greig Smith recognised cases where the kidney and surroundings were so much altered that the kidney "could not have been removed from the body by any proceeding claiming to be regarded as surgical."

Mr. Henry Morris admits cases "in which it was difficult even to dissect the organ away in the post-mortem room," in which it is difficult or impossible to identify where kidney tissue begins and scar tissue ends (if, indeed, they are not inseparably blended), and in which, further, the closure of a urinary sinus is "sometimes impossible"; and other similar opinions could be quoted.

(4) *Nature of case for which renal vessel ligation performed.*—It was upon a case of this nature that the operation which I devised was successfully performed. The patient was a short, thick-set, strong young man, otherwise healthy. Nine months before he came under my care, he had sustained a rupture of the left kidney, his very short subcostal space was filled with scar tissue, and a sinus through which pus and urine were freely discharged. I made four attempts to perform nephrectomy, but had to abandon the operations partly on account of furious hæmorrhage, partly on account of the impossibility

of distinguishing renal from surrounding scar tissue. (Notes of the case are given below).

(5) *No precedent to go upon.*—I have not been able to find any reference to ligature of the renal vessels as an operation to secure a definite surgical result *per se*.

(6) *Experimental ligation of renal vessels.*—In various physiological publications and text-books references are made to atrophy of renal epithelium and cessation of urinary secretion as a result of ligature of either the renal arteries, or the veins. Dr. Beddard's experiments on the frog ('Journal of Physiology,' vol. xxviii, 1902), even allowing for differences between batrachian and mammalian renal blood-supply, lend support to the view that by ligature of the renal vessels the glomeruli of the whole kidney are cut out of the circulation, that complete ligature prevents spontaneous secretion of urine, and that "cutting off the arterial blood-supply of the kidney causes the epithelium of the tubules to degenerate rapidly." De Souza ('Journal of Physiology,' vol. xxvi, 1900–1901) supports Heidenhain's contention "that the amount of urine secreted is directly proportional to the velocity of the blood-flow through the kidneys," and suggests that probably a similar result might be obtained by tying only the renal veins.

(7) *Deductions from laboratory experience.*—Reasoning on these lines I concluded that if the renal artery and vein were ligatured complete atrophy of the renal secreting tissue would be ensured, and supposing the presence of urine to be the sole hindrance to healing, that if once the secretion of urine within the limits of the abscess ceased, then healing by normal granulation would follow. And this, indeed, was found to be the case; the sinus healed, and I had a letter only a few days ago, nearly two years after the operation, in which the patient said he remains quite well, is earning a living, and that the sinus has never reopened.

(8) *Significance of blocked ureter.*—In the notes of the case it will be seen that evidence pointed to the conclusion that the ureter had become blocked. It seems

obvious that when this is so a renal fistula can never heal spontaneously; it would be quite fortuitous that a blocked ureter (excluding, of course, the case of a removable, or movable, calculus) should ever again become patent. It is very seldom advisable to undertake any surgical proceeding with the object of re-establishing patency of a blocked ureter in old suppuration cases; for a successful result, even if achieved, is not likely to remain a permanent one.

(9) *The presence of, and final cessation of, urine in the discharge from sinus.*—At this point I may say that it remains a matter for regret that I did not have some estimate made from time to time during the healing period of the urinary constituents in the discharge from the sinus, for the purpose of comparison with similar analyses made before operation. The presence of urine in the discharge was only too obvious before the operation. After the operation, for the first few days, the discharge was scanty and smelt faintly of urine. Then, on the fifth day, a large amount of discharge came away in which the smell of urine was not apparent; nor was it ever again present, the discharge appearing as a clear, odourless serous fluid. Healing was complete on the fifty-seventh day after operation. Apparently, then, atrophy of urine secreting epithelium occurred within five days or so.

(10) *Pathology of the cessation.*—It must remain a matter for speculation whether infarction of the whole kidney occurred, or whether a more gradual process of atrophy took place. The urine appearing in the discharge for the first few days may well have been a residuum remaining in the various recesses of the abscess.

Having stated the indications for, and the object of the procedure adopted, one or two points remain to be considered as to (1) technique, (2) advantages, (3) limitations of the operation.

(11) *Technique.*—(*a*) The technique is rather obvious, but remembering the possible existence of accessory or abnormal renal afferent vessels, a search should be made along the aorta to a limited but reasonable extent for

these; Zondek found accessory arteries in five out of fifteen subjects examined.

(*b*) Asepsis can only be assured by avoiding the suppurating area at all costs; this is effected by keeping as far from the kidney as possible, and as close to the aorta and vena cava as is safe to apply a ligature.

(*c*) Access to the renal pedicle, in abdominal nephrectomy, is claimed to be safer when made through the outer layer of the mesocolon by reason of less danger to the colic blood supply; to follow this route, however, would almost certainly open up the septic area, and moreover the colon may be found inseparably united to the kidney. In my case no evil resulted from approaching the renal vessels through the inner mesocolic layer, and this notwithstanding that a search was made for possible renal afferent vessels.

(12) *Advantages.*—Properly carried out there is little or no risk either from hæmorrhage, shock, or sepsis. As regards (*a*) *hæmorrhage*, this method is in great contrast to any form of piecemeal nephrectomy; (*b*) it is an *aseptic* operation which nephrectomy would certainly not be; (*c*) subcapsular or piecemeal nephrectomy is at best *a very severe operation*; ligature is not so, and shock should not occur; there is inappreciable risk as regards possible damage to the colon or other surrounding organs and serous cavities (septic infection).

(13) *Limitations.*—Ligature of the renal vessels may be a very simple proceeding or may be insuperably difficult, and of course there may occur cases where the kidney is so closely adherent to the aorta or the vena cava as to make it impossible to apply a ligature to the vessels. Greig Smith mentions a case where "the aorta and vena cava were so adherent to the kidney that it was found impossible at the autopsy to dissect them apart." I take it that such a condition would be easily recognised through a sufficiently free opening in time to save a blunder. But in the case of the right kidney it might still be possible to ligature the artery by getting at it

My gratitude to these gentlemen is not easily expressed.

Conclusion.—In conclusion, though in the first instance this operation was performed when nephrectomy had been attempted and found impossible, I should not hesitate to employ renal vessel ligation in the future as a substitute for nephrectomy where this latter operation promised great difficulty, and the likelihood of such severe hæmorrhage as might suffice to turn the scale between life and death in an exhausted patient. Moreover, as a preliminary to a subsequent nephrectomy (a septic operation), aseptic ligation of the renal vessels would be sound surgery, though I claim that nephrectomy would then be unnecessary as the ligature would be followed by rapid and permanent cure. Ligature of the renal vessels is surely a great improvement on any form of subcapsular piecemeal nephrectomy or morcellement. The former is a simple, the latter a very serious procedure, and both effect the same result.

While quite conscious of the imperfect support that a single successful case affords to a new method, the very rapid, satisfactory, and permanent cure so obtained seems to warrant my bringing the subject before the notice of the Fellows of the Society with some confidence that it may be deemed worthy of criticism at least, and possibly, of some degree of approval as to its suitability in cases of a similar nature.

Brief history of case.—C. M—, aged 19.

September 14th, 1902.—Admitted. Kicked at football previous day. Severe pain in left loin.

October 2nd.—Large abscess opened close to left iliac crest; let out pus and urine. Diagnosis: Rupture of ? kidney or ureter.

January 21st, 1903.—Extensive empyema evacuated; previous abscess still discharging pus and urine.

April 4th.—Abscess explored. It extended forward amongst abdominal muscles to near umbilicus, upwards to left pleural cavity, backwards and upwards to left kidney

area, downwards into pelvis. Pus in vesical urine. Diagnosed ruptured kidney.

June 8th.—Sent to Dublin for nephrectomy.

June 21st.—Pyuria; pus and urine discharging from sinus in loin.

July 17th.—*Operation.*—Attempted nephrectomy; abandoned owing to profuse hæmorrhage; removed five calculi.

August 6th.—Scar in eighth intercostal space opened spontaneously.

November 10th.—Skiagraph; shadow of calculus in kidney.

November 12th.—Empyema scar again opened spontaneously, having remained closed six weeks.

December 1st.—*Operation.*—Attempted nephrectomy; abandoned owing to inadvertently opening peritoneum and inability to define limits of kidney tissue; three calculi removed; very profuse hæmorrhage.

March 6th, 1904.—Skiagraphed; further shadow of calculus.

March 15th.—*Operation.*—Two calculi removed; profuse hæmorrhage, therefore gave up attempt at piecemeal nephrectomy; unable to clamp pedicle; collapsed and critical condition for many hours.

September 26th.—Cathelin's separator. Right ureter urine acid, clear, no pus or blood, no albumen. Left ureter urine neutral, opaque, "smoky," pus and blood-cells.

November 17th.—No pus in urine; free discharge of pus and urine from sinus. *Operation.*—Attempted nephrectomy; began by peeling peritoneum from scar tissue; opened peritoneum inadvertently, at once closed; further separation impossible; removed large calculus from ? pelvis of kidney, having a rounded process of shape and size of ureter, one third of an inch long; kidney-tissue and scar quite inseparable; very severe hæmorrhage. Saline transfusion adopted.

November 18th.—Sudden profuse hæmorrhage; threatening life; condition very critical for some hours; hæmorrhage controlled by gauze packing.

January 29th, 1905.—Urine (vesical), clear, neutral, no trace of albumen; no pus or blood cells.

May 2nd.—Urine clear, normal; no shadow of calculus visible; abundant urine and pus from sinus. Cathelin's separator. Right ureter, urine clear, normal; none collected from left ureter.

June 13th.—*Operation.*—Laparotomy by Lennander's incision. Some difficulty in finding renal vein, owing to enlarged lymph glands, vein ligated, then renal artery tied and divided. No accessory afferent vessels found.

August 9th.—Sinus in loin completely closed.

August 15th.—Discharged quite well.

May 24th, 1907.—Letter to say patient remains quite well. Sinus has never reopened or given any trouble.

Until the last operation it had been necessary to apply every day four or more very voluminous dressings; even these were often quite insufficient to take up all the urine and pus, his bed and clothes occasionally becoming soiled. There was not the least tendency to heal, nor did the amount of discharge and urine ever tend to lessen, until after the final operation.

Postscript.—Since writing the above I have come across the following ('Trans. American Surg. Assoc.,' vol. xiv, 1896, p. 312): Mr. Keen, in a paper on "Traumatic Lesions of the Kidney," when referring to hæmorrhage in subcutaneous rupture of the kidney, says: "Bobroff has advised to arrest the hæmorrhage by simple ligature of the renal vessels without nephrectomy, basing his conclusions on experimental grounds, which show that necrosis does not necessarily follow. I am not aware of any case in which this has been carried out in the human subject, and I should certainly not be disposed to recommend it. *If the vessels are ligated the kidney will probably not remain functional, and is at least liable to necrosis or atrophy.*"

DISCUSSION

Mr. A. E. Barker: It would be a pity to allow Major Holt's very valuable paper to pass without comment, although, as a new method, one cannot claim any experience in it. But all of us who have done many nephrectomies must have often regretted having had no alternative to so severe an operation. Personally I can recall many such, and particularly one comparatively recent case. It was that of a young man who sustained a rupture of the left kidney from a fall. The result was a large false hydronephrosis of the loin, which I opened and drained. Ultimately I attempted nephrectomy, but in the presence of extensive and very tough adhesions with the diaphragm and adjacent organs had to abandon the operation. Again, as I heard, nephrectomy was attempted by another surgeon but ended fatally Here Major Holt's operation would have been invaluable, and I think we should hail it as a most valuable addition to our surgical procedures.

Mr. Stephen Paget said it is surely an advantage of the operation that clean tissue is left behind after the operation, and tissue which, in respect of some internal secretion, might be of value to the organism.

CHANGES OCCURRING IN THE NUMBER OF RED CORPUSCLES AND THEIR HÆMOGLOBIN CONTENT IN CASES OF PULMONARY TUBERCULOSIS UNDERGOING SANATORIUM TREATMENT

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THE following observations were made upon a number of consumptives who had been under our care from time to time in the Banchory and Mundesley Sanatoria. We are indebted to Dr. Lawson, of the Banchory Sanatorium, for the after-histories of several of the patients referred to in this paper.

Our object in making these observations was to determine:

(1) The number of the red blood-corpuscles and the variation in their hæmoglobin index in various types of pulmonary tuberculosis.

(2) The changes that occur in the red cells in patients undergoing sanatorium treatment.

METHOD OF OBSERVATION.

Blood was drawn from the finger without pressure The hæmoglobin was estimated with Fleischl's hæmometer,

and the red blood-count was made with Thoma Zeiss' apparatus. One of us obtained the specimens of the blood, and the other estimated the hæmoglobin and counted the red cells without knowing the source of the blood until the results were recorded.

Re the number of red cells and hæmoglobin index in normal individuals.—The average count for the normal adult man is usually given as 5,000,000 cells per cubic millimetre, for women 4,500,000. In a healthy young man, however, it is more common to find from 5,000,000 to 6,000,000 (Emerson).

Hæmoglobin index.—By the colour index or hæmoglobin content is meant the percentage of hæmoglobin divided by the percentage of red cells. What is the colour index reckoned in the above manner for the average normal man and woman? Emerson states that 5,000,000 is only in very general terms the normal figure for the red cell count, and few hæmoglobinometers read normal blood at 100 per cent.

The index in fairly normal persons varies from ·80 to ·88 (Emerson). "In the case of our students (192 of whom were examined), as estimated with the Fleischl instrument, the men had an index of ·84 and the women an index of ·87" (Emerson).

We used the Fleischl instrument in all our observations, both on the normals and on the consumptive patients, so that this work of Emerson is of particular interest to us. It would seem, then, that we may take as the standard of the normal hæmoglobin index any figure between ·80 and ·90.

Observations upon Normal People living in a Sanatorium.

As a matter of interest, and to obtain a standard for the blood of normal individuals living under the exceptionally good conditions obtained by residence in a sanatorium we examined the blood of six normal people in residence in the Banchory Sanatorium.

Of these six people, four were members of the sanatorium staff, doing a hard day's work, but living under general sanatorium conditions; they were taking, however, only ordinary amounts of food, though of the same highly nutritive quality as that taken by the patients. The other two normals were healthy people, who, for some time, had been under the full sanatorium *régime*. The following are the results of our observations made upon these six people.

TABLE I.

(A) *Members of the Staff.*

	Hæmoglobin percentage.	Red cells.	Hæmoglobin index.
Nurse L—	90	5,496,000	·92
„ W—	95	5,560,000	·85
„ K—	95	4,976,000	·95
Dr. B—	104	5,600,000	·93
Average	96	5,408,000	·91

(B) *Normal Persons Living the Full Sanatorium Régime.*

	Hæmoglobin percentage.	Red cells.	Hæmoglobin index.
Mr. A—	115	5,600,000	1·03
Miss B—	105	4,976,000	1·05
Average	110	5,280,000	1·04

Average Records of the Six Normals.

Hæmoglobin percentage.	Red cells.	Hæmoglobin index.
100	5,368,000	·95

These results are very striking evidence of the beneficial effect of fresh air and good food. The blood of the four people doing a hard day's work in a sanatorium was most satisfactory, the average hæmoglobin index of ·93 being well above the normal average. The blood of the two normal people living the full sanatorium *régime* was exceptionally

rich in hæmoglobin, the percentage hæmoglobin being 115 and 105 respectively, and the hæmoglobin index 1·03 and 1·05. *Apropos* of these figures we may add that we have been constantly impressed by the rapid way in which people with some degree of anæmia, such as maids and nurses, improve under sanatorium conditions, and this without the use of iron and other drugs commonly given in anæmia. Taking the blood of these six normal people together, we get the following average:

	Hæmoglobin percentage.	Red cell count.	Hæmoglobin index.
Average of six normals	100	5,368,000	·95

From these observations we conclude that the standard of hæmoglobin index for normal people living in sanatoria is a high one—a good deal higher than the standard for normal people living and working in a town.

We would put the standard for the hæmoglobin index for normal people living in a sanatorium at anything between ·90 and 1.

For the purpose of comparison we give Emerson's figures and our figures for normal individuals.

	Average hæmoglobin percentage.	Average number red cells.	Average hæmoglobin index.
176 normal male students	92	5,000,000	·84
16 normal women students	87	4,800,000	·87
6 normal persons living in a sanatorium	100	5,368,000	·95

Observations upon Patients Suffering from Pulmonary Tuberculosis.

We have made a great number of observations upon consumptive patients, and of these we now publish records of sixty individuals. We can conveniently divide these patients into several clinical groups, viz.:

(1) Patients with early and limited disease with little or no fever.

(2) Patients with fairly recent but somewhat extensive and active disease, for the most part with well-marked fever, and other symptoms of active disease.

(3) Patients with chronic disease of long standing, *e.g.* a year or more, with a varying degree of activity of the disease.

In the majority of cases we give the condition of the blood of these patients when leaving the sanatorium on completion of their course of treatment; in one series of cases we also record the condition of the blood on admission, and in some other cases we give observations made at intervals throughout the course of treatment. We also give, in parallel columns, a brief summary of the clinical condition of each patient on admission and on discharge, and also a note with regard to their after-histories. The following are definitions of the terms we have used to describe the condition of the patients on discharge from the sanatorium.

Complete arrest: General health completely restored in every respect, and lung disease completely arrested (apparent cure), there being no physical signs present, or only those which are compatible with a completely healed lesion.

Incomplete arrest: (*a*) General health completely restored, but physical signs, 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. No improvement or disease extending.

Observations on Patients with Early Disease.

In Table II we give the results of our observations on fourteen patients, who were admitted with early disease of a few months' duration. Nearly all these patients were typical cases of early and fairly limited disease, associated with little or no fever, or other symptoms of tuberculosis. In the case of the first six patients in the table we are able to give the condition of the blood, both on admission and on discharge, and in several cases also the condition of the blood at intervals throughout the course of treatment.

TABLE II.—*Type: Cases of Early Disease (Pathologically) with little or no Fever or other Constitutional Disturbance.*

Name.	Duration of disease.	On admission.					No. of weeks under treatment.	On discharge.					After-history.
		Clinical condition.		Condition of blood.				Clinical condition.		Condition of blood.			
		No. of lobes.	Fever.	Hb. per cent.	No. of red cells.	Hb. index.		Lung disease.	Gain of weight.	Hb. per cent.	Red cells.	Hb. index.	
1	6 mos.	3	*Nil*	107	6,360,000	·84	40	Incomplete arrest A	9 lbs.	115	5,380,000	1·06	Alive and well after 5 years.
2	4 ,,	1	,,	103	5,200,000	·99	14	Complete arrest	10 ,,	115	5,380,000	1·06	Lived 4½ years.
3	2 ,,	2	,,	108	7,100,000	·76	13	,, ,,	12½ ,,	115	6,740,000	0·86	Quite well when lost sight of.
4	3 ,,	1	,,	96	6,300,000	·76	15	,, ,,	20 ,,	110	5,544,000	1	Well after 5 years.
5	Indefinite	1	,,	110	6,300,000	·87	5	,, ,,	16 ,,	110	5,920,000	0·92	Quite well when last heard of.
6	4 mos.	2	Present	73	5,400,000	·67	33	Incomplete arrest A	19 ,,	106	5,650,000	0·93	Fairly well after 5 years.
Average	4 mos.	2		99	6,112,000	·82	20	—	14 ,,	112	5,743,000	0·97	—
7	3 mos.	2	*Nil*	—	—	—	19	Complete arrest	11 ,,	115	5,760,000	1	Well after 5½ years.
8	3 ,,	1	Slight	—	—	—	16	,, ,,	1 ,,	115	5,400,000	1·06	,, ,, 5 ,,
9	4 ,,	5	High	—	—	—	32	,, ,,	24 ,,	113	5,400,000	1	,, ,, 5 ,,
10[1]	6 ,,	2	*Nil*	—	—	—	40	,, ,,	22 ,,	115	5,620,000	1·03	,, ,, 5½ ,,
11	6 ,,	4	,,	—	—	—	36	Incomplete arrest A	15 ,,	115	5,480,000	1·05	,, ,, 5 ,,
12	3 ,,	2	,,	—	—	—	17	Complete arrest	16 ,,	107	5,360,000	1	,, ,, 5½ ,,
13	4 ,,	2	Slight	—	—	—	33	,, ,,	3 ,,	115	5,210,000	1	,, ,, 5 ,,
14	7 ,,	2	Definite	—	—	—	29	,, ,,	26 ,,	96	5,280,000	0·90	,, ,, 5½ ,,
Average of 14 patients	4½ mos.	3	—	—	—	—	24	—	13 ,,	111	5,580,000	0·99	—

[1] Case of pleurisy with effusion.

From the table it is seen that three out of these six patients had, on admission, some degree of anæmia, the hæmoglobin index being ·76, ·76, and ·67 respectively. In two of these patients this diminution of hæmoglobin index was associated with an increase in the number of red cells, the counts being 7,100,000, 6,300,000, and 6,300,000 respectively.

The remaining three patients had a hæmoglobin index within normal limits—viz. ·84, ·99, and ·87, but in two of these patients the red cell count was abnormally high—viz. 6,300,000 and 6,360,000. From these observations we conclude, then, that in the case of patients suffering from early pulmonary tuberculosis with little or no fever—(*a*) the red cell count is usually increased somewhat, and sometimes considerably so, *e. g.* up to 7,000,000; less commonly the red cell count is normal; (*b*) the hæmoglobin index may be normal (50 per cent. of our cases) or it may be somewhat reduced (50 per cent. of our cases).

Condition of the blood in patients admitted with early disease, and discharged from the sanatorium apparently cured.—The condition of the blood of these six patients with early disease already referred to, and of eight more patients of the same type, on their discharge from the sanatorium apparently cured, is of considerable interest. These fourteen patients had, on discharge, been under treatment for an average period of twenty-four weeks, and as a result of this treatment eleven of them had left apparently cured, viz. with no physical signs incompatible with a completely healed lesion, and in normal general health. The remaining three patients had only slight signs of disease remaining, and were also restored in general health. The average gain of weight in these fourteen patients was 13 lbs.

The condition of the blood in these fourteen patients was most satisfactory, the average for the fourteen being—

Hæmoglobin percentage.	Red cells.	Hæmoglobin index.
111	5,580,000	·99

The hæmoglobin percentage was very high—much above the normal standard of Emerson—in eight patients above

115. The hæmoglobin index was also very high—in nine patients 1 or more. The red-cell count, in a large majority of these patients, was normal, viz. between 5,000,000 and 6,000,000, the average for the fourteen patients being 5,580,000. In only one patient was the count as high as 6,000,000. The condition of the blood of these fourteen patients on discharge was, in fact, better than that of the majority of the six normal people living in a sanatorium; this is shown in the following comparison:

	Hæmoglobin percentage.	Red cells.	Hæmoglobin index.
Average of fourteen patients	111	5,580,000	·99
Average of six normals living in the sanator'm.	100	5,368,000	·95

On comparing the condition of the blood in these six patients with early disease, on admission and on discharge, we see that the improvement which took place in the condition of the blood as the tubercular disease became cured, consisted in an increase in the percentage hæmoglobin and a diminution in the number of red cells, the result being an improved hæmoglobin index. The following are the figures:

	Hæmoglobin percentage.	Red cells.	Hæmoglobin index.
Average of six early cases on admission	99	6,112,000	·82
Average of the same cases on discharge	112	5,743,000	·97

The rate of improvement of the blood in these cases is well shown by the following series of observations in two of them.

	Hæmoglobin percentage.	Red cells.	Hæmoglobin index.
(1) On admission	96	6,300,000	·76
After one month	108	6,240,000	·87
After three months	115	5,780,000	1·00
After five months	110	5,544,000	1·00

	Hæmoglobin percentage.	Red cells.	Hæmoglobin index.
(2) On admission	107	6.380,000	·84
After two and a half months	106	5·616,000	·95
After four months	115	5·380,000	1·06

The after-histories of these fourteen patients have been extremely satisfactory, no less than twelve of them being in good health at the present time; one has been lost sight of and the fourteenth lived for four and a half years.

Observations upon patients with comparatively recent but somewhat extensive lung disease, with considerable fever and acute symptoms generally.—In the next table (Table III) we give the condition of the blood in sixteen patients with this type of disease on their discharge from the Sanatorium. All these patients had, on admission, well-marked signs of active disease, and for the most part they did not do very well under treatment, an average gain of weight of 10 lb. not being associated with arrest of the tubercular disease. From the table it is seen that no less than thirteen of these patients were discharged with their lung disease either extending or incompletely arrested, and with general health still bad. Patient No. 14 was discharged, however, in very fair general health, although the physical signs in his lung were still well marked.

The characteristics of the blood in these fourteen patients were a high red-cell count, with a low total percentage hæmoglobin, giving a low hæmoglobin index—viz. ·78 on the average. The high red-cell count is very noticeable, for no less than nine of these patients had a count of 6,000,000 or more; in three cases it was 6,500,000. The low hæmoglobin index is also noticeable; in one patient it was as low as ·66. In two patients, Cases 9 and 11, the condition of the blood was one of true anæmia—viz. with a low red-cell count and a low hæmoglobin index. The condition of secondary anæmia, which is generally associated with progressive pulmonary tuberculosis of a chronic type, is very persistent in many cases,

and alters very little despite some temporary improvement in general health. The following are good examples:

CASE 56.—*Extensive Active Disease of Left Lung with Considerable Fever, Emaciation, etc.*

	Hæmoglobin percentage.	Red cells.	Hæmoglobin index.
After 4 months' treatment	80	6,350,000	·63
„ 5 „ „	83	6,500,000	·64
„ 6 „ „	87	6,300,000	·69
„ 7 „ „	78	6,100,000	·64
„ 8 „ „	74	5,630,000	·66
„ 9 „ „	75	5,920,000	·63
„ 10 „ „	70	5,320,000	·66
„ 12 „ „	70	5,920,000	·60

CASE 48.—*Very extensive Active Disease in the Left Lung with Considerable Fever.*

	Hæmoglobin percentage.	Red cells.	Hæmoglobin index.
After 2 months	97	6,350,000	·76
„ 3 „	108	6,100,000	·88
„ 5 „	100	6,360,000	·79
„ 9 „	87	5,192,000	·81

CASE 46.—*Extensive Active Disease in the Right Lung with Considerable Fever.*

	Hæmoglobin percentage.	Red cells.	Hæmoglobin index.
After 1 month	104	7,000,000	·74
„ 2 months	107	7,500,000	·71
„ 5 „	100	6,000,000	·83
„ 6 „	95	6,600,000	·72
„ 8 „	82	6,536,000	·64

Towards the end of life a condition of the blood, characterised by a falling of hæmoglobin percentage and hæmoglobin index, with a reduction in the number of red cells, may be met with, *e. g.* case of very extensive chronic disease, with terminal relapse.

	Hæmoglobin percentage.	Red cells.	Hæmoglobin index.
After 18 months' treatment	70	4,880,000	·73
1 month later	55	4,880,000	·56
2 months later	60	5,400,000	·55

Died two months later.

In striking contrast with the records of these fourteen patients we note the condition of the blood of the two patients in this group who after nine months' treatment were discharged, one with lung disease apparently cured and with general health restored, and the other with general health restored and the physical signs very largely cleared up.

The blood in both these patients was very satisfactory, the average for the two being:

Hæmoglobin percentage.	Red cells.	Hæmoglobin index.
106	5,513,000	·96

The red-cell count in case No. 16, viz. 5,840,000, was still high, but the hæmoglobin, viz. 113, was proportionately high.

A comparison of the blood of the sixteen patients in this group with our normal standard is shown in the following:

Patients with Recent and Extensive Active Disease.

	Hæmoglobin percentage.	Red cells.	Hæmoglobin index.
Average of 14 patients discharged with the disease still active	90	5,823,000	·78
Average of 2 patients discharged with arrested disease	106	5,513,000	·96
Average of 6 normals	100	5,366,000	·95

From this comparison it is seen that the two patients in this group who left after a long course of treatment with their disease well arrested had their blood restored to a normal condition. Compared with Emerson's standard their blood was considerably better than normal, and though not quite up to the standard of our patients with early disease who were discharged apparently cured it was somewhat better than the blood of the six normal

people living in a sanatorium. The records of these two patients, then, show that arrest of pulmonary tuberculosis and restoration of health in the case of patients suffering from somewhat advanced disease is associated with the restoration of the blood to its normal condition with regard to the number of red cells and the hæmoglobin.

GROUP 3. *Patients with chronic slowly-progressive disease of long standing (a year or more).*—This group of twenty-two patients (Table IV) we have divided into two sub-groups, viz. (1) those who were discharged with lung disease still actively progressing and in bad general health, (2) those whose disease was, to some extent, arrested, and whose general health was, to some measure, restored.

Reviewing, first, the eleven patients in sub-group 1, namely, those discharged with their disease still actively progressing, we see that they nearly all had the condition of secondary anæmia which we have already described as existing in patients with recent actively-progressing disease, viz. a high red-cell count with a low hæmoglobin content.

The average figures of these eleven patients were :

Hæmoglobin percentage.	Red cells.	Hæmoglobin index.
84	5,812,000	·72

No less than six of these eleven patients had a red-cell count of over 6,000,000 ; the low hæmoglobin index was also noticeable, in two patients being below ·60.

Reviewing the blood of the eleven patients in sub-group 2—viz. those discharged with a varying degree of arrest of the disease and in fair general health, we note that the condition was much more satisfactory. The red-cell count was either within normal limits or somewhat increased, the average of the 11 patients being 5,630,000.

The percentage hæmoglobin was in every case normal, or above normal, and the hæmoglobin index was, with one exception, up to or above Emerson's normal standard, the average for the eleven patients being ·88.

TABLE IV.—*Type : Cases of Chron*

(A) *Patients discharged with signs and sy*

No. of case.	Duration of disease.	On admission. Clinical condition. No. of lobes.	On admission. Clinical condition. Fever.	No. of weeks under treatment.	On discha Clinical condit Gain of weig
1	2 years	4	99–101°	6	8 lbs.
2	2 „	3	*Nil*	11	6 „
3	2 „	3	99–100·6°	76	Lost
4	1 „	2	99–100·8°	13	Nil
5	3 „	5	Slight	14	7 lbs.
6	2 „	5	„	8	Nil
7	5 „	5	98·6–100·2°	3	5 lbs.
8	3 „	5	98·4–100°	33	24 „
9	2 „	5	102·2–104°	20	Lost
10	2 „	4	99–100°	27	25 lbs.
11	2 „	4	Slight	4	Nil
Average . .	2¼ years	4	99–101°	·24	12 lbs.

(B) *Patients discharged with disease considerably arrested*

12	10 years	4	*Nil*	21	15 lbs.
13	8 „	3	„	15	19 „
14	3 „	2	„	36	Nil
15	6–8 months	2	„	12	13 lbs.
16	7 years	2	„	48	3 „
17	4 „	2	99–100°	40	Nil
18	1 „	5	98–101°	21	11 lbs.
19	5 „	5	97·8–99·8°	14	10 „
20	2 „	3	Slight	19	28 „
21	2 „	4	„	16	24 „
22	4 „	5	*Nil*	43	7 „
Average . .	4 years	3	—	26	11½ lbs.

Progressive Disease of Long Standing.

ptoms of active disease still well marked.

On discharge.				
Clinical condition.	Condition of blood.			After-history.
Lung disease.	Hb. per cent.	No. of red cells.	Hb. index.	
Incomplete arrest B	62	5,280,000	0·58	Died 2 months later.
„ „	80	4,800,000	0·82	„ 3 weeks „
Disease extending	87	6,300,000	0·69	„ 9 months „
„ „	98	6,240,000	0·79	„ 2 weeks „
Incomplete arrest B	85	6,880,000	0·62	Lost sight of.
„ „	95	6,000,000	0·79	Died.?
Died in sanatorium	63	5,600,000	0·59	Died within 1 month.
Incomplete arrest B	95	6,000,000	0·79	„ 9 months later.
Died in sanatorium	70	4,880,000	0·73	„ 7 „ „
Incomplete arrest B	100	6,560,000	0·76	—
„ „	87	5,400,000	0·80	Died 2 years later.
—	84	5,812,000	0·72	—

and with general health to some extent restored.

Incomplete arrest A	113	5,280,000	1·06	Fairly well 4 years later.
„ „ B	100	5,520,000	0·91	Well 4 years later.
„ „ A	115	5,976,000	0·96	Very ill 5 years later.
„ „ B	110	5,560,000	1	Died 18 months „
„ „ B	100	5,520,000	0·99	Died 1 year „
„ „ A	97	6,000,000	0·83	In fair health 5 years later.
„ „ A	95	5,840,000	0·81	Died 2 years later.
„ „ B	85	5,970,000	0·71	„ „ „
„ „ B	95	5,580,000	0·84	In fair health 5 years later.
„ „ B	86	5,680,000	0·75	Died 10 months later.
„ „ B	100	5,600,000	0·89	„ 5 years „
—	99	5,630,000	0·88	—

The difference clinically between these two groups of chronic cases is that, whereas the eleven patients in the first group all left with physical signs and symptoms of the disease well marked, those in the second group were discharged with their disease to a considerable extent arrested. The difference between the condition of the blood of the patients in these two groups is very striking, for whereas the blood of those with active disease was characterised by a high red-cell count with marked diminution of hæmoglobin, the blood of the patients discharged with quiescent disease was within normal limits compared with Emerson's normal standard, although still below our standard for normal people living in a sanatorium.

It seems to be clear, then, that the greater the degree of activity of the disease and the longer the duration of this activity, the more marked will be the changes in the blood we have described—viz. increase in the number of red cells and diminution in hæmoglobin content, and the greater the degree of arrest of the disease, and the longer the disease remains arrested, the closer will the condition of the blood approximate to the normal.

Summary of Results.

	Hæmoglobin percentage.	Red cells.	Hæmoglobin index.
Averages of:			
Eleven chronic cases of long standing discharged with active disease	84	5,812,000	·72
Fourteen cases of recent extensive and active disease discharged with active disease	90	5,823,000	·78
Eleven cases of chronic disease discharged with some degree of arrest	99	5,630,000	·88
Two cases of recent and extensive active disease discharged apparently cured	106	5,513,000	·96
Fourteen cases of early and limited disease discharged apparently cured	111	5,580,000	·99
Six of the above early cases on admission	99	6,112,000	·82
Average of six normals living in a sanatorium	100	5,368,000	·95

Conclusions.

From these observations we conclude that (1) cases of early and limited pulmonary tuberculosis with little or no fever have (*a*) the number of red cells and their hæmoglobin content within normal limits, or (*b*) a slight anæmia due to absolute diminution in the amount of hæmoglobin, the red-cell count being either normal or considerably higher than normal—*e. g.* between 6,000,000 and 7,000,000; (2) patients with extensive and active pulmonary tuberculosis associated with considerable fever, etc., but without complications, nearly always have some degree of anæmia due to diminution of hæmoglobin. This anæmia is found both in cases of extensive active disease of comparatively recent origin, *e. g.* six months or so, and in cases of acute relapse occurring in the course of chronic disease of long standing. The number of red cells in these cases is nearly always increased somewhat, the average number found in twenty-five patients of this type being 5,800,000, but in no less than fifteen, or 60 per cent. of these patients, the count was 6,000,000 or more. Less commonly, the red-cell count is within normal limits, but with a definite diminution of hæmoglobin. The hæmoglobin index is reduced, and is generally something between ·70 and ·80. The average of twenty-five patients with extensive active disease was ·75; the lowest ·58 and the highest ·88.

Compared with the average hæmoglobin index found by Emerson in normal individuals who lived in a city, viz. ·80–·90, the hæmoglobin content found in our cases of active tuberculosis was appreciably diminished in a large majority of cases, and, compared with our standard of normal individuals living in a sanatorium, the hæmoglobin content was very low. In two cases only we found a diminution in hæmoglobin associated with a diminution in the number of red corpuscles. The observations on one of these two patients were taken during a terminal relapse occurring in the case of a patient with very extensive disease of long standing.

(3) The greater the activity of the lung disease, and the longer its duration, the more marked are the changes in the blood—viz. reduction in the percentage hæmoglobin, increase in the red-cell count, and diminution in the hæmoglobin content. Towards the end of life, in patients dying of pulmonary tuberculosis, there is sometimes a gradual diminution of hæmoglobin and hæmoglobin content, which is associated with a reduction in the red-cell count. (4) Gradual arrest of the tubercular process is associated with a gradual increase in the percentage hæmoglobin and a diminution in the red-cell count, with a resulting increase of hæmoglobin content. (5) Complete arrest (apparent cure) of the lung disease is associated with restoration of the blood, as regards the red-cell count and hæmoglobin to its normal condition. (6) The blood of patients with completely arrested disease and of normal people when living in sanatoria becomes very rich in hæmoglobin, the hæmoglobin content in both cases usually being considerably higher than that of normal people living in towns.

Comments.

A very interesting question is how to account for the red-cell count in patients with active and extensive pulmonary tuberculosis being nearly always normal, or considerably in excess of normal—*e. g.* as high as 7,000,000. Some consider that it is due to the concentration of the blood, due to sweating, diarrhœa, or vomiting, but these conditions do not, in our experience, always account for it, as the high red-cell count is found sometimes when none of these symptoms have been present. Others consider the hypercythæmia to be a compensatory feature for the dyspnœa, since dyspnœa, due to any cause, produces a hypercythæmia; others admit an anæmia which is covered by an oligæmia, and autopsies suggest a diminution in the total volume of blood. Von Limbeck considers the changed water metabolism the important point, the general drying of the tissues concentrating the blood. Grawitz

considers the absorbed products of caseous globules to have a lymphagogue effect, thus concentrating the blood (Emerson). In our opinion, the high red-cell count found in cases with extensive active disease seems most likely to be due to the concentration of the blood as a result of a diminution of the total volume. We do not think, however, that the high red-cell count sometimes found in patients with early and limited disease, with little or no fever, is to be accounted for in the same way. The high red-cell count in these patients is rapidly reduced as the disease becomes arrested, and it seems possible that this increase of red cells may be due to a real increase due to generous dieting. Such an increase in the red-cell count is sometimes noted as a first change in the blood of patients who are doing very well.

We have noted this in the case of five patients, four of whom were cases of recent infiltration and one of chronic disease considerably arrested and without fever. The following are the records of these patients:

On admission.	After one to three months' treatment.	
5,499,000	6,000,000	Associated with a large increase in hæmoglobin content.
5,200,000	5,500,000	
5,100,000	5,680,000	
5,520,000	6,024,000	Associated with a small increase in hæmoglobin content.
5,580,000	6,400,000	

The next change in the blood of all these five patients was a diminution in the red-cell count, associated with a further increase in hæmoglobin content.

This increase of the red-cell count, associated with a steady increase in hæmoglobin, has a good significance, but is only a temporary condition. One of the most interesting points which we noted, and one which we have not seen described hitherto, is the gradual improvement which occurs in the condition of the blood of patients with active pulmonary tuberculosis as the morbid process becomes arrested. This improvement, as previously stated, always consists in a gradual reduction of the red cells until normal

limits are reached, and with this is associated a gradual increase in the hæmoglobin content.

The Practical Value of Observations upon the Red-cell Count and the Hæmoglobin in the Treatment of Consumption.

Apart altogether from the purely scientific or academic value of these observations on the condition of the blood of consumptive patients, a knowledge of the condition of the blood of any patient as to the hæmoglobin content is of distinct practical value, since it gives a very good indication of how the patient is reacting to treatment, and how completely the tubercular disease has been arrested. In the first place, we do not consider any consumptive to be in a thoroughly satisfactory condition unless his blood is approximately up to the normal standard, and, in the case of patients under treatment in a sanatorium, up to the standard for normal people living in a sanatorium. On reference to the after-history columns in our tables, it is seen that, with very few exceptions, all the patients who were discharged from the sanatorium with their blood in a normal condition have done very well. This is due, of course, to the fact that the restoration of the blood to its normal condition is associated with complete arrest of the tubercular disease; indeed, it can be taken as reliable evidence of the fact of arrest of the disease and restoration to health. In our experience, cases of early disease who are doing very well soon show an improvement in their blood condition, and, on the average, four months' treatment restores the blood up to the standard of normal people living in a sanatorium.

When a patient of this type—viz. with early disease—does not rapidly improve in this way it is an indication that the patient will not so readily throw off the disease, and further, when improvement in general health and physical signs are not associated with a restoration of the blood to the high standard which we expect in the case of patients of this type, the prognosis is not so good. It is

an indication that the disease is only quiescent and not eradicated. The following are records of patients doing thoroughly well:

	Hæmoglobin percentage.	Red cells.	Hæmoglobin index.
On admission	96	6,300,000	0·76
After 1 month	108	6,240,000	0·87
After 3 months	115	5,780,000	1
On admission	107	6,380,000	0·84
After 2½ months	106	5,616,000	0·95
After 4 months	115	5,380,000	1·06

It will be noted that the blood of both these patients was restored to a very high level in three and four months respectively. Both these patients left apparently cured, and have remained so ever since. Compared with these two records, the following observations on two patients of a similar type, clinically speaking, are of interest.

	Hæmoglobin percentage.	Red cells.	Hæmoglobin index.
On admission	73	5,400,000	·67
After 1 month	98	6,000,000	·81
After 4 months	103	6,160,000	·84
After 5 months	110	6,160,000	·89
After 6 months	106	5,650,000	·93

This patient had only a single apical lesion, and was discharged apparently cured, but as seen from the above records his blood improved comparatively slowly, and did not reach the same high standard as that of the other two patients. This particular patient has not done anything like so well subsequently, as he relapsed within twelve months of discharge, and his disease has gradually progressed, with intervals of arrest ever since.

Miss W—. Case of Early Disease of the Right Apex.

After six months' treatment: No physical signs except impaired resonance, no fever for five months, gained 14 lb. in weight; general health better than it had ever been in her life; walking seven to eight miles daily.

CONDITION OF BLOOD.

Hæmoglobin percentage.	Red cells.	Hæmoglobin index.
74	5,100,000	·73

This poor condition of the blood associated with such great improvement in health, and so far as physical signs indicated, complete arrest of the tubercular disease, is very unusual and of bad significance. This patient, as a matter of fact, relapsed soon after discharge, went steadily down hill, and died within twelve months.

Of course, the rapidity with which the blood improves must always be considered along with the amount of disease present in any particular case. In the case of patients with only a limited infiltration, the improvement, as we have already said, is very rapid, and reaches an abnormally high standard.

When the disease is more extensive in character, the improvement is slower, although if the treatment is continued long enough to secure a thorough arrest of the disease the blood will eventually reach the same high standard as that of patients with limited infiltration only. The following are examples:

Patients with somewhat Extensive Disease, with Fever.

CASE 1.

	Hæmoglobin percentage.	Red cells.	Hæmoglobin index.
After 6 months' treatment	100	6,160,000	0·81
After 10 ,, ,,	112	6,160,000	0·91
After 14 ,, ,,	110	5,280,000	1·94

CASE 2.

	Hæmoglobin percentage.	Red cells.	Hæmoglobin index.
After 6 months' treatment	104	5,920,000	0·88
After 8 ,, ,,	105	5,840,000	0·90
After 11 ,, ,,	110	5,560,000	1

In both these cases arrest of the lung disease and restoration of the general health were slow.

It would appear also that in the case of patients with chronic disease of long standing associated with, in many cases, some permanent damage to the general constitution, it is impossible to raise the hæmoglobin content to the same high level as is possible in the case of patients with limited early disease, even though the disease becomes quiescent and all symptoms of active disease clear up.

An hæmoglobin index of 1, even after a long course of treatment, is not often noted in these cases, the average being more commonly ·90. An hæmoglobin index of ·90 is, of course, very satisfactory, and above the normal standard of the average person living in town. It is only low relatively in comparison with the hæmoglobin content found in patients with completely arrested disease and in normal people living in sanatoria. The fact that the hæmoglobin content cannot be raised to such a high level is, in our opinion, merely an indication that tubercular disease, in chronic cases of long standing, cannot often be thoroughly eradicated.

Comparison of our Results with those of Other Observers.

Grawitz has divided cases of pulmonary tuberculosis into three groups. Group 1 with a slight involvement of the apex without fever showing clear signs of anæmia, some with a normal count, others with a slight reduction. Some early cases have almost normal blood. Whether there is a group in which the blood signs precede the physical signs may depend upon the care with which the signs are sought.

Group : 2 cases of chronic phthisis with cavity formation but without other complications, the temperature slight; the blood picture is remarkably normal as regards count and hæmoglobin. Group 3 : cases of hectic fever, supposed by some to be due to a secondary infection, but by others to pure infection of the tubercle bacillus. In these cases

there is true anæmia, a diminution of the count, sometimes rapid, and of the hæmoglobin, an anæmia which progresses often until death with evidence of true blood destruction (Emerson).

A very similar description of the blood in pulmonary tuberculosis is given by Coles ('Diseases of the Blood').

On inquiry we find that Dr. Coles obtained his information from von Limbeck's work, second edition, 1896, p. 337, and von Limbeck himself quotes Grawitz as his authority.

Our results agree very closely with these observations of Grawitz, viz. as to the slight degree of anæmia found in patients with early pulmonary disease, and the remarkably normal condition of the blood (as compared with standard normal of people living in a town) in regard to the red-cell count and hæmoglobin content found in patients with chronic disease with only slight fever, or, in other words, with a considerable degree of arrest of the disease.

The Group 2 of Grawitz we consider to be comparable with Sub-group 1 of our group of chronic cases.

So far as we can determine, Grawitz has not noted the condition of the blood in cases of fairly recent active disease with considerable fever, but without definite excavation. With regard to Grawitz' third group, viz. cases with hectic fever, we have only the records of one patient whom we can classify under this heading. The observations on this patient, which show a progressive fall of hæmoglobin with reduced red-cell count, agree with the observations of Grawitz.

A more recent work upon the blood in pulmonary tuberculosis is that by Ullom and Craig, published in the 'Transactions of the National Association for the Study and Prevention of Tuberculosis in America,' 1906. These writers adopt the same system of classification of pulmonary tuberculosis as Grawitz—a system which we consider to be unsatisfactory. Their conclusions are as follows: the hæmoglobin in Group 1 is quite constantly moderately reduced, averaging 74·8 per cent. In Group 2 we find a very slight increase in the amount of hæmoglobin, in the

general average, to 75·6 per cent. In Group 3 we find a decrease in the average to 70·6 per cent. It would have been more marked if it were not for the three fatal cases which had a very high hæmoglobin percentage.

Red corpuscles.—In considering the number of red cells we find that in Group 1 the eight cases ranged between 3,920,000 and 4,930,000, with an average of 4,510,000. The eighteen cases in Group 2 varied between 3,250,000 and 5,550,000, averaging 4,630,000. In Group 3, thirteen cases gave from 2,950,000 to 5,870,000, averaging 4,297,000. We find, therefore, in Group 1, a mild anæmia, in Group 2, the average is higher, and that in Group 3 we have two distinct divisions of cases, those with a distinctly low count mainly made up of "stationary cases," and a group in which the counts are quite high, almost entirely made up of "fatal cases." The average for this group is lower than either of the preceding groups. In only one instance was a single normoblast noted.

Hæmoglobin content.—The study of the colour index or the relation of the red corpuscles to the hæmoglobin gives very striking figures. In all three groups the colour index averages practically the same, and this average is distinctly below normal.

In Group 1 none of the cases reach normal, ranging between ·73 and ·95, averaging ·826. In Groups 2 and 3 we have a much wider range in the figures, three cases going above 1, and three falling below ·70, the average for Groups 2 and 3 being ·823 and ·831 respectively.

When comparing the results of our observations with those of other observers it is seen that several of our chief conclusions are in accord with the conclusions arrived at by other writers. The slight degree of anæmia often found in patients suffering from pulmonary tuberculosis has generally been noted, also the fact that the red-cell count in cases of chronic disease is often normal, or higher than normal—the hypercythæmia of von Limbeck and Grawitz. There is one noticeable difference, however, between our results and other published results, viz. that

our figures, both for the red-cell count and hæmoglobin are, for the most part, considerably higher than those hitherto recorded. The difference, indeed, in this respect, is sufficiently marked as to suggest there being some fallacy in our method of observation. We believe that our figures are correct, and that the difference is to be accounted for by the fact that our observations, with very few exceptions, were made upon patients who had undergone a long course of sanatorium treatment, whereas the other observations quoted were made upon hospital patients untreated or not undergoing any special form of treatment. The influence of the sanatorium *régime* in increasing the red-cell count and, markedly, the hæmoglobin, is well shown by our observations on normal people living the full sanatorium life, and we have very little doubt that a long course of sanatorium treatment influences the condition of the blood in cases of consumption in a similar way. Our records, in short, represent the condition of the blood in consumptive patients after sanatorium treatment.

There is another factor which tends to make the average of all our observations on the high side. The type of consumptive which has the lowest red-cell count and percentage hæmoglobin is the very acute case running a terminal course; of this type there is only one representative in our records. Again, the type of consumptive which most usually shows a normal red-cell count or an abnormally high count is the patient with chronic disease of some standing; the patients whom we observed included a large number of this type. We may add that a colleague of one of us (Dr. Burra, Assistant Medical Officer, King Edward VII Sanatorium) is now carrying out a series of observations upon the blood in consumptive patients, and that the results he has obtained so far confirm the results which we have recorded in this paper.

Bibliography on the Subject.

The literature on the subject has been well summed up by Ullom and Craig.

"*Hæmoglobin.*—As this was the first component of the blood to be studied quantitatively for clinical purpose we find a large series of estimations of this element. The methods varied greatly, from the spectroscopic to the purely chemical, which renders the results hard to correlate. That the hæmoglobin is constantly reduced in bone tuberculosis has been shown by Vierordt, Lacker, Bierfreund, and Dane. That, on the other hand, this reduction is not constant in pulmonary tuberculosis of various grades is shown by Leichtenstern, Gnezda, Barbacci, and Oppenheimer, who found fairly numerous cases in which the hæmoglobin was above normal. Quinquaud, Wiskeman, Laache, Neubert, Reinert, v. Noorden, Biernacki, Strauss, Rohnstein, and Stevens found a decrease in hæmoglobin of varying degree in all cases. In those instances in which the cases have been grouped according to Grawitz' system we find the hæmoglobin is given as follows: Group 1. v. Limbeck, Ewing, and Swan, decreased; Appelbaum decreased or normal; Cabot, increased or normal. Group 2: Cabot and Swan, reduced; v. Limbeck, Appelbaum, and Ewing, normal, or approximately so. Group 3: all the writers found it reduced. The majority, therefore, found it reduced in Group 1, normal in Group 2, and reduced again in Group 3. The amount of reduction varied greatly in the different reports. In regard to the high hæmoglobin finding in Group 2, we will refer again in considering the red corpuscles.

"*Red cells.*—Andral, Sorenson, Pick, v. Noorden, Zappert, Biernacki, Strauss, and Rohnstein, and Claude and Zaky found a constant reduction of the red corpuscles in tuberculosis of various stages. Becquerel, Melassez, Laache, Neubert, Reinert, Cabot and Da Costa found the decrease was not constant, as in some of the cases the count was normal or above normal. The most marked decrease was in a case reported by Melassez, in which the red cells were as low as 980,000. Oppenheimer believes the count is normal in this condition and only reduced in hæmoptysis. Dane, in bone tuberculosis in children, found the reds, as a

rule, not reduced. Where the cases are classified we find the following: Group 1: v. Limbeck, Strauer, Grawitz, Halbron, and Swan give a reduction in the red cells; Stevens, Appelbaum, and Ewing believe they are reduced frequently, but may be normal. Group 2: all the observers give a normal or increased count, except Swan and Halbron, the former, however, finding it sometimes increased; Halbron found it constantly decreased. In Group 3 all the above investigators found the red cells reduced, except Swan, who found them generally much reduced, but increased in three out of fifteen cases. The evidence in regard to the red corpuscles points to the fact that in cases of infiltration or cavity formation, with hectic fever, especially the latter, they are decreased in number. That in cases with cavity formation without fever or complications, and occasionally, also in the other two groups, we have a normal or even increased count." (From 'Examination of the Blood in Pulmonary Tuberculosis,' Ullum and Craig.)

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ON THE DE NOVO ORIGIN OF BACTERIA, BACILLI, VIBRIONES, MICROCOCCI, TORULÆ, AND MOULDS, IN CERTAIN PREVIOUSLY SUPERHEATED SALINE SOLUTIONS, WITHIN HERMETICALLY SEALED VESSELS

BY

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During the present year (1906), after a very long interval, I began again to occupy my leisure time in making experiments with different superheated saline solutions, in order to ascertain whether any evidence could be obtained of a *de novo* origin of living matter therein. I was induced to deal with such solutions for various reasons.

It seems clear that if living things originally appeared upon this earth solely as a result of natural processes, they must have taken origin from some such materials. Then, again, it seemed likely that such solutions would be less deteriorated than others containing organic matter by subjection to high temperatures. I was further tempted to use such solutions after discovering, last year, that Bacteria of different kinds would grow and freely multiply in a simple solution of ammonium tartrate when exposed to diffuse daylight, even better than when the solutions were kept at a much higher temperature but in the dark.[1]

[1] "The Simplest Kind of Protoplasm," 'Knowledge and Scientific News,' August, 1905, p. 199.

The recognition of this latter fact was something altogether new. It was well known previously that direct sunlight proves destructive to Bacteria, but diffuse daylight was also supposed to be injurious.[1] We shall find, however, that this is very far from being true.

It is clear that no experiments of this kind can be deemed conclusive until a general agreement has been arrived at as to the thermal death-point of such micro-organisms as are apt to be found within the experimental vessels. Fortunately, after years of research by workers in different parts of the world, there is at last unanimity in regard to this important point.

It is now well recognised that all microbes (including Torulæ and Fungus spores generally), when in their ordinary condition of active growth, are killed by exposure for a minute or two in fluids that are raised to the boiling point, 212° F.—almost all of them being, in fact, killed at distinctly lower temperatures, ranging from 140°—167° F. (60° to 75° C.).

The spores of Bacilli, however, constitute a well-known exception to this rule. Still, those of the hay-Bacillus and of others occasionally to be found in tap-waters have been declared by Chamberland, the present sub-director of the Pasteur Institute, to be all destroyed when immersed in fluids for a minute or two at a temperature of 115° C.[2]

There are certain "Thermophilic Bacteria," found in soils and in some other sites, which are more resistant still; but these have never been met with in tap-water, and are certainly not to be found in freshly distilled water, such as has been employed in all the experiments of which I am about to speak.

[1] In a lecture on "The Effects of Physical Agents on Bacterial Life" at the Royal Institution in June, 1900, Dr. Allan Macfadyen is reported to have said (the 'Times,' June 11th): "Direct sunlight was a most deadly bactericidal agent, and diffuse light was also injurious, though slower in action." See also 'Proceedings of the Royal Institution,' vol. xvi, p. 451.

[2] Comptes rendus, 1879, vol. i, p. 659.

Very full details concerning this part of the subject, which is of the greatest importance in reference to these experiments, will be found in a new work shortly to be published, entitled 'The Evolution of Life.'

Trials were at first made with several different solutions containing ammoniacal and other salts, either alone or in combination with certain acids; but attention was finally concentrated upon some solutions very similar to those with which I had performed a few tentative experiments in 1871, in which carbon was ostensibly absent, though its close chemical ally silicon was always present.

My reason for making trials at that time with such solutions may be best explained by the following quotation from my work 'The Beginnings of Life' published in 1872 (Vol. II, Appendix A, p. ix):

"Living matter being the result of a chemical combination of a certain kind, there is no absolute improbability in the supposition that the carbon usually existing in the living compound might be replaced by some other element. With the hope of throwing some little light upon this very difficult subject, I made several tentative experiments with saline solutions containing—in addition to nitrogen, oxygen, and hydrogen—some other element in the place of carbon. The element with which the carbon was replaced was either silicon, boron, chromium, aluminium, or iron.[1] Except in those in which carbon was replaced by silicon, no living things have been met with in any of these solutions (after they have been boiled and the necks of the flasks have been sealed during ebullition). This result—taking it merely for what it is worth—is extremely interesting and suggestive, since silicon is certainly the element which most closely resembles carbon, and which might therefore best replace it in compounds otherwise similar to those which constitute the basis of living matter. A silicon alcohol and ether has in fact

[1] As I have already stated, these experiments were merely tentative. It is not supposed that solutions were employed free from all trace of carbon, existing as an impurity.

been produced by Professor Wöhler, in which the carbon of the ordinary compounds is replaced by silicon. It is therefore deemed quite possible that silicon may take the place of carbon in certain forms of living matter. No absolute proof of this, however, can at present be advanced. What follows must be taken merely as an indication of the possibility of such an occurrence."

Reference was then made to an observation of my own, in which a mass of Mould was found growing luxuriantly on the surface of some silicate of soda solution, contained in a corked bottle which had been unopened for about six months. Attention was also called to the previous observations of Messrs. Roberts (afterwards Sir Wm. Chandler Roberts) and Slack on solutions of hydrate of silica, as recorded in the 'Quarterly Journal of Microscopical Science' for 1868 (pp. 105–108), in which they say: "All the specimens of silica solution supplied by Mr. Barff to Mr. Slack, whether kept in bottles nearly full and corked, in bottles containing much air, or in open vessels, exhibited the mildew threads in the course of a week or ten days." After relating some further observations and experiments, they say in conclusion, "The preceding experiments show the facility with which moulds will grow in a solution of pure silica in distilled water."

These observations proved plainly enough that solutions of silica were favourable nourishing fluids for the *growth* of living things; and this induced me to see whether any evidence could be obtained tending to make one believe that an actual *origination* of living matter could take place in such solutions.

Some few experiments were made at that time, and the results were such as to induce me to make further trials during the early months of this year; and these experiments soon convinced me that evidence of a remarkable kind was to be obtained.

I propose, therefore, now to refer to some of the results of experiments with three different solutions containing

silica, two of which are closely alike. Their composition is as follows :

(A) Sodium Silicate, two minims
Ammonium Phosphate, four grains
Dilute Phosphoric Acid, four minims
Distilled Water, one fluid ounce.

(B) Sodium Silicate, four minims
Ammonium Phosphate, four grains
Dilute Phosphoric Acid, four minims
Distilled Water, one fluid ounce.

(C) Sodium Silicate, six minims
Liquor Ferri Pernitratis, eight minims
Distilled Water, one fluid ounce.

About half an ounce of one or other of such solutions has been used in each experiment. The tubes employed have been mostly composed of soft German glass, about 1 inch in diameter and 3 inches long, with a tapering shoulder rather more than an inch in length beyond the body of the tube.

During the process of making, such tubes would, of course, be thoroughly sterilised, and the narrow extremity of each was always closed at this time. In this sterilised condition the tubes were delivered, and were thus always retained by me till they were about to be used.

In the case of the A and B solutions the ammonium phosphate is first allowed to dissolve, with the aid of a little heat, in the distilled water ; the acid is then added and next the sodium silicate.[1] Such a solution is clear, and shows no precipitate. It is introduced into one of the tubes, which has just been opened by filing off its extremity ; and the tube is then at once securely re-sealed in the flame of a Bunsen's burner.

In preparing the C solution a small amount of a pale

[1] This latter ingredient (sold as a dense liquid) is best kept for use diluted with an equal bulk of distilled water, as in that condition the proper quantities to be used can be more easily measured.

yellowish precipitate of silicate of iron is produced as soon as the solution is made.

Four or five of the sealed tubes are introduced into a can of water, if they are to be heated to 100° C. only. The temperature of the water is gradually raised to the boiling point, and the boiling is prolonged for ten minutes, when the tubes are taken out with a wooden forceps and allowed to cool.

When temperatures between 115° and 130° C. (239° and 266° F.) have been employed a calcium chloride bath has been prepared which boils at the desired temperature, and a thermometer is fitted to the can so that its bulb is immersed in the mid-strata of the fluid. The tubes are placed in the bath as before when cold. The temperature of the bath is then raised to the desired point, and with a little care can be easily maintained at that temperature for ten or twenty minutes, as may be desired.

When the tubes have cooled somewhat they have been cleaned externally, labelled, and then either exposed to diffuse daylight, or else placed in an incubator at 35° C. (95° F.).

Most of these experiments were performed during the warm and bright weather which occurred during the summer and up to nearly the end of September in 1906; and from a comparison of their results with those of some other experiments made during the months of September and October (in which exposure to light was had recourse to as the favouring stimulus) the advantage derivable from warm and bright weather was very plain.

As a rule, tubes containing the A or the B solution should be exposed to light or else to heat in the incubator for four to six weeks; while, when the C solution is being used they should be exposed to light or to heat for distinctly longer periods—that is, for from two to four or even six months, if the best results are to be obtained.

These are, in themselves, facts of great significance in regard to the interpretation of the results; since when living organisms are found to have been taken from the

tubes, the questions to be determined must always be, (1) have the organisms descended from others pre-existing within the experimental vessels, that have not been destroyed by the previous heating to which the tubes and their contents have been submitted? or (2) are they organisms that have been actually engendered within the experimental vessels, after the process of heating to which they and their contents have been exposed?

In reference to these questions it must be steadily borne in mind that where the organisms found are Bacteria, Micrococci, Torulæ, or Moulds, and the sterilised experimental vessels together with their contents have been exposed to temperatures of 100° C. and upwards for ten minutes, we have no right to suppose that any survival could occur. And, similarly, where the organisms found within such tubes have been Bacilli, and the tubes and their contents (looking to the nature of the latter) have been exposed to 115° C. and upwards for ten minutes —and still less when they have been exposed to 130° C. for twenty minutes—we should have no right, in the existing state of knowledge, to suppose that Bacilli or any organisms could survive such an ordeal.

This fact of the admitted thermal death-point of the organisms in question is the first all-important consideration, in reference to the interpretation of the experimental results when living organisms are found. But another consideration which, in the light of existing knowledge, is of scarcely less importance is this. In the A and B solutions, after the process of heating, more or less silica is deposited, leaving the fluid above quite clear; while in the C solution the amount of the pre-existing deposit is slightly increased. Now if within an hour or two, or a day or two, after these deposits have undergone the process of heating, some of the tubes are opened, as "control" experiments, and portions of the flakes of silica are withdrawn and carefully examined, no trace of organisms is to be found; though a similar careful examination of the sedi-

from other tubes that have been favourably ex-

posed, during an interval of several weeks or months, may reveal the fact that in such sediment different kinds of living organisms are more or less plentifully present.

The force with which such facts tell in favour of *de novo* origination as against mere survival of germs is extremely great, when we consider that in the case of an organic infusion, if organisms appear as a result of contamination, the fluid in the course of a *few days* becomes cloudy or actually turbid, owing to the rapid growth and multiplication of the Bacteria. Yet in these sodium silicate solutions the supernatant fluid invariably remains perfectly clear, *month after month*, even though Bacilli, Micrococci, or Torulæ may be swarming in or upon the flakes at the bottom of the tube—where at first, and for some long period, no such organisms are to be found.

FIG. 1.

Appearance of a tube after it has been heated.

Much more time, therefore, seems needed for origination of living matter to occur than suffices for mere growth and multiplication of pre-existing germs.

Another very important point is this. In the organic infusions a large proportion of the organisms found will exhibit very active movements, but in the sodium silicate solutions the organisms are *invariably motionless*, scattered through or upon the flakes, and apparently originating where they are found. This holds good for Torulæ as well as for Micrococci and Bacilli, and must be regarded as a very significant fact when we see these motionless organisms occurring singly, as well as in small groups, throughout the flakes. When hundreds, and often thousands, of such motionless organisms are thus to be seen; and when it is borne in mind that no organisms whatever are to be found within other of these tubes (serving as "control" experiments) if their contents are

examined within a day or two after they have been heated, what conclusions are we to draw? If *organisms are not there at first, after the process of heating, and if, after an interval, they are there in abundance and are invariably stationary, clearly they must have developed in the sites where they are found.*

Is there any chance of contamination having taken place after the heating? None. The procedure has been this. After an interval of weeks or months, the end of the tube has been cut off, and if the orifice is narrow some of the deposit is shaken out on to a clean microscopic slip, which has just been sterilised by passing it four or five times through the flame of a spirit lamp; and the deposit is then covered by a cover-glass, which has been similarly treated. If organisms are found, they are generally photographed at once; or else after they have been stained by drawing a drop of a solution of eosin or of gentian violet beneath the cover-glass. In case the specimen is reserved for future more careful examination, or with a view to see whether the organisms found will develop further, the cover-glass is at once surrounded with some paraffin melting at about 105° F.—which will sometimes prevent evaporation for several weeks, and allow Bacteria or Torulæ to multiply, or the latter to develop hyphæ: and thus remove any lingering doubt as to whether the organisms are really living.

Where the aperture of the tube is larger, the only variation is that some of the deposit is withdrawn from the tube by a pipette, which has just been sterilised in the flame of the spirit lamp, and from it transferred to the sterilised microscope slip.

With the solutions which I have described, when the periods of exposure to light and heat have been long enough, organisms will generally be found in the first one or two specimens taken from the tube. As I have said, the organisms will all be motionless, but their numbers, looking to the character of the fluids employed, will

clearly testify to the fact that they must have developed within the tube after it had been heated—moreover, in the specimens mounted as I have described, the organisms will often be found to further multiply or develop beneath the cover-glass.

A few examples of the results obtained in these experiments may now be given, as illustrated by photomicrographs taken at the time when the tubes were opened.

In Fig. 2 a number of twisted Bacilli or Vibriones are shown that were taken from a tube containing some of the B solution which had been heated to 100° C. for ten minutes, and which was subsequently exposed to light for only three weeks, though during bright and warm weather.

Fig. 3 shows a multitude of Micrococci and Bacteria, together with a few long Bacilli, taken from another tube containing a B solution which had been heated to 115° C. (239° F.) for ten minutes, and subsequently kept in an incubator for five weeks. On the edge of another of the flakes of silica from this tube the Micrococci were found that are represented in Fig. 4.

Two incipient Moulds, such as have been very frequently met with, are shown in Fig. 5, which were found in a B solution that had been heated to 120° C. (248° F.) for ten minutes, and which was subsequently exposed to light during six weeks. Several of such Moulds were found in the one or two samples of silica taken from this tube, some of them being in a very early stage of development, and with filiform extremities—showing no evidence of commencement from a Torula corpuscle or other definite spore.

In Fig. 6 some remarkable thread-like organisms are shown that were taken from a tube which had been heated to 130° C. (266° F.) for twenty minutes five months previously. During the first three months it, and a companion tube prepared at the same time, had been exposed to light for three months, when both of

FIG. 2 (× 500).

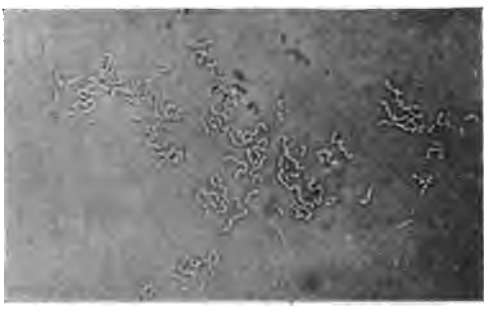


FIG. 3 (× 700).

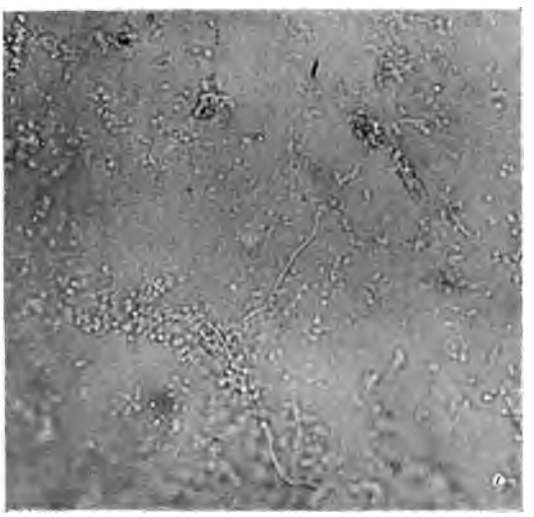
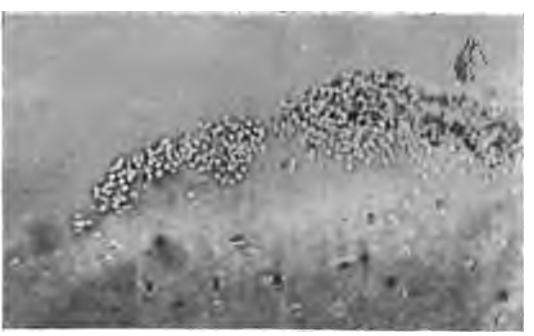


FIG. 4 (× 700).



Organisms taken from previously closed tubes which had been heated to 100° C. and 115° C. for ten minutes.

Fig. 5 (× 400).

Fig. 6 (× 500).

Fig. 7 (× 700).

Organisms taken from previously closed tubes which had been heated to 120° C. for ten minutes and to 130° C. for twenty minutes.

A large group of Micrococci, partly stained with eosin, is shown in Fig. 9, which was taken from a tube containing some of the C solution that had been heated to 130° C. for twenty minutes, six months previously. It was exposed to light for three months, and was subsequently kept in the dark for another three months. This was the longest time that any of the tubes had been left; and the organisms were more abundant and more varied than in any of the others whose contents I have examined. The groups of free Micrococci were very numerous, and they were mixed with a smaller number of others within loculi, together with the Spirillæ, Torulæ, and some well-developed Mould.

In Fig. 10 an early stage of the loculated Micrococci is shown—a group of three. Single loculi, each containing from 5 to 7 Cocci, have often been seen, as well as larger groups of such bodies, which ultimately tend to grow into comparatively large masses in which the walls of the loculi are no longer distinct, such as is shown in Fig. 10. In both the small and the large masses some of the Cocci seem to give origin to twisted filaments like those to be seen in Fig. 11. Similar filaments I have also found growing from some of the groups of free Micrococci. The filaments are more like those of an Actinomyces than of a Mould, but I have been able to find no description of anything like them—that is, having such an origin —in any of the bacteriological works consulted.

The loculated Micrococci are comparable with Leuconostoc and the "frog-spawn"-like masses, to which *L. mesenteroides* gives rise, though the contents of the loculi in this latter case are always Streptococci. The organisms represented in Figs. 10 and 11 were taken from a vessel which had been heated to 100° C. for ten minutes, though almost exactly similar organisms have also been found in two tubes that had been heated to 130° C. for twenty minutes. The photographs of the organisms from the latter tubes were not quite so satisfactory, however, as those that have been here reproduced. This organism also seems to be one hitherto unknown. But it is interesting to find

FIG. 8 (×700 and 500).

FIG. 9 (× 700).

FIG. 10 (× 700).

Organisms taken from previously closed tubes which had been heated to 130° C. for twenty minutes and 100° C. for ten minutes.

that it, like the other organisms that I have met with in these solutions containing sodium silicate, is to be met with in fluids which have been exposed to very high

FIG. 11 (× 700).

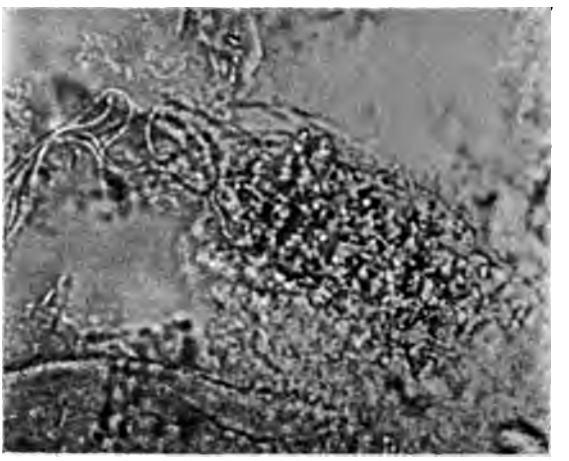
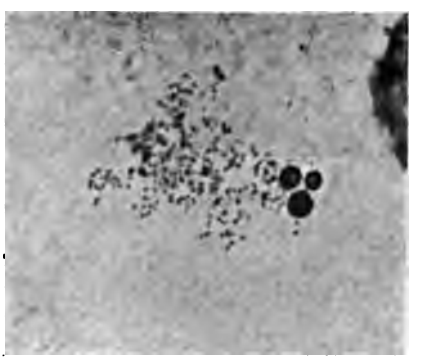


FIG. 12 (× 700).



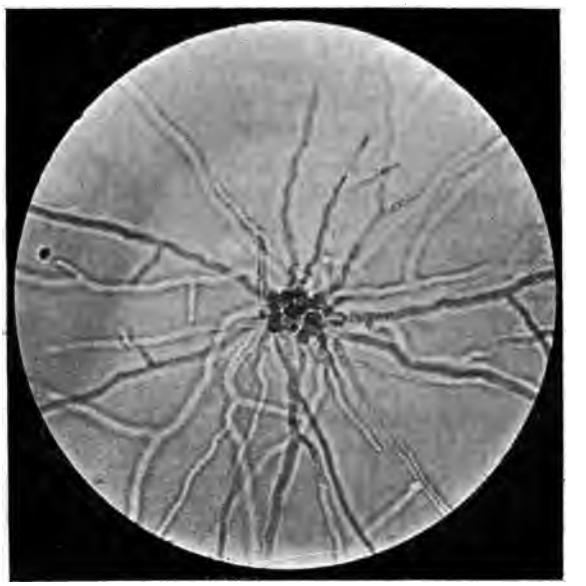
Organisms taken from previously closed tubes which had been heated to 100° C. for ten minutes and 130° C. for twenty minutes.

temperatures, as well as in those that have been heated to 100° C. only.

In another of the tubes containing a C solution which

had been heated to 130° C. for twenty minutes, and had subsequently been exposed to light for six weeks only—though in very warm, bright weather—I found, in the first sample of sediment taken from the tube, many groups of Micrococci, both free and within loculi, some of the former especially giving origin to long twisted filaments. One very large group of the loculated organisms was also found as well as a number of Torulæ. In Fig. 12 a group

FIG. 13 (× 375).



Organisms taken from previously closed tubes which had been heated to 130° C. for twenty minutes.

of small free Micrococci from this tube is shown, together with three Torula corpuscles, all deeply stained with eosin.

After two drops had been taken from the tube it was re-sealed on July 13th, and left in the dark at the ordinary temperature of the air for the next six weeks. On my return to London, after this interval, I examined the contents of this tube again, and found in the sediment a great increase in the Torulæ, so that in places there were groups

of 20–30 or more some of which were developing hyphæ. A small patch of such Torulæ and Mould, partly stained with eosin, is shown in Fig. 13, though I found very many other patches—some larger and some smaller. The Torulæ shown in Fig. 12 would correspond with those shown under a much lower magnification in Fig. 13, and there can be no doubt but that they are some of the same organisms.

Before making some final statements concerning the major problem with which we have hitherto been concerned, a subordinate though highly interesting question in reference to these experiments in which silicon has been present and carbon has been ostensibly more or less completely absent, must again be referred to.

The question is, whether we are to consider that silicon, whose chemical properties are so very closely allied to carbon, and which, like carbon, first appears, as Sir Norman Lockyer has shown, in some of the hottest stars, can take its place, wholly or in part, in protoplasm.[1]

I do not pretend that my experiments afford any distinct proof that such a substitution is possible—since carbon may have been present in the solutions, in the way of some accidental impurity, either in the distilled water or in the chemicals employed.

Still, the experiments recorded in the previous pages and elsewhere are very suggestive that such a substitution may be possible, when we bear in mind (*a*) the freedom with which Moulds will grow in and on the surface of colloidal silica, as originally observed by Roberts and Slack, and subsequently by myself; and when (*b*) we recognise that minute Moulds and different varieties of Micro-organisms

[1] According to Ostwald ('Inorganic Chemistry,' translation, 1904, p. 425), "The largest part of the earth's surface is composed of silicon dioxide, or of its compounds: over a quarter of the solid crust of the earth is formed by silicon." And in regard to the silicates of the alkali metals, which go by the name of *water glass*, he says (p. 427): "These salts are readily obtained by fusing quartz with the hydroxides or carbonates of the alkali metals."

will appear within closed tubes containing the solutions in question, though these have previously been exposed to high temperatures. It may be said that in such cases the organisms, if from no other source, have been capable of taking their carbon from the CO_2 of the air within the tube; but, against this supposition, in the case of the sealed tubes there is the fact (*c*) that the organisms are invariably found away from the air, in or upon the flakes of silica which collect at the bottom of the tube; and that for month after month they are to be found there only, and never free in the strata of water intervening between the flakes and the air. There is the further fact (*d*) that the organisms appear almost, if not quite, as freely in tubes from which the air has been expelled by boiling as in those containing air. Taken together, these facts seem to me to make out a strong presumptive case in favour of the view that silicon is capable, to some extent, of taking the place of carbon in protoplasm.[1]

Interpretation of the Experimental Results.

In regard to the cardinal fact of the appearance of organisms in the experiments that have just been recorded, this seems only capable of receiving one interpretation, namely, that the organisms have been evolved within the tubes. All the old objections to such an interpretation have been fairly met. The organisms found have undoubtedly been living, and they have occurred in tubes which have been *flambés*, previous to being again superheated together with their contents.

It is now generally admitted, as we have seen, that all micro-organisms in their ordinary condition are killed by the briefest exposure in fluids to 100° C.—to say nothing of lower temperatures.

[1] Many organic compounds, in which silicon wholly or in part replaces carbon are referred to in my forthcoming work 'The Evolution of Life,' p. 279, Note 1.

The spores of Bacilli are recognised as the only products of micro-organisms capable of resisting such an exposure. Yet, as we have found, various forms (other than Bacilli) which are admitted to be killed at 100° C. can, and do, constantly appear within closed vessels that have been heated to this temperature for ten to twenty minutes. This applies, among other less known forms, to Bacteria, Vibriones, Micrococci, Streptococci, Torulæ, and other germs of Mould. When these organisms appear within sealed tubes under such conditions, we can, therefore, only conclude that they must have been evolved *de novo*.

In regard to ordinary Bacilli, the position, as we have seen, is different. These particular forms possess "spores," which, at all events after they have undergone desiccation, are capable of resisting a higher degree of heat. The extent of their powers of resistance has been differently estimated, but after prolonged researches in the Pasteur Institute, the point was formerly fixed by the master himself at 110° C., and later, after two years' work, could only be raised, as we have seen (p. 2), by his then assistant to 115° C. At this point, it is proclaimed, all the spores of ordinary Bacilli are completely and rapidly killed.

Noting by the way that even this temperature is much higher than Bacilli spores, in my direct experiments, were shown to be capable of withstanding (see *loc. cit.* pp. 70–84), and, further, that such bodies are little likely to be found in the freshly distilled water, or in either of the chemicals made use of in my experiments, let us, for the present, accept this temperature of 115° C. as needful to destroy all the products of ordinary Bacilli. But, as we have seen, such bodies, as well as Bacteria, Vibriones, Micrococci, Streptococci, Torulæ and other germs of Fungi, have appeared within our experimental vessels when they have been heated, for from ten to twenty minutes, to temperatures ranging from 115° to 130° C. These organisms which we have seen to be living—which developed and multiplied—must, therefore, have been

evolved *de novo*. What other answer is it possible to give?

All the bacteriologists throughout the civilised world during the innumerable researches they have carried on for the last five-and-thirty years have only been able to find certain so-called "Thermophilic Bacilli" in the soil that are capable of resisting higher degrees of heat than 115° C. Yet these low organisms, which are said never to be found even in tap-waters, and *a fortiori* not in recently distilled waters or in either of our chemicals, have, according to Christen[1] never shown signs of life after they have been exposed to "125° to 130° C. for five minutes and longer; to 135° C. for one to five minutes; or to 140° C. for one minute."

The moist resistant forms of these spores of the soil Bacilli, examined by W. H. Park, succumbed, however, at temperatures rather lower than this. He says they were destroyed "by exposure for twenty-five minutes in steam at 113° to 116° C., and in two minutes at 127° C."[2]

It comes to this, then, that all the organisms found in my experiments, with the exception of Bacilli, are such as would be killed at 100° C.; that these latter, so far as they could by any possibility be found within my tubes, should have been killed in one or two minutes at 115° C., yet Bacilli, as well as Bacteria, Vibriones, Micrococci, Streptococci, Torulæ and other Fungus-germs, dying under 100° C., have been taken in large numbers from tubes that had been heated to 115°–130° C., for ten to twenty minutes.

Elsewhere, I have said,[3] "we have in reality two distinct and more or less independent methods for attacking the problem as to the present occurrence of Archebiosis—that (*a*) by *experiment* with superheated fluids in closed flasks;

[1] See Lehmann and Neumann's 'Principles of Bacteriology,' Transl. 1906, p. 53.

[2] 'Pathogenic Micro-organisms,' 2nd Edition, 1906, p. 45.

[3] 'The Nature and Origin of Living Matter,' 1905, p. 158

and (*b*) another less recognised method, that of mere *observation*, aided by high microscopic powers, of what occurs in thin films of suitable, unheated organic fluids. This latter method calls attention to, and thoroughly exposes the fallacy of the common belief that, the occurrence of 'spontaneous generation' is opposed to the universal experience of mankind. It shows that what is supposed to contradict this common experience lies altogether outside, and, of necessity, completely beyond the range of ordinary human experience. Nobody with unaided eyes could ever have witnessed the birth from fluids of invisible particles, by which 'spontaneous generation' of living matter must always commence, if it commences at all. And, even when aided by the most powerful microscope, nobody could decide when the *minimum visible* particles appear in the field of view, that such particles have proceeded from invisible germs of pre-existing organisms, rather than from a primordial synthesis of living units. But, in regard to this latter point, I have already shown that absolutely no logical or consistent reason exists for a disbelief in the present occurrence of Archebiosis." There is, as was stated, "no vestige of evidence to show that under favourable conditions the process may not be continually taking place all around us."

But what is assumed to be continually taking place in free nature—in ponds, lakes, rivers, the sea, and in innumerable other sites—must be regarded as a process far more easily brought about than can ever be possible under the very restrictive conditions which can alone exist in our experimental vessels, where we have to do with small quantities of fluid, more or less degraded by a preliminary heating, and enclosed within small glass vessels. Yet, as I have endeavoured to show by all the evidence recorded, in Chapters X–XII of my forthcoming work 'The Evolution of Life,' concerning the thermal death-point of living organisms and their germs, taken together with many old and these new experiments, the evolution of living

things is capable of occurring even under such unfavourable conditions.

We seem bound to conclude, in fact, that there must be a natural proclivity to the formation of living matter. We are almost driven to such a conclusion when we see the freedom with which it can be produced in a simple inoculated solution of ammonium tartrate in distilled water.[1]

There we have the mere growth and multiplication of very varied micro-organisms occurring under conditions so simple as to be almost incredible. Now, again, we are confronted with facts, hard to be believed and difficult to understand—yet incontestable—when we find varied forms of Bacteria and Torulæ appearing plentifully in hermetically sealed tubes which have been previously submitted to a scathing degree of heat far more than is sufficient to destroy all known forms of like kind. The organisms are not there at first after the tube has been heated: after a time they are to be found in abundance. But they are invariably motionless, and must, therefore, have developed in the sites where they are found.

The wonder too is enormously increased when we think of the simplicity of the media in which this life-giving process occurs. The simplicity of the conditions, in fact, for the "origination" of life in my latest experiments, is only equalled by the simplicity of the conditions sometimes sufficient for the "growth and multiplication" of already existing living things—as shown by their rapid increase in the simple ammonium tartrate solution in distilled water, in which at least seven different kinds were found growing side by side.[2]

Then, again, in my work, 'The Nature and Origin of Living Matter,' Chapter IX, many instances of what I believe to be the *de novo* origin of Bacteria by heterogenesis are cited; and in all such cases what can be seen is this—particles becoming visible in the midst of more

[1] See 'Knowledge and Scientific News,' August, 1905, p. 199.

[2] *Loc. cit.*, p. 200.

or less homogeneous protoplasm, *such particles being invariably motionless*, but followed soon by their development into definite Bacteria or their allies—recognisable as such by their shapes and modes of collocation. There is, therefore, in all such cases an appearance altogether different from that of *adult organisms in a state of activity*, such as would be seen if we had really to do with cases of infection.

The similarity in the mode of origin of the Bacteria and Torulæ within the flakes of silica in my recent experiments, to that which occurs in these processes of heterogenesis is therefore very striking—we have always the minute motionless specks, gradually becoming visible and taking on characteristic shapes, just as crystals do when they separate from their parent media, and just as the doctrine of evolution would seem to require in the case of micro-organisms.

And, in reference to this fact of the new-born units developing into *well-known forms*, the following remarks made elsewhere may be quoted :[2]

"There would be, in fact, just as much reason why the new-born organism should develop into the form of one already in existence, as there would be that the crystal of sulphate of soda which forms to-day in a solution of that substance should resemble that which formed under similar conditions twelve months or a hundred years previously. He who believes in the uniformity of natural phenomena could anticipate no other result. Living matter, which we believe to be now produced *de novo*, speedily shapes itself into some well-known form ; and so also new crystalline matter, which may have been produced synthetically by the chemist in his laboratory, falls habitually into one or other of the known crystalline systems."

"It seems, therefore, no more wonderful that the simple Mould that develops *de novo* to-day should resemble another which develops from the spore of a pre-existing organism, than that a crystal forming independently to-day in a

[1] 'The Nature and Origin of Living Matter,' 1905, p. 287.

saline solution should resemble another which is capable of arising by the growth of a fragment detached from a similar pre-existing crystal. In all these cases there is a similarity of product, because the crystalline or organic form produced is to be regarded as the physical expression of the harmonious actions which have led to its production—because the forms are the results of a physical necessity, and not of a mere blind chance."

The remarkable constancy of the shapes and other properties of crystals, in accordance with their molecular composition and structure, is an interesting fact, when we consider that crystals are always born anew—that heredity has nothing whatever to do with their properties.[1] Similarly, I would say that the Bacteria and Torulæ that are ever being born anew can owe nothing to heredity, but that their shapes and properties must also be, in the main, due to their ultimate molecular composition.

There is one important difference, it is true, between the two kinds of units. The simpler molecular composition of the crystal, which is a statical aggregate, does not predispose to change; while the infinitely more complex molecular architecture of the living unit distinctly favours those changes in form and in property, under varying external conditions, which bacteriologists have long ago found to exist—though without such change in surrounding conditions the organisms will, of course, continue to "breed true."

[1] In an interesting article on "The Study of Crystals" A. E. H. Tutton says (the 'Times' Engineering Supplement, September 19th, 1906): "One of the main results of recent investigation has been to prove that the beautiful exterior shapes of crystals, as defined by the geometrical angles between the numerous truly plane natural facets, rather than by the relative development of those planes, is an invariable and characteristic property of the substance composing the crystal, by which that substance can be identified from all the countless other substances which are known to exist." Moreover, he adds, "The refractive power and the behaviour of the 'crystal' in polarised light afford immediate evidence of identity almost as valuable as the angles between the natural facets."

DISCUSSION

Professor STARLING said that he believed that most physiologists would be in sympathy, in principle at any rate, with the main thesis which it was Dr. Bastian's object to prove. The doctrine of continuity had, during the past quarter of a century, extended its sway over all realms of intellectual activity, and the dictum that "natura non per saltum agit" had been accepted as applicable in all departments of biological study, so that at the present time the existence of a direct line of descent from the lowest to the highest forms of life had become to most biologists a logical necessity. If, therefore, man himself could be regarded as produced by the action of natural selection on variations arising spontaneously in some lower type, and all grades were to be found between him and the lowest organised being, it seemed difficult to believe that the lowest organisms of all, the establishment of the life of which, in many cases, was a subject of difficulty, were not produced by a similar continuous process from those constituents of the world which we were accustomed to regard as inorganic and lifeless. To every physiologist the creation of living matter was a familiar process. Every day, in all of us, the lifeless material of the food was being taken up and formed into the living constituents of our own bodies. The growth of the living matter of a man from the microscopic fertilised ovum involved the addition of a huge amount of living material, which is only got at the expense of lifeless matter. In this process, however, of vivification of the material of the food, one factor was present which would have to be dealt with in any attempt to form a conception of the origination of life itself. In the process of assimilation the food which was built up and endowed with life was in each case moulded by the pre-existing living material. It was a well-known fact that, on account of this necessity of conforming to the living mould, striking differences existed between the food value of substances which are chemically almost identical. Thus, of the sugars only dextrose (d-glucose) and its immediate derivatives—fructose and mannose—were of value as food, the corresponding molecules derived from lævo-glucose, which were the very mirror images of our familiar food sugars, being quite useless to the plant or animal organism. The first step in the utilisation of the life-creating energy of the sun's rays was the assimilation of carbon from the CO_2 of the atmosphere by the green parts of plants. This process could be imitated, in part at least, in our laboratories; under the action of chlorophyll or other fluorescent substance we could catch the energy of the sun's rays and utilise it for the formation of formaldehyde from

the carbon dioxide of the atmosphere. The formaldehyde could be converted with the greatest ease into a mixture of sugars, but in the plant this process was constrained by the living mould, so that the sugar formed was always of the one type—namely, d-glucose. Practically every product of the vital activity of organisms was endowed in a similar way with the optical activity, which was the physical result of its one-sided or spiral formation. Why living matter must always be built in this spiral, and what the significance of this mode of construction is for the processes of life, it was not possible as yet to determine. This riddle, however, would have to be solved before we could try to form an adequate idea of the processes involved in biogenesis. The many difficulties which beset the problem could not, however, alter the firm belief of many physiologists that just as there is no break in the chain of life, so there is no real gap between the inorganic and the organic worlds. The formation of living material, as it begins in the green parts of plants, was an endothermic process, and involved the absorption of energy, which, in this case, is furnished by the sun's rays. It was possible that in the early stages of the world's history, under the influence of the higher temperature then obtaining on the earth's surface, what had been called a "fortuitous concourse of atoms" resulted in the endothermic formation of some substance, with a tendency to continuous polymarisation. Such a substance, though it would grow, would not be living, since it would possess no powers of adaptation, and the smallest change in external conditions would result in its destruction, with the evolution of the energy involved in its birth. The first beginning of living stuff would take place when such a substance was formed with two conditions of stability, so that, *e.g.*, while growing continuously by polymarisation at higher temperatures, the disintegration occurring on a small fall of temperature would be limited and not involve total destruction of the molecule. Such a substance would represent a substratum on which natural selection could work, so that of the many compounds formed by its interaction with the surrounding medium those would tend to persist which had the greatest range of stability. The range of adaptation, however, would be, for many ages, extremely limited. Even in the lowest organisms at the present time we find an extreme susceptibility to the slightest changes in the environment. It was only necessary to transfer a loopful of bacteria to another culture medium, approximating as nearly as possible in composition to that in which they have grown, to destroy a large number of the living organisms, the fresh cultivation being formed by the multiplication of the minority of the bacteria which survive the change. If such a spontaneous formation of something on which natural selection might work with the production of organised functions could take place once it could take place

many times, and we must assume, not that life was introduced on to this planet at one moment of geological time, but that such a substance was continuously being formed and destroyed by various cosmic events. At the present time we must speak, not of one protoplasm, but of many protoplasms, corresponding to each individual and to each cell of each individual, and just as there is a multiplicity of protoplasm at the present time, so one must assume a multiplicity in the births of the living matter which was the ancestor of the existing forms of life. If, therefore, it was to be assumed that many creations of living matter had occurred on the earth's surface, was there any reason why a similar spontaneous generation should not occur at the present time? Although the temperature of the earth has actually cooled, and the interatomic play at its surface is not so violent as is assumed to have been the case at an earlier period, the energy of the sun's rays is still available for, and made use of by, the organic world in the creation of life after the pre-existing forms or moulds, and could therefore furnish the energy for the endothermic production of a new form of living material, apart from the co-activity of existing life. Dr. Bastian was therefore justified in carrying out his experiments under the influence of sunlight. There are, however, two considerations which must seriously prejudice the success of any researches carried out at present on this subject. In the first place, a spontaneously formed living matter must be of extremely low organisation and unstable in the highest degree. It has long given place in the world's history, as the result of natural selection, to organisms which, relatively to it, must be regarded as highly differentiated, presenting chromatin, cell-wall, cytoplasm at least. Any such newly-formed living matter would, therefore, probably even before it were detectable by ourselves, serve simply as a pabulum to the myriads of organisms existing and struggling for a living on the world's surface. Only in a lifeless world, such as that contained in Dr. Bastian's sterile tubes, would it be possible for any life to come into existence without at once becoming the prey of the organisms of the present day. But even then the question would arise how such a low form of life would be capable of recognition as living. The chief criterion at the present day for the living nature of any minute organism is its power of multiplication. Supposing the forms described by Dr. Bastian were really alive, it should be possible, by the means familiar to the bacteriologist, to separate them into their varieties and cultivate them on artificial media. The difficulty at once presents itself, however, that the absence of such a test would not at once exclude the living character of these forms, owing to the fact that the more primitive the nature of the living substance the less resistance would it present to changes of environment, and the trifling manipulation necessary to carry out the

test, the slight change of medium involved, might suffice to destroy the glimmer of life possessed by these forms. Dr. Bastian would probably inform the meeting later on as to the nature of the tests which he had applied to the contents of his tubes in order to convince himself of the living character of the forms he described. The subject was one of extreme interest to everyone concerned with research into living functions, and the speaker could not but admire the courage with which Dr. Bastian had attacked the solution of this most difficult of all problems.

Dr. E. W. Goodall observed that as Dr. Bastian's paper was the result of a great amount of work, it had necessarily been put before his audience in a somewhat condensed form. He might have misunderstood some of the points which, perhaps, were explained in detail in the unabridged paper. But he asked, with respect to the sterilisation of the tubes, whether control experiments had been made. Had similarly constructed tubes, containing known organisms growing in culture media, and sealed up, been submitted to the same process as the tubes containing Dr. Bastian's salt solutions? He also requested to be informed whether the organisms observed by the author of the paper had been sub-cultured? Some of them appeared to resemble organisms already well known; and it would have been a simple matter to have determined their nature. He understood that it was a fact known to chemists that sterile solutions of certain salts would, under certain conditions, yield microscopic appearances which bore a close resemblance to certain micro-organisms. It was impossible to say merely from the inspection of photographs projected on the screen what the organisms seen by Dr. Bastian really were. Hence it was of importance to know whether any attempt had been made to ascertain, by bacteriological methods, whether they were living.

Dr. Bastian, in reply, said there was absolutely no room for doubt that the organisms found by him in the saline solutions were living organisms. The comparison of the "control" examinations with the final examinations showed that; moreover, the organisms had often been seen, as stated, to multiply and develop beneath the cover-glass. In regard to the question whether his tubes were thoroughly sterilised, he pointed out that the glass-blower in making them would be compelled to make them red-hot, and that the tubes were sealed in this condition. They were only opened to be charged with the experimental fluids, and were then immediately re-sealed in the flame of a Bunsen's burner. Independently of this preliminary heating, however, the subsequent heating of the tubes and their contents to 115°, 120°, 125°, or 130° C., would, in either case, have been deemed by Pasteur of itself to have been an adequate sterilisation. It was not for him to make cultivations of the organisms found. His object was to show that organisms were produced

under such and such conditions. Individually, he cared little about the nature of the organisms. Let the bacteriologists repeat the experiments, when the cultivation of the organisms might be much better undertaken by them.

IDIOPATHIC CYANOSIS DUE TO SULPH-HÆMOGLOBINÆMIA

(ENTEROGENOUS CYANOSIS)

BY

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AND

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For our knowledge of the peculiar forms of cyanosis with which this paper deals we are indebted almost entirely to the work of a few observers in Holland.

The first case was recorded by Stokvis, of Rotterdam, in 1902, under the title of "Autotoxic Enterogenous Cyanosis" (methæmoglobinæmia ?). He showed the presence of the methæmoglobin spectrum-band in the blood. He also found that the urine contained a substance which gave a red colour with hydrochloric and other acids, and transformed oxyhæmoglobin into methæmoglobin. He referred the condition to absorption of toxic substance, produced by putrefactive changes in the intestines.

Talma, of Utrecht, in the same year reported three cases and showed that the methæmoglobin was intracorpuscular and not, as in the case of drug poisoning, in the blood plasma.

Van de Bergh, of Rotterdam, in 1905, showed that Stokvis' substance in the urine was nitrites and a

decomposition-product of normal urine. He showed also that the band in his own patient's blood was not that of methæmoglobin, but corresponded with that of sulph-hæmoglobin. He referred to a simple means of distinguishing readily between the two by the addition to the blood of sulphide of ammonium.[1] This caused the immediate disappearance of the methæmoglobin band, but not that of sulph-hæmoglobin. He concluded that the condition was due to H^2S absorbed from the intestines, but he was unable to show the presence of H^2S in the blood.

He also isolated H^2S-forming organisms from the stools, but attached no value to it, as this could be easily done with normal stools.

In the same paper he also recorded a case of methæmoglobinæmia, and noted that this condition cleared up in twenty-four to forty-eight hours upon a milk diet, but returned in four hours after an ordinary meal.

In his next paper he reported that after an operation for stricture of the rectum the patient who was the subject of the first case recovered completely.

He also briefly described three other cases of sulph-hæmoglobinæmia, all associated with obstinate constipation and recovering completely when the constipation was cured. In two cases of methæmoglobinæmia also mentioned in the same paper he proved the presence of nitrites in the patient's blood.

Douglas and Gibson in July, 1906, recorded a case of methæmoglobinæmia, from whose blood they stated that they isolated the *B. coli communis*, and they entitled their paper "Microbic Cyanosis."

The following is the account of the present case:

Catherine S—, aged 37, unmarried, was admitted into the hospital for debility and cyanosis.

Although extremely feeble, she was able to walk into the ward. The most striking feature in the case was the colour of the skin, which was of a leaden hue, resembling

[1] This is an old test and is mentioned by MacMunn.

in tint that of silver staining, which made her look when asleep as if she was moribund. The cyanosis was universal, well marked in the face, cheeks, lips, ears, and nose, and under the finger- and toe-nails. The tongue too showed the purple colour well. In all parts it was easy to demonstrate that the colour was due to the blood and not to pigmentation, as pressure with the finger, or by bending the nails, showed the skin when emptied of blood to be of the normal yellow hue. The fingers were not clubbed. The pulse was regular, feeble, not rapid, and of low tension. The respirations were 20, regular, and quiet.

Physical examination revealed nothing. The cardiac dulness was normal. There were no cardiac murmurs. The lungs expanded well, were of normal size, and neither they nor the mediastinum presented any abnormal physical signs.

The abdomen was normal and the spleen not enlarged. The urine had a specific gravity of 1015, contained neither albumen nor sugar, and was of the ordinary colour. The appetite was fair and digestion good, the bowels habitually constipated, but kept open daily with medicine. The patient slept well.

The only objective sign about the patient, except her feebleness, was the cyanosis, and nothing had been discovered so far which would explain it. There was no sign of congenital morbus cordis nor of emphysema.

The absence of dyspnœa put out of court any pulmonary disease or mediastinal pressure.

A preliminary examination of the blood in the wards showed the number of red cells to be 4,800,000, of the white 3800, while the hæmoglobin was 90 per cent. The case was therefore not one of polycythæmia, which was on general grounds unlikely because of the patient's pallor.

To confirm the results another examination was made a few days later in the pathological laboratory by Dr. Andrewes, who reported the red blood-cells to number 5,050,000, white cells 3400, and the hæmoglobin per-

centage to be 115. The differential count was normal, viz. polymorphonuclear 72 per cent., lymphocytes 24 per cent., large mononuclear 4 per cent., eosinophile none.

The red cells were normal in size and shape, and there were no nucleated red cells to be seen.

For a spectroscopic examination the patient was taken into the physiological laboratory, and there, with the help of a strong arc light, a spectrum was obtained by Dr. Edkins through the web of the thumb. This showed what appeared to be the spectrum of methæmoglobin.

Subsequent examination by Dr. Langdon Brown confirmed this, and the spectrum was easily demonstrated in the blood placed in a microscope cell, so as to obtain films of varying thickness, the two oxyhæmoglobin bands in the green being easily seen in thin films and the band in the red being best seen in a thicker one. By regulating the thickness of the film the three bands could be easily demonstrated in the same specimen.

The presence of methæmoglobin in the blood led to a more careful examination of the urine. Its colour, as stated, was not abnormal, and Dr. Hurtley and Dr. Garrod pronounced the methæmoglobin spectrum completely absent from it.

Some blood was now drawn from the patient and centrifugalised, so as to separate the serum and cells. Spectroscopically the serum showed no trace of methæmoglobin bands, but they were distinct in the portion containing the red cells.

So far, then, the case appeared to be one of intracorpuscular methæmoglobinæmia.

Most of the cases of methæmoglobinæmia recorded have occurred as the result of the action of certain drugs—chlorate of potassium, nitrite of amyl, permanganate of potassium, phenacetin, antipyrin sulphonal, veronal, trional, and others.

Consequently the sisters and nurses were ordered to watch the patient and her friends closely, but nothing

was discovered. The patient herself denied absolutely that she had ever taken any medicines except what the doctor had prescribed, and the doctor stated that except for occasional doses of bromide and iodides he had given nothing for a long time more than a tonic of iron with arsenic.

As the patient lived in an isolated part of the country, from which the nearest chemist's shop was some miles away, drugs must have been sent by post if they were to reach her privately. No evidence, however, of the frequent receipt of letters or packets by the patient could be obtained either from the family or from the postman. So that all evidence of the obtaining and taking of drugs was lacking. During her stay in the hospital she certainly had no such drugs, and her condition after some weeks was not materially changed. Drugs were therefore dismissed as the cause of the condition.

The following history of the case was furnished me by Dr. Steele, of Stokeferry:

The patient has been an invalid for ten years or more. She was very neurotic, and suffered chiefly from neuralgia and sleeplessness, with dyspepsia and constipation, and, as a consequence, she became anæmic. Her first illness began in July, 1904, when she had a curious nervous attack with delirium and severe pain in the left side of the chest. Previously to this attack she had had bromide of potassium and chloral (30 gr. of each) occasionally at night to relieve the pain in the chest and secure sleep. The attack came on suddenly without any apparent cause. There was no rise of temperature and no paralysis. She used to see objects (animals, etc.) in the room, and was at times semi-comatose, though she could always be roused. The attack was not alcoholic.

She had for a time hypodermic injections of morphia frequently to give sleep, but after a time the morphia lost effect and was given up. She was ill for about two months, and then gradually improved. When convalescent

from this attack she was in much the same condition of health as before it.

The present illness was of a different character altogether, and commenced in August, 1905. When she became cyanosed gradually and lost strength the cyanosis at first was observed only during exertion, but gradually became persistent. The weakness slowly increased until she was quite unable to exert herself at all. She became an in-patient for a time in the Lynn Hospital, where the diagnosis, she says, was made of thoracic aneurysm.

No drugs appeared to do any permanent good. Iron, in various forms, as well as iodides, bromides, and arsenic were all tried at different times. The only combination that seemed to benefit her at all was iron, digitalis, and strychnine.

In October, 1905, I saw her myself when I happened to be making a visit in Norfolk. The cyanosis was considerable. She was so feeble and ill that I did not like to disturb her by much examination, for she looked almost as if moribund. My visit was a casual and hurried one, and I was unable to make any diagnosis. I fully expected she would die shortly, and when Dr. Steele wrote to me a year later saying that she was still in exactly the same condition, and that he would like me to admit her into the hospital, my interest was greatly excited, and I gladly took her in.

Dr. Wood Clarke now undertook to make some further examination of the urine and blood, and succeeded in showing that what appeared to be the spectrum of methæmoglobin was not that of met- but of sulph-hæmoglobin, and that the case, therefore, was one of intra-corpuscular sulph-hæmoglobinæmia.

The following is Dr. Wood Clarke's account of his investigations:

The laboratory investigations were directed to the blood, the urine, and the fæces.

As cyanosis had been described in connection with two

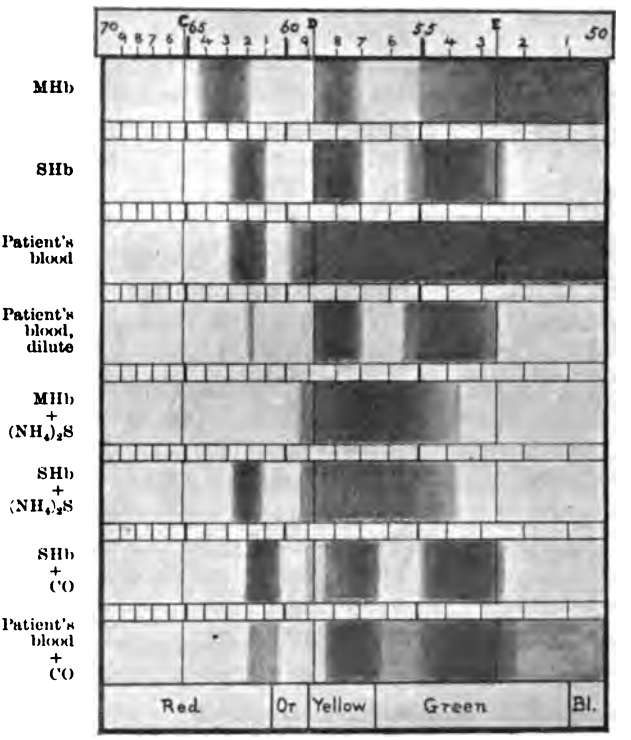
different forms of hæmoglobin—viz. met- and sulph-hæmoglobin—a preliminary study of them seemed desirable before starting upon investigations into the present case. Accordingly a preparation of methæmoglobin was made by treating a few c.c. of defibrinated ox-blood with potassium ferricyanide, and others of sulph-hæmoglobin by bubbling H_2S through portions of blood for a time or by adding a weak, watery solution of H_2S directly to them. Accurate spectroscopic examination of each was now made. Each showed a dark band in the red, but their positions were different. The methæmoglobin band extended from λ 620 to λ 645 on the scale, shading off to λ 615 on the one side to λ 650 on the other. The sulph-hæmoglobin band extended from λ 610 to λ 625 and in concentrated solution to λ 630.

On December 22nd and again on December 29th specimens of the patient's blood were obtained—on the first day from the finger-tip and on the second from a vein in the arm. A few drops were diluted with water and filtered. Each gave a definite band in the red, ranging in the one case from λ 613 to λ 629, in the other from λ 612 to λ 628. In both cases the band was faint, so that it was necessary to use a solution of such strength that the oxyhæmoglobin bands were blotted out. This made it clear that the patient's blood contained, not met-, but sulph-hæmoglobin. This was confirmed by the test referred to by van der Bergh, which consists in the addition to the blood of a little ammonium sulphide; in the case of methæmoglobin the band in the red disappears immediately, while with sulph-hæmoglobin it persists, and is, indeed, somewhat intensified; in both cases the oxyhæmoglobin bands slowly merge to form the band of reduced hæmoglobin.

During the course of the investigation it was noticed that the passage of CO through sulph-hæmoglobin solutions, provided the gas was free from acids, caused a definite change in the spectrum, all the bands being shifted slightly towards the violet end of the spectrum.

The two oxyhæmoglobin bands are replaced by those of carbon-monoxide hæmoglobin, while the band in the red changed from λ 610–λ 625 to λ 605–λ 620. This test was applied to the patient's blood, and the typical change in the bands was noted. The passage of CO through

CHART.



methæmoglobin causes no change in the spectrum. Further investigations upon this property of sulph-hæmoglobin are at present being carried on in this laboratory.

An attempt was now made to ascertain if there were any free H^2S in the patient's blood. For this purpose a

modification of the method used by van der Bergh was employed.

Two wash-bottles were so connected with a suction pump that air could be passed through the solution they contained in series. In the first bottle was placed the solution to be tested for H^2S, in the second some distilled water containing ammonia. After air had been drawn through the solution for ten minutes the ammonia solution was tested with sodium nitro-prusside. By this means as little as 0·0002 grm. of H^2S could be detected with ease from a watery solution, and it was found that in the ten minutes all the H^2S in the water in the first bottle had been carried over into the second.

5 c.c. of ox blood were now taken; to it were added 0·0002 gr. of H^2S and the test repeated. The presence of free H^2S was easily proved, but the spectroscope showed no sulph-hæmoglobin band.

To another 5 c.c. of blood a much stronger solution of H^2S was added; the blood then showed the typical band and continued to do so after all free H^2S had been drawn off. This band was still distinct after the blood had been exposed (*a*) for eighteen hours to the air and (*b*) for half an hour to CO.

Some blood was now treated with H^2S and kept for some hours in a stoppered bottle. From this no H^2S could be obtained.

Lastly, it was found that on the addition of a strong solution of H^2S to the blood the sulph-hæmoglobin band did not appear immediately, but only after standing for five minutes.

The observations show that the combination of H^2S with hæmoglobin occurs rather slowly, that before the combination has taken place the free H^2S can be removed by a stream of air, but not afterwards. This test tried on the patient's blood gave negative results, from which it may be inferred that if there was any free H^2S at all in the blood, it must have been in extremely minute quantities.

It now seemed desirable to obtain some idea as to the quantity of H^2S required in order to form sulph-hæmoglobin in sufficient quantity to be detected by the spectroscope.

A fresh aqueous solution of H^2S was therefore titrated and found to contain 2·856 grm. per litre; 10 c.c. of this solution was repeatedly diluted by halving and replacing with water until a series of solutions was obtained, which represented from 0·01428 to 0·00003 gr. per 5 cm. 10 c.c. of blood were added to each tube, the mixture shaken and divided into two equal portions; one set of tubes was placed in the incubator at 37° and the other left in the cold room. After twenty-four hours the specimens of blood were examined with the spectroscope. It was then found that in the blood left in the cold room the band was not visible in the tubes of the fifth dilution, which represented 0·0009 H^2S, while it was clear, though faint, up to the last dilution in the tubes which had been placed in the incubator, representing a dilution of $\frac{1}{512}$ or less than ·00003 H^2S in the 10 c.c. of mixed blood and water.

From these experiments it will be seen that the amount of H^2S required to cause an appreciable change in the blood-pigment at body temperature is far below that which can be detected by any chemical means, and consequently that it is no wonder it cannot be demonstrated in the patient's blood.

Blood-culture.—Blood was taken from a vein in the patient's arm by Dr. Elmslie on December 29th; with this various media were inoculated but no organism grew.

The urine.—The urine was carefully examined on several occasions. There was never either albumen or sugar present. Indol, by the colorimetric methods of Jaffé and Bouma, was decreased. Ferric chloride gave no reaction. The urine was acid when passed, and gave a negative reaction with HCl. Nitrites were present as an extremely faint trace when the urine was acid, and in considerable amounts when alkaline. The ethereal sul-

phates were greatly reduced below the normal, the ratio between these and the mineral sulphates on three separate days being 1 : 24, 1 : 35, and 1 : 26. The total sulphates were normal in quantity. An amyl alcohol extract after the addition of acetic acid showed that there was no increase of hæmatoporphyrin, and only the faintest possible band of urobilin. No blood-pigments were present. Benzoylation of a litre of fresh urine gave no precipitate.

The stool.—On December 29th the patient was placed on the Adolf Schmidt test diet for examination of the stools. At this time the constipation suddenly ceased, and under the influence of magnesium sulphate the stools became copious and watery. It seemed that during this period the cyanosis became less marked.

The stool obtained, which probably also contained urine, was large and fluid, and had a decidedly offensive odour. It was slightly alkaline in reaction. On being rubbed in a mortar and examined on a black plate a few small shreds of connective tissue and mucus were found. Meat-digestion was complete. On treating with Lugol's solution no undigested starch granules were to be found. The addition of 30 per cent. acetic acid and heat demonstrated that there was no excess of fat in the stools. No fatty acids were seen. A few grammes of stool placed in a Strassburger fermentation apparatus and left in an incubator at 37° C. did not develop an appreciable amount of gas in forty-eight hours. At the end of this time the reaction was about as strongly alkaline as at the beginning. Testing the stool after forty-eight hours with ammonia and sodium nitro-prusside only a slight pink colour was obtained, while the water in the collecting tube of the apparatus was quite free from H_2S.

A second specimen of stools, obtained on January 5th, was soft, dark green in colour, and of unpleasant odour. Microscopic examination showed the same findings as before except for a few undigested starch granules. A faint trace of H_2S was proved after fermentation.

In the attempt to determine whether there were an increase in the sulphuretted hydrogen-forming organisms in the patient's intestines the following method was used at the suggestion of Dr. Andrewes, and under his direction: One gramme of solid stool was weighed in a sterile watch-glass, thoroughly ground up in a sterile mortar, and washed into a flask containing 99 c.c. of sterile water. This was then diluted decimally through ten flasks and 1 c.c. of the fluid in each was added to a bouillon tube containing 0·5 per cent. lead acetate. The cultures were grown at 37° C. for forty-eight hours anaerobically and forty-eight hours aerobically. With our patient's stool blackening from lead sulphide appeared up to a dilution of 1 : 10,000,000—*i. e.* 1 c.c. of the fluid contained 0·0000001 grm. of stool, while with four controls taken from other patients on the same diet and drugs it varied from 1 : 10,000 to 1 : 1,000,000,000. The experiments were too few to be conclusive, but they suggest that in this patient there was no increase of H_2S-forming organisms in the intestines at the time of the examination.

The accompanying table gives an analysis of thirteen cases, eight of methæmoglobinæmia and five of sulph-hæmoglobinæmia. In the first four cases recorded of methæmoglobinæmia the exact nature of the condition is a little uncertain, as at that time the distinction between met- and sulph-hæmoglobinæmia was not recognised.

Taking the two groups together, we find that eight of the patients were men and five women, but of the five sulph-hæmoglobinæmic cases four were women and only one a man. All the cases were adults except two, a boy aged 9 and a girl aged 17. With these exceptions the ages ranged from 29 to 67.

The chief symptoms beside the cyanosis were headache and general weakness. The weakness was often extreme, and in two cases was described as a "feeling of paralysis

of the arms and legs." The cyanosis was general, but especially conspicuous in the mucous membranes.

There were no respiratory or cardiac symptoms other than the weakness would account for.

Clubbing of the fingers was observed in two cases. The symptoms were usually of gradual onset, but occasionally developed very suddenly.

The duration of the cases varied from six months to eight years. Of six cases in which the result is recorded five got well and one died. In the fatal case the only lesions discovered at the autopsy were ulcerative enteritis and chronic parenchymatous nephritis.

The blood-count is given in five cases. In four the red cells were 5,000,000 or slightly more, in one 3,360,000, so that in spite of the pallor there is no real anæmia; the white cells 7900 in two cases, 10,000 in one, 20,000 in one, and 3400 in another.

The hæmoglobin percentage was given in four cases as 90, 96, 70, and 115.

The differential blood-count in one case was normal.

The urine showed the presence of albumen in three cases as a trace only except in the fatal case, in which chronic parenchymatous nephritis was found post mortem. Indol was in excess in four cases and in defect in two cases. The etherial sulphates were increased in one case and diminished in one case. The pigments were not abnormal either in amount or nature.

Contrasting the two groups, it would appear that the methæmoglobin cases were associated with diarrhœa, and improved greatly on a milk diet, while the sulph-hæmoglobin cases were associated with constipation and did not improve till this was relieved.

In van der Bergh's first case of sulph-hæmoglobinæmia, that of a boy, aged 9, the constipation depended on a

Case.	Author.	Sex.	Age.	Symptoms.	Duration of cyanosis.	Bowels.	Stools.
1	Stokvis	M.	33	Diarrhœa 6 years; sudden collapse, cyanosis, clubbed fingers, heart and lungs normal	4 years	D.	Fluid, acid, odorous, flagellata
2	Talma	M.	41	Headache, dizziness, cyanosis, œdema, systolic murmur at apex, palpitation and dyspnœa, diarrhœa	1 year	D.	Fluid, odorous
3	Talma	M.	29	Dysentery, cyanosis, swelling of glands, heart enlarged, sounds weak	—	D.	—
4	Talma	M.	30	Headache, weakness, cyanosis, backache, glands enlarged	¼ year	R. and D.	—
5	Von der Bergh	M.	9	Cyanosis, rectal stricture, urethro-rectal fistula, tympanitis, clubbed fingers and toes, heart and lungs normal	2 years	Retention	Fluid, alkaline, stinking
6	Von der Bergh	M.	25	Sudden weakness, œdema, cyanosis, heart and lungs normal	7 years	D. and R.	Normal
7	Von der Bergh and Gutterink	F	67	Exudative pleuritis, obstinate constipation	—	C.	—
8	Von der Bergh and Gutterink	F	60	Post-apoplectic hemiplegia, constipation	—	C.	—
9	Von der Bergh and Gutterink	F.	17	Retro-peritoneal tuberculosis, constipation	—	C.	—
10	Von der Bergh and Gutterink	M.	51	Diarrhœa 4 years, headache, feeling of paralysis, collapse	4 years	D.	Fluid, odorous, acid
11	Von der Bergh and Gutterink	M.	31	Dysentery, headache, feeling of paralysis of legs and arms, cyanosis, heart and lungs normal	8 years	D.	Fluid, odour of butyric acid, anguillula stercoralis
12	Gibson and Douglas	F.	36	Weakness, headaches, cyanosis, diarrhœa	3 years	D.	Offensive nitrites
13	West and Clarke	F.	37	Headache, weakness, cyanosis, constipation	1½ years	C.	Offensive

[1] The cases 1, 2, 3, and 4 are entered as MHb cases, though the fact that that they were the latter condition.

Blood.						Urine.				Result.	Remarks.
Red cells.	White cells.	Per cent. Hb.	Spectrum.	Nitrites.	Cultures.	Albumen.	Indol.	Etherial sulphates.			
—	—	—	MHb[1]	—	—	+	—	+ +	Red with acids	Died	*Autopsy.* — Ulcerative enteritis, chronic parietal nephritis.
5,000,000	7900	90	MHb[1]	—	—	—	+ +	—	—	Unimproved	—
—	—	—	MHb[1]	—	—	—	+ +	—	—	Cured	—
—	—	—	MHb[1]	—	—	—	+ +	—	—	—	—
5,200,000	20,000	—	SHb	—	—	—	+ -	—	—	Cured	Operation on rectal stricture.
—	—	—	MHb	—	—	+	—	—	—	—	Condition cleared up temporarily on milk diet.
—	—	—	SBb	—	—	—	—	—	—	Cured	Cases 7, 8, and 9 cleared up as soon as the bowels were kept open.
—	—	—	SHb	—	—	—	—	—	—	Cured	
—	—	—	SHb	—	—	—	—	—	—	Cured	
5,200,000	7900	96	MHb	+	—	+	—	—	—	—	Condition cleared up temporarily on milk diet.
—	—	—	MHb	+	—	—	— —	—	—	—	Ditto.
3,360,000	10,000	70	MHb	+	*B. coli com.*	Trace	—	—	—	—	Previous history of the use of aniline drug.
5,050,000	3400	115	SHb	—	Sterile	—	— —	— —	—	—	—

sulph-hæmoglobinæmia had at that time not been described makes it possible

rectal stricture which resulted from a operation for imperforate anus. A second operation which relieved the stricture and constipation was followed by recovery.

A Summary of Previous Investigations.

The first case recorded by Stokvis was that of a soldier who had been cyanotic for five years, the condition being associated with chronic diarrhœa. The patient died, but the autopsy showed only ulcerative enteritis and chronic parenchymatous nephritis. Spectroscopic examination of the patient's hand and tongue showed distinctly the band of methæmoglobin. As the action of drugs could be excluded, Stokvis concluded that the condition was due to intestinal absorption, and he turned his attention to the urine as the most likely source of information as to the etiology of the methæmoglobin-forming substance.

On adding 1 c.c. of defibrinated ox blood to 25 c.c. of the patient's urine he was able to obtain methæmoglobin in ten minutes. He further found that the addition of hydrochloric, phosphoric, sulphuric, and nitric acids to the urine produced a deep red colour. These two reactions occurred only when the urine was alkaline. An examination of the etherial and mineral sulphates showed that the former were greatly increased over the normal, the average ratio being 1 : 2·79. The amount of indican was in excess. From these findings he concluded that the substance in the urine which gave the red colour with acids was a product of intestinal putrefaction, and was the toxic material which caused the change of oxyhæmoglobin to methæmoglobin in the blood. Repeated efforts to isolate this substance failed completely.

Talma's three cases of methæmoglobinæmic cyanosis were associated with long-standing diarrhœa. He added to our knowledge of the condition by proving that the methæmoglobin was in the blood-corpuscles, and not in the plasma, as is usually the case in drug-poisoning. As in the urine of all his cases he found an increase of indol

and skotol he accepted Stokvis' opinion as to the cause of the disease.

By Hijmans van der Bergh's researches considerable light was thrown on the subject. In his first paper he reports two cases. The first was a boy, aged 9, who suffered from a rectal stricture and urethro-rectal fistula following on early operation for imperforate anus. After proving that the altered blood-pigment was intra-corpuscular, he also turned his attention to the urine. Referring to recent work of Steensma and older work of Schönbein and Röhmann, he showed that normal urine in the process of putrefaction gives off nitrites—powerful methæmoglobin-forming substances—and is turned red by the addition of strong hydrochloric acid, and that, furthermore, the addition of a small amount of sodium nitrite to a normal acid urine gives it the property of being turned red by acids. He therefore concluded that the substance which Stokvis put forward as the cause of the disease was nitrites, and as a normal constituent of putrefying urine could not be considered of pathological significance. Van der Bergh next turned his attention to the stools, and found that 1 c.c. added to 10 c.c. of oxyhæmoglobin solution produced a band in the red end of the spectrum in from ten to twelve hours. He was, however, struck by the fact that this band was not quite the same as that formed by the action of the patient's urine on ox-blood. Careful comparisons enabled him to ascertain that he was dealing with two definite spectra; the one showing a band in the red, the middle of which represented a wave length of λ 631·3 was given either by a known solution of methæmoglobin or by the action of putrefying urine on ox-blood, and the other, with a band centred at λ 617·8 or definitely more towards the blue end of the spectrum, was produced by the action of the patient's stool on ox-blood. The latter coincided exactly with the spectrum obtained from the blood of the patient. He, therefore, concluded that the substance in the patient's blood which gave the dark

band in the red end of the spectrum was not methæmoglobin but some new substance, and he set about to discover what this might be. Having tried all known drugs which act on oxyhæmoglobin, he came to the conclusion that sulphuretted hydrogen was the only substance which could act on the blood so as to produce the spectrum which he had found in the blood of his patient. He, therefore, decided that the foreign substance in the blood was the so-called sulph-hæmoglobin studied by Hoppe-Seyler and Harnack, and he called his case "sulph-hæmoglobinæmia." Attempts to prove the presence of sulphuretted hydrogen in the blood of his case by chemical methods failed, but the author was able to show that, if ammonium sulphide were added to methæmoglobin, the two bands of oxyhæmoglobin were changed to the broad band of reduced hæmoglobin, and the band in the red immediately disappeared, while in the case of sulph-hæmoglobin, although the oxyhæmoglobin was reduced, the band in the red remained unaltered. He also isolated sulphuretted hydrogen-forming organisms from the stools, and by inoculation of ox blood produced sulph-hæmoglobin. As this can also be done in normal stools it was not considered of great importance.

In van der Bergh's second case, which proved to be methæmoglobinæmia, he added but little to our knowledge of the condition, except to find that on a strict milk diet the condition clears up in forty-eight hours, to return again within four hours after a full meal. In his second article, published in conjunction with A. Gutterink, he states that his first sulph-hæmoglobinæmia case recovered completely after an operation on the rectum which enabled a proper evacuation of the bowels. He also refers to three more cases of sulph-hæmoglobinæmia associated with obstinate constipation, in which the cyanosis and spectroscopic picture cleared up when the bowels were kept open. Two more cases of chronic diarrhœa showing the methæmoglobin band enabled the authors to prove the presence of nitrites in the patient's blood.

In Gibson and Douglas's case of methæmoglobinæmic cyanosis two loopfuls of diluted blood were inoculated into nutrient agar and the *B. coli communis* grown. These findings were not confirmed, and the authors expressly state that they should not be taken as final. That they themselves put some weight upon them is shown by the title they gave to their article—viz. "Microbic Cyanosis."

General Remarks and Conclusions.

A review of the facts observed in connection with this case show that the cyanosis is due to a change in the blood-pigment, which in every way corresponds to the sulph-hæmoglobin of Hoppe-Seyler and Harnack. The case therefore belongs to the class described by von der Bergh as sulph-hæmoglobinæmia.

The causes of this condition are far from clear. In this case a specific microbic infection of the blood seems hardly probable from the duration of the illness and the negative results of the blood-culture. Increased formation of sulphuretted hydrogen in the intestines seems not likely to be the sole cause, considering the frequency with which this gas occurs in the normal intestine. In this case in the stools, after forty-eight hours' incubation, not enough H_2S gas was generated to show more than a trace in the fermentation apparatus, and the urinary findings usually associated with intestinal putrefaction were lacking. It is possible that the cause may lie more in the intestinal walls than in their contents, and that the error may be a hyperabsorption of H_2S rather than an hyperformation. Still another hypothesis which should be mentioned on purely theoretical grounds is brought to our minds by the old work of Claude Bernard and Hoppe-Seyler and the more recent investigations of Harnack. Bernard found that sulphuretted hydrogen given to animals intravenously was much less toxic than when given by the arteries, and the two latter authors have shown that H_2S will not combine with blood

saturated with CO_2, that CO_2 acts so as to protect the blood from the attack of the H_2S. It may therefore be possible that the defect is one which prevents the H_2S from being excreted by the lungs and enables it to pass over from the venous blood, where, owing to the excess of CO_2, it is a more or less inert substance, and to enter the arterial system, where, finding oxyhæmoglobin with no protecting CO_2, it can attack it with toxic force.

Some authors hold that what has been described as sulph-hæmoglobin is not a definite chemical compound formed by the combination of sulphur and hæmoglobin, but some intermediate product of the destruction of oxy-hæmoglobin by sulphuretted hydrogen. Whether this be so or not is immaterial, from a clinical point of view, for the occurrence of sulph-hæmoglobin in the patient must be taken, as far as we at present know, as evidence of the action of H_2S upon her blood. The point of chief clinical interest, then, becomes the source of the sulphuretted hydrogen, and, if in the intestines, the explanation of its toxicity to one patient and not to another. On this question we have been able to throw no light, but we trust that as the condition becomes more widely recognised further investigation will solve the problem.

The chemical investigations have been carried out in the Laboratory of Chemical Pathology at St. Bartholomew's Hospital, and we express our sincere thanks to Dr. Garrod and to Dr. Hurtley for their co-operation and help, as well as to Dr. Andrewes, Dr. Simpson, and many others who have shown their interest in the subject by their willing assistance.

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DISCUSSION

Dr. A. E. GARROD thought it would be interesting to know whether the cyanosis was due to deficiency of oxygen-carrying power of the hæmoglobin or to a changed colour of the blood.

Dr. WEST, in reply, said that this point, owing to circumstances, could not be determined. As compared with the polycythæmia cases, the patient showed extreme pallor, but a blood-count did not show any anæmia.

ON THE CONDITION OF THE BLOOD-VESSELS DURING SHOCK

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I WISH again to bring forward the question of the conditon of the blood-vessels during shock because of its great practical importance, and because of the divergent views which have been advanced on the subject.

In 1893 I urged that the theory then taught was not in accord with the conditions observed at the bedside and in the operating theatre. My dissatisfaction with the teaching of that day seems now to be amply justified, for the views recently advanced by Dr. Geo. W. Crile have obtained a wide acceptance, and his theory differs much more than mine does from that formerly regarded as orthodox. It was taught for more than fifty years, and most text-books still state that shock is caused by a paralysis of the vessels in the splanchnic area. Dr. Crile wrote that "in shock the essential phenomenon is a diminution of the blood pressure;"[1] and he argued that the cause of this fall of pressure "must be an exhaustion of the cardiac muscle, of the cardiac centres, of the blood-vessels, or of the vaso-motor centre."[2] He arrived

[1] 'Blood Pressure in Surgery,' p. 401.

[2] Ibid.

at the conclusion that it was due to "an exhaustion or breakdown of the vaso-motor centre."

He recorded a large amount of experimental work which he interpreted according to his theory, but it seems to me that he altogether failed to give a satisfactory explanation of several important facts which were put forward by himself or which are generally recognised.

For example, if it is assumed that a fall of blood pressure in the carotid is necessarily accompanied by a relaxation of the arteries generally, it follows that the blood pressure must at the same time be raised in the smaller arteries and in the capillaries, or, on the other hand, the teaching that the arterioles, by their contractions and relaxations, exercise a regulating power over the blood pressure—a stopcock action as it has been called—must be set aside as physiologically inexact.

It is quite certain that neither the capillaries nor the small vessels are distended during shock, and no evidence has been put forward to explain why it is that their distension does not occur as a consequence of the paralysis of the muscular coats of the latter which is said to exist.

Again, Dr. Crile stated that the first action of alcohol and other vaso-dilators administered during shock was to increase the volume of the radial pulse, but no explanation was offered as to how vaso-dilators increased the volume of the blood in an artery which was declared to be already paralysed by an exhaustion of its nervous supply.

Dr. Crile also asserted that when an animal was in a state of profound shock he could not, by introducing fluid into its veins, raise and sustain the blood pressure beyond a certain time even during the infusion.[1] The solution escaped from the vessels at a rate fairly proportional to the rate of infusion, and he offered no explanation of this. It is certain, however, that the vessels are

[1] Ibid., p. 269.

not generally dilated, and they should therefore be capable of accommodating a very large quantity of saline solution if their nerve-supply is paralysed.

Another point of importance is that Dr. Crile found that the blood pressure in the portal vein was raised during shock. If it be a good argument that because the blood pressure in the carotids is lowered therefore the vascular system must be relaxed, it would seem also to hold good that the rise of blood pressure in the portal vein indicates a contraction of the vessels in the portal system.

According to this view, there would be a complete reversal of the old theory that the vessels of the portal area were relaxed during shock, and it would be necessary to believe that an area in the interior of the body has its vessels contracted, whilst elsewhere the arteries are paralysed. But if the pressure in the portal vein is raised, and if the arteries generally are paralysed, it is exceedingly difficult to understand why the blood does not flow freely into the paralysed systemic vessels, in accordance with the universal law that moving fluids flow in the direction of least resistance. In this way the raised pressure in the portal area should rapidly be reduced, but we are told that the portal rise of pressure develops simultaneously with the peripheral paralysis of vessels. This combination of phenomena seems to me mechanically impossible.

It has also been shown by Cobbett and Roy, Sherrington and Monckton Copeman, that the blood of an animal in a state of shock has its specific gravity raised. An explanation as to how this can occur, when the vasomotor system is exhausted, has not been given. If the vessels are paralysed, their tendency should be to dilate, and serous fluids under these circumstances should pass from the tissues into the vessels. As a consequence of this, the specific gravity of the blood would be lowered, because the serum of the blood has a lower specific gravity than that of the blood as a whole.

Shortly then, before the view that the vessels generally are paralysed during shock can be accepted, an explanation should be given:

First, as to why the blood does not flow freely into and distend the capillaries, which assuredly it does not.

Second, as to how the first effect of vaso-dilators can be an increase in the volume of blood in an already paralysed vessel, as is asserted by Dr. Crile.

Third, as to why the vessels which are said to be relaxed, but are evidently not dilated, will not accommodate saline fluids, as is asserted by Dr. Crile.

Fourth, as to how the blood pressure is raised in one area, as Dr. Crile asserts; and

Fifth, as to how the specific gravity of the blood becomes raised if the pressure on it is lowered.

A theory which gives rise to so many and such important difficulties should be investigated very critically, and it seems to me that a careful examination of the argument shows that a link in the chain of evidence is altogether wanting.

Dr. Crile asserted that the lowered blood pressure in the carotid "must" be due to a cardiac or vascular loss of power. Apparently it did not occur to him that a contraction of the vessels might cause or accompany a lowering of blood pressure. His omission to discuss this part of the question is the more remarkable because, with reference to animals reduced to such a degree of shock that "the vaso-motor mechanism was wholly inactive, except in response to most heroic stimulation," he stated that "a number of extensive dissections were made to observe the condition of the vessels at this stage. The arteries were quite empty, the tissues pale, but the venous trunks everywhere were full, alike in the somatic and in the splanchnic areas. There was a manifest transference of the greater bulk of the blood from the arteries and capillaries to the veins" ('Surgical Shock,' by Dr. Geo. W. Crile, 1899, p. 140). This observation is not inconsistent with the theory that certain veins may

be paralysed during shock. It is much more in harmony with the views I advanced that the vascular system is in a state of tonic contraction during shock, and that therefore the more powerful smaller arteries force the blood into the vessels which yield most easily, namely, the venous trunks and large arteries in the central, warmer parts of the body. The observation seems to me diametrically opposed to what we would expect if the vaso-motor centres were paralysed. Dr. Crile should at least explain the relationship of this condition of the arteries to his theory. Yet in his argument he altogether overlooked the possibility that a lowered blood pressure might be caused by a tonic contraction of the vessels, and also the fact that by his own showing "the arteries were quite empty." So completely did he ignore this aspect of the condition of shock, that I have not found that he offered any proof whatever, either argumentative or experimental, of the statement above quoted, that the fall of blood pressure *must* be due to a relaxation of some part of the vascular system.

Moreover, he was able to fix on paralysis of the vaso-motor centres as the cause of the lowered blood pressure only by a process of exclusion. He showed that the fall of pressure was not due to a paralysis of the heart muscle, of the cardiac centres, or of the muscle of the vessel walls; therefore, he argued, the vaso-motor centres must be paralysed. But he put forward no evidence whatever that they were paralysed during shock, and there are conclusive reasons for excluding paralysis of the vaso-motor centres also from the causes of the fall of blood pressure during shock, for Lord Lister has recorded that he never failed to obtain a contraction of vessels by stimulating the brain and spinal cord of the frog until both were completely destroyed.[1] This observation proves that an injury cannot paralyse the vaso-motor centres except by destroying them, and therefore that surgical

[1] 'Phil. Trans.,' vol. clxviii.

shock cannot be due to a paralysis of the vaso-motor centres.

I therefore venture to maintain that Dr. Crile has not made good his contention that a lowering of blood pressure in the carotids must depend on a relaxation of some part of the vascular system, and I would further argue that it is not an universal law that a lowered blood pressure in the carotid is necessarily caused by a relaxation of arteries generally or even of the systemic vessels.

The ordinary mechanics of moving fluids do not support

Dr. Crile's view. The blood stream may be compared to that of a river with many channels. If we assume that the accompanying figure represents diagrammatically a river with a long island in its middle, and therefore with two courses approximately of equal size, there can be no doubt that if the channel (A.B.) were filled in so that a mere trickle of water passed along it, the pressure of the water in this channel would be reduced, and by filling in the channel altogether the pressure of water would, of course, be completely removed. At the same time, the flow on the other side of the island would be increased in volume and in force, it being assumed that the water could not overflow the banks. During the process of gradually filling in and contracting the bed of the channel

(A.B.), lateral pressure would, of course, be applied to the flowing water, and there might be very great difficulty in filling in the water course; but the result, except perhaps just at first, would not be an increase of the pressure of the water against the banks of the contracted channel. On the contrary, in it the pressure of water would inevitably (after a variable time) be lowered, and the process of filling in the channel would become progressively more easy. The rapidity and ease with which the flow of water could be transferred to the other channel might vary, but under all circumstances, if it were possible to fill in one channel, the water would be forced into the other, and, if the process were carried sufficiently far, the increase of pressure would be in the overfilled, and *not* in the contracted channel. In the latter the pressure would be reduced, and finally would cease to exist.

If the water courses on each side of the island were rubber tubes, the effects of compression of one and of dilatation of the other would be exactly the same as in the water stream, so far as the incidence of outward and onward pressure exerted by their flowing contents would be concerned.

So when a vessel or a series of vessels in the human body is contracted, the blood-pressure in the affected area is lowered, as a rule, whilst the flow becomes more free, and the pressure rises in some other part or parts of the vascular system.

There is a well-recognised law that, by a stopcock action, a contraction of the arteries causes a rise of blood pressure, and their relaxation is accompanied by a fall. But this is only a partial statement, and the law, when fully stated, does not support Dr. Crile's theory. The essential point in this stopcock mechanism is that the contraction raises the pressure in the large vessels, whilst at the same time it lowers the pressure in the small ones beyond the point where the stopcock is supposed to act. A relaxation of arterial tension has, of course, converse effects.

Hence, it is quite as true to assert that a relaxation of the vessels raises the blood pressure (in the smaller arteries) as that it lowers the pressure (in the larger arteries).

Dr. Crile's whole argument depends on the idea that a relaxation of the arteries must cause a fall of blood pressure. But it is certainly more correct to say that, in individual vessels, a lowering of the blood pressure is accompanied by a contraction of their walls than that it is accompanied by a relaxation. When the blood pressure in the aorta is lowered that vessel contracts on account of its elasticity, and when the pressure in it is raised the vessel is dilated. In this case contraction accompanies a fall of blood pressure. Again, when small arteries contract, the volume and pressure of blood within them are reduced, and the lumen may even be occluded so that the blood pressure is completely removed. When these small vessels relax, a fuller blood stream and a higher blood pressure ensue in them. Thus in these vessels also contraction accompanies a fall of pressure.

During digestion the fuller blood flow in the splanchnic area is associated with a dilatation of the vessels of that area, and an increased blood pressure in them, whilst the lowered blood pressure in the systemic vessels is associated with a contraction of the superficial vessels, and a lessened flow of blood to the surface.

Thus in the large and in the small vessels, and in almost all conditions, a lowering of blood pressure in any particular artery or vascular area is accompanied by a contraction and not by a relaxation of the vessels concerned.

It is only when no vascular area will yield or when long continued irritation causes chronic hypertrophy of the vascular tissues throughout the body, that a general rise of blood pressure is produced by vascular contractions. But the artery in which there is a chronic high tension is not necessarily a contracted vessel.

Thus a consideration of the subject shows that it may

be argued that Dr. Crile has not sufficiently substantiated his assertion that the cause of a fall of blood pressure in the carotid "must be an exhaustion of the cardiac muscle, of the cardiac centres, of the blood-vessels, or of the vaso-motor centres." But his thesis depends upon the accuracy of this statement.

A fall of blood pressure in the carotid *may* be due to a relaxation of some part of the vascular system or of the vessels generally, but no evidence has been brought forward sufficient to prove that the fall of blood-pressure, which in shock affects both the large and the small arteries is associated with a relaxation of these vessels. On the contrary, the evidence seems to me to prove that a lowered blood pressure *throughout* the systemic arteries *must* be due to a contraction of these arteries, a contraction which Dr. Crile found actually to exist. This view affords the best explanation of the vascular phenomena of shock. All observers agree that the first effect of a stimulation of a sensory nerve is a contraction of the small arteries, and as I read the evidence the conclusion is inevitable that in shock there is an extreme development of this contraction which extends to the medium-sized and to many of the large vessels. The narrowing process progresses until it may be difficult to find a pulse anywhere. I have never seen any indication that the intense contraction changes to a relaxation unless the state of shock tends to pass off. Lord Lister's description of the effects of stimulation of the nerve-centres negatives the possibility of the occurrence of an exhaustion of their energy except by their destruction, and Dr. Crile has not put forward any evidence that a relaxation occurs other than the fact that the blood pressure in the carotid is lowered, which, as I have shown, does not prove his point.

If there is an intense and extensive contraction of the arteries during shock the question arises as to where the blood goes. Undoubtedly some part of the vascular system must relax or the bulk of the blood must be re-

duced. That the latter happens I have no doubt, but it cannot take place quickly to any great extent. Hence some part of the vascular system must relax. The blood is not found in the arterioles of the central parts of the body. On the other hand, I am convinced from clinical observation, and I shall give a good reason for mentioning the mere conviction, that the larger internal vessels spout more forcibly if divided when the patient approaches a condition of shock than they do if the operation is not severe. I have noted this in the cystic branch of the hepatic artery, in the renal arteries, and in the larger mesenteric arteries, and I believe that it is due to the fact that these large central vessels are unusually full.

I venture to put forward this belief which is founded on clinical observations, and not on exact measurements, because Dr. Crile has recorded, as a fact, that the pressure in the portal vein is raised during shock. I have already pointed out that the explanation of this is difficult—I think it impossible—if the arteries generally are believed to be paralysed. But a rise of blood-pressure in an internal area is fully accounted for if the vessels throughout the body tend to contract, and if the superficial vessels are more tensely contracted than the central ones. A difference between the superficial and the central areas in this respect ought to exist, for a low temperature acts powerfully in contracting the vessels, and the superficial temperature falls markedly in the state of shock. Thus the evidence shows that the blood collects towards the centre of the body.

Much stress has been laid on the effect of adrenalin on the vessels. This drug induces a powerful contraction of their muscular walls, and it raises the systemic blood pressure during shock. Its action in thus raising the blood pressure is said to afford a proof that the vasomotor centres are paralysed. But the rise of pressure in the carotids would be quite as certain to be induced by adrenalin if, as I believe, the blood is mainly collected in the larger internal vessels. Any conditions which power-

fully contracted these internal vessels when thus dilated would tend to redistribute the blood and force some of it into the systemic vessels, exactly as pressure upon the abdomen may improve the radial pulse when a state of shock exists. Of course the extended muscle fibres of a dilated vessel have a greater range of contraction than fibres already firmly contracted. Hence the action of adrenalin does not prove that the vessels generally are dilated. According to the interpretation of the action of this drug which I have suggested, the beneficial effects gained by powerfully contracting the dilated internal vessels must be accompanied by a very greatly increased resistance to the propelling force of the heart, and the risks of cardiac failure would therefore be considerable.

In this connection the result of one of Dr. Crile's experiments [1] is very interesting. In a dog which had been reduced to a state of profound shock after severance of its accelerantes and vagi the blood pressure in the carotid was raised by the administration of adrenalin, and, as the pressure was rising, the animal died suddenly, but before the initial blood pressure was restored. No explanation of this death was offered, and it is very difficult to understand why the dog died if the arteries were paralysed, and if that condition was being successfully counteracted by the administration of adrenalin. On the other hand, if the adrenalin was merely squeezing the bulk of the blood out of the dilated internal vessels and forcing it in part into the contracted superficial ones, it must have added very materially to the already increased amount of work required from the heart, and thus a cardiac failure and sudden death would be easily accounted for.

To sum up :—The theory that the vessels are intensely contracted during shock offers a full solution of all the difficulties that I have pointed out as arising when the vessels are assumed to be paralysed. It accounts for the pallor of the skin, for the improvement in the pulse

[1] 'Blood Pressure in Surgery,' Experiment 106.

induced by vaso-dilators, for the difficulty of injecting fluids into the veins, for the rise of pressure in the central vessels, and for the rise of specific gravity of the blood.

The view of this question that I have put forward differs very slightly from that founded on Goltz's observation that tapping the abdomen of a suspended frog caused a paralysis of its splanchnic vessels. According to that view the blood collected in the splanchnic area because the vessels of that area became paralysed. The view I advocate accounts for the distribution of the blood by the occurrence of a contraction of the arteries generally and of the superficial ones more particularly, by which the blood is forced into the central areas, in which the vessels must, of course, dilate. According to both these views the blood flows to the centre of the body because of the relatively greater resistance in the superficial vessels as compared with that in the central vessels. But I maintain that the relaxation of the larger internal vessels which occurs is brought about chiefly by intravascular pressure. Probably they yield in part also because of a physiological necessity for an accumulation of the blood in the warmer parts of the body. But the vessels in these parts are certainly not paralysed during shock. It is easy to understand how, if my view is right, a large quantity of blood may pass into the veins of the splanchnic area after death, and so a condition which formerly was relied upon as a conclusive evidence that these vessels were paralysed may be produced. Indeed Dr. Crile has shown that even during life the venous trunks everywhere were full and the arteries were quite empty. He said that "the relative proportion of blood in the arteries and capillaries and that in the venous trunks gave a rough estimate of the depth of the shock at that time."[1] The fact that the capillaries were not distended shows clearly that the smaller veins had not lost their tone, and, therefore, that the vaso-motor centres presiding over them were intact and active. This observation seems to

[1] 'Surgical Shock,' Crile, loc. cit.

me to prove conclusively that the blood is forced into the central parts of the body, not drawn into them.

The old view that the vessels of the splanchnic area were paralysed during shock became untenable when it was possible to observe the internal vessels during operative procedures, for as the condition of shock develops the arterioles do not dilate, the parts remain pale, and unless large vessels are divided operative procedures cause little hæmorrhage.

Observations on shock or on conditions resembling those of shock, produced by prolonged exposure of the peritoneum to the air, must of course be complicated by evidences of inflammation of that membrane, a source of error which has not always been eliminated.

Practical Conclusions as regards Treatment.

The above considerations might be regarded as of purely speculative and academic interest, but they have a very direct bearing upon the treatment of shock, and the effects of treatment also help to support my view. Warmth and stimulating vaso-dilators have long been used for combating this state, and they are theoretically beneficial if my theory is right. If Dr. Crile's theory is correct vaso-constrictors and, theoretically, an ice pack should be employed. But he definitely states that exposure to cold is prejudicial. There can be little doubt that, in practice, injections of strychnia are useful during shock, probably by strengthening the whole vascular system. But Dr. Crile found that a condition which he regarded as one of shock, and in which the blood pressure in the carotid was lowered, could be rapidly induced by giving strychnia in large doses to dogs. As it produced this condition its administration was not considered good treatment for shock. There is no evidence that this strychnia shock did not arise in exactly the same way as

traumatic shock, namely, by an exaggerated effect of its primary constricting action on the vessels.

I have already referred to the action of adrenalin. Any treatment which helps to tide a patient over a short time until an operation can be finished or until a state of shock passes off may be useful, but according to my theory adrenalin administered for shock can be beneficial only at a considerable risk.

According to Dr. Crile's statements saline injections are useless when shock is intense, because the fluid if injected is at once exuded into the tissues. Before this stage is reached, saline injections, by tending to keep up the bulk of the blood, may act beneficially, but again at some risk of throwing an extra burden on the heart and on the pulmonary vessels, and of causing an œdematous condition of the tissues. It seems to me possible that intravenous injections may help to save life during an operation and yet at a later date may be a cause of mischief, especially in the lungs, the circulation in which is likely to be most directly disturbed.

During recovery from shock, if, as I believe, the vessels are relaxing, saline injections should be useful, and it is well known that at this stage fluid will find its way into the vessels from the rectum almost as quickly as by direct injection.

Theoretically, and I think practically, injections into the cellular tissue are to be preferred to those into a vein, if some such treatment is considered desirable during shock, because if the fluid does not find its way into the vessels from the cellular tissue, an attempt to force it into a vein is likely to be not only useless but also harmful.

The most important treatment of shock is preventive. When there is time before an operation, the patient should be got into the best condition possible by the administration of strychnia, by attention to the condition of the bowels, by judicious feeding, and by the treatment of any complication that may exist. During an operation

everything should be done to maintain a flow of blood to the surface and to the head, and to keep the patient alive by warmth, by strychnia, by vaso-dilators, and by mechanical means, such as lowering the head and compressing the abdomen. The surgeon must reduce traumatism by getting over the operation quickly, but with the least possible loss of blood at the time and afterwards. These are the methods that are generally most successful, and they are exactly those which are suggested by the view that the vessels are contracted. I submit therefore that methods founded on the theory that the vaso-motor centres are paralysed, especially those concerning warmth and stimulants, should not be adopted unless the accuracy of this view can be supported by more convincing evidence than has at present been put forward.

DISCUSSION

Mr. J. P. Lockhart Mummery said that the theory that shock was due to exhaustion of the centres in the medulla and cord controlling the vaso-motor mechanism, and that the great fall in blood-pressure which occurred was due to the relaxation of the blood-vessels throughout the body, was founded upon carefully ascertained clinical and experimental facts. Dr. Crile had done a great deal of careful experimental work upon the physiology of shock, and our knowledge of that condition was in large measure due to him. Dr. Harvey Cushing, Dr. Mayo, himself, and others, had confirmed this work both clinically and experimentally. In the Hunterian lectures for 1905 he brought forward clinical observations in support of Crile's theory, and for nearly two years Dr. Symes and he had been carrying out a series of experiments in connection with the physiology of shock. The theory that shock was due to exhaustion of the vaso-motor centres was no mere hypothesis, but a true theory founded upon well-established facts, and if it was to be upset it must be by something more than assertions unbacked by experimental facts. Mr. Malcolm had brought forward no new facts nor any experimental evidence in support of his views. With regard to Mr. Malcolm's contention that the arteries were constricted in shock, he could positively assert the contrary. He had repeatedly examined the arteries of animals suffering from all stages of shock. When shock had commenced, as evidenced by the blood-pressure falling, the arteries could be seen to dilate. Vessels which before could not be seen became obvious, and all the small arteries could be seen to relax and become filled with blood. When surgical shock had been fully established all the arteries, arterioles, and veins could be seen to be relaxed fully, This appeared throughout the circulation, and with it there was a fall in blood-pressure to a fatal level. The capillaries were, however, for the most part empty, as there was not sufficient pressure to maintain the circulation through them, the circulation having almost ceased except in the larger vessels. If, while an animal was in a condition of severe shock, constriction of the arteries was produced, as it could be by the intravenous injection of adrenalin, the blood-pressure rapidly rose and the circulation was temporarily restored. Adrenalin acted directly on the walls of the vessels, as it caused constriction of the vessels, and raised the blood-pressure, even after the whole central nervous system had been removed. The very condition, therefore, which Mr. Malcolm claimed was the cause of shock was the condition which prevented its occurrence or counteracted it. These facts were entirely opposed to Mr. Malcolm's views, and he could not

agree with him that his views were in any way compatible with Crile's facts.

Dr. LEONARD WILLIAMS: It seems to me that Mr. Malcolm's paper is an attack not only on Dr. Crile's work, but also upon the teaching of all modern physiologists on the subject of the behaviour of the blood-vessels in the ordinary variations of blood-pressure. Mr. Malcolm appears to have overlooked the fact that the exhaustion of the medulla and the consequent paralysis of the peripheral blood-vessels is partial only. If the exhaustion were complete, the paralysis of the vessels would be complete, and these vessels would be dilated to their widest capacity. Now, as the total capacity of the blood-vessels is about three times as great as that of the contained blood, it follows that in total paralysis the animal would immediately die. Some measure of tone must therefore remain in the vessels. Now we know that the main object, or at any rate one of the main objects, of the circulation of the blood is to keep the medulla bathed with this fluid. If the medulla is starved the animal dies at once. The circulation in the brain must therefore be maintained at or above a certain level. The phenomena of shock are produced by the fact that the circulation in the brain falls to this level, and sometimes even below it. There is no difficulty, as Mr. Lockhart Mummery has pointed out, in explaining the pallor of the skin and the rise of pressure in the portal system, if the blood-pressure in the brain is not to fall below the point at which death immediately takes place; for, although the general condition of the vessels is one of relaxation, if there were not a relative constriction in some parts the pressure in the brain could not be maintained at anything approaching its proper height. Mr. Malcolm's views on the effect of the dilatation of vessels upon the blood-pressure would not, I think, be accepted by Leonard Hill, E. H. Starling, and those other workers to whom we owe our present knowledge of the vascular phenomena. Now if we are to admit that there are some points which still await explanation on the theory that relaxation of the vessels is what obtains in shock, I think it will be difficult for Mr. Malcolm to show that there are not much greater difficulties to be explained, if, as he suggests, the condition of the vessels in shock is that of constriction. We know how easy it is for the starved medulla to cause death, but it is difficult to see how the medulla, which is supplied with blood at a very much higher pressure than normal, can give rise to the phenomena under discussion. Mr. Malcolm's appeal to the results of treatment does not seem to me in any way to favour his contention. The administration of stimulating vaso-dilators (I suppose he refers to ammonia) is valuable because of their stimulation of the respiratory centre, which may conceivably transfer some of that stimulation to the vaso-motor centre. It cannot have any

effect in dilating blood-vessels which are already dilated. The application of warmth is necessary, not only as being agreeable to the patient but to prevent undue loss of heat, which from various causes is very liable to take place. The proper treatment in cases of shock is to put the patient on his back and empty his abdominal vessels, so that the heart may be adequately supplied with blood which it can send to the medulla.

Dr. A. MORISON: Shock is admittedly a term intended to convey the belief in a powerful impression made upon the nervous system, the governing system of vital function, and of producing a disturbance of such function directly by inhibition of it and indirectly by disturbing its blood supply. Mr. Malcolm's paper deals with the latter form, which cannot in truth be detached from the direct inhibition of any function, because functional activity, while tripartite, is also triune. That is, cell, nerve, and blood supply are equally essential to continued and normal activity. Clinically, both in medicine and surgery, we observe two chief kinds of effect upon the vascular system by shock. Firstly, either the heart is brought to a sudden standstill, at times from comparatively trivial causes, with cessation both of circulation and respiration and of every other function, including consciousness; or, secondly, the cardiac force is profoundly depressed, the ventricular output is small per systole, while the heart rate is notably increased. The pulse, therefore, is small and quick in the latter case, and the surface cold, pale and clammy, or bathed in perspiration. Owing to this important part played by the heart as the central organ of circulation under the then circumstances, I prefer the term "cardio-vascular" depression to "vaso-motor" change, which directs more attention to the periphery. Whether trauma be due to operative procedure, to the pain of calculous colic, to angina pectoris, or to the action of a poison, these two chief varieties of shock are observable both by physicians and surgeons. Physicians also, perhaps more frequently than surgeons, see intermediate stages between these extremes, in which there may be for a time some active contraction of the peripheral vessels. Such active narrowing, as in some cases of angina pectoris, may eventuate in relief and the quick resumption of normal action, or it may proceed to graver collapse and rapid dissolution. When the full effect of shock is rapid and the heart's action is suddenly arrested and the patient dies, the heart is found post-mortem in complete diastole, the left as well as the right chambers being filled with blood, the arteries, of course, empty, and the veins engorged. When the vascular effects of shock are gradual and the patient dies, the left ventricle is usually found comparatively empty, and the right, together with the general venous system, surcharged, while the nerve-centres, like the rest of the arterial periphery, are blanched. In considering

narrowed arteries in shock we must, therefore, bear in mind the elastic recoil of so-called "empty" vessels as distinguished from their neuro-muscular narrowing. We know that the venous system is capable of containing all the blood in the body, and we know, also, that cyanotic conditions observed during life disappear after death until the advent of post-mortem lividity. To produce cyanosis a certain active turgescence is necessary. When venous turgescence is adynamic, as it is in shock, cyanosis need not be noted, although the blood is chiefly contained in the veins and largely in the deep and abdominal veins. This adynamic venous plethora is like the pale stasis of death, on the confines of which shock trenches. The facts, therefore, criticised by Mr. Malcolm, namely, pallor of skin, difficulty of injecting fluids into the veins, some rise of pressure in the portal veins, and an increase in the specific gravity of the blood, do not seem difficult to account for on the assumption of a more or less progressive venous turgescence from disablement of the propulsive power of the heart. As to the fact, if fact it be, that the first effect of shock is to increase the volume of blood in the radial artery, I cannot speak from direct observation otherwise than to deny it, except in the intermediate variety to which I have referred. I know from personal observation that the ordinary methods of determining blood-pressure, as by the Riva-Rocci instrument, at times indicate a satisfactory figure in cases in which the arterial pressure, judged by the finger and the observation of the patient as a whole, is undoubtedly low. I have only been able to account for this apparent contradiction by assuming that the encircled arm under these circumstances registers venous turgescence as well as systolic force. The condition of the arterial system in shock, therefore, appears to me to be an empty, if shrewd, view rather than of actively contracted arteries, and I am forced to agree with the more orthodox conception of the cardio-vascular state in shocks expressed by Mr. Crile rather than with the theory advanced by my friend, Mr. Malcolm.

Mr. WILLIAM SHEEN: Mr. President and Gentlemen, I wish to thank the President and Council of the Society for kindly inviting me to take part in this discussion, and to say with what interest I have listened to Mr. Malcolm's paper. As your kind invitation, with Mr. Malcolm's abstract, only reached me this morning, I have had no opportunity of giving this subject much immediate consideration, and I must ask you to pardon me if my remarks are somewhat fragmentary. My interest in surgical shock dates from the time when I read Mr. Malcolm's and Mr. Mummery's very conflicting papers, the one stating that the arterioles were contracted, the other that they were dilated, in shock. I venture to suggest that they are neither contracted nor dilated, but *retracted*. By retraction I mean contraction

followed by relaxation, but not by extension. Retraction is the condition of the uterus when the child has been delivered and the muscle has relaxed without the cavity enlarging, and of the biceps when after active contraction it is relaxed, the elbow remaining flexed. My view of shock,—for example, from stimulation of the central end of the divided sciatic nerve—is that first a pressor effect is produced, vaso-constrictor impulses reaching the arterioles, the blood at the same time accumulating in the internal veins, particularly those of the splanchnic area, the condition being comparable to that which obtains after a meal, in fear, cold, sleep, and certain other conditions, owing, as Mr. Malcolm says, "to the physiological necessity for the conservation of heat." After a variable period, which may be of some duration, the pressor effect is exhausted, and vaso-dilator impulses reach the arterioles and they relax, but do not dilate because there is no blood to fill them, the blood being accommodated in the splanchnic and other internal veins; their condition is then one of *retraction*. Mr. Malcolm will ask me why, at this juncture, in accordance with ordinary physical laws, the blood does not flow towards the arterioles to equalise the pressure again? This is a question I cannot completely answer, but I would quote Mr. Malcolm himself in a recent paper where he says that the "dynamics of the system are very complex," and would point out that we do not know what the action of the liver may be in acting as a "stop-cock" to the splanchnic veins. I do not agree with Mr. Malcolm that the arterioles continue contracted until death occurs: surely at some time their condition must become one of relaxation. Mr. Mummery's explanation of the rise of the specific gravity of the blood with contracted arterioles I cannot understand, whereas Mr. Malcolm's, that the more fluid parts of the blood are squeezed out of the contracted vessels, appears reasonable and satisfactory. I should like to draw attention to the fact that in cases of very severe injury—for example, crush of both legs necessitating double amputation—there may be for some little time none of the ordinary symptoms of shock. The individual is rational, moves uninjured parts actively, talks, and perhaps smokes. This corresponds, in my opinion, to the stage of well-contracted arterioles with a well maintained or even raised blood-pressure. Similarly, the first effect of any operation is often a rise of blood pressure, but this early period is here shortened by the anæsthetic. I think that the subject is not as yet thoroughly worked out, and that the combined efforts of physiologist, physician, surgeon, and therapeutist, with even the physicist, are necessary for full elucidation. May I point out how different the "shock" of the physiologist is from the "shock" of the surgeon. In Schäfer's 'Text-book of Physiology' we find: "'Shock' . . . a term more used by the physician than the physiologist, . . . a term somewhat ill-defined in scope. In some forms of the

clinical condition, circulatory disturbance and inspissation of the blood play a part, but as understood by the physiologist 'shock' is primarily a nervous condition." After this somewhat contemptuous dismissal of the clinical interpretation of the term, there follow rather more than two pages dealing with shock as the result of various modes of transection of the spinal cord. I can imagine the confusion of the medical student who passes in his studies from one description to the other. In conclusion, sir, I thank you again for permitting me to take part in this discussion.

Dr. A. P. BEDDARD thought the primary thing to explain in shock was the fall in the general blood-pressure. Physiologists would admit that the only other factor was the peripheral resistance which depended on the condition of the microscopic arterioles. The result of the underfilling of the arteries was that the small visible arteries contracted up and became invisible. As to the pallor of the surface, that depended on the accumulation of the blood in the abdominal veins, even the volume of a limb diminished in shock. The prime factor was the diminished peripheral resistance in the splanchnic area. The application of heat in shock was a fundamental thing, the muscular tone was reduced and the main source of heat removed, and thus the subject became really a cold-blooded animal. Shock, however, was not merely a vascular phenomenon.

The PRESIDENT: I think it very useful to have our attention called to so important a subject as that of shock, which has both a physiological and clinical interest; and Mr. Malcolm's paper and the discussion that has followed has shown that there is considerable difference of opinion as to the causation of the phenomena of shock and a good deal that still remains to be discovered concerning it. The conditions observed in shock appear to be less disputable than their explanation, and while the explanation of these conditions is still unsettled it will probably be wise for those of us who have to treat the condition to base our treatment chiefly upon the lessons of experience. In spite, therefore, of what has been said in this discussion, I shall still venture to apply warmth and to administer stimulants to those suffering from the effects of shock. The term "shock" is not very well defined, though its effects are usually sufficiently manifest, and it must be remembered that its causes and concomitants are various, and its effects by no means confined to the vascular system.

Mr. MALCOLM: In reply to Mr. Mummery's contention that there is not sufficient blood-pressure to fill the capillaries, I would point out that, according to Dr. Crile, the power of the heart is not necessarily weakened during shock. Dr. Crile recorded that "in experiments in which the cardio-accelerator fibres had been previously severed, and the heart in consequence beat slowly until death, a descending curve in the blood-pressure,

showing the final vaso-motor break-down, was practically the same as in experiments in which the heart ran rapidly until the end."[1] Moreover, Dr. Crile asserted that the great venous trunks, both splanchnic and somatic, became more and more full whilst the arteries became "quite empty"[2] as shock deepened, and that "the output of the heart does not depend at all upon the height of arterial pressure, but is in direct proportion to the venous pressure."[3] If, then, the heart's power is not necessarily lessened during shock, if the large venous trunks become increasingly full, if the heart's output depends on the pressure in these enlarged venous trunks, and if the arteries are empty but paralysed, the explanation that the blood is kept from dilating the capillaries by a want of pressure does not seem altogether satisfactory. On the contrary, the conditions described by Dr. Crile are eminently calculated to facilitate a free arterial and capillary circulation, or at least a free flow of blood into the arteries and capillaries. It is true that Dr. Crile in another passage stated that during shock "the venous pressure had sunk so low that the heart received but little blood upon which to act."[4] But this assertion was made without any evidence being put forward to support it, and without any stated recognition or explanation of its apparent inconsistency with the facts detailed. Mr. Mummery's contention that the pressure is sufficient to dilate the arteries, arterioles, and veins, whilst the capillaries remain empty, is also very difficult to understand. Again, the effects of vaso-dilators seem to me irreconcilable with the idea that the capillaries are empty from want of blood-pressure during shock. Mr. Mummery contends that these effects until recently were misleading. In his lectures at the Royal College of Surgeons he said that, until the Riva-Rocci instrument was introduced a fallacy had existed, and a delusive appearance of an improvement in the pulse had been observed after the administration of alcohol. He added that, on the contrary, "by observing the blood-pressure with the sphygmomanometer after the administration of alcohol to a patient, or to myself, I have found that the blood-pressure, instead of being raised, is actually lowered and that the apparent improvement in the pulse is due to vaso-dilatation and an increased flow through the vessel."[5] That is the effect in health, but there can be no doubt that the blood-pressure is raised, and not lowered, in shock. With the Riva-Rocci instrument the observer estimates the pressure on the brachial artery which is necessary to obliterate the radial.

[1] 'Surgical Shock,' pp. 153–154.

[2] *Loc. cit.*, p. 140.

[3] *Loc. cit.*, p. 138.

[4] *Loc. cit*, p. 140.

[5] 'The Lancet,' 1905, vol. i, p. 280.

Hence, if Mr. Mummery had applied the sphygmomanometer to a patient whose pulse at the wrist was absent from traumatism, he could not possibly have found by means of that instrument that there was any blood-pressure at all in the radial. But, as often happens, if the pulse was again felt, as a consequence of giving alcohol, it would immediately become possible to record a certain pressure as necessary to obliterate it. Whether this pressure would be great or small does not matter. The fact is indisputable that a rise of pressure from nothing to a positive quantity must, under these circumstances, be recorded. The improvement is, therefore, not imaginary. If, then, during the state of shock the blood-pressure is raised in the radial by the administration of a vaso-dilator, it is clear that a lowered pressure caused by vaso-motor paralysis cannot be the cause of the deficient capillary circulation. A vaso-dilator can have no effect on a paralysed vessel. That a fall of blood-pressure in the radial follows the administration of alcohol to a normal person is altogether beside the question. The difference of the effects is due to the fact that in the state of shock the stop-cock action of the vascular contractions reduces the flow of blood in vessels larger than the radial so that relaxation permits a more free flow of blood *into* the radial, and thus causes a rise of blood-pressure in it, whereas, in health, the tone of the vessels acts as a stop-cock, shutting off the blood only from vessels distal to the radial, and thus relaxation allows the blood to flow more easily *away from* that vessel, and the blood-pressure in it is therefore lowered. Mr. Mummery says there is nothing easier than to put saline solution in large quantity into an animal's veins. In reply, I will only say that my argument is based on Dr. Crile's statement that the deeper the degree of shock, the more difficult it becomes to introduce saline fluid into the veins.[1] The rise of blood-pressure in the portal vein is said by Mr. Mummery to be very slight, but, if its existence is acknowledged, the point I have raised as to why the bulk of the blood flows from the vessels in which the blood-pressure is falling into those in which it is rising is still unexplained. Mr. Mummery says that he finds the rise of the specific gravity of the blood very difficult to explain. I venture to assert that it is impossible to explain this change as a consequence of a vascular relaxation, whereas it naturally follows if arterial tension exists. The vascular changes described by Mr. Mummery as characteristic of the state of shock are never observed in the course of operations on human beings when the peritoneum is not inflamed. Mr. Mummery's observation that the arteries are dilated during shock is also irreconcilable with Dr. Crile's statement that they are "quite empty" in that condition. In his lectures at the Royal College of Surgeons Mr. Mummery stated that shock could be induced

[1] 'Blood-pressure in Surgery,' p. 269.

by prolonged exposure of the peritoneum. But a state of shock induced by prolonged exposure or manipulation of the tissues in the splanchic area must be accompanied by a peritonitis just as the removal of the eyelids must be followed by a conjunctivitis. The vascular changes thus induced have no bearing on the condition of the blood-vessels during shock. Dr. Crile himself has said that "autopsies in experiments in which the splanchnic area was not involved did not reveal a condition of vascular distension in this area different from that in any other area,"[1] that is to say, the tissues were pale and the arteries empty. Mr. Mummery urges that I have not brought forward any new facts or experimental work, and that these are necessary if the theory he supports is to be upset. This comment seems particularly irrelevant if it is recognised that I accept all Dr. Crile's statements of facts as the basis of my argument that his conclusions are unproved. Moreover, if experimental work is alone to be considered as satisfactory evidence, it is, I think, allowable to demand that those supporting the same theory should state clearly the facts on which they base their opinions. The superior importance which appears to be claimed for experimental work is not demonstrated when Mr. Mummery's statement that the arteries are dilated during shock is contrasted with Dr. Crile's assertion that they are "quite empty" in that state. Dr. Crile's description of the state of the vessels would almost appear to be a "new fact" to Mr. Mummery, as I am sure it will be to many who have formed their views of his theory without a thorough investigation. I would expect the vessels to be as described by Mr. Mummery, if the vaso-motor centres were paralysed, but Dr. Crile says it is not so, and Dr. Crile's statement agrees in every detail with the conditions which I have observed in the operating theatre and at the bedside. In reply to Dr. Leonard Williams, I would say that I have never stated that relaxation of vessels is not a cause of a fall of blood-pressure. Without doubt, a relaxation of small vessels generally lowers the blood-pressure in the large arteries, but I maintain that a sufficient contraction of an individual vessel also always lowers the blood-pressure in the particular vessel concerned, provided that some other vascular area will yield so as to accommodate the displaced blood. Of course, the vascular changes are not the cause but the accompaniments of shock. They help to explain some of the symptoms. Dr. Morison prefers to speak of a cardio-vascular paralysis. But Dr. Crile has shown, and I also have published the conclusion,[2] that cardiac failure is not a necessary accompaniment of shock. With others, Dr. Morison omits to explain how a fall of arterial

[1] 'Surgical Shock,' p. 139.

[2] 'The Physiology of Death from Traumatic Fever,' p. 66. J. and A. Churchill.

blood-pressure and an empty but relaxed condition of the somatic arteries can be associated with a rise of pressure in the portal veins. Mr. Sheen's suggestion that the vessels are paralysed and retracted seems to be the only possible explanation of the association of empty arteries with the alleged loss of their nervous control. But an empty paralysed artery is a physiological anomaly, and it is especially so when in another area the blood-pressure is raised. Dr. Beddard's statement that the blood-pressure in the carotid is merely the lateral pressure in the aorta seems hardly correct. The carotid has a muscular coat, and if it is contracted sufficiently the pressure in it may be reduced, even if that in the aorta is unaltered. It is generally recognised that during digestion the blood-pressure in the splanchnic area is raised, whilst that in the somatic area is lowered. According to my view the relative changes of blood-pressure found during digestion are exactly paralleled by those observed in shock, although in the latter condition the changes are greater, and the vascular system generally is in a state of unusual tension. Dr. Beddard attributes the peripheral resistance entirely to the contraction of the arterioles. But arteries of the size of the radials may certainly contract almost to obliteration. I have seen a patient without a perceptible pulse at the wrist as a result of anæsthetics and traumatism at the end of a severe operation, from which pulse condition a perfect recovery took place in a few hours. Associated with the absence of radial pulse there was a cardiac beat of 48 to the minute, and the pulsations in the carotids were very distinct and forcible. Such a marked case of absence of radial pulse associated with a slow, strong heart action is rare, but on several occasions I have noted the radial pulse absent, whilst the heart-beats and the carotid impulses were very forcible, and from 80 to 90 to the minute. This combination of phenomena is absolutely irreconcilable with Dr. Crile's theory, and it seems to me to be obviously due to a narrowing of the arteries by contraction, those as large as the radial being affected to such a degree that the pulse in them cannot be felt, although the heart is beating with unusual power. Dr. Beddard's statement that the whole of a limb is reduced in size during shock seems to me in itself almost a proof of, and is at least a fact which strongly supports, the view that the vessels are contracted. In conclusion, I would urge that the only physiological explanation of the vascular phenomena observed in shock is to be found in the well-recognised fact that stimulation of a sensory nerve causes a contraction of the arteries. On the other hand, Dr. Crile's theory is founded on the deduction that a fall of blood-pressure *must* be caused by a vascular relaxation—a deduction which is distinctly opposed to physiological teaching, although it holds good in certain common but strictly limited circumstances.

THE CAUSES OF COLITIS, WITH SPECIAL REFERENCE TO ITS SURGICAL TREATMENT

WITH AN ACCOUNT OF 36 CASES

BY

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A GREAT deal of attention has been paid within recent years to the subject of colitis, more especially, perhaps, on the continent, and in 1905 Dr. Hale White brought the subject prominently before the profession in this country by the publication of his study of 60 cases. Although colitis is generally looked upon as belonging to the province of the physician rather than the surgeon, many cases of colitis come under the care of the surgeon.

Colitis has been described under many different names by different authors. Such names as "colica mucosa," "tubular diarrhœa," "membraneous colitis," and "muco-membraneous entero-colitis" are only a few of the names which have been used to describe what is, without doubt, the same condition. My cases do not differ in symptomology in any important particular from those recorded by other writers under one or other of these different names. The essential symptom in all cases is the presence of excess of mucus in the stools in the form of casts (complete or broken up), shreds, or large glairy masses. In addition,

bleeding, intestinal sand, diarrhœa, and acute attacks of abdominal pain are common accompanying symptoms.

From the study of my own cases I have been driven to the conclusion that colica mucosa, by whatever name it is called, has no claim to be described as a disease any more than has diarrhœa or hæmatemesis, but that it is a symptom or condition which may result from a number of different diseases of the colon of widely different characters. This is not, I think, a new hypothesis, as although the point has not been specially emphasised before, it is the logical conclusion to be drawn from such collections of cases as those of Dr. Hale White and some of the German writers on the subject. I shall attempt to show that, in the great majority of cases, a lesion or lesions can be demonstrated which it is only reasonable to suppose is the cause of the condition.

Until a few years ago our only means of knowing the condition of the colon in cases of colitis was by a study of the symptoms, and an examination of the abdomen and of the discharges, aided, sometimes, by what was seen of the outside of the bowel when an operation was performed, or of its interior when the patient had died from some other cause when suffering from colitis. Within the last four or five years, however, the technique of examining the lower bowel by means of instruments has so much improved that we have been enabled to accurately examine the interior of the lower bowel in all those cases where symptoms of colitis were present, and in this way much has been learned as to the causes of colitis which was hitherto unknown.

The most important advance in our knowledge of these cases has been due to the invention of the electric sigmoidoscope. This instrument enables us to see the condition of the mucosa in the rectum and sigmoid flexure and to compare the condition found in health with that found when symptoms of colitis are present. It has rendered untenable the theory of Professor Nothnagel, that colitis is a neurosis unaccompanied by local lesions of the large bowel, for in a large proportion of the cases definite local lesions can be found to explain the symptoms.

Before going further it will, perhaps, be advisable for me to define what I mean by the sigmoid flexure, as its anatomical limits are somewhat indefinite. By the sigmoid flexure, as referred to in this paper, is meant that portion of the large intestine which forms a freely movable loop between the termination of the descending colon and the upper end of the rectum. The junction between the rectum and sigmoid flexure is readily definable from the interior of the bowel as there is a well-marked valve or sphincter where these two portions of bowel join, which can be easily seen during an examination with the sigmoidoscope. The distance to which the instrument can be passed varies in different subjects, but it is usually possible to see as far as the centre of the sigmoid loop, and often for another three to four inches beyond that. The distance as measured from the anal margin is from 20 to 30 cms.

A point of no little importance which a systematic sigmoidoscopic examination of the bowel in colitis cases has brought out is that the causes for the condition are, in many cases, confined to the lower part of the sigmoid flexure and the rectum. This fact was pointed out by Tuttle and Kelly, and I have been able repeatedly to verify the truth of their observations. It is, after all, not surprising that this should be so, as the portion of the large intestine which is most subjected to abnormal conditions is the sigmoid flexure and rectum; it is here that stagnation and pressure occurs in constipation, and there can be no doubt that constipation is by far the commonest antecedent of colitis. It is here that any irritant material, be it solid or liquid, will collect and tend to stagnate if present in the large bowel, and it is here that the greatest amount of traumatism from the passage of hardened fæcal masses will be felt.

In fact, those portions of the large bowel which are most liable to injurious influences are the two dependent ends, the cæcum and the sigmoid flexure, and it is here that disease, when it attacks the large intestine, is most frequently found. We should, therefore, expect that the

chief lesion would be situated either in the cæcum or in the sigmoid flexure, and, as I shall attempt to show, this is the case.

Symptoms.

Diarrhœa was a prominent feature in the majority of the cases and in some it was most severe.

In Cases 16, 20, and 23 there were at one time as many as twenty to twenty-five stools *per diem*.

The diarrhœa was usually the symptom which first caused the patient to seek medical advice.

In almost all the cases there was an antecedent history of constipation quite irrespective of whether an inflammatory lesion or a new growth was found at the examination.

Bleeding.—Bleeding occurred in twenty-two out of the thirty-six cases.

In most of the cases in which there was bleeding this could easily be accounted for by the fact that definite ulceration was present in the bowel. In some, however, no definite cause for the bleeding was discovered.

In two of the cases of new growth, Nos. 18 and 19, there was no bleeding at all. This shows, I think, that bleeding cannot be considered characteristic of new growth.

Mucus was present in the stools in all the cases in sufficient quantities to draw the patient's attention to it. In some cases very large quantities of mucus were passed so that the stools consisted sometimes of little else.

The mucus was in some cases thin and almost fluid, in others thick and gelatinous or in shreds and strings.

Large mucous casts were present in the stools in eight of the cases, and I would especially draw attention to the fact that casts were seen in two of the cases, Nos. 3 and 21, which were due to malignant disease of the bowel. These two cases are, I think, worth recording in detail.

Case 3 was a woman who for five years had suffered from acute attacks of pain in the abdomen, followed by the passing of large mucus casts *per anam*. There had been some bleeding at times during the last twelve

months. On examining her I found that she had a large growth in the sigmoid flexure. The growth was quite inoperable and she died a few months after I saw her.

Case 21 was a man. He had only had symptoms of bowel trouble for six months. On several occasions he passed mucus casts several inches in length. On examining him I found a large malignant growth in the sigmoid flexure and a mass of enlarged glands in the meso-rectum. No operation was possible in his case.

Loss of weight was quite as common in the non-malignant as in the malignant cases. It was most marked in those cases associated with severe diarrhœa.

Intestinal sand was seen in the stools in two cases (Nos. 2 and 20). In both cases it was a fine yellowish-red sand when passed, but it became black if left for some days. The sand was analysed in both cases and found to consist of carbonates and phosphate of calcium and ammonium, but no cholesterin. It is an interesting fact that Case 2 also occasionally passed uric acid gravel in the urine.

In both cases very severe attacks of abdominal colic were associated with the appearance of the sand.

Enterospasm.—This interesting condition was present in one of my cases (Case 20).

Enterospasm is a localised contraction of the bowel, often resulting in a temporary intussusception. It most usually occurs in the descending colon or sigmoid flexure, and is accompanied by extreme pain localised to that portion of the abdomen in which the contracted portion of bowel lies.

The spasm may involve a large length of bowel, according to some authorities, but in those cases in which I have met with it only an inch or two of bowel has been contracted. In my patient there were attacks of extreme abdominal pain, and a hard tumour about two inches in length could be felt in the sigmoid flexure. I examined the patient very carefully, as I suspected that her colitis might arise from a growth in the sigmoid flexure. She had constant diarrhœa, with stools consisting of blood and

mucus. The tumour might have easily been mistaken for a growth, but I found that it always disappeared when the pain ceased, and was always present when a fresh attack of pain occurred. The contracted portion of bowel always occupied the same position, and I came to the conclusion that the spasm resulted from an ulcer in that portion of the bowel, as there was tenderness over that spot when the contraction was not present. The patient recovered as the result of a careful dieting and irrigation.

My colleague at St. Mark's, Mr. Swinford Edwards, had a most interesting case of the same sort. He opened the abdomen to explore for a tumour in the sigmoid flexure, and on drawing out the bowel found a contracted band about two inches in length resembling a local intussusception. While he was watching, it disappeared and again reappeared.

Enterospasm appears to result from some local irritation within the bowel. The irritation may result from an ulcer within the bowel, or from a hardened fæcal mass, or concretion.

The Examination of the Patient.

A patient suffering from colitis cannot, as a rule, be properly examined in the consulting-room or out-patient department of a hospital. The sigmoid flexure is normally full of fæcal material, and special means are necessary to empty it. The patient should be confined to bed, and the rectum and sigmoid flexure thoroughly emptied by aperients and enemata administered by a skilled nurse. The examination with the sigmoidoscope is not painful, and, as a rule, causes but little discomfort, so that an anæsthetic is not required in most cases. The greater part of the sigmoid flexure can be readily examined and the condition of the mucosa observed. The sigmoidoscope, like the cystoscope or laryngoscope, requires practice before it can be properly used, but in skilled hands it is perfectly safe, and the view of the parts obtained with it is all that could be desired.

The abdomen should, of course, be examined, especially the appendix area and the left iliac fossa.

The stools should also be examined, and in some cases a microscopical or bacteriological examination made of the abnormal constituents.

Cases of Colitis in which Lesions are present within the Bowel.

I shall attempt to show that some lesion, either within the bowel or external to it, but involving it, can be demonstrated in the majority of cases of colitis, and that the recent improved methods of examining the bowel have proved that what is generally called colitis is a symptom of some local lesion of the bowel. I do not wish to assert that whenever we find an abnormal condition of the mucosa we are necessarily, at once, to look upon it as the cause of the colitis. I do not think that the gravity of the lesion in some of the cases is sufficient to justify this view, and it may be that the appearances are but secondary to some more serious lesion at a higher level. That this may occur is evidenced by the following case which I saw in consultation with Mr. Mayo Robson. The patient was an elderly gentleman who for many months had been passing large quantities of blood and mucus in his stools. An examination with the sigmoidoscope was made and revealed the presence of numerous small follicular ulcers, scattered about on the mucous membrane of the rectum and sigmoid flexure. There seemed to be sufficient ulceration present to account for his symptoms, and he was treated by irrigation and injections upon this assumption. After a few weeks, however, although the ulcers had healed, he was still passing blood and mucus in considerable quantities. He was again examined with the sigmoidoscope, and on this occasion a cancerous ulcer was detected very high up in the sigmoid flexure. This was subsequently excised by Mr. Mayo Robson. This case shows the importance of a thorough and careful examination, and that the presence of definite

ulceration in the bowel does not exclude the possibility of a more serious lesion at a higher level.

It will be observed by reference to the table of cases, that in the majority of cases of colitis the lesions discovered, on examination, were of a chronic inflammatory nature. This is opposed to the views expressed by Nothnagel, who believes colitis to be, in most cases, a neurosis.

In my series of cases a definite inflammatory condition of the mucosa was found in 24 out of the total of 36 cases, or in about 71 per cent. The appearance of the inflammatory condition found varied very considerably in the different cases and varied sometimes in the same case at different times.

In all the cases the inflammation was of a chronic character and appeared to involve only the mucous membrane or submucous cellular tissue.

The most usual appearance was for the mucous membrane to be injected and bright red; the normal mucous membrane is of a pale pink colour. The surface was dull, having lost its normal glistening appearance, and often granular. This granular condition closely resembles that seen in granular pharyngitis. In many of the cases the mucous membrane looked as if its surface had been rubbed off with sand-paper.

It bled readily if touched with the instrument and bleeding spots could often be seen apart from this.

Scattered about on the mucous membrane there were irregular patches of yellowish-white adherent mucus which often gave a very characteristic appearance to the bowel.

Often a very extensive area of bowel was affected and in some cases the whole of the visible portion of the sigmoid flexure was involved. The inflammation was always most marked at the flexures and on the valves of Houston.

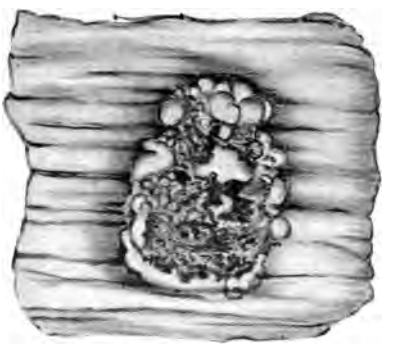
In some of the cases, more especially when an examination was made during acute attacks, the condition resembled an extensive superficial ulceration of the mucosa.

In addition to the granular condition of the mucous

membrane there was definite ulceration in seven of the cases. The ulcers were irregular in outline, quite shallow, and with a granular base. These ulcers were often very numerous, and sometimes extended throughout the entire sigmoid flexure and rectum (see plate).

To turn to the cases in which ulceration is found: The degree of ulceration which may be found in a case with symptoms of colitis varies very considerably. Thus, in some cases I have seen a few shallow ulcers at irregular intervals situated chiefly in the rectum, or in the lower portion only of the sigmoid flexure.

In other cases these ulcers were so numerous as to cover



Specimen of cancer of the sigmoid excised in Case 35.

most of the surface of the rectum and sigmoid, the normal mucosa standing out in contrast to the background of ulceration. Even in such cases, however, the ulceration may be so shallow as to involve the superficial layers only of the mucosa, and it is important to distinguish between this variety and that in which the ulcers are numerous and deep. The latter is a much more serious condition; the ridges of healthy mucous membrane between the ulcerated areas here appear like polypi, so prominently do they stand up from the surrounding ulcerated areas.

As a rule, ulceration is found in those cases of colitis where severe symptoms exist, but this is not always the case, and I have seen well-marked ulceration in cases where

the symptoms were mild, and no ulceration in cases where the symptoms would lead one to suppose the severe ulceration was present. The ulcers, as a rule, in the more chronic cases are found at those points where local irritation or traumatism is likely to occur, thus, at the recto-sigmoidal junction, in the bottom of the sigmoid pouches, and on the surfaces of the rectal valves.

These shallow ulcers must be distinguished from the ulceration found in cases of acute ulcerative colitis or dysentery. In the latter conditions the whole thickness of the mucosa is destroyed and if healing occurs there is of necessity considerable scarring.

In one case I had the opportunity of examining a patient who had recovered from a severe attack of dysentery and the white stellate-looking scars left by the healed ulcers could be seen all over the rectum and sigmoid flexure.

In the shallow form of ulceration, however, which I have seen associated with chronic colitis, the ulcers readily heal with suitable local treatment and no scarring results.

The symptoms during an acute attack may, however, be very severe, and it is important to distinguish this form of ulceration from the deeper and more serious ulceration, as the prognosis, while good in the former, is not so favourable in the latter.

Case 36 is of particular interest. The patient was a gardener, aged 41, a patient of Dr. Baynton Forge's. He had always been a healthy man, and with the exception of one or two slight attacks of diarrhœa had had no serious illness until lately. In July, 1906, after a period of overwork, he was suddenly taken ill with severe diarrhœa. This got worse and he began to pass blood and mucus in considerable quantities. In spite of rest and treatment he steadily got worse. He was for some weeks in Maidstone Hospital but there was no material improvement in his condition. When I first saw him, in February of this year, he was passing eight or nine stools in the day, and there was much mucus and blood. He had lost two stone in weight, and his complexion was pale and sallow. I took

him into St. Mark's Hospital and examined him with the sigmoidoscope under an anæsthetic. The whole of the mucous membrane of the rectum and the lower part of the sigmoid flexure was rough and very coarsely granular in appearance, suggesting the appearance seen in a very well-marked case of granular pharyngitis. There was extensive ulceration, the ulcers being irregular in outline and shallow, involving only the superficial layers of the mucosa. Above the centre of the sigmoid the ulcers were much less numerous. On digital examination some enlarged glands could be felt in the meso-rectum, and abdominal palpation revealed a distinct thickening in the left iliac fossa. He was treated by large oil injections into the colon and irrigation with potassium permanganate 1–1000 for a fortnight. Under this treatment the ulcers healed and all the symptoms cleared up, but as there was still a distinct thickening in the left iliac fossa I did not feel certain that there was not a growth high up in the sigmoid flexure which could not be reached with the sigmoidoscope. I therefore thought it advisable to do an exploratory laparotomy. On opening the abdomen I found there was no growth, but the walls of the sigmoid flexure were very much thickened, so much so that the bowel in places felt like a thick india-rubber tube; there were also numerous enlarged glands in the meso-sigmoid. In the centre of the sigmoid flexure there was a thick band of fibrous adhesion about three-quarters of an inch broad binding down the sigmoid flexure into the left iliac fossa and causing a kink in the bowel (see plate). The transverse colon was normal as were also the cæcum and appendix. All the other organs were normal. I divided the fibrous band and closed the abdomen. After he had got over the operation the previous treatment was continued, and he was discharged apparently quite well at the beginning of April. With the sigmoidoscope it could be seen that all the ulcers had healed, though the surface of the mucosa was still rough and granular, but decidedly better than when he was first examined.

In four cases there was a chronic hypertrophic catarrh associated with œdema of the submucous tissue. On examining such a case with the sigmoidoscope the mucosa is seen to be much paler than normal and to be thrown into numerous folds or concentric rings. The mucous membrane bulges forward into the bowel lumen, giving the appearance of a partial intussusception at the point examined.

The mucous membrane is thickened and œdematous, though its surface retains its normal shiny appearance. The sigmoidoscopic appearances are very characteristic, and cannot be mistaken.

This condition was associated in all my cases with excessive secretion of gelatinous mucus.

Severe pruritus was present in two of the cases, and Case 6 consulted me on this account. This patient got better after regular irrigation of the bowel with an alkaline lotion and the use of astringent injections of krameria, and his pruritus, which had existed for many years, was cured.

I have seen this hypertrophic catarrhal condition of the mucosa in two cases of pruritus, in which the patient did not give a history of colitis.

Cancer as a Cause of Colitis.

In seven (Cases 3, 18, 19, 21, 32, 34, 35) out of my series of thirty-six cases the colitis was due to a tumour within the bowel.

In all of these cases there was considerable doubt about the diagnosis previous to the discovery of the tumour, and several of the patients had previously been under treatment for colitis. In looking through my cases of cancer of the rectum I find there are several in which the symptoms were those of colitis rather than cancer, but I have not included them as there was at no time any doubt about the diagnosis, as a digital examination revealed the true state of affairs.

It is impossible, from the symptoms alone, to distinguish

those cases of colitis which are due to cancer from the other forms. The presence of blood in the stools is of no value. In Cases 18 and 19 there was no blood, and only a very small amount of blood on two occasions in Case 21.

Definite mucus casts may be passed in cases of cancer, as is shown by Cases 3 and 21. In both these cases typical mucus casts had been passed on several occasions.

The age of the patient does not help us much, as in Case 18 the patient was only forty-two, and only two of the others were over fifty-five; while the average age for all the cases is forty-three.

The time during which the symptoms have existed will not exclude cancer, as in Case 19 symptoms had existed for one and a half years, and in Case 3 for five years.

Case 3 is a remarkable one. The patient had had symptoms of a severe colitis for five years; she had passed mucus casts of the bowel after acute attacks of abdominal pain. She had laparotomy performed twice. What was found on the first occasion I do not know, but at the second operation an adherent ovarian cyst was removed. When I saw her she had a malignant growth at the upper end of the sigmoid flexure. A short-circuiting operation was subsequently performed by another surgeon, but she died from intestinal obstruction shortly afterwards.

In Case 18 a large growth in the sigmoid flexure was discovered on examining the patient with the sigmoidoscope. I subsequently performed colotomy, after finding that the growth could not be removed.

In Case 19 I removed the growth, which was a large papilloma becoming malignant at its base, and the patient has remained well since—a period of over two years.

In Case 21 the growth was inoperable.

In Case 32 the growth was seen high up in the sigmoid flexure through the sigmoidoscope. The patient, unfortunately, refused operation.

In Case 34 the growth was situated 22 cm. from the anus. It was seen through the sigmoidoscope and could not have been diagnosed otherwise. This patient had

numerous small ulcers scattered over the mucous membrane of the rectum and sigmoid flexure below the growth. Mr. Mayo Robson subsequently excised the growth from the abdomen and anastomosed the ends of the bowel.

Case 35 is of particular interest. The patient was an exceedingly neurotic man, aged 46, who was sent to me by Dr. Forge, of West Malling. For nearly two years he had been under treatment by different doctors for ulcerative colitis, and during that time he had been passing blood and mucus in his stools almost continuously. There was no constipation and no difficulty in keeping the bowels open at any time. He had been putting on weight slowly for about two months before I saw him. On examining him with the sigmoidoscope I found a small malignant growth, about the size of a shilling, growing from the anterior wall of the sigmoid flexure about six inches above the upper end of the rectum. The mucous membrane above the growth was inflamed and resembled the condition seen in non-malignant cases of colitis.

I excised the growth and a V-shaped portion of the adjacent mesentery from the abdomen and joined the ends of the bowel end to end by simple suture; the abdomen was closed without drainage. He made a good recovery and returned home in good health about one month after operation. A microscopical examination of the growth showed that it was an ordinary medullary carcinoma.

Among the rarer causes of chronic colitis which have been recorded mention must be made of actinomycosis of the colon, two cases of which are recorded by Beck.

Another rare local condition is tuberculosis, of which seven cases are recorded by Beck.

Multiple Adenomata of the Large Intestine.

This is a very curious condition and only a comparatively small number of cases have been recorded. These adenomata are present in great numbers; in some cases the entire mucosa of the large bowel is covered over with

them; in some of the cases only a portion of the bowel is affected, the commonest situations being the sigmoid flexure and rectum. The symptoms are those of a severe colitis. There is often much blood in the stools, but in some of the cases there has been none.

One of the most remarkable cases of this disease was that reported by Lilienthal; the whole colon was affected, and the patient was ultimately cured by excision of the entire large intestine.

A case was reported recently by Dr. Mabyn Read, and the specimen of a case was shown at the Leicester Meeting of the British Medical Association in 1905 by Mr. Stanmore Bishop. Mr. Carey Coombs has also recently reported a case.

Causes of Colitis Outside the Bowel.

Colitis may result from adhesions due to an acute pericolic inflammation. Cases of acute pericolitis are not uncommon, and several well-authenticated cases have been described, notably by Dr. Rolleston and Mr. D'Arcy Power.

In one of my cases the patient had an acute attack of abdominal inflammation, and a large abscess in connection with the sigmoid flexure and rectum was found and opened through an abdominal incision. Shortly afterwards she commenced to suffer from well-marked symptoms of mucous colitis, which increased up to the time I saw her three years after the operation. Everything pointed to the adhesions around the sigmoid flexure and binding it down into the pelvis as the cause of her symptoms.

Uterine displacement may be a cause of colitis by interfering with the action of the sigmoid flexure and the evacuation of its contents into the rectum. A retroflexed uterus acts like a ball-valve in blocking up the posterior pelvic outlet, and causes a very considerable degree of obstruction in some cases. In Case 27, in which laparotomy was performed for colitis, the retroflexed uterus could be seen to block the posterior pelvic outlet almost completely,

and the sigmoid flexure above the point of constriction by the uterus was greatly distended and hypertrophied. Ventro-fixation of the uterus resulted in considerable improvement in this patient's condition.

B. V. Beck, out of 500 cases of chronic colitis, found chronic inflammatory lesions in the neighbourhood of the colon in 394.

Colitis and Appendicitis.

It is difficult to say exactly what is the association between appendicitis and colitis. The most usually accepted view is that colitis may result from a chronically inflamed appendix. Dr. Caley has expressed the view that appendicitis often results from colitis. The probability would seem to be that both appendicitis and colitis result from the same causes, namely, incorrect dieting and constipation.

That appendicitis can be the cause of colitis is, I think, unquestionable. The evidence in favour of this view is very strong.

A certain degree of colitis is usually present in all cases of acute appendicitis, though no definite symptoms of the conditions may show themselves.

In exactly what way appendicitis causes colitis we do not know, but it seems probable that it may produce it in several ways.

(1) Appendicitis is generally accompanied by inflammation of the ascending colon and cæcum, and in some cases this inflammation may spread into the transverse colon and even reach the sigmoid flexure. As Mr. Lockwood has pointed out, it is not uncommon during the operation for appendicitis to find that the inflammation has spread into the ascending colon.

(2) Appendicitis may also cause colitis by the formation of adhesions between the appendix or cæcum and the sigmoid flexure. These adhesions, by interfering with the lumen of the gut, either through direct constriction or by causing abnormal flexures and angles, may result in a local inflammatory process being set up, and the symptoms

of colitis subsequently develop. The first result of such adhesions is usually constipation, to be followed in a shorter or longer time by diarrhœa and the passage of mucus. This occurred in Case 17. On opening the abdomen I found the appendix adherent at its tip to the sigmoid flexure, and several adhesions binding down the sigmoid and preventing it from rising out of the pelvis. Considerable improvement followed removal of the appendix and separation of the adhesions.

(3) Thirdly, it would seem probable that a chronically inflamed appendix may cause symptoms of colitis by the frequent discharge of septic material into the bowel. It has become customary to describe the colon and rectum as septic channels, though there is no justification for this. As a matter of fact, septic organisms are not at all unusually numerous in the large intestine, and the pathogenic flora of the mouth is far more numerous than that of the rectum and colon. Even the skin is a more common habitat for septic organisms than the colon.

It is not surprising, therefore, that a chronically inflamed appendix which is continually discharging septic organisms into the bowel, may be the cause of colitis, just as we know carious teeth and septic conditions of the mouth can cause gastritis.

We have very positive evidence that removal of an inflamed appendix will, in many cases, lead to a complete cure of colitis.

In Mr. Lockwood's paper already referred to he records three such cases where the symptoms of colitis cleared up entirely after the removal of an inflamed appendix.

Dr. Hale White, in his valuable paper on membranous colitis, records four cases in which the appendix was removed for symptoms of colitis. In only one of these, however, did a cure of the colitis follow the operation. It is noteworthy, however, that the successful case is also the only one of which it is recorded that the appendix was found to be diseased.

Hr. Beck found a chronically inflamed appendix to be the

cause of colitis in a considerable number of cases of chronic colitis, and believes it to be a common cause of colitis. Tuttle records five cases of chronic colitis cured by the removal of an inflamed and adherent appendix.

Ohmsted, of Ontario, records a case of severe mucous colitis in which rapid improvement followed the removal of a congested appendix, and Amatt, of Ontario, records two similar cases.

Adhesions of the Bowel as a Cause of Colitis.

There is every reason to believe that adhesions binding down the large bowel and interfering with its normal movements are an important factor in the causation of colitis. In a great many cases of severe and intractable colitis for which an exploratory laparotomy has been performed, adhesions binding down the sigmoid flexure, and more rarely the cæcum or splenic flexure, have been found, and their division has been followed by a cure of the condition; though not infrequently the adhesions have re-formed at a later date with a recurrence of the old symptoms. These adhesions have been supposed to arise from long-continued constipation, but it is difficult to understand how adhesions can arise from constipation alone, and it seems much more probable that the presence within the bowel of hard, retained fæcal masses has set up a local inflammation, or definite ulceration, and that the adhesions to neighbouring parts has resulted from the local peritonitis thus caused. This seems all the more likely, as adhesions are usually found in those portions of the bowel, such as the sigmoid, ascending colon, and cæcum, where retention of fæcal material most easily occurs.

I believe local, simple ulceration of the mucosa of the large bowel is much commoner than is generally supposed. I have often observed simple ulcers in the flexure of the sigmoid with the sigmoidoscope in cases of chronic constipation, though no symptoms of ulceration were present.

The adhesions interfere with the normal peristaltic move-

ments of the colon, and a chronic catarrhal inflammation of the mucosa is set up at the points where adhesions have formed.

A floating kidney has been supposed to cause colitis, and Tuttle records two cases apparently cured by fixation of an exceptionally mobile kidney. In those cases of colitis associated with movable kidney which have come under my observation I have seen no reason to suppose that the condition of the kidney was in any way the cause of the colitis.

It is interesting to observe that symptoms of colitis may occur in cases of abdominal aneurysm. In such cases the colitis apparently results from the pressure of the aneurysm upon some contiguous portion of the large bowel. Tuttle records six cases of abdominal aneurysm in which there was well-marked and intractable mucous colitis.

In six of my cases no local cause for the colitis could be discovered, and in two cases (Cases 25 and 30) the local lesion did not seem sufficient to account for the symptoms.

In Case 29, in which no local lesion was found, the subsequent history of the patient pointed to the presence of a malignant growth high up in the colon, but I have since heard that he is still alive, though not any better, one and a half years after he was examined.

In Case 25 it seemed probable that the chief lesion was higher up in the sigmoid flexure than we were able to see.

There are thus only six cases out of thirty-six in which there was no sign of any local lesion. And when we consider the very large part of the colon which cannot be examined it seems at least probable that there was a definite lesion in that part which could not be examined.

Treatment.

The most important factor in the treatment of colitis is undoubtedly to ascertain first of all the real cause of the condition. And the reason the treatment of colitis has

often, in the past, been unsatisfactory, is undoubtedly due to inaccurate diagnosis, and treating as a disease what is only a symptom.

In the inflammatory conditions rest in bed is advisable at the commencement of treatment. This is more particularly of value in enabling the patient to be kept under close observation.

Irrigation of the bowel seems to be the best method of treatment if carried out properly, and the inflammatory condition as a rule quickly subsides.

A skilled nurse who understands how to get the irrigating fluid right up into the colon is essential.

The irrigation must be done with a douche and not with a syringe. The reservoir should never be raised more than one or two feet above the anus and the fluid must be admitted slowly, with the patient in the semi-prone position.

If the condition is at all acute it is better not to begin with irritant injections, an alkaline lotion, such as glycerine thymol co. 15 per cent., or sodium bicarbonate one drachm to the pint, is very good, or if there is much ulceration hydrogen peroxide.

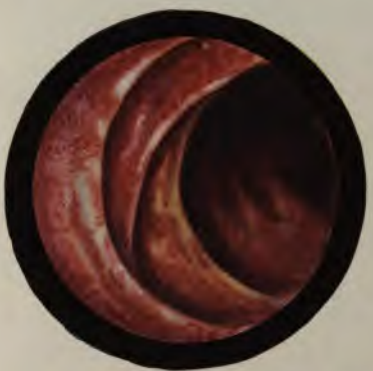
Later, argyrol 1 per cent. or potassium permanganate 1 in 2000 are very useful. Large injections of olive oil, carefully administered and retained for as long as possible, I have found most useful in some of the ulcerative cases.

Irrigations are, however, quite useless unless care is taken to see that they reach the affected area—by no means always an easy matter.

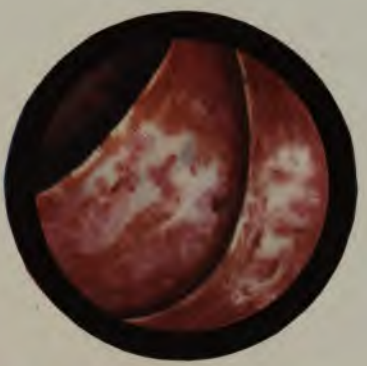
In some cases where there is local ulceration the ulcers can be painted with nitrate of silver or ichthyol through the sigmoidoscope.

Dieting is also very important, and the great thing is to get the bowels to act normally. Von Noorden's diet seems the best in most cases.

The most difficult cases to treat would seem to be those in which there is hypertrophic catarrh. They are easily made better, but not easily cured. Stimulating injections

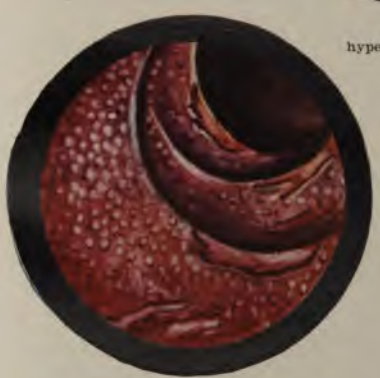


Chronic granular colitis.

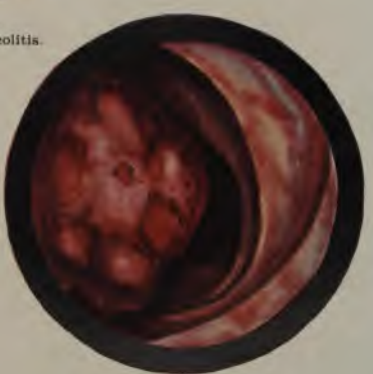


Chronic catarrhal colitis.

Chronic hypertrophic colitis.



Chronic granular and ulcerative colitis.



Ca ıcer in the sigmoid flexure, causing symptoms of chronic mucous colitis.

The interior of the sigmoid flexure as seen through the sigmoidoscope in cases of chronic colitis.

Leonardson & Co.

For other illustrative drawings, etc., see 'Medical Annual,' 1906; Allbutt's 'System of Medicine,' vol. iii; 'Clinical Trans.,' vol. xxxviii (1905), p. 282.

are of the most value in these cases, and treatment must be continued for some time.

In the malignant cases operation is, of course, indicated where possible.

In those cases where colitis is associated with a thickened and tender appendix removal of the appendix, so as to get rid of a source of infection, is certainly indicated.

In some of the cases of severe ulcerative colitis giving rest to the bowel by establishing a temporary artificial anus may be necessary, but I do not think this will often be the case if the local treatment of the colon by irrigations and applications through the sigmoidoscope is carefully carried out. Before a left inguinal colotomy is performed for colitis a careful examination with the sigmoidoscope should always be made to ascertain whether it is possible to open the bowel above the diseased area.

The chief advantage of appendicostomy is that it enables the patient to readily perform irrigation of the colon for himself.

In those cases of colitis in which no local cause can be found to account for the symptoms, and in which no material improvement in the symptoms has followed a careful and thorough course of medical treatment, I think surgical interference is indicated. Laparotomy should be performed and a thorough examination made of the large intestine; any adhesions which may be found binding down the bowel should be divided; if the sigmoid mesentery is unduly lax, allowing partial volvulus to occur, it should be shortened by stitching. If there is a retroflexed uterus it should be fixed in the normal position, and if any thickening or ulceration of the bowel wall can be detected the advisability of resecting that portion of the bowel or of short-circuiting it must be considered.

I do not think that the operation of ilio-sigmoidoscopy is advisable except in a few exceptional cases. When very extensive adhesions or ulceration in the large bowel are discovered, which it does not seem probable can be permanently remedied, this operation should be considered,

	ge.	Sex.	Symptoms.	Duration.	Condition found.	Remarks.
	50	Male	Mucus, bleeding, attacks of pain	3 years	*Nil*	—
	46	Female	Severe attacks of pain, mucus casts, intestinal sand	9 months	*Nil*	—
	51	„	Severe attacks of pain, mucus casts, bleeding last twelve months	5 years	Cancer of sigmoid flexure	Inoperable, died.
	46	„	Mucus and diarrhœa	Some years	*Nil*	—
	22	„	Continuous diarrhœa, mucus	10 years	Mucous membrane red and congested, upper limits not seen	—
			Excessive mucus in stools, pruritus	6 „	Hypertrophic catarrh, œdema of submucosa, upper limits not defined	—
		„	Blood and mucus	3 months	Hypertrophic catarrh, upper limits not seen	—
	78	„	Blood and mucus, severe diarrhœa	1 month	*Nil*	Relapse 5 months later.
9	26	„	Bleeding, mucus, and diarrhœa, severe pain at times	3 years	Hypertrophic catarrh and ulceration	Had been operated on for piles three times within 2 years.
10	35	Female	Large mucus casts, mucus constantly present in stools	2 „	Adhesions binding down sigmoid flexure; 2½ years ago a large abscess in the back of the pelvis was opened and drained from the abdomen; a normal appendix had previously been removed	—
1	45	„	Mucus casts, bleeding	4 „	Granular chronically inflamed condition of mucous membrane, most marked in rectum, but upper limits not defined	—

No.	Age.	Sex.	Symptoms.	Duration.	Condition found.	Remarks.
12	23	Male	Bleeding, diarrhœa and spasmodic pain, much mucus	6 years	Mucous membrane granular and inflamed, several small polypi seen high up in sigmoid	Very difficult patient to examine properly.
13	35	„	Diarrhœa and much mucus	3 months	*Nil*	—
14	17	Female	Mucus and bleeding	3 years	Hypertrophic catarrh confined to rectum and lower part of sigmoid	—
15	32	Male	Constant diarrhœa, much mucus	8 months	Mucous membrane inflamed and granular, numerous shallow ulcers, mucosa normal above middle of sigmoid	Relapse 5 months later.
16	32	Female	Diarrhœa and large quantities of mucus, bleeding occasionally	3 years	Inflamed and granular condition of mucous membrane, upper limits not defined	Rapid recovery, relapse 7 months later.
17	29	„	Pain, diarrhœa, and mucus	1 year	Mucosa much inflamed, narrowing of bowel seen high up, laparotomy revealed adhesions and appendix adherent to sigmoid	Patient had suffered from gonorrhœal proctitis previously.
18	42	„	Diarrhœa, large quantities of mucus	3 months	Cancer of sigmoid flexure	Inoperable.
19	63	Male	Diarrhœa, much mucus, no bleeding	1½ years	Papilloma high up in rectum, becoming malignant at base	Excision.
20	39	Female	Diarrhœa, mucus casts, entero-spasm, intestinal sand	10 „	Catarrhal inflammation of mucosa, upper limits not defined	—
21	52	Male	Much mucus, mucus casts, bleeding on one or two occasions	6 months	Cancer in sigmoid flexure	—
22	65	„	Diarrhœa, much mucus, some bleeding	6 „	Mucous membrane congested	—
23	30	„	Diarrhœa, bleeding, much mucus	3 years	Extensive shallow ulceration, most marked in rectum and lower sigmoid	—

No.	Age.	Sex.	Symptoms.	Duration.	Condition found.	Remarks.
24	28	Male	Diarrhœa, much mucus, bleeding	3 years	Shallow ulcers, mucosa granular, upper limits not defined	—
25	45	„	Diarrhœa, much mucus, bleeding occasionally	Several years	Catarrhal inflammation in rectum and sigmoid, bowel narrowed by adhesions	Local condition hardly accounts for symptoms.
26	40	„	Diarrhœa and mucus	1½ years	Granular condition of mucosa, catarrhal inflammation	—
27	39	Female	Mucus casts	3 „	Hypertrophic catarrh, retroflexed uterus	Ventro-fixation.
28	37	„	Mucus casts, bleeding	15 „	Catarrhal inflammation of rectum and sigmoid	—
29	58	Male	Diarrhœa and mucus	8 months	*Nil*	—
30	41	„	„ „	2 years	Slight catarrhal inflammation of mucosa in sigmoid	Condition hardly accounts for symptoms.
31	40	„	Diarrhœa, mucus casts, bleeding	3½ „	Chronic inflammation and ulceration in rectum and lower part only of sigmoid	—
32	53	„	Diarrhœa, mucus, and bleeding	6 months	Cancer in sigmoid flexure	Operation refused.
33	50	„	Ditto	9 „	Catarrhal inflammation of sigmoid and rectum, piles	—
34	65	„	Ditto	1 year	Ulceration in sigmoid flexure, and cancer higher up in sigmoid	Excised.
35	44	„	Ditto	2 years	Cancer of sigmoid flexure	Growth excised.
36	41	„	Diarrhœa, mucus, and bleeding (severe)	7 months	Ulceration in sigmoid, mucous membrane very granular, the walls of the sigmoid were very much thickened	Exploratory laparotomy revealed stout adhesion band holding down sigmoid and enlarged glands in mesentery, no growth.

DISCUSSION

Dr. HALE WHITE: I think the first point that calls for criticism in Mr. Mummery's paper is the name of it. He uses the word "colitis" without any reference to the fact that there are many kinds of colitis, such as acute, chronic, ulcerative, and follicular, but from what he said he rather inferred that all the cases he described were what is commonly known as membranous colitis. But that certainly is not so; membranous colitis is a definite clinical entity: the patients are nearly always women—more than half Mr. Mummery's cases were men. These women pass long tubes or casts from the bowel; very few of Mr. Mummery's cases did this. Then, also, these women frequently have some variety of enteroptosis, commonly a floating kidney. They frequently suffer from disease of the pelvic organs, and often have that curious complaint, membranous dysmenorrhœa. I think that more than one variety of colitis is included in Mr. Mummery's series, and that very few of them are of the membranous variety. I do not think that the membranous variety is specially confined to the sigmoid as he suggests, for there is frequently a good deal of pain and tenderness along the whole colon. The mere presence of mucus in the stool certainly ought not to be regarded as evidence that the patients are suffering from membranous colitis, and probably several varieties of inflammation of the rectum and sigmoid are included in Mr. Mummery's series. It seems to me, however, a great advantage to us that he has brought forward the use of the sigmoidoscope. It is a very easy instrument to use—much more easy than the ophthalmoscope or the laryngoscope; and further, I quite agree with him that it is very necessary always to see in any case of colitis, of whatever variety, whether there is not some organic disease which sets it up. With most cases of membranous colitis no such organic disease will be found, but sometimes, as I have often seen, grave organic disease is present and is frequently overlooked.

Sir DYCE DUCKWORTH: I find myself in agreement with Dr. Hale White as to the somewhat specific character of these cases, which occur chiefly in women, and are attended by the formation of mucous casts, sometimes in large quantity. In these cases there is commonly an associated condition of nervous instability. I have always regarded these cases as dependent on catarrh of the large bowel, somewhat akin to those of plastic bronchitis. The disorder is certainly not limited to the sigmoid colon, but probably involves all the large bowel. The association of mucous casts with cancer, as described by Mr. Mummery, must be infrequent, but I believe that it has not hitherto been recognised.

It should be borne in mind that there are various grades of intensity of the process which determines this formation of mucous casts and their consistency. The more severe of these are rebellious to all treatment, others yield to a general improvement of health, and the employment of large enemata of borax solution. Many of these cases are benefited by the treatment of ascending douches as practised so well at Plombières. I quite agree with Mr. Mummery that in prolonged, unyielding cases the bowel should be examined, and physicians will only too gladly avail themselves of the new instrumental aid which he has so well described to-night.

Dr. F. PARKES WEBER: I have listened with great interest to Mr. Lockhart Mummery's paper. The point, however, which I wish specially to remark on, is the great frequency of colitis in the broadest use of the term. I believe that some degree of chronic catarrh of the colon is of as frequent occurrence as chronic nasal catarrh, and this is supposing the frequency to be very great. I doubt whether the cause of chronic colitis is generally a local one in the surgical sense. Conditions of the local circulation of blood, and naturally the state of the blood-vessels and lymphatics in the wall of the bowel, have probably much to do with the presence or absence of chronic colitis, and thus also the frequently beneficial effects of improving the local and general circulation by muscular exercise and massage can readily be understood. It seems to me that some forms of chronic colitis may be compared to the disturbed nutrition of the skin (pigmentation, "varicose eczema," and chronic ulceration) of the legs resulting from varicose veins and imperfect circulation in the lower extremities. Recurrent attacks of "biliousness," whether connected with constipation, dietetic errors, or climatic influences (for instance, a visit to some relaxing sea-side locality [1]) are often, I believe, to be regarded as exacerbations of chronic colitis, the increased activity of the intestinal flora leading to symptoms of auto-intoxication. In all such cases the great and generally-recognised value of aperients containing calomel is probably in great part to be attributed to the action of calomel as an intestinal antiseptic. On the other hand, in cases of chronic colitis with constipation energetic action of the bowels induced by purgatives is often at first followed by a feeling of lassitude and great nervous irritability.[2] This preliminary period of depression has, according to some observations,

[1] *Cf.* F. Parkes Weber, "On the Biliousness sometimes induced by Sea-air"; 'Treatment,' London, January 11th, 1900.

[2] Hence some more or less neurasthenic persons, who are accustomed to have their bowels opened by aperients on alternate days, say that they feel at their best, both more capable for work and with less of their usual mental condition of "irritable weakness," on the intermediate days, that is to say, on the days when their bowels are not opened.

been found to correspond to a temporary increased absorption of putrefactive products of intestinal origin (evidenced by examination of the urine), immediately following purgation. In certain cases in which "biliousness" is accompanied by pyrexia and influenza-like symptoms, I suspect that auto-intoxication from the absorption of toxic products of bacterial action is accompanied by an actual emigration from the large intestine of some of the bacteria themselves, a kind of true temporary "septicæmia" of intestinal origin. In regard to the passage of intestinal bacteria from the intestine into the blood-stream, Garnier and L. E. Simon ('Société de Biologie,' Paris, June 1st, 1907) have proved its occurrence as a result of certain dietetic experiments in animals. In conclusion I wish to emphasise my belief that the subject of chronic colitis embraces a much wider field than the class of cases specially referred to in Mr. Lockhart Mummery's interesting communication.

Dr. P. J. CAMMIDGE: I have listened to Mr. Mummery's paper with great interest, and particularly to that portion of it in which he referred to the symptomatology and differential diagnosis of the conditions with which chronic colitis may be associated, as that is the aspect of the subject with which I am best acquainted, and of which I have had the most practical experience. In speaking of the symptoms, Mr. Mummery referred to the microscopical and bacteriological examinations of the stools, but omitted to consider—what in my opinion is of far greater importance—the chemical examination of the fæces. In my experience, now extending to some forty cases of colitis, the results of a quantitative chemical examination afford very conclusive and reliable data on which to work, while microscopical and bacteriological investigations, although often supplying confirmatory evidence, are frequently inconclusive or even misleading. The fæces in cases of colitis are always alkaline in reaction, often very strongly so, and a gravimetric estimation of the fats, by the method I described in the 'Brit. Med. Journ.,' October 28th, 1905, p. 1102, shows that while the total amount of fat may or may not be in excess of the normal, the relation between the "neutral fats" and "combined fatty acids" is nearly always considerably disturbed, the combined fatty acids being in excess. A still more important point, however, is the presence of a high proportion of inorganic ash. In normal persons, and in most diseases not involving the lower bowel, this rarely exceeds 15 per cent. of the dry weight, but in colitis it generally lies between 19 per cent. and 25 per cent., and may even be as high as 44 per cent., as I found it in one of my cases, which, however, it is interesting to note, was not associated with any gross deposit of intestinal sand. I cannot agree with Mr. Mummery that blood is not constantly present in the fæces in malignant disease of the colon. It is true that it is frequently

not to be discovered by inspection, or on microscopical examination, but in all the specimens from five cases of cancer of the colon that I have recently examined occult blood was found by chemical means. The discovery of blood in the fæces is not, of course, diagnostic of cancer, for it may originate from simple ulcers, piles, etc., but the blood-flow from these is intermittent, whereas in cancer every sample of the fæces contains some blood. The constant presence of occult blood in the stools is, I think, strongly suggestive of malignant disease of some part of the gastro-intestinal tract, and when this is associated with the other characters of the fæces that I have mentioned the probability is that the growth is situated in the colon, as my cases have shown. I should like to ask Mr. Mummery whether in those cases in which blood was said to be absent he was relying on naked-eye inspection or microscopical examination alone, or whether a chemical analysis for blood was made.

Dr. Sidney Phillips: I think a good many of Mr. Mummery's cases might be termed cases of growth in the colon rather than cases of colitis, though they may be accompanied by secondary colitis; and the first point in the diagnosis of any case is to ascertain if possible if any growth be present. The cases recorded by Mr. Mummery are rather cases of sigmoiditis than of ulcerative colitis generally, and though the sigmoidoscope will be of no service in the diagnosis of those cases in which the inflammation is in the transverse or ascending colon, the transverse colon lies in close contact with the lower border of the stomach, and ulceration or disease of this part of the colon produces symptoms easily referable to the stomach. I think that ulceration of the colon is attributed to constipation more frequently than is justified, and information is greatly needed as to other causes of colitis.

Dr. H. A. Caley said: I, too, consider that some of the alleged causes of colitis are probably consequences, *e.g.* the hypertrophic condition of the mucous membrane. The evidence that adhesions are a cause of colitis is unsatisfactory; they are probably the effect of colitis and of a subsequent pericolitis. The balance of evidence seems in favour of the view that appendicitis is more often the sequel than the antecedent of colitis. The paper is, however, of great value in helping to establish the view that local lesions are often associated with colitis.

Mr. Mummery, in reply, said: I would again emphasise the point that in some cases of malignant disease of the colon the symptoms are like those of membranous colitis. None of my cases were acute as ordinarily understood. No chemical or microscopical examination was made in the cases where no blood was seen. There was no evidence in my cases of a syphilitic nature of the ulcers. The presence of intestinal sand seems to make the case much more serious. As the ulcers heal up under

irrigation, the symptoms disappear and do not return unless ulcers again form. Adhesions probably act rather in maintaining the condition than in causing it. The removal of an appendix not curing the colitis does not prove that it is not the cause of the colitis.

ON
VOLVULUS OF THE ENTIRE SMALL INTESTINE, CÆCUM, AND ASCENDING COLON
OPERATION AND RECOVERY
WITH AN ANALYSIS OF NINE OTHER CASES OF COMPLETE VOLVULUS

BY

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THE cases of this form of volvulus, though few in number, are of special interest. They represent the greatest amount of twisting of the intestine than can occur, and the condition is only rendered possible by an extremely loose attachment of the commencement of the large intestine to the posterior abdominal wall. This condition of floating cæcum and ascending colon is certainly congenital, and it is noteworthy that most of the cases of complete volvulus have occurred in young subjects, eight out of ten of the patients being under twenty-five years of age. The floating condition of cæcum and ascending colon cannot, however, be said to cause the volvulus, although the former must exist to allow of the occurrence of the latter, just as torsion of the spermatic cord can only occur in patients who have a

free mesorchium. It is well known that the great majority of cases of torsion of the cord and testis are seen in young lads, but now and then it is met with in adults. So, whilst three of the recorded cases of complete intestinal volvulus were in infants under four weeks old, a fact which points strongly to the favouring influence of the congenital looseness of cæcum and colon; one patient was fifty-five years of age, and another no less than seventy-two. This last patient and my own, a young man aged twenty-two, were the only ones who recovered out of the ten cases hitherto recorded.

It will be understood that by the term complete volvulus is meant an axial rotation of all the intestine that can possibly become twisted, *i. e.* from the duodenum to the ascending colon; this rotation amounts usually to 360 degrees from right to left. By this rotation the cæcum is carried behind the mesentery into the left side of the abdomen, where it may be found in the iliac fossa, or as in my case and several others, as high as the spleen. This remarkable position of the cæcum may be accompanied by an extraordinary amount of distension, the twisted coils of small intestine becoming full of fluid, which can pass on into the cæcum, whilst their weight drags them into the pelvis and causes their stretched mesentery to act as a tight band which crosses the displaced colon, preventing the contents of the cæcum from passing any further.

With regard to this complete volvulus it is the displacement and distension of the cæcum that deserves special mention, distinguishing it from all other forms of volvulus, and rendering both diagnosis and treatment at the time of the operation of special difficulty.

Case 1.—A lad, aged 22, was sent to me at the London Hospital by Dr. Spencer Wicks in September, 1901, on account of a large cyst at the back of the abdomen (developing in the lesser sac of the peritoneum). On the very day that I had arranged to operate on this cyst it

ruptured spontaneously, and laparotomy was deferred. Next day symptoms of intestinal obstruction commenced and rapidly developed, there was frequent vomiting and increasing abdominal distension. This distension was most marked in the left hypochondrium, where there was a large resonant mass which proved to be distended cæcum. Enemata had no result on the obstruction. A median laparotomy was performed. On opening the abdomen a quantity of yellowish fluid was let out, and a remarkable condition of the intestines was revealed. The small intestine was congested and greatly distended, the cæcum (enlarged to at least eight or nine inches in diameter) was found in the left hypochondriac region, having thus crossed the abdomen from below upwards and to the left. In so doing it had passed under the mesentery of the small intestine, by which the colon was constricted as though by a band. Distended and inflamed coils were drawn up from the pelvis, and between the bladder and rectum was a quantity of soft yellow fatty material like custard, several ounces of this were removed. It seemed at first impossible to bring the cæcum back to its normal position, and it was so distended as to threaten rupture. A fair-sized trocar and cannula were inserted into the cæcum, and a large quantity of fluid and gas let out. After this had been evacuated the aperture was sewn up with fine silk, and the small intestine being drawn outside the abdomen the mesentery and intestines were untwisted through a complete circle, and the cæcum brought back to its place. (See Fig. 1, p. 630).

One difficulty and danger still remained, the very distended condition of the small intestines. The only prospect of relieving this appeared to be the formation of an artificial anus, which was accordingly made in the ileum near the ileo-cæcal valve by means of Paul's tube. Through the opening a large quantity of fluid stuff was The abdominal wound was then sewn up, with n of the artificial anus in the middle line. ntioned that the peritoneal cavity was

cleansed with sponges as far as possible, but it was not advisable to prolong the operation by searching for the remains of the ruptured cyst. It was quite certain that, without evacuating the cæcum and drawing the intestine outside the abdomen, the obstruction could not have been relieved.

The after-progress of the case was tedious, but may be compressed into a few words. The Paul's tube came away on the fifth day, but the aperture would not close spontaneously. On the 23rd October and on the 20th November operations were performed to close the opening by means of double rows of fine silk sutures, the loop of intestine being freed from the wound. The second operation was done under eucaine; neither was successful. The abdominal skin became very sore from the irritation of the discharge. Early in January a third and more radical operation was performed; the coil was completely freed, sutured, and dropped back into the abdominal cavity.

This was successful. The three operations undoubtedly caused some diminution in the calibre of the intestine, but not, I hope, sufficient to lead to trouble in the future. An end to end resection was contemplated, but not performed, on account of its risk.

Several months after the patient had recovered from his intestinal trouble he returned on account of the original cyst having refilled. A second laparotomy was performed above the umbilicus, and I found the cyst to be a large one developed in the lesser sac of the peritoneum. It pushed the stomach upwards, the transverse colon downwards. The cyst was freely drained and washed out; in the course of a few weeks it became obliterated. During the last three years I have seen the patient on several occasions, and he continues to enjoy excellent health.

This case was operated on in 1901, and it appears that the only other successful one that has been recorded was operated on by Brentano three years later, in 1904. The

following account is condensed from an article by Dr. W. Danielsen,[1] of Marburg.

CASE 2.[2]—A decrepid old woman, aged 72, had suffered from vague abdominal pains, with occasional constipation, for about a year. At the end of this time acute obstruction came on. She had a double hernia, which was, however, reducible, and could not have anything to do with the obstruction. Beyond the existence of the latter with some ascites no diagnosis could be made, and at the operation the greatest difficulty was found in making out the condition. The fluid in the peritoneal cavity was blood-stained, the coils of small intestine appeared to be all twisted on their mesenteric axis, and there were many abnormal bands present between the coils. In the centre of the intestinal mass was a hard lump, which proved to be a swollen mesenteric gland with caseous degeneration. Reduction or untwisting of the volvulus was exceedingly difficult, and was only partially effected after division of the bands. It was then found that the cæcum and vermiform appendix had taken part in the twisting, and both lay in the left hypochondrium close to the spleen. When the whole mass was lifted out of the abdomen the volvulus could at last be unravelled, the cæcum being brought over to the right side again from under the mesentery. The coils soon recovered their normal colour, and it was not thought necessary to empty them by puncture. The abdominal wound was sutured without drainage, and the patient made an excellent recovery. This case was operated on by Brentano, and will be referred to under his name.

These two cases, Brentano's and my own, seem to be the only ones in which recovery has followed operation. It is obvious that the chief reason for this fact, apart from the considerable mortality attending any kind of volvulus,

[1] Danielsen, 'Beiträge zur Klinischen Chirurgie,' January, 1906, pp. 100 to 117.

Möhring, 'Deutsche Med. Wochenschrift,' 1905, No. 7.

is the difficulty the surgeon has in recognising the condition even when the abdomen is opened.

One important lesson from the two cases is, that unless the whole mass of distended coils is brought outside the abdomen it is impossible to recognise the twisting on which the obstruction depends and to reduce it. It should be noted that in Brentano's case neither cæcum or small intestine were punctured, whilst in my case I thought it impossible to effect reduction without this procedure.

In the following three cases of complete volvulus an operation was performed, but in only one of them was the true condition determined before post-mortem examination.

Case 3. Prof. Küttner, of Marburg (case narrated by Danielsen, *loc. cit.*).—A woman, aged 55, had suffered from symptoms suggesting gall-stones, and some concretions had been passed which were found to be intestinal and not biliary. She was seized with a sudden attack of intestinal obstruction, which was at first mistaken for biliary colic. She had constant vomiting, and the abdomen was much distended, especially in the left lower quadrant, where a distended coil the size of a man's fist was especially noticed; this coil proved at the operation to be the cæcum. Obvious peristalsis was present, and indican was freely present in the urine. After a fruitless trial of enemata for a few hours abdominal section was performed. Many distended and cyanotic coils of small intestine were found surrounding and overlapping a huge purple cæcum; attempts at unravelling only made the obstruction greater, until the whole was brought out of the abdomen in warm towels. It was then found that the small intestine, which was provided with an exceedingly long mesentery, had undergone torsion through 360 degrees from the duodenum downwards, and that the cæcum, also provided with a very long mesentery, had been twisted through over 180 degrees.

After division of certain mesenteric bands it was ultimately possible to reduce the volvulus. The intestine

was not punctured, the abdominal wound being completely sewn up. The patient did badly, there being signs of pneumonia and peritonitis within a few days of operation.

On the fifth day an artificial anus was made in a loop of small intestine, but without any advantage, the patient dying on the eighth day.

At the post mortem sanguineous fluid was found in the peritoneal cavity, the intestine was distended, and there were numerous scars at the back of its mesentery. The cæcum was not in its usual place, but lay in front of the lumbar vertebræ, the vermiform appendix touching the sigmoid flexure. It would, therefore, appear that reduction had not been completely effected at the time of operation, or that the volvulus had partly recurred.

Case 4. Fröhlich ('Bull. et mém. de la Soc. de Chir. de Paris,' May 15th, 1901).—A boy, aged 3, was seized with intestinal obstruction, accompanied by ascites. At the end of three days' constant vomiting, etc., laparotomy was performed, but, largely owing to the desperate condition of the patient, no real diagnosis could be made during the operation. An artificial anus was made, the patient dying two days later. At the post mortem it was found that a complete torsion of the intestine had taken place, but, strange to say, in the reverse direction to that which happened in all the other cases. The sigmoid flexure lay on the right side under the ribs, with the small intestine all packed in this region; the mesentery formed a tight band in front of the hepatic flexure of the colon. Hence both cæcum and ascending colon were greatly distended, and their walls thickened to 1 centimetre. It will be understood that in this case the cæcum was not out of place, a rotation having occurred from left to right, but in this, as in all the other cases, the mesentery formed an obstructing band.

Case 5. Hausmann ('Centralblatt für Chir.,' 1900).—A soldier, aged 22, had suffered four attacks of intestinal

obstruction, from which he had recovered; in a fifth typical attack he was operated on on the fourth day. The greatly distended cæcum and ascending colon were found to have a mesentery continuous with that of the small intestine. The cæcum was out of place, but it is impossible to gather from the account of the operation exactly what was found. The cæcum was replaced in the right iliac fossa, when the contracted small intestine filled again with air. The patient died the day after operation. At the post-mortem it was evident that the volvulus had affected the whole of the small intestine as high as the second part of the duodenum, there being no distinction between the descending and third parts of this organ. The mesentery of the small intestine compressed the ascending colon which lay beneath it, the cæcum being situated on the left side close to the splenic flexure. It is obvious that withdrawal of the twisted intestines from the abdomen at the time of operation would have given a better chance of complete reduction.

In the remaining cases no operation was performed, and the exact condition was only determined post mortem.

Case 6. Bednar ('Die Krankheiten der Neugeborenen und Säuglinge,' Vienna, 1850).—A boy, aged 10, when suffering from variola and pneumonia, developed on the day before his death uncontrollable diarrhœa and vomiting. Post mortem it was found that the ascending colon was unconnected with the posterior wall of the abdomen, and its mesentery blended with that of the small intestine. Both had twisted round so that the cæcum lay in the left flank, the latter was greatly distended. The small intestine was collapsed, and there were extravasations of blood into its mesentery and lymphatic glands.

Case 7. Epstein-Soyka ('Prager. Med. Wochenschrift,' 1878).—An infant, 4 days old, had constant vomiting, and blood mixed with the fluid stools. The upper part

of the abdomen was much distended, and the obstructed coils of intestine could be felt beneath the abdominal wall. At the post mortem it was found that a similar condition existed to that described in the other cases.

A complete volvulus had occurred from right to left, so that the cæcum lay in the right iliac fossa. Both cæcum and ascending colon had their mesentery continuous with that of the small intestine.

Case 8. Wandel ('Mitteilungen aus den Grenzbieten, etc.,' Band 11). A boy, aged 7, had been subject since infancy to attacks of sickness, followed by constipation. Fourteen days before his death he fell and sustained a fracture of the left femur, six days later vomiting came on, with abdominal distension and other signs of obstruction. No operation was done. Post mortem a complete volvulus was found, the ascending colon and cæcum lying in the left lumbar and iliac regions. The intestine had rotated 360 degrees, it was so remarkably free that the right half of the transverse colon, and the ascending part of the duodenum had shared in the rotation.

Case 9. Wandel (Ibid).—A young man, aged 20, after jumping from a landing stage to a steamer was seized with abdominal pain and extreme collapse. He died a few hours after being admitted into the hospital, but it is not stated how long time intervened between the onset of symptoms and admission. Post mortem the peritoneal cavity contained much blood and thick milky stuff. A complete volvulus from right to left was present which could be easily reduced.

Case 10. Pescatore ('Deutsche Zeit. für Chir.,' Band 68).—An infant, 19 days old. A balloon-like viscus was found just below the ensiform cartilage at the post-mortem; it was first thought to be stomach, then colon, but it proved to be an enormously distended duodenum. The ordinary horseshoe outline of the duodenum was

absent, and the organ looked like a stomach with greater and lesser curvature. It contained five times as much fluid as the stomach; below the duodenum a complete axial rotation of the small intestine and cæcum had occurred, one and a half times from right to left. Where the volvulus began in the duodenum the intestine suddenly contracted to the size of a pencil. The position of the cæcum is unfortunately not stated, though presumably it lay in the left half of the abdomen.

Such is the abstract of hitherto recorded cases, though doubtless others have been met with, especially in hospitals for children, which have not been published. The points of resemblance are of much interest, though the features are not quite constant.

The invariable condition, without which complete volvulus cannot occur, is the floating condition of cæcum and ascending colon. This is due to a congenital arrest of development, and it is perhaps more frequent than is generally supposed. Legueu estimates it at 8 per cent. in infants; Tuffier estimates it at 2 per cent. in adults. Wandel, from post mortem observations made at Bâle and Kiel, puts the frequency as high as 10 per cent.

One striking fact that comes out is this, whenever the cæcum and ascending colon are free and have their mesentery continuous with that of the small intestine, a similar congenital abnormality may be expected in the duodenum. As is well known in normal subjects, the third part of the duodenum is more closely anchored than any other part of the small intestine, whereas in these cases it is not fixed to the spine, but is directly continuous with the second, from which indeed it cannot be distinguished. Further, the hepatic flexure of the colon in several cases shares in the floating condition.

Of course, this congenital floating condition of the ascending colon does not imply that volvulus must of necessity occur, any more than the persistence of Meckel's diverticulum means inevitable trouble.

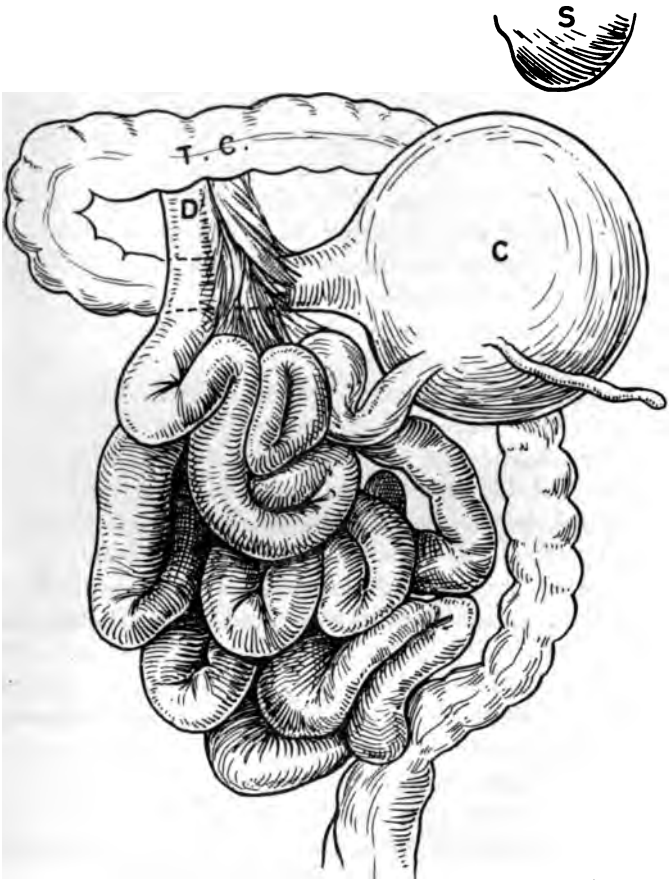
As in cases of more limited volvulus, I mean twisting of the sigmoid flexure, etc., a trauma or sudden abdominal strain may act as the immediate cause.

In Cases 1, 8, and 9 the volvulus had followed respectively rupture of an intra-abdominal cyst, a severe fall, and a violent strain in jumping. In two of the remaining cases reducible herniæ existed, and Danielsen attaches more importance to this fact than seems to be justified.

The existence of inflammatory and contracting bands in connection, for example, with tuberculous glands in the mesentery, may undoubtedly lead to volvulus. It should be noted, however, that in several of the cases no cause could be assigned besides the floating condition of the cæcum, etc. I once operated for radical cure of a large left inguinal hernia, and found within the sac the whole of the cæcum and the vermiform appendix. The floating cæcum in this case had wandered across to the left iliac fossa; we have only to imagine it slipping behind the mesentery of the small intestine, and becoming thereby compressed, to see how further peristalsis may lead to complete volvulus. It is evident that in at least four of the ten cases such a condition had led to mild attacks of obstruction, spontaneously recovered from, before the chief attack which led to the patient's death. One interesting symptom occurred in Cases 1 and 9, viz. the presence of curd-like material in the peritoneal cavity. In my own case I attributed this to the rupture of a pseudo-pancreatic cyst, but this explanation must be erroneous, since no cyst had existed in the other case. It is possible that the volvulus, by suddenly obstructing the lacteals, leads to pouring out of lymph just as it causes intra-peritoneal extravasation of blood.

Out of nine cases of complete volvulus in which the sex was noted seven were males and two females. It is, therefore, probable that the congenital malformation which leads to its occurrence is much more common in males. Three of the patients were infants, five were children or young adults, whilst two were elderly. In nine out of

Fig. 1.



T.C. Transverse colon. D. Duodenum. C. Cæcum, greatly distended and situated below S The spleen. The whole of the small intestine, together with ascending colon, have undergone torsion.

the ten cases the rotation of intestine seems to have occurred from right to left; in only one did it occur in the reverse direction.

The importance of early operation is obvious, and so complex and puzzling is this form of volvulus that only a very free incision of the abdominal wall, and lifting all the intestines forwards, will enable the surgeon to diagnose and relieve the condition. Danielsen states that complete volvulus is generally a chronic disease, and that the history of preliminary attacks distinguishes it from the more common cases of limited volvulus. On the other hand, in my own case and five others the onset was as sudden, and the symptoms were as acute, as in most examples of acute intestinal obstruction. Moreover, the occurrence of previous slight attacks have often preceded obstruction by a band or a volvulus of a limited portion of gut, so that their history has but little diagnostic importance. During the operation the most important sign of complete volvulus is the presence of the distended cæcum on the left side of the abdomen, although this is not absolutely constant.

The anatomical condition which favours complete volvulus is of special interest. As already noted it consists in an arrest of development by which the end of the duodenum, the whole of the small intestine, the ascending part of the transverse colon are provided with one and the same mesentery. Dr. Arthur Keith kindly informs me that he has observed between ten and fifteen examples of this condition, most of them were in the bodies of children, but two were in adults. As shown in Fig. 2, the superior mesenteric artery supplies the whole of the intestine which becomes twisted in complete volvulus, in fact, as Dr. Keith says, "the artery is the axis on which the twist takes place."

It may be noted that the terminal branch, the ileo-colic of the superior mesenteric artery, is the axis round which a well-known form of partial volvulus occurs, this is illus-

FIG. 2.

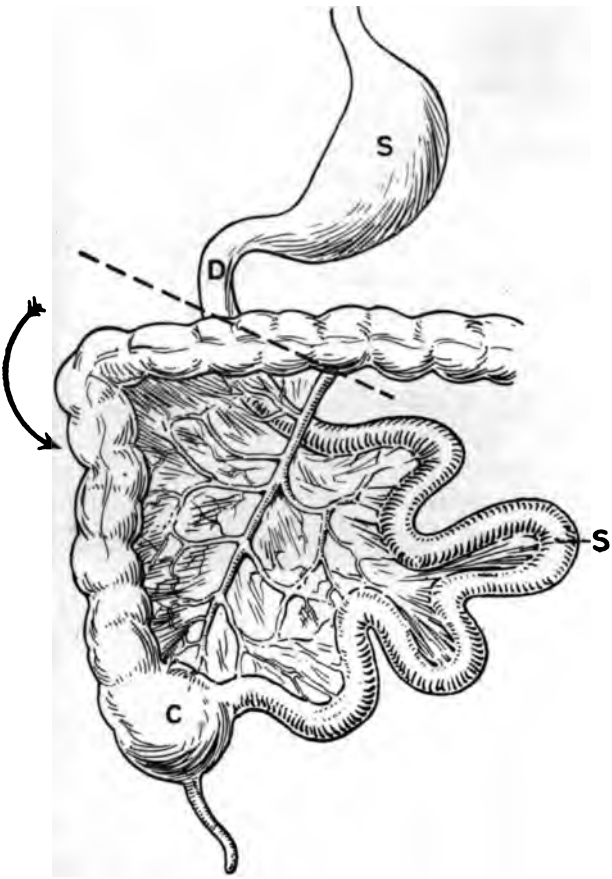


Diagram showing the amount of intestine concerned in complete volvulus (below the dotted line). The arrow indicates the direction in which the twist almost always occurs. Note the peculiar form of the duodenum (D), and the relation of the superior mesenteric artery to the parts involved in the twist. S. Stomach. C. Cæcum. S.I. Small intestine.

trated in Fig. 2. The stage at which the development has stopped in these cases is the following, the main intestinal loop consisting of small and large intestine undergoes rotation, whereby the cæcum and colon are carried over to the right, the duodenum twisted to the left, but neither cæcum or ascending colon are yet anchored. This arrangement is a common one among the Carnivora.

Dr. Keith has described the further changes (which should occur) in detail in lectures published in the 'Lancęt,' March 14th, 1903, p. 712. He notes that the process of adhesion of the ileo-colic segment begins at the end of the third month of fœtal life, and is not completed until the second year. Sir F. Treves has also drawn attention to the subject in his lectures on the Anatomy of the Intestinal Canal, etc., and in Morris' Anatomy (1893 edition, p. 1033) he describes the arrest of development predisposing to complete volvulus, in the following words: "In these instances the whole of the small intestine, together with the cæcum and the ascending colon, were slung from a common mesentery, the attachment of which to the posterior parietes was by a narrow neck giving passage to the superior mesenteric artery, and bounded by the duodenum and the transverse colon. In these subjects there was no trace of the mesentery proper as it is found in the normal human body."

Note.—In its development the intestine is always described as rotating from *left to right* on the axis of the superior mesenteric artery. It is of interest to note that in all but one of the recorded cases of complete volvulus, the rotation was really a continuation of this movement, but inasmuch as the cæcum passes behind the mesentery to the left side of the abdomen, it has been referred to in this paper as a *right to left* rotation.

DISCUSSION

Mr. BARKER: At the risk of mentioning a matter not quite in accord with the subject of Mr. Hutchinson's paper, I should like to allude briefly to a case of my own, already published in the 'Lancet' of November 5th, 1904. In so far as it was a volvulus of the whole of the small intestine from the first part of the jejunum to within a few inches of the ileo-cæcal valve, it was like the case now alluded to, though not quite the same. It was also twisted on its axis through 360° from left to right "against the hands of the clock." My experience in this case leads me to a different conclusion to that put forward by the reader of the paper. I have a rooted dislike to "eviscerating" operations, and find it possible in my work almost entirely to avoid the practice of withdrawing the intestines from the abdomen. In the case in point I was able, without allowing this very large volvulus to escape through the wound, to unroll it completely. The patient, who was four months' pregnant, made a perfect recovery, and bore a healthy child at full term. If permitted I should like to throw on the screen a figure which I made immediately after the operation. [Demonstration given.]

Dr. ARTHUR KEITH: In my opinion Mr. Hutchinson is right in distinguishing the form of volvulus he has described as belonging to a class by itself. Torsion which involves the intestine from the duodeno-jejunal junction to the hepatic flexure of the colon could happen only if there were a complete developmental arrest of the usual fixation of that tract of the gut to the posterior abdominal wall. The case described by Mr. Barker did not belong to Mr. Hutchinson's group; there was no evidence in his drawing or description of any defect or abnormality in the attachment of the gut. In my Hunterian Lectures on Glenard's disease ('Lancet,' March, 1903) I dealt with the manner in which the primitive mesentery of the bowel in man became applied and fixed to the posterior abdominal wall during embryonic and post-natal life, and the reason for its extensive attachment. In the great anthropoid apes the attachment of the bowel, both as regards form and extent, exactly corresponds to what is found in man, but in no other form of animal is the attachment so extensive or so firm. In the anthropoids, as in man, the body is habitually carried in the upright posture, and I have always regarded this extensive fixation of the bowel as an adaptation to the upright posture. Even at birth the process of fixation is not complete; that point is not reached until a child has reached its third year and the cæcum finally attained its permanent position in the iliac fossa. In the type of volvulus described by Mr. Hutchinson the process of fixation was arrested

in the stage to be seen at the end of the third month of development. The duodenal loop is partly free; the torsion of the U-shaped loop had taken place, the cæcum being thus thrown in front of the right kidney, but the posterior aspect of the mesentery of this loop, instead of being fixed to the posterior abdominal wall in the right lumbar region, remained free. The superior mesenteric artery forms the axis of the embryonic U-shaped loop of the bowel, and in Mr. Hutchinson's type of volvulus the torsion occurred round an axis formed by this artery. A complete failure in the process of mesenteric attachment was rare; hence volvulus of this loop was rare, but a lesser degree of arrest was common. In these cases the lower part of the ascending colon and lower coils of the ileum were supported by a common sheet of mesentery, in which the superior mesenteric artery terminated. A volvulus formed by the cæcum and lower part of the ileum was thus possible, and a number of such cases had been described. Such belonged to the same genus as Mr. Hutchinson's form. From Mr. Hutchinson's cases I conclude that the extensive attachment of the human intestine is evidently to prevent a volvulus from taking place. There is, in the Museum stores of the London Hospital, such a case of volvulus as Mr. Hutchinson describes. It occurred in a child, aged 20 days, which was under the care of Dr. Percy Kidd.

Mr. GERALD S. HUGHES: I have lately had the opportunity of seeing a case of volvulus of the small intestine very similar to the one described by Mr. Barker. The patient was a single woman, aged 38, admitted into the Bolingbroke Hospital under the care of Mr. D'Arcy Power. The history that was elicited from the patient's friends was that she had always been a healthy woman, but from babyhood she had suffered from occasional attacks of severe abdominal pain, accompanied by vomiting. On December 3rd, 1905, three days before her admission to the hospital, she was taken ill with vomiting and abdominal pain. The vomit was bilious in character. She was examined by her medical man, who found the abdomen quite flaccid and soft, no distension being present and no tenderness on palpation. The next day she was still vomiting, but no distension of the abdomen was noticed. On December 5th an enema was given and the bowels were well moved for the first time since the commencement of the illness. On December 6th she was found to be greatly distended, and, as she was in a critical condition, she was sent to the hospital. On admission the patient was found to be extremely collapsed and she died half an hour after admission. At the post-mortem examination she was found to have a volvulus of the whole of the small intestine from the duodenal-jejunal flexure down to within six inches of the ileo-cæcal valve. The mesentery was completely twisted on itself at its root, the twisting being clockwise. The small intestine was

enormously distended and the mesentery greatly thickened, so much that it was found impossible to untwist it, even after the evisceration of the bowel. The cæcum and ascending colon were in their normal positions. From the situation of the twist right back against the back wall of the abdomen and from the thickening of the mesentery, which was especially well marked about the twist, it is difficult to believe that this volvulus only dated some three days; it seems more probable that the twisting of the mesentery was of long standing, perhaps even from birth, and that the sudden acute termination was caused by some irregularity in peristalsis causing a little extra twisting and so obstruction to the circulation.

Mr. HUTCHINSON, Junr.: Dr. Keith has made clear in his remarks the fact which I hoped to emphasise—the peculiar dependence of complete volvulus upon a congenital arrest of development involving the attachments of intestine from the duodenum to the transverse colon. In the case adduced by Mr. A. E. Barker, his own drawing shows that both duodenum and colon were normally fixed by the peritoneum, that no such arrest of development had occurred. Hence, his case of partial volvulus, although extensive, could not possibly have gone on to become complete. Neither Mr. Barker's case nor the one reported from the Bolingbroke Hospital, belonged therefore to the group described and illustrated for the first time in this country on the present occasion. It was of interest to note, as attention had not previously been called to it, that in examples of unattached colon and cæcum, etc., the vermiform appendix is quite peculiar. It is long (sometimes eight inches or more) and whip-like, with symmetrical fringes of fat towards its extremity; moreover, it directly continues a tapering cæcum, such as is seen in the carnivora. Mr. Barker has used the unfortunate and repellent term "evisceration"; there is, of course, no such surgical procedure as true evisceration. I do, however, reassert the importance, in these rare cases of complete volvulus, of drawing the whole of the twisted intestines well forwards into the wound. In this way alone, in the two hitherto successful operations for this condition, was a correct diagnosis effected, and was untwisting rendered possible. The cases reported from the hands of most able German surgeons, in which neither diagnosis nor reduction was secured at the time of operation, are emphatic endorsement of my proposition.

FRACTURES OF THE ODONTOID PROCESS OF THE AXIS

BY

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(COMMUNICATED BY MR. C. A. BALLANCE)

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THE paper is divided into two parts; the first dealing with proved examples of the fracture, the second with cases in which the diagnosis must remain doubtful. The objects with which it is written are to bring together all that is known at present of the clinical and pathological aspects of fractures of the odontoid process of the axis; in the light of this knowledge to discuss some new ideas on the mechanism of its production, to show that it is by no means necessarily fatal or accompanied by spinal cord symptoms such as paralysis or anæsthesia, that the lesion is a good deal more common than is thought, that a good skiagraph will usually indicate its presence, and Case XVI is brought forward as an example of its successful recognition and treatment.

I.

The records of forty cases of fracture of the odontoid process form the basis upon which the following remarks are made. The examples have not been merely culled

from the literature, but have been diligently sought out and examined in the various pathological museums of the British Isles. These forty cases may be arranged as follows:

	Examples.
Fracture of the odontoid process alone .	16
„ with forward dislocation of the atlas .	3
„ „ backward „ „ .	2
„ „ rotatory „ „ .	5
„ „ a portion of the body of the axis .	6
„ „ fracture of the atlas . .	7
„ „ „ „ and axis .	1
	40

In every case except one (Case XVI), in which it was proved by a skiagraph, the anatomy of the injury was certified by a *post-mortem* examination. In twenty-four, 60 per cent. of the above, the injury to the odontoid process was accompanied by another dislocation or fracture; in sixteen, or 40 per cent., it occurred alone. The most frequent additional injury was fracture of the atlas, eight cases, followed closely by rotatory dislocation of the atlas, seven cases.

The injury is by no means necessarily fatal. The interval which occurred between the accident and the date of death varied from a second or nothing to forty-two years (specimen in the Yorkshire College, Leeds). The length of this period can be studied in two classes, namely, fracture of the odontoid process alone and in combination with other injuries. In the museum specimens the clinical history is very frequently deficient or wanting.

(A) Fractures of the Odontoid Process alone.

In three instances (Friedlowsky [I], Stephen Smith [VI], and the London Hospital, 206 [XIV]) the process was found broken off during the dissection of a subject after death, showing that they had recovered and lived; in two instances (Macnamara [III] and St. Bartholomew's Hospital, 1136 *a* [XV]) death was instantaneous. The others died respectively after 4 days, 3 weeks, a "few weeks," 5 months, 6 months, 8 months, and 2 years. Of these twelve cases, of which a clinical account was examined, only two died at once. Of the remaining nine, six presented no paralysis or anæsthesia; in two, these spinal symptoms came on later; and in only one, was there instantaneously "some loss of power," as it is indefinitely described.

The length of time lived after the accident, and the frequent absence of spinal cord symptoms in the recorded cases (66 per cent.) render it probable that some recover altogether undiagnosed. Vanderpoel's [IX] case is an example of the penalty which may be exacted on account of the injury not being known. Seven and a half months after the accident the man who had been working for some time, having had no spinal symptoms, suddenly displaced the fragments; complete paralysis ensued, and he died in three days.

(B) Fracture of the Atlas with other Injuries.

The most quickly fatal complication is backward dislocation of the atlas; Caboche's case [XXXVIII] died in two days, Swan's [XXXIX] at once. The least fatal complication is forward dislocation of the atlas; Debenham and Hutchinson's case [XXXV] died after two years, Flint's [XXXVI] after "several months," and the Leeds specimen [XXXVII] was obtained forty-two years after the accident. In only two cases, Dupont's [XXVII], and Smith and Clegg's [XXXII] was death immediate, and in only one,

Broca's [XXV], was it found by chance post mortem. Both the former of these were complicated by rotatory dislocation of the atlas.

The others died respectively after 2½ hours, 3 days, 5 days, 8 days, 14 days, 21 days, 23 days, 34 days, 11 weeks, 47 weeks, 101 days, 14 months. In thirteen cases it is possible to say from the clinical history whether or not there were spinal symptoms. In five there was paralysis immediately consequent on the injury; in five there was no paralysis, 38 per cent.; in three paralysis came on slowly some time after the accident. These cases possess a marked difference in their clinical history to those in which the odontoid process was broken by itself, 38 per cent. showing no cord symptoms against 66 per cent. of the latter.

From the length of time lived, and the not infrequent absence of spinal symptoms (38 per cent.), it may be argued, as it has been in the previous section, that some cases are overlooked altogether. Mr. Cuthbert Wallace's case (XXXIII), which died from sudden displacement of the fragments from injudicious moving on the eighth day, and Gibson's (XXXI), which fell back dead from suddenly sitting up on the twenty-third day, illustrate the penalty that may be exacted for overlooking the lesion.

Comparing these two classes together, it may be said that fracture of the odontoid process with complicating injuries is more likely to be fatal, and more frequently followed by spinal symptoms than when the process alone is broken. This is precisely as would be expected.

Fractures of the odontoid process generally heal by fibrous union, 97 per cent. of cases which have recovered. Bony union can take place, as is shown by the specimen 1636 in the Pathological Museum of Trinity College, Dublin.

Mechanism.

Of the recorded fatal cases 60 per cent. showed the existence of a gross complicating injury, and 40 per cent.

fracture of the odontoid process alone. The character of the complicating injury indicates that the process may be easily broken off by the sweeping guillotine-like rotatory movement of the displaced bones, somewhat in the way that the executioner's axe decapitates criminals. The absence

FIG. 1.

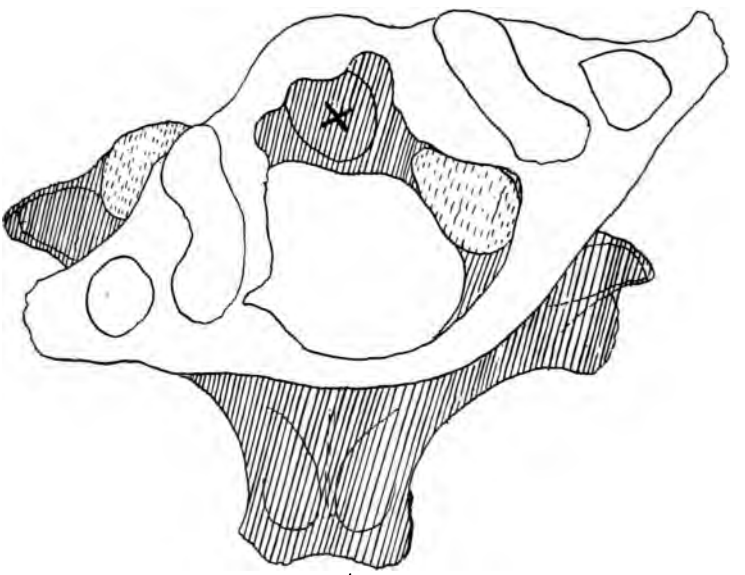


Diagram of the atlas and axis to show how the odontoid process can be "mown off" in unilateral rotatory dislocations or subluxations of the atlas. The atlas is unshaded, the axis is shaded, and the odontoid process is marked with a cross.

of any displacement post mortem is no evidence that there had been none; the bones are known to spring back into place when the complicating injury is less than a complete fracture or dislocation of the vertebral column.[1] Moreover, the post mortem manipulations might conceal or exaggerate it. Therefore, it seems probable

[1] "Spinal Concussion," 'Lancet,' September 22nd, 1906.

that a similar "mowing" displacement of the atlas on the axis may account for the fracture of the odontoid process in these cases. In six out of eight fatal cases of rotatory dislocation of the atlas, the process is known to have been broken.[1] Rotatory dislocations and subluxations of the atlas[2] are by no means necessarily fatal, and to these rotatory movements we must look principally for the mechanism of fracture of the odontoid process by itself. In complicated cases the anterior arch of the atlas was broken alone three times, once the posterior arch alone, three times both arches of the atlas, and once one of its lateral masses.[3] In the production of the fracture of the odontoid process it is the anterior arch of the atlas which suffers most, and almost always to one side or other of the anterior tubercle, hardly ever in the middle line. The anterior arch is broken in the "mowing" off of the process, and the posterior arch by the subsequent displacement. Phillips' [XIX] wonderful case is the only example of a complicating fracture of the posterior arch alone. The atlas breaks before the transverse ligament, which then protects the spinal cord from injury by the odontoid process.

One of the most remarkable anatomical points in fractures of the odontoid process is the site of the fracture, which in far and away the majority of cases takes place transversely at the neck which joins the process to the body of the axis. In criminal executions by decapitation, a blow is given to the neck severing the head from the body. In fracture of the neck of the odontoid process the mechanism is not the same, the line of cleavage being produced by indirect violence, not direct. The great constancy with which the fracture occurs at this place indicates the common method of the mechanism which

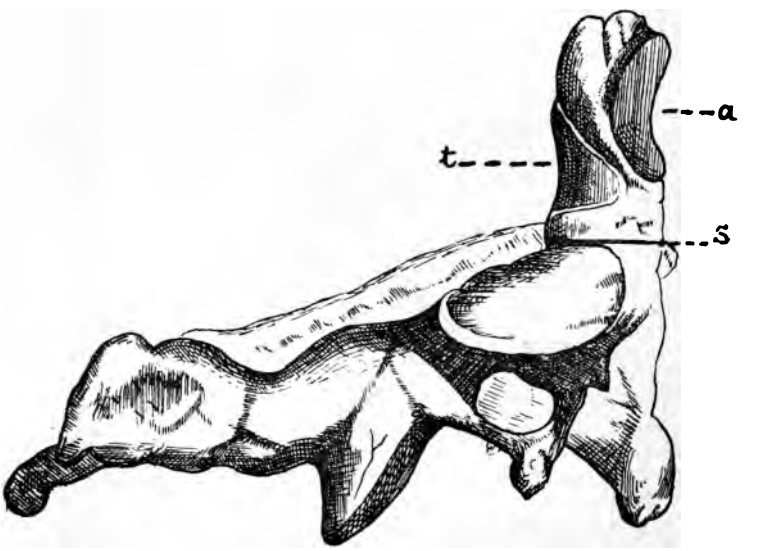
[1] "Unilateral Rotatory Dislocations of the Atlas," 'Annals of Surgery.'

[2] "Subluxation of the Atlas," 'Trans. Clinical Society,' November 9th, 1906.

[3] 'Fractures of the Atlas' (not yet published).

produces it. The "snapping off" of the process can be explained easily. If the process is looked at sideways (Fig. 2) it will be seen that the articular surface for the anterior arch of the atlas on the front is above and does not correspond with that for the transverse ligament on the back of the process. The anterior facet is nearer the free end of the process than the posterior. These arti-

FIG. 2.



Showing the situation of the facets on the odontoid process: *a*, for the atlas; *t*, for the transverse ligament; *s*, for the line marking the site of the fracture.

cular surfaces show the places at which force is applied in fractures by indirect violence. In consequence all movements of extension and flexion of the head will exert a "couple" of forces which will tend to snap the odontoid process through the neck, its weakest part. In fact, the mechanism of fracture of the neck of the odontoid process is the same as breaking a wooden match, fixed at one end,

with the forefinger and thumb. From this it will be easily seen that the very great frequency of fracture at this point is explained from its causation by the common movements of extension and flexion of the head in severe degrees. Fractures of the base of the process formed 81 per cent. of this series, in 4 per cent. only the tip or upper part of the process was broken off, and in 15 per cent. part of the body was detached with the odontoid process.

Fig. 3.

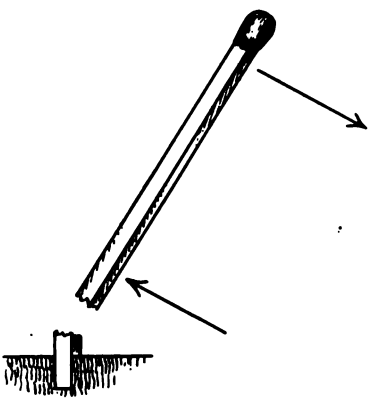


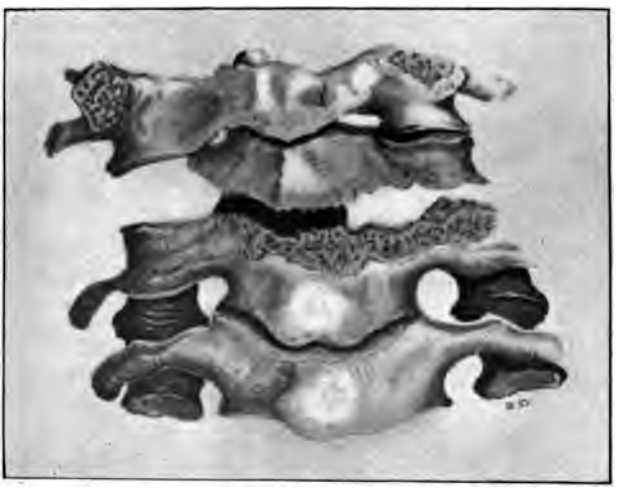
Diagram to illustrate the action of a couple snapping a match between the finger and thumb. The upper arrow indicates the direction of the force applied by the finger; the lower, that of the thumb.

The fracture of the tip of the odontoid must be produced by severe flexion (or extension ?) of the head, when the "check ligaments" will tend to snap off the tip of an unusually long process. Owens College Museum, 738, and Friedlowsky [1].

The mechanism of separation of the odontoid process with part of the body of the axis is more complicated and less easy to explain. It is not due to any modification of the mechanism by means of which the process itself is broken through its base. But it is due to the violence being transmitted through one or both occipital

condyles and lateral masses of the atlas to the axis; much in the same way as a chisel transmits the violence from the hammer and separates a flake of stone in sculpture, or as the "core" of prehistoric man knocked flakes off the flint when our ancestors used flint implements. If the force is transmitted through one condyle by reason of the head being rotated or laterally flexed to

FIG. 4.



Photograph of a specimen in the St. Thomas's Hospital Museum of the separation by fracture of the odontoid process and part of the body from the rest of the axis vertebræ. Wallace's Case xxxiii.

that side, then the odontoid process and part of the body of the opposite side of the axis will be struck off. For instance, in Wallace's case [XXXIII] the left superior articular facet and the odontoid process were broken off on account of violence being applied obliquely to the right superior facet of the axis through the right side of the atlas and the right occipital condyle.

If the force is applied obliquely through the two con-

dyles to both sides of the axis, the odontoid process and a portion of both sides of the body may be chipped off. Eve's case [XXIX] is an illustration, and also that in Professor Pirie's collection at Aberdeen, numbered 280 (XXXIV).

In these fractures it is uncommon to find the fragment of the body attached to the odontoid process perfectly symmetrical to it.

Examples of fracture of the odontoid process alone.

I. Friedlowsky. 'Medicinische Jahrbuch,' xv, 233. Fracture of the odontoid process.—In a subject in the dissecting-room only the lower half of the odontoid process was found, which had worn for itself a new facet on the posterior surface of the anterior arch of the atlas. The author considered it to be the result of a traumatic fracture in early life as there were no signs of disease. There had been fibrous union between the fragments.

II. Humphrey. 'Provincial Medical and Surgical Journal,' 1850, 575. Fracture of the odontoid process.—A man, aged 59, fell from a considerable height. Afterwards the neck was fixed by muscular contraction and the chin poked forward. He cried out when taken unawares or when his head was suddenly moved; and on one occasion, crepitus being heard, he exclaimed, "That's in my neck." With care he could nod and turn his head from side to side, the muscles of the neck being firmly contracted. There was no paralysis of any sort. He gradually became worse, and died on the fourth day.

Post mortem.—The transverse and other ligaments remained almost entire, and prevented much displacement of the bones. No sign of fracture could be felt on introducing the finger through the foramen magnum; but on examination it was found that the odontoid process was broken off at its base, but there was no appa-

rent displacement. Other injuries were present in the body.

Death on the fourth day.

Cambridge University Pathological Museum Catalogue, 1334.

III. Macnamara. 'Dublin Medical Press.' Fracture of the odontoid process.—An old woman was knocked down by a blow on the side of the head from the buffer of an engine. Death was instantaneous.

Post mortem.—The head was extremely movable on the trunk. The odontoid process was found to be fractured across its base.

IV. Malgaigne. 'Fractures,' ii, 327. Fracture of the odontoid process.—A man, between 30 and 40, fell whilst carrying a heavy weight on his shoulders. He continued working for a month. He then sought advice for a deformity of the neck. Death resulted from convulsions a few weeks after the accident.

Post mortem.—A transverse fracture of the base of the odontoid process was found.

Death a few weeks after the accident.

V. Parker. 'New York Medical Journal,' 1853, x, 164. Fracture of the odontoid process.—A man, aged 40, was thrown from a cart which came in contact with a tollgate, alighting on his head and face about fifteen feet away. He was seen the following day when he was in a restless state, with pain in the back of the neck, inability to rotate the head, neck swollen, no paralysis. On the fifth day an irregularity in the region of the atlas and the axis could be made out. Improvement was gradual, and he went about his business. About four months afterwards he felt a scraping in his neck, and died a few days later.

Post mortem.—The parts were unusually vascular. The "occipito-axoideum" ligament had been destroyed

in part. The odontoid process was broken off, and the lower extremity inclined backwards. Doubtless some unfortunate movement of the head had caused pressure on the cord.

Death five months after the accident.

VI. Stephen Smith. 'American Journal of Medical Science,' 1891, October. Fracture of the odontoid process. —1. A man, aged 49, who had had syphilis, had a tumour on the upper cervical vertebræ which resulted from a blow a few months previously.

Post mortem.—There was a fracture of the base of the odontoid process with displacement of the atlas forwards. The author did not regard it as syphilitic but traumatic.

VII. Stephen Smith. Ibid. Fracture of the odontoid process.—2. A man who had, three months previously, received a blow on the neck, after doing his work for six weeks, noticed a gradually increasing loss of power in his arms and back. At the end of six months he died.

Post mortem.—There was a transverse fracture of the base of the odontoid process.

Death after six months.

VIII. Spangenbern. 'Edinburgh Medical Journal,' lxiv, 527. Fracture of the odontoid process.—A man, aged 28, fell from a horse, remounted, and rode two and a half miles, when his neck gave a snap, and he fell insensible. After this he lost so much power in his legs that it was only "later" that he could walk. He died of "hectic" two years later.

Post mortem.—Fracture of the base of the odontoid process, which the author regarded as traumatic.

Death after two years.

IX. Vanderpoel. 'Medical Press and Circular,' 1877, xxiv, 171. 'Archives of Clinical Surgery,' June 15th, 1877. Fracture of the odontoid process.—A man, aged

17, two weeks before admission to the hospital, fell on the back of his head from a waggon. There were no paralytic symptoms, and he continued his work. A peculiar deformity of the neck resulted which led the surgeon to diagnose a fracture-dislocation of the fourth cervical vertebra. Seven and a half months after the accident, whilst at work, he was seized with pain in the neck. In three days' time the paralysis was complete; it began as a right hemiplegia.

Post mortem.—The odontoid process was found fractured across its base. There was no dislocation, all ligaments were intact. Death was due to some meningitis, secondary to some septic bone disease, which the author thought was in all probability consequent on the injury. (There was no mention of the existence of any sign of this problematic bone disease, which seems to have been a mere surmise put forward by the author.)

Death after eight months from meningitis.

x. University College, Liverpool. Pathological Museum. Fracture of the odontoid process.—N. 303. "The bristles point to the odontoid process, broken off, but still retained in position by its ligaments. The broken surfaces are covered with lymph. Behind, by looking through the foramen magnum the posterior common ligament is seen to be uninjured. In the cord, which has been laid open, no extensive laceration or softening can be discovered. A bale of cotton which was being hoisted into a warehouse, fell on to his head, and he was immediately paralysed to some extent. In the course of a week the paralysis was complete, and in three weeks he died."

xi. Museum of the Royal College of Surgeons, Ireland. —Ea. 513. Specimen without history showing a fracture through the base of the odontoid process.

XII. Trinity College, Dublin. Pathological Museum.—1636. "Fracture of the odontoid process, union with bone."

XIII. Same Museum as XII.—127c. A cast of the fracture of the odontoid. The original specimen does not exist. The cast was made by Dr. R. Smith.

XIV. London Hospital Pathological Museum.—206. The odontoid process was connected as usual with the occipital bone by the perpendicular and moderator ligaments, but was attached to the second vertebra by means of two slender ligaments which passed from it to their insertion nearthe superior articular facet of the latter. The transverse ligaments, larger than usual, flattened horizontally and polished above and below, formed a distinct interarticular ligament between the odontoid process and the body of the second vertebra. (Mr. Curling's description.) The apposing surfaces of these bones presented facets.

From a male, aged 38, who slipped and fell, in descending a ladder, a few feet backwards, striking the hind part of his head and neck. Sudden death on the third day. Mr. Curling added that the injury is an old one, and had preceded the accident narrated. 'London Hospital Reports,' i, 142.

XV. St. Bartholomew's Hospital Museum Catalogue. Fracture of the odontoid process.—1136A. From a groom, aged 35, who had been exercising a horse, and was found in the road dead, without any sign of violence except a slight extravasation on the back of the head.

Post mortem.—There was a fracture of the base of the odontoid process. The lower part of the medulla had been destroyed by pressure, and there was extravasation of blood in the cord.

XVI. Battle. 'St. Thomas's Hospital Reports,' 1905. 'Transations of the Medical Society,' 1906.—A man had

an accident and fractured his odontoid process, as shown in a skiagraph. There were no paralytic symptoms. The man recovered. The case was shown at the Medical Society of London, 1906.

Examples of Fracture of the Odontoid Process and the Anterior Arch of the Atlas.

XVII. Macarthy. 'Transactions of the Pathological Society,' London, 1874, xxv, 201. Fracture of the atlas and odontoid process.—A man, aged 33, whilst unloading a ship fell forwards down the hold. There was complete paralysis of all extremities and loss of sensation below the third rib. On the following day the motor paralysis was unchanged, but he had regained sensation over his upper limbs, his trunk, and the upper part of his thighs. Death occurred on the third day.

Post mortem.—On examination it was found that the atlas was broken into five pieces, and the odontoid process broken off at its base. There was no displacement of the fragments, which were held together by ligaments or periosteum. There was no evidence of compression of the cord, the symptoms must have been due to some concussion of the cord, the fractures of the atlas and the axis being merely accidental complications.

XVIII. Melicher. 'Oesterreichische Medicinische Wochenschrift,' 1848, 934. Fracture of the atlas and odontoid process.—A woman, aged 47, fell and was temporarily stunned. Paralysis came on "later" and not immediately. Death occurred on the thirty-fourth day.

Post mortem.—There was a fracture of the middle of the anterior arch of the atlas, and another of the base of the odontoid process.

Death on the thirty-fourth day.

XIX. Phillips. 'Medico-Chirurgical Transactions,' 1837, xx, 78. Fracture of the odontoid process and atlas, with

anterior dislocations of the latter.—A man, aged 32, fell off a hay-rick on to the back of his head. Afterwards he suffered from a stiff neck and was unable to rotate his head. Over the second cervical vertebra there was a small tumour which was slightly painful on pressure. There was a projection in the pharynx corresponding with the position of the body of the second cervical vertebra. He had pleurisy, later anasarca, and died forty-seven weeks later of "hydrothorax."

Post mortem.—The condyles of the occiput rested upon the articulating facets of the axis, and it was found that the whole of the atlas, in front of and including the lateral masses, had been separated from the posterior segment and displaced forwards in front of the body of the axis. In this way apparently two spinal canals were formed, the true one through the axis, and the other through the anteriorly dislocated portion of the atlas.

The odontoid process had also been broken off through its base. The man seemed to experience little discomfort from the injury, and died forty-seven weeks afterwards.

Casts of the specimen are described in the following Museum Catalogues :

Charing Cross Hospital, 556.

Middlesex Hospital, 729.

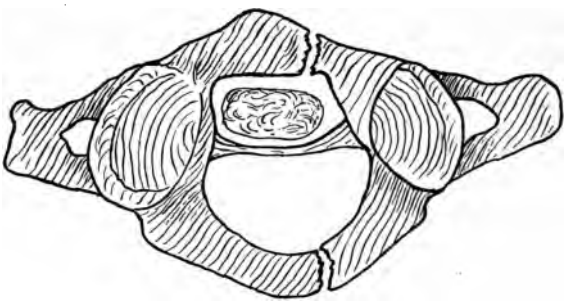
Royal College of Surgeons, Edinburgh, iii, 65.

xx. Scott. 'British Medical Journal,' 1904, 247. Fracture of the atlas and odontoid process.—A man, aged 23, was attacked and hit across the back of the neck with a heavy stick. Both his arms were broken with the same implement. The neck showed nothing particular, and could be rotated forcibly. On the eighth day he died from tetanus.

Post mortem.—On rotating the head crepitus was felt. All joints and ligaments were intact, but there were fractures of the odontoid process at its base and two of the

atlas. The first of the latter was situated on the anterior arch, just to the left of the tubercle. The second was on the right side of the posterior arch. The ligaments had withstood the force of the movements of daily life,

FIG. 5.



Fracture of atlas; Dr. Scott's case.

and also forcible movements on the autopsy table when the muscles were out of action.

Death from tetanus about a fortnight after the assault.

XXI. Owens College, Manchester. The Pathological Museum. Fracture of the atlas and odontoid process.—738. A dry specimen consisting of an atlas and part of an axis. The left half of the posterior arch of the atlas is fractured obliquely, and the anterior arch is fractured close to the left lateral mass. The tip of the odontoid process is broken off. There is no evidence of repair about the specimen, which is old and without any clinical history.

XXII. The London Hospital Museum Catalogue. Fracture of the atlas and odontoid process.—204. An atlas and axis. The central part of the anterior arch of the atlas is separated by a fracture on either side of the tubercle. The posterior arch is also severed by fractures just behind the inferior articular facets on each side. The odontoid process has been broken through at its base and remains attached to the atlas. No clinical history.

of rotation. During life the man had carried his head a little obliquely, and the neck was a little stiff. The right articular process of the axis had been displaced into the spinal canal so that the little finger could be hardly inserted. The author regarded it as due to traumatism.

XXVI. Cortes. 'Malgaigne's Fractures,' ii, 329. Fracture of the odontoid process with rotatory dislocation of the atlas.—A youth, aged 15, was thrown to the ground and received several blows on his head and neck. He was well for nine weeks, when he lost the use of his limbs and died in the eleventh week.

Post mortem.—It was found that the atlas was dislocated forwards, with the right side more advanced than the left. The odontoid process was fractured across its base and lay almost horizontal, there was no bony union.

Died in the eleventh week.

XXVII. Dupont. 'Bulletin de la Société Medicale de la Suisse,' 1876, x, 65. Fracture of the odontoid process with unilateral rotatory dislocation of the atlas.—A man in delirium tremens leapt from the fourth story of a building. Death resulted in a few hours. Upon examination there was considerable separation between the atlas and the axis. The latter was dislocated backwards, and pivoted on the left facet which remained in its proper place. The odontoid process was fractured at its base. But owing to the fact that the ligaments remained intact there was no displacement of the process. The vertebral artery was also ruptured.

XXVIII. Lambotte. 'Annales et Bulletin de la Société de Medicin d'Anvers,' 1894, lvi, 031—133. Fracture of the odontoid process with unilateral rotatory dislocation of the atlas.—The fracture was produced by a simple movement of extension of the head whilst the young woman was sewing. After this she suffered from pains in the head and a stiff neck. A year after the accident

she began to suffer from paralysis in the upper limbs, imperfect anæsthesia, exaggerated reflexes, etc. Death occurred about fourteen months after the injury.

Post mortem.—The odontoid process was found to be fractured across its base transversely. The fracture was repaired by some fibrous tissue. The atlas was dislocated forwards on the right side only. The transverse and check ligaments were intact.

Examples of Fracture of the Odontoid Process with part of the Body of the Axis.

XXIX. F. S. Eve. 'St. Bartholomew's Hospital Reports,' 1877, xiii, 237. Fracture of the upper part of the body of the axis, with separation of the odontoid process.—A man, aged 20, was struck on the head by an empty packing case, which fell from a crane. He was immediately paralysed, and died shortly after.

Post mortem.—The neck was extremely loose and movable, but no displacement of the bones could be felt. A fracture was found through the body of the axis at the base of the odontoid process. The line of the break passed very obliquely from behind downwards and forwards, beginning posteriorly at the point where the nutrient artery enters the bone, and terminating at the greatest concavity of the body of the bone. The atlas was dislocated forwards with the odontoid process. The spinal cord was nipped between the posterior arch of the atlas and the sharp fractured edge of the body of the axis. It was softened and ecchymosed, but not lacerated.

Death occurred two and a half hours after the accident.

XXX. Fracture of the upper part of the body of the axis, separating the odontoid process; also fracture of the body of the fifth cervical vertebra.[1] South. Chelius,

[1] "Dual and Distinct Fractures of the Spine," 'St. Thomas's Hospital Reports.'

i, 534. (Quoted by Stephen Smith, loc. cit.)—A man fell down stairs, and died on the fifth day.

Post mortem.—The atlas was found to be broken in two places, the line of fracture being diagonal and through the left vertebral foramen. The odontoid process of the axis was broken off, together with a piece of the body of the axis. The body of the fifth cervical vertebra was crushed, and the spinal cord disorganised at this spot. Death on the fifth day.

XXXI. Fracture of the upper part of the body of the axis, with unilateral rotatory dislocation of the atlas. Gibson. 'Lancet,' 1885, ii, 429.—A man, aged 58, rolled down a bank in the evening and lay there all night. Upon rising he was too unsteady to walk, and had to be assisted home. His head was very much set forward, the chin resting on the sternum; it was held rigidly in this position. He said that he was suffering from a pain of a burning character. There was a great prominence at the back of the neck just below the occiput. The highest cervical spine was two inches from the occiput. A diagnosis was made of a displacement between the atlas and the axis. There was no paralysis. He was laid on the bed and steady traction applied to the head, when the dislocation was suddenly reduced with a snap. Crepitus was also felt, indicating that there was a fracture present. The prominence of the spines had disappeared, and the head went naturally into line with the body. A week later he was seized, after eating some bread and butter, with abdominal pain. Whereupon, in spite of efforts, he started up, and almost immediately fell back dead.

Post mortem.—Considerable separation was found between the atlas and the axis. The cord was tightly stretched, and pulled against the anterior surface of the canal. There was no damage to the cord. The odontoid process, with part of the body of the axis, was broken off

The transverse ligament was intact. No damage to the cervical cord was apparent.

Death on the eighth day after the injury.

The specimen is in the St. Thomas's Hospital Museum Catalogue under the number 188*a* (Fig. IV.)

XXXIV. University of Aberdeen, Surgery Museum Catalogue. Fracture of the odontoid process of the axis. 280. Professor Pirie's collection.—The line of fracture is through the base of the process well below the neck. There are no signs of healing, and there is no history.

Examples of Fracture of the Odontoid Process and Forward Dislocation of the Atlas.

XXXV. Debenham and Hutchinson. 'Clinical Lectures and Reports of the London Hospital,' 1867–78, iv, 210. Fracture of the odontoid process with forward dislocation of the atlas.—A man, aged 20 years, was wheeling a loaded barrow which weighed half a hundredweight, the handles being supported in part by a strap round his neck. The wheel passed over a large stone, jerking him severely. He did not fall, but his chin dropped on to his chest. Next day he walked several miles, supporting his head between his hands. He gradually became emaciated and died.

Post mortem.—The atlas was dislocated forwards, with the odontoid process about half an inch and a little to the left. The process was broken through its base. The lower articular facets of the atlas, pushed forwards and downwards, are anchylosed to the front of the axis, and the axis and the third cervical vertebra are also anchylosed. The cord must have been compressed.

Death two years after the accident.

London Hospital Museum Catalogue, 207.

XXXVI. Flint. 'New York Medical Journal,' 1869, ix, Fracture of the odontoid process with forward dis-

location of the atlas.—A man, aged 40, received a blow on the neck some months previous to death. It is not known from what he got the blow. At first he complained of pain in his right leg, later he became paralysed. The paralysis passed off, and he got better; but a further increase of the dislocation led to its recurrence and his death.

Post mortem.—The first cervical vertebra was dislocated forward, the odontoid process being broken off and carried forward by the atlas. The ligaments between the atlas and the axis were much stretched. The left articular process of the second cervical vertebra was broken off. The left posterior half of the body was not seen. There was a good deal of reparative material about the site of the fracture. The second vertebra was abnormally movable on the third, though there was no definite dislocation.

Death several months after the accident.

XXXVII. Yorkshire College, Leeds, Pathological Museum. (Specimen unmounted.) Fracture of the odontoid process with forward dislocation of the atlas.—The cervical vertebræ, of which the first, second, and third are anchylosed. The bodies of the second and third preserve their relative positions. The atlas is displaced downwards and forwards in front of the bodies of the second and third. The odontoid process is tipped over and adherent to the axis and the atlas. The fourth vertebra is free, and the sixth, fifth, and seventh are anchylosed. The patient's head was pushed forwards whilst he was washing a sheep in 1854, and he died in 1896, forty-two years afterwards! He had pain down the spine and a stiff neck ever after the accident. He was not rendered unconscious; he had remained in bed for the next three weeks. On admission in 1896 he could not rotate his head, though he could flex it fairly well. Extension was limited. There was considerable prominence at the top of the cervical spine posteriorly to the right side of the middle

line. On the left side at the same level there was a defect or depression, as though there may have been lateral displacement of the axis.

Death due to cancer of the lower jaw. At the post-mortem examination it was found that the foramen magnum was much contracted by the displacement of the atlas. There was a fracture of the base of the odontoid process with forward dislocation of the atlas.

Examples of Fracture of the Odontoid Process, and Backward Dislocation of the Atlas.

XXXVIII. Caboche. 'Bulletin de la Société Anatomique,' Paris, 1898, lxxiii, 5th series, xii, 779. Fracture of the odontoid process with backward dislocation of the atlas. —A woman, aged 54, whilst carrying two pails of water downstairs fell backwards. She complained that she had hurt her neck, and of attacks of pain in that region. The chin was in the middle line, and the neck slightly inclined forwards. There was complete paralysis of the limbs. A lump in the pharynx was reduced by pressure and traction on the head, but as soon as the tension was relieved the lump reappeared.

Post mortem.—The atlas and the odontoid process which was broken off at its base was dislocated backwards on the axis. The transverse ligament was intact, whilst the atlanto-axoid ligaments were partially destroyed.

Death occurred two days after the accident.

XXXIX. Swan. 'Boston Medical and Surgical Journal,' 1877, xcvi, 226. Fracture of the odontoid process with backward dislocation of the atlas.—A man, aged 77 years, fell downstairs on his forehead. Death was instantaneous.

Post mortem.—The spinal cord just below the medulla was converted into a reddish-grey pulp over an extent of nearly one inch in length. The skull and atlas were

fracture of the odontoid process, sometimes even when complicated with other and severe injuries. It may, therefore, be of interest to quote four cases which have been reported from twenty to sixty years ago of recovery after the injury. In the first two there was only pain and stiffness of the neck; crepitus also was felt in the second. In the third, besides pain and stiffness of the neck, some temporary paralysis appeared at the end of fifteen months. In the fourth, there was obviously gross injury to the neck, paralysis of both arms and part of one leg. In the last case these paralyses came on immediately after the injury, and were doubtless due to hæmorrhage into the cord as the condition improved. In fractures at the top of the spine it is impossible to get severe injuries to the cord without producing instantaneous death, such as occurs in other regions. Hence the early paralyses consequent on injuries to the upper part of the spine are due to hæmorrhages, which may produce irregular scleroses and paralyses, as in Sinckler's case.

1. Bernhuber. 'Deutsche Klinik,' 1852, No. 52, 589. Fracture of the odontoid process; recovery.—A man, aged 18, received an injury to his neck which resulted in pain and limitation of movement. There were no other signs. The diagnosis was fracture of the odontoid process. Recovery.

2. Huber. 'Oesterreichische medicinische Wochenschrift,' 1846, 1342. Fracture of the odontoid process and forward dislocation of the atlas.—A man, aged 19, fell and hurt his neck badly. The doctor in charge distinctly felt crepitus. The case was diagnosed as fracture of the odontoid process and forward dislocation of the atlas. Recovery.

3. Kuster. 'Arch. für klin. Chir.,' 1884, xxxi, 218. Fracture of the odontoid process and the atlas; recovery. —A woman, aged 18, slipped, fell, and knocked the back

of her head. There was no paralysis, and she went on with her work. Later she complained of pain in her neck, and went early to bed. Fifteen months later she entered hospital for paralysis. Recovery resulted from rest and medical treatment. The diagnosis made was "fracture of the anterior arch of the atlas and the odontoid process."

4. Sinckler. 'Philadelphia Medical Times,' 1875, v, 418. Fracture of the odontoid process; recovery.—A man, aged 22, jumped off a train in motion, and was thrown violently to the ground; he thinks that he was struck between the shoulders by the end of the car, but does not know on what part of his body he fell.

The head was strongly flexed, the chin resting on the sternum and immovable. Both arms were paralysed There was also complete loss of power in the right leg as far as the knee. There was a large prominence in the cervical region of the neck, not painful on pressure, but somewhat so on movement of the head. About four months after the accident he was able to walk, but the right arm was powerless. The swelling on the neck just below the occiput was noticeable, and the chin rested on the sternum. This condition improved somewhat, but to what extent it is impossible to say, as the man was lost sight of.

Bernhuber's case is a mere statement, but was, apparently, received by his contemporaries. In both Huber's and Kuster's cases there was evidently some gross injury which may quite well have been a fractured odontoid process. Sinckler's case is most fully described; it leaves little doubt that the man recovered from a very serious injury to his upper cervical spine, though in these pre-skiagraph days it was impossible to be sure of its exact nature.

DISCUSSION.

The PRESIDENT said: One thing that comes out very clearly from the paper is the importance of recognising the frequency of this condition which is much greater than is usually held. It is a remarkable fact that a patient may sustain a fracture of his odontoid process, and after suffering merely a little discomfort for a few days resume his active occupation, and then after some months suddenly die. There is one case recorded, not referred to, I think, in the paper, of a milkman who, after a few days' pain, went about actively doing his work for five months, and then died suddenly, and I know of another case who similarly was engaged actively in his work for months before his sudden death occurred. I am reminded, too, of the case of a child who had extensive tuberculous disease of the odontoid process, of which the only symptom was wry-neck. This passed away under treatment and rest, but death occurred eventually from tuberculous meningitis, and at the necropsy a mere spicule of bone was found to be representing the odontoid process. The moral of the paper is, I think, that a skiagraph should be taken in doubtful cases and particularly that it should be taken through the mouth.

Dr. ARTHUR WHITING said: I am interested in the paper not so much from its surgical aspects as in relation to the effect of fracture-dislocations of the cervical spine in producing injury to the central nervous system. The group of twelve cases referred to in the paper may probably be taken as typical of others, and of these it is remarkable that, although two died at once, and one three days later from displacement of the fragments of bone, in only one of the ten remaining were there any nervous symptoms at the time, and that was a mere vague weakness. Leaving out of account the possibility of sudden death, the fracture-dislocations in the upper cervical spine seem to contrast very favourably in their effects when compared with those in the middle and lower cervical spine; in the one there seems to be more usually at the most mere discomfort, in the other with total transverse lesion of the cord the condition of complete paralysis of all four limbs and trunk muscles excluding the diaphragm, loss of all sensation, paralysis of sphincters, and the rest is nothing short of tragic. It would seem, therefore, that in the event of a broken neck the patients with fracture in the region of the upper vital part of the cord are in better case, even allowing for the risk of occurrence of sudden death, than in fracture of the lower cervical spine. It seems probable that the transverse ligament of the atlas may be an important means of giving immunity from injury to the upper cord in this kind of accident. One plain lesson of the paper is that with such an injury the absence of all nervous symptoms does not negative the danger of sudden death at a later date.

EXPERIMENTS ON PROLONGED PROTEIN FEEDING

WITH

SPECIAL REFERENCE TO THE THYROID GLAND AND THE OSSEOUS SYSTEM [1]

BY

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[COMMUNICATED BY DR. E. I. SPRIGGS]

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A CONNEXION between meat-eating and the production of gout has long been maintained, and attempts to induce the disease in animals and birds by prolonged protein feeding have been made from time to time. The most recent experiments in this direction were undertaken by Chalmers Watson, who fed eight fowls on meat alone for periods ranging from four to sixteen months. His results were recorded in a paper read before this Society in 1904 (8). No condition suggestive of gout was found in any of the birds, but, on the other hand, alterations in the long bones and hypertrophy of the thyroid glands were described and attributed to the excessive meat diet. These changes were so remarkable that it was thought desirable to repeat the experiments. This has been done during the past two to three years and the object of the present paper is to record the results obtained.

[1] The expenses of this investigation were [illegible]t from the Royal Society to Dr. Pembrey.

Experiments.

The experiments began in October, 1904. Two cocks, two hens and a cockerel were obtained, but shortly afterwards one of the hens was accidentally killed, and during the remaining period four birds were under observation. Except in the matter of food every care was taken to place them in healthy surroundings. They were kept in a commodious run, part of which was indoors under cover, protected from rain and wind, and part out-of-doors, facing south-west and enjoying ample sunshine. The cage was kept clean, and constant supplies of gravel, dust-baths, and fresh water provided. The protein diet was not commenced at once, but a gradually increasing quantity of raw meat was added to a gradually decreasing quantity of grain. For the first week the food was wholly grain; for the next two weeks three parts grain to one of meat; from the third to the fifth weeks equal parts of grain and meat; in the sixth week one part of grain to three of meat; while from the latter date (December 8th, 1904) until the termination of the experiments no food except meat was allowed. By this gradual change to an abnormal diet it was hoped to avoid any initial digestive troubles, the animals being afforded an opportunity to accommodate themselves to their new food. In the earlier part of the experiments the meat given was the ordinary butcher's trimmings, but later this was discarded on account of its cost and half-raw horse-flesh substituted.

Until the beginning of February, 1905, no alteration was observed in the birds, but at this date they became listless and moping, standing about their runs without evincing any interest in their surroundings. They ceased to scratch over the gravel, and two of the cocks penned with the hen lost all pugnacity towards each other and their sexual activity failed. Whereas their crowing had been frequent, they now for over a month remained silent. All four birds developed diarrhœa which soon became

extreme, the droppings being watery, and passed at intervals of a few minutes. Their appetites suffered no diminution, and each readily ate one and a half pounds of meat a day, but as this appeared not to satisfy their hunger the amount was increased to two pounds for each bird. Simultaneously with the diarrhœa there developed an excessive thirst, the large drinking fountains having to be replenished twice or thrice a day, and the birds repeatedly buried their widely-opened beaks beneath the surface of the water, drinking greedily. No nervous symptom or joint trouble of any sort was noticed.

The acuteness of the attack lasted some two to three weeks, but the droppings remained liquid till the end of the summer. By the middle of March the animals were bright and active again, and their weights, which had not increased during their illness, began to rise. From now until their deaths they remained in the best of health except for the usual malaise associated with moulting. In November, 1905, three of them (the fourth having been recently killed) were shown at the Physiological Society, and nothing in any way wrong was detected in their appearance. The hen, during the two summers she was under observation, laid occasional eggs. They were of normal size, the yolks were pale, and the flavour excellent.

A record was kept of the weights and is shown in the table. Cock A and Cock B lost weight during the first two months, but thereafter rapidly increased, their progress being checked only by the acute attack described above. Cock C (a cockerel), which was at a period of rapid growth, remained constant in weight for two months, and then increased until at the end of twelve months it had more than doubled its size (Fig. 1).

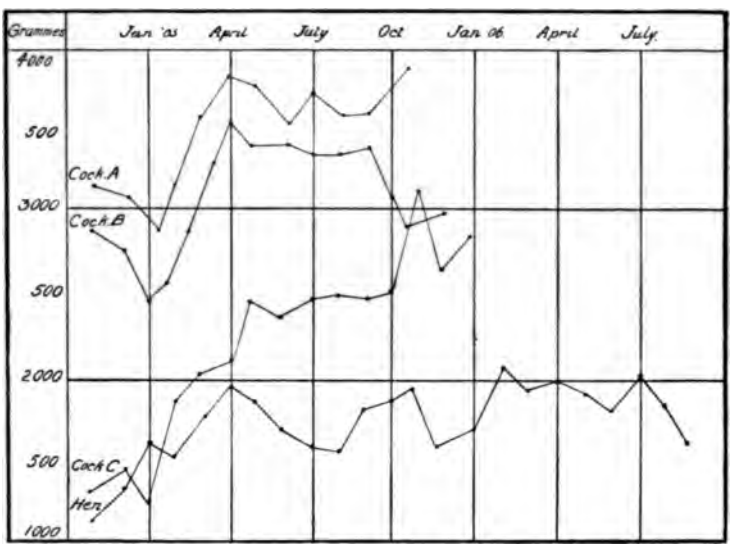
Post-mortem Examinations.

Each bird was killed by coal-gas and a complete post-mortem examination at once made. In each case microscopic sections were prepared of the following organs:

thyroid, parathyroid, suprarenal, pituitary, spleen, liver, small intestine, kidney, pancreas, bone-marrow, lymphatic gland, and testis or ovary. Specimens of these organs were fixed in 10 per cent. formalin in normal saline, prepared by the paraffin process and stained with hæmatoxylin and eosin.

Cock A.—An old bird nearing the end of a period of

FIG. 1.



Weights of fowls.

moulting, killed October 20th, 1905, after being on the protein diet for more than ten months. The body was very well nourished with a copious deposit of fat, especially at the root of the neck and in the abdomen. In the subcutaneous tissue covering the sternum were found numerous deposits of a chalky character varying in size from a millet seed to a small bean. Others occurred beneath the skin of the right thigh and one large deposit lay at the back of the neck. Their chemical examination

was kindly undertaken by Dr. Forster Morley, who found them to consist of calcium phosphate with just a trace of oxalate but no uric acid. The thyroid glands found at their normal site at the root of the neck on or near the origin of the carotid vessels were pear-shaped and somewhat translucent bodies of a pale red. The right weighed 0·20 gr., the left 0·28 gr. Posterior to the right and quite separate from it was a small parathyroid weighing 0·026 gr., while in a corresponding place on the left side were two parathyroids, one anterior to the other, and together weighing 0·036 gr. The spleen was firm and the size of a small horse-chestnut. The bone-marrow was soft and very dark red, almost brown, in colour. The joints of the extremities were examined, but no gouty deposit was present, and the articular cartilages were smooth, glistening, and well lubricated by synovia. The long bones were normal in every respect (shape, density, thickness, and regularity of shaft). No gross pathological change was noted in any portion of the body.

Cock B.—Killed December 15th, 1905, after being on the protein diet for over a year. Body well nourished with a fairly ample deposit of fat. One or two calcareous nodules found subcutaneously. The thyroid glands, in the usual position, were pale pinkish-white in colour and not translucent. The right weighed 0·22 gr. and the left 0·21 gr. Close to the posterior pole of each lay a parathyroid, that on the right weighing 0·031 gr. and that on the left 0·050 gr. The bone-marrow was dark in colour. The joints were healthy and the long bones normal. No gross pathological change was found in any viscus.

Cock C.—Killed January 9th, 1906, after thirteen months of meat-feeding. The body was well nourished with ample deposits of fat, especially at the root of the neck. No subcutaneous calcareous bodies were present. The thyroids were pinkish-red and translucent, and the weights of the right and left 0·21 gr. and 0·18 gr. respectively.

Just posterior to the left thyroid were two parathyroids weighing 0·039 gr., while in contact with the posterior pole of the right was a bilobed parathyroid weighing 0·055 gr. The bone-marrow was dark red—very much like blood-clot. The pancreas, for nearly half its length, presented a curious mottled-green appearance. All the other structures, including the joints and long bones, were normal.

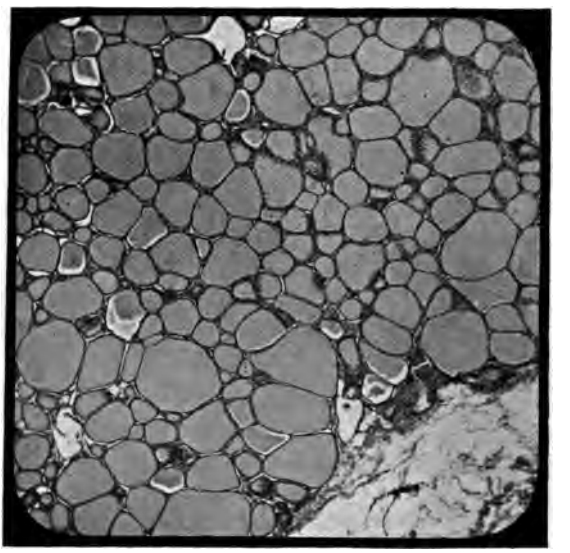
Hen.—Killed September 22nd, 1906, after being on a protein diet for one year and ten months. The feathers were straggling, owing to moulting. The body was very well nourished with a rich deposit of fat everywhere. A few calcareous concretions were found under the skin. The thyroid glands, in the usual position, appeared normal. They presented the customary shape, were reddish-brown, and weighed 0·08 gr. and 0·11 gr. on the right and left side respectively. Posterior to the right, but not in contact with it, was a small parathyroid weighing 0·018 gr., while posterior to the left were two parathyroids, the anterior being minute, and both together weighing 0·021 gr. The bone-marrow was dark red. The joints were healthy, and no change was observed in the long bones. The pituitary was pale brown. No gross pathological change was noted in any viscus.

Microscopical Examination.

Thyroid and parathyroid glands.—The thyroids of all four birds were similar in appearance on microscopical section. The glands were divided into small compartments by delicate connective-tissue trabeculæ enclosing thyroid vesicles. These presented the usual variations in size and were lined by a single layer of flattened epithelial cells arranged round masses of colloid secretion. Intervesicular glandular tissue was almost entirely absent, as is often the case in the thyroids of birds. Occasionally the cells approached the outline of a squat cubical epithe-

lium, and some vesicles were lined partly by the flattened variety, partly by the squat cubical. There was no folding of the lining epithelium, nor had any proliferation of the secreting cells occurred (Fig. 2). The parathyroid cells consisted of the usual clumps of polyhedral, embryonic-looking cells, the protoplasm of some being clear and of

FIG. 2.



Thyroid of fowl after prolonged protein-feeding, showing the normal structure of the avine thyroid.

others being oxyphile and granular. No detail that was not normal to the avine thyroid was present.

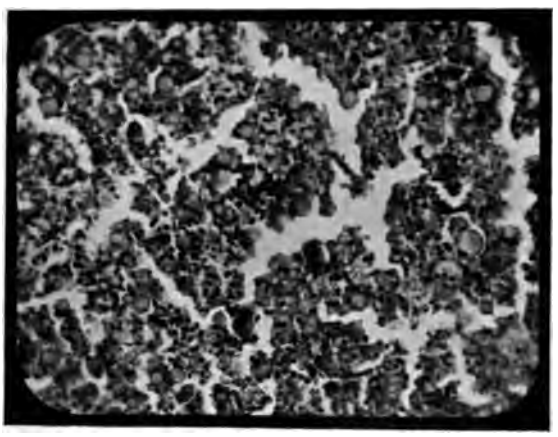
Pituitary gland.—All four pituitary glands showed the usual cellular structure, but there was a remarkable increase in the quantity of colloid present in the bodies. Drops of this secretion were scattered to excess through every section (Fig. 3).

No change was observed in any of the other viscera.

Remarks.

All the birds thrived on the diet, increasing considerably in weight and laying down plentiful fat. It is difficult to say to what extent, if at all, the special feeding was responsible for the attack of diarrhœa. The illness did not appear until some weeks after the dietetic treatment had commenced, and, although no modification was made in the character of the food, the symptoms passed off and never returned. Poultry are known to be subject to epidemic attacks of diarrhœa (4). On the other hand it

Fig. 3.



Pituitary gland of fowl after prolonged protein-feeding, showing many drops of colloid.

was noted as long ago as 1660 (5) that "the flux in poultry commeth with eating too much moyst meat." My impression was that the diarrhœa in these experiments was not directly attributable to the character of the food.

With a view to comparison with the above results Chalmers Watson's experiments may be briefly summarised.

Eight fowls were dieted for from fourteen to sixteen months. Two died from "acute nervous derangements" at the fourth and seventh months respectively, and two

at the end of six months were returned to the poultry-yard in apparently perfect health. Of the four remaining, one (No. 4), which at the post-mortem examination was found to be tuberculous, gained in weight, the others (Nos. 3, 5, and 6) lost weight. The tuberculous fowl suffered from an attack of opisthotonos shortly before death. Another fowl presented attacks of "paralysis" from which it recovered without any change in the *régime*. The two remaining birds, both cocks, showed motor symptoms, one being paretic, the other paralysed. In all there was a striking absence of the clinical features of gout. The post-mortem findings were as follows :

The two fowls that succumbed at the fourth and seventh months repectively revealed no gross lesions. Their thyroid and parathyroid glands were not examined microscopically. Fowl No. 4 presented tuberculous ulceration of the ileum and a liver studded with caseous tuberculous nodules. Its thyroids were normal. Of the fowls Nos. 3, 5, and 6 the only changes common to all were found in the thyroid and parathyroid glands, which were recorded as much enlarged in Nos. 3 and 6, and enormously enlarged in No. 5. Histological examination showed (*a*) increase in size of vesicles which were filled with colloid, (*b*) diminution in inter-vesicular glandular tissue, (*c*) increase in the number of cells in the walls of the vesicles. Sections of the parathyroids were normal. Changes in the long bones were observed in Nos. 3 and 6. The bones were increased in thickness and irregular on the surface and the femora were markedly curved. The marrow had almost entirely disappeared, being replaced by a fairly dense, non-cellular tissue presenting some of the characters of an osteoid tissue. A similar appearance was seen in the outer part of the bone, and apparently represented much altered Haversian spaces. Chemical examination showed a great increase in the proportion of organic matter.

Comparing the results now being recorded with those above quoted, they will be found to agree only in the absence of any changes suggestive of gout. The following

points may be specially emphasised: (1) There were no nervous or motor symptoms at any time; (2) the health of all four birds was excellent at the time they were killed; (3) the thyroid and parathyroid glands were not enlarged (*vide infra*), and their sections presented the normal features of the avine thyroid; (4) the shafts of the long bones were not increased in thickness, nor were they irregular or curved; (5) on the other hand, there was a striking increase of colloid in the pituitary glands—organs not examined by Chalmers Watson.

These results show that fowls fed for a long period on an exclusive protein diet, and at the same time kept under healthy conditions, including a supply of lime and other bases, present no loss of weight, no changes in the thyroid or parathyroid glands, and no changes in the osseous system.

The two most important points raised by the above observer are the changes in the osseous system and the hypertrophy of the thyroid glands.

(1) *The changes in the osseous system.*—These have already been detailed, but there is ample ground for regarding them as due to other causes than excessive meat diet. The context provided shows that the changes should be ascribed to an insufficient supply of calcium and other bases. The necessity of supplying fowls with ample lime is recognised by all poultry-keepers, and a deficiency of this material produces a condition known to them as "leg-weakness." Wright, in his 'Book of Poultry' (10), which I am informed is a standard work on the subject, says that leg-weakness "may arise from . . . a deficiency of bone-matter; and the symptoms . . . consist of a more or less constant squatting on the ground, instead of standing or walking." Chalmers Watson says of his birds that they "showed a greater tendency to sit, and would rest on the ground for much longer periods than was natural." The possibility suggested by Wright's statement that Chalmer Watson's birds were really suffering from "leg-weakness" is supported by this latter's remark

that "the fowls showed a marked tendency to pick the lime off the walls, and continued doing so for some time," and that chemical analysis of the bones "showed a great increase in the proportion of organic matter." The date at which the symptoms of "leg-weakness" became marked is not specifically given, but can be calculated from the context, where it is stated that a tendency to lameness which had affected one of the birds was not manifest "after the fifth month," and that "a little later" the increased tendency to squat on the ground appeared. This fixes the onset of the symptoms at or about the sixth month. During the first six months the birds, with the exception of one cock (No. 6), were kept in the country in a hen-run, the floor of which was composed of natural soil, but at the end of six months they were transferred to iron cages in the Physiological Laboratory of Edinburgh University. The symptoms of "leg-weakness" did not, therefore, show themselves so long as the birds were kept in surroundings where a natural supply of grit was to be obtained.

It may be further mentioned that in a later paper, read before the Pathological Society in December, 1906, Chalmers Watson described effects on the bones of rats of prolonged protein-feeding, but in the subsequent discussion it was pointed out by Spriggs (7) that an exclusively protein diet implied a deficiency of calcium salts and of bases generally, and that the osseous changes recorded were such as would result from a deficiency of these substances. Chossat (1), in 1842, showed that pigeons fed on grain freed from any accompanying grit were unduly liable to fractures, and that post-mortem the bones were soft. These changes did not occur if lime was added to the grain.

The conclusion seems warranted that the changes in the bones described by Chalmers Watson should have been attributed, not to the excess of meat, but to a deficiency of lime and other bases.

(2) *Hypertrophy of the thyroid glands.*—The weights of these bodies, as given by Chalmers Watson, are as follows:

No. 1.—No weights recorded.

No. 2.—No weights recorded.

No. 3.—Weight of one thyroid 1·25 gr., weight of second not recorded. Body-weight 1680 gr.

No. 4.—Weights of thyroids not recorded—stated to have been normal. Body-weight 2625 gr.

No. 5.—Weights of thyroids 9·5 gr. and 3·0 gr. respectively. Body-weight 2540 gr.

No. 6.—Weight of one gland 1·15 gr., weight of the other gland not recorded. Body-weight 1640 gr.

The average weight of a thyroid gland is given as 0·14 gr.

It will be seen that of the six fowls that were examined post-mortem, three showed no change in the thyroids, one showed great hypertrophy of both lobes, and in the remaining two one lobe was enlarged, but no record was made of the condition of the other. This omission to record both weights in three cases and one weight in each of two cases is to be regretted, as it detracts from the value of the conclusions obtained. (*a*) Fowls vary in weight between very wide limits, and an amount of thyroid tissue, normal to a large bird, would represent hypertrophy in a small. Numbers 4 and 6, differing so much in weight, would be expected to possess thyroids differing correspondingly in weight. In my own series one bird was more than twice the weight of another, and it would obviously be fallacious to judge of the degree of hypertrophy by comparison of the absolute weights of the organs. The only trustworthy standard for comparison is the amount of thyroid gland per kilogram body-weight, and in only one of the above examples can this figure be ascertained. (*b*) The thyroid glands in birds, as in mammals, may be of unequal size (3), and a record of the weight of one gland alone is no index to the total amount of thyroid tissue; for example, in No. 5 one gland was more than three times the weight of the other.

As regards parathyroid glands my experience has been (3) that they possess no constancy of size, but vary between wide limits.

An examination of the thyroid gland of two full-grown healthy fowls gave the following figures :

Body-weight in grammes.	Weight of thyroids.	Total weight of thyroid.	Thyroid per kilogram body-weight.
(1) 1871 ...	0·14 0·15	0·29	0·155 } Average 0·164
(2) 1899 ...	0·15 0·18	0·33	0·174 }

The following table gives the weights obtained from the four birds of my own series :

	Body-weight in grammes.	Weight of thyroids.	Total thyroid.	Thyroid per kilogram body-weight.
Cock A ...	3890	0·20 0·28	0·48	0·123 } Average 0·131
Cock B ...	2980	0·22 0·21	0·43	0·144 }
Cock C ...	2805	0·21 0·18	0·39	0·138 }
Hen ...	1617	0·08 0·11	0·19	0·118 }

If the weights of the thyroids were considered apart from the body-weight, three out of four would appear to represent hypertrophy. The fallacy of the conclusion appears when the weights per kilogram body-weight are obtained. These figures show that in none of the series did the thyroid hypertrophy.

It is probable that in Chalmers Watson's examples where only one lobe was weighed it was the larger lobe that attracted attention. The increase in size in these two specimens was less than one-fourth that in the larger lobe of the remaining fowl. We therefore see that the factor or factors, whatever they may have been which lead to hypertrophy of the gland, operated unequally on three birds and not at all on the other three.

Two additional points of great importance must be borne in mind. In the first place enlargement of the thyroid gland is known to occur in many animals, including birds, quite apart from any question of diet. Sloan

(6) has described goitre in crows, and Fischer in 1896 (2) stated that domestic fowls are subject to the same condition. The possibility that fowls Nos. 3, 5, and 6 were really the subject of pathological goitres has not been excluded, and the ascription of the hypertrophy to excessive protein-eating appears premature. This is the more probable seeing that apart from my own experiments many other investigations on the effect of protein-feeding have been conducted, but as Chalmers Watson states (9) in none has any change in the thyroid glands been recorded.

The second point to which due weight must be given is that each of the three fowls in which the thyroid was affected presented evidence of disease elsewhere. The heart of one was hypertrophied, its duodenum was dilated, and before death its blood presented a very striking deficiency of white blood-corpuscles; in the second there were very pronounced atrophic changes in the intestinal tract and the duodenum was dilated; while the third showed profound atrophic changes in the intestinal canal, a great increase of pigment in the liver, and before death a very pronounced leucopenia. If, further, it be accepted that these birds had so suffered from a want of lime and other bases as to develop in each instance "leg-weakness," and in two out of the three instances definite changes in the bones, it is obvious that it is well-nigh impossible to trace the influence of an excess of protein among so many morbid conditions, and any conclusions in this direction must be received with the greatest caution.

On the Thyroid Gland in Birds of Prey.

In connexion with the foregoing the comparative histology of the thyroid glands in birds is of great interest. There is a large class of birds—the raptores or birds of prey—feeding exclusively on meat, and the condition of their thyroids should provide evidence bearing on the

relation between the glands and meat-eating. I cannot find any published records of the structure of the thyroids of these birds, but I have recently, by the courtesy of Mr. Beddard, the Prosector to the Zoological Society of London, had opportunities of examining post-mortem representative types of the eagles, vultures, owls, falcons, and kites, as well as many non-raptorial birds. Altogether more than thirty species were examined, but no obvious difference in sizes between the thyroids of carnivorous, omnivorous, and graminivorus birds was noted.

Accepting for the moment the conclusions of Chalmers Watson it would be expected that the thyroid glands of birds of prey would show to a marked degree the features he has ascribed to excessive protein-feeding. It should be found (1) that the thyroids of these birds consist of large vesicles filled with colloid, and that the intervesicular glandular tissue is diminished; (2) that the thyroids of non-raptorial birds when contrasted with these show vesicles of comparative small size, a not over-abundant secretion of colloid, and an average amount of intervesicular glandular tissue.

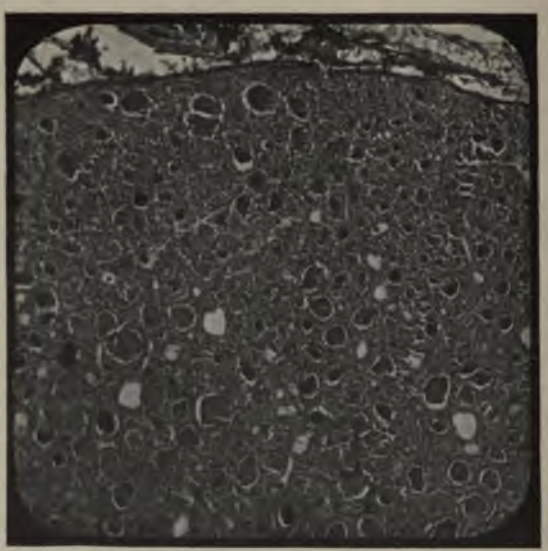
In the specimens I have examined I find that this relation does not exist and that not only were the structural characters, which we are assuming to be associated with meat-feeding, not specially marked in carnivorous birds, but in some instances the exact contrary was pronounced. Further, these characters were strikingly marked in many species which subsist chiefly or exclusively on vegetable food. Micro-photographs of a series of avine thyroids will illustrate these points. The specimens are arranged in three groups, first, a group representing the carnivorous birds of prey, next, a group of fish-eating birds, and lastly, a group of graminivorous birds.

(1) *Carnivorous birds of prey.*—These are represented by the short-toed eagle (*Circaëtus gallicus*), the barn owl (*Stryx flammea*) and the Elenora falcon (*Falco elenoræ*).

FIG. 4.

Thyroid of Elenora falcon (*Falco elenoræ*)—a carnivorous bird—showing entire absence of vesicles and only a slight amount of colloid, which is restricted to lymphatic spaces in the connective-tissue trabeculæ.

FIG. 5.



Thyroid of herring-gull (*Larus argentatus*)—a fish-eating bird—showing vesicles, for the most part small, and a sparse secretion of colloid. Intervesicular glandular tissue is present to considerable amount.

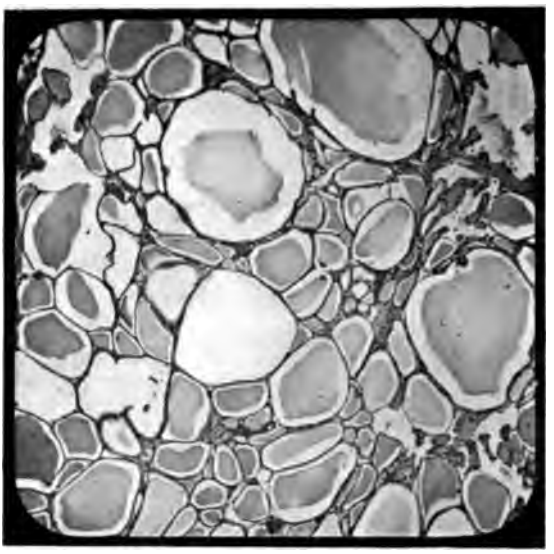
In the thyroid of the eagle the vesicles are of good size, and contain plenty of colloid; intervesicular tissue is absent. In the owl many of the vesicles are quite small, and not a few contain no colloid and intervesicular tissue is present to considerable amount. The Elenora falcon possessed a striking thyroid in which no vesicles at all are present, the polyhedral cells still retaining their primitive arrangement into solid clumps; colloid secretion is present only to a very slight extent, and is found occupying chiefly the lymphatic spaces in the connective-tissue trabeculæ (Fig. 4).

(2) *Fish-eating birds.*—The examples of these are the guillemot (*Lomvia troile*) and the herring gull (*Larus argentatus*). In the guillemot the vesicles are not small and the sections show a lavish secretion of colloid and a minimum of intervesicular tissue. The thyroid of the herring-gull contained no large vesicles anywhere and the secretion of colloid is sparse. It will be noticed that the vesicles are separated from each other by an unusually large amount of intervesicular tissue (Fig. 5).

(3) *Graminivorous birds.*—As types of this group are the ostrich (*Struthio camelus*), the crowned crane (*Balearica pavonina*), the white-backed piping crow (*Gymnorrhina leuconota*) and the tataupa tinamou (*Crypturus tataupa*). In the ostrich the vesicles vary much in size, but throughout the greater part of the section they are large. There is a free secretion of colloid, and intervesicular glandular tissue is practically absent (Fig. 6). In the crane the vesicles of the thyroid are large, the secretion of colloid copious, and intervesicular glandular tissue almost entirely absent (Fig. 7). The piping crow shows vesicles for the most part small, though here and there a large follicle appears; the amount of colloid is moderate and intervesicular tissue is present, though not to a very great amount. Lastly, in the tinamou no vesicles are present, the cells of the entire gland retain their primitive arrangement, and only at rare intervals is colloid found (Fig. 8).

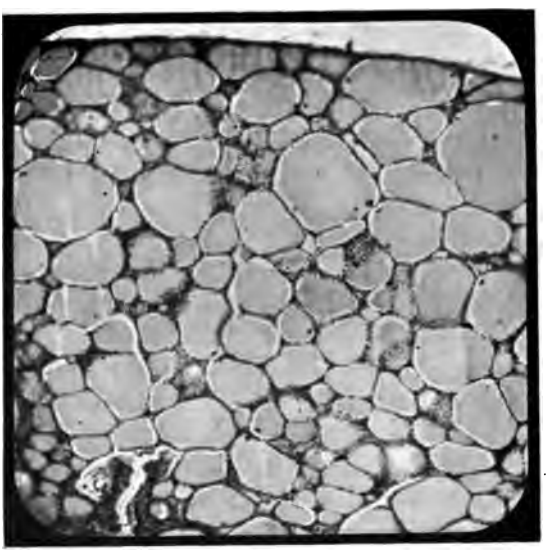
These few specimens show all varieties of structure

FIG. 6.



Thyroid of ostrich (*Struthio camelus*)—a graminivorous bird—showing vesicles, for the most part large, a free secretion of colloid, and no intervesicular glandular tissue.

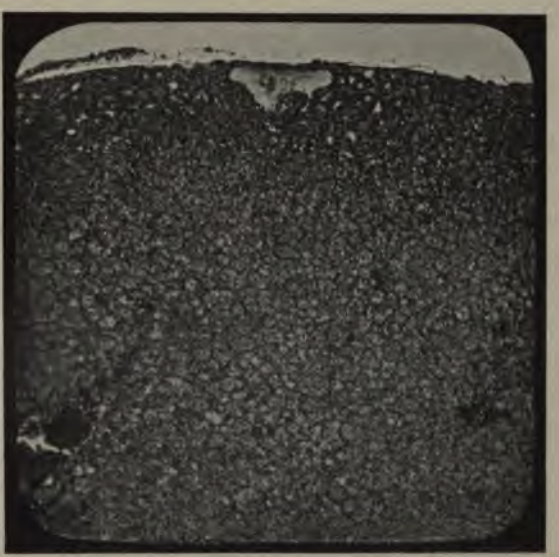
FIG. 7.



Thyroid of crowned crane (*Balearica pavonina*)—a graminivorous bird. Vesicles on the whole large, plentiful colloid, and little or no vesicular glandular tissue.

from the thyroid with large vesicles, much colloid and little intervesicular glandular tissue, to the thyroid in which vesicles are completely absent, the whole gland consisting of tissue identical with that which constitutes intervesicular glandular tissue. Both the extreme types are represented in the carnivorous and in graminivorous birds alike, and it must be concluded that the structure

FIG. 8.



Thyroid of tataupa tinamou (*Crypturus tataupa*)—a graminivorous bird. No vesicles are present; the only colloid is that in a lymphatic beneath the capsule. The resemblance of this thyroid and that of the falcon to a parathyroid gland is marked. All the figures represent the same degree of magnification.

of the normal avine thyroid is not modified by the diet-habits of the birds.

CONCLUSIONS.

(1) Fowls kept for long periods on a protein diet but otherwise under healthy conditions showed no hypertrophy or microscopic change in the thyroid glands.

(2) Their bony tissues are not affected provided the deficiency of calcium and other bases in the meat-diet is made up by a supply of lime.

(3) In none of the birds was any change indicative of gout observed.

(4) Each of the four fowls maintained under the above conditions showed an unusual amount of colloid in its pituitary gland.

(5) The thyroid glands of carnivorous birds of prey are not obviously larger, proportionately, than those of other classes of birds, nor are there any peculiarities of microscopical structure in the glands of carnivorous and graminivorous birds referable to differences in their natural foods.

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3. Forsyth.—Journ. Anat. and Physiol., vol. xlii, p. 141.

4. Klein.—Grouse Disease, 1892.

5. Markham, G.—Cheape and Good Husbandry for the well-Ordering of all Beast and Fowles, and for the general Cure of their Diseases, 10th ed., 1660.

6. Sloan.—Lancet, 1887, vol. i, p. 1082.

7. Spriggs.—Lancet, 1906, vol. ii, p. 1779.

8. Watson, Chalmers.—Med.-Chir. Trans., 1894, vol. lxxxvii.

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10. Wright.—Book of Poultry. Cassell and Co.

DISCUSSION

Dr. E. I. Spriggs: This subject is of importance to dietetics, especially with respect to the alleged ill-effect of an excessive meat diet. Dr. Forsyth's experiments indicate clearly that when a sufficient amount of suitable inorganic material is supplied the bones are not affected in fowls by such a diet. The evidence which he has put before us, both from his own fowls and from a study of comparative anatomy, that the thyroid body is also not definitely influenced in any constant way by such a diet, is very strong. After hearing Dr. Chalmers Watson's paper on the effects of an excessive meat diet on rats, read before the Pathological Society, I suggested, as Dr. Forsyth has mentioned, the defect of lime as a possible cause of the bone changes, and I have since been conducting experiments on rats with the object of obtaining evidence on this point. I have brought these animals here this evening in order to show the condition of their coats. It will be remembered that changes in the fur were a marked feature described by Dr. Chalmers Watson in his rats when upon a meat diet.

[Dr. E. I. Spriggs exhibited three series of rats fed upon different diets. The first series were fed on meat and distilled water, the second on meat with lime salts (calcium phosphate) and distilled water, and the third (the control series) on bread and milk. The coats of the first series were rough and dirty. The coats of the second series were good, but showed a just perceptible difference from those of the third series, which were normal. The conclusion drawn was that the change observed in the fur of these animals when placed upon a meat diet is very much less marked if lime salts are supplied in suitable quantity.]

Dr. F. Parkes Weber: It is very interesting that Dr. David Forsyth in his experiments found none of the "leg-weakness," which formed such a striking symptom in the fowls kept on a meat diet by Dr. Chalmers Watson. In a paper on some cases of polyneuritis of obscure origin (shortly to be published in the 'International Clinics'), I have incidentally suggested that the leg-weakness observed in Dr. Watson's fowls was probably due to a "polyneuritis gallinarum," similar to that experimentally produced by Eijkman, Maurer, and others, by feeding fowls on rice (*without the husk*). Polyneuritis in poultry has been also produced by Grijus and Holst,[1] by feeding the fowls with sterilised or highly-cooked meat; how much of it is due to deficiency of lime-salts and how much to oxalic acid poisoning

[1] *Cf.* Prof. Axel Holst's paper "On Polyneuritis of Poultry" at the Epidemiological Society, London, May 31st, 1907.

has still to be settled. Oxalic acid appears to produce polyneuritis like arsenic, carbon monoxide, and carbon bisulphide. Both Maurer and Treutlein have recently produced a "polyneuritis gallinarum," similar to that of Eijkman, but obtained by giving the fowls oxalic acid. Treutlein thinks that oxalic acid, introduced as such, or produced in the animal's crop by decomposition of the rice-meal, causes a separation of calcium from the tissues. He was able to arrest the bad symptoms (including the polyneuritis) by administering an excess of calcium carbonate, thus probably neutralising the oxalic acid.

Dr. DAVID FORSYTH, in reply, said: In the first place, sir, I should like to thank the Society for the kind reception they gave to my paper. The chief contention I have submitted to-night—that the changes in the bones which had been ascribed to an excess of meat were due to a deficiency of lime and other bases—is supported in a striking manner by the very interesting series of rats which Dr. Spriggs has just shown. The differences in appearance between his meat-fed animals and his meat- and lime-fed animals was obvious to us all, and the ill-conditioned state of the former when contrasted with the comparative sleekness of the latter was an object-lesson in itself of the importance of the lime factor in these diet researches, and of the necessity of giving full attention to this point in conducting all such investigations. I shall await with much interest the final results of Dr. Spriggs' experiments. Dr. Parkes Weber spoke of polyneuritis as a possible result of meat-feeding. This condition was certainly not present in any of my own fowls, in none of whom was there at any time any symptom suggestive of nerve trouble. The relation between diet and polyneuritis is interesting, but the disease has been described as a result, not only of a meat diet, but also of a carbohydrate diet. Marchoux and Salibeni have recorded [1] an outbreak of polyneuritis among a number of monkeys fed on boiled rice. The symptoms passed away when cooked maize was substituted, reappeared on a return to the rice, and finally cleared up on again employing maize.

[1] 'Compt. Rend. Soc. de Biol.,' 1903, vol. lv, p. 1251.

DEMONSTRATIONS

Demonstration (by Epidiascope and Microscope) of the Bodies described by him in 1895 as "Protozoa" in Syphilitic Lesions, with subsequent observations.

By J. Jackson Clarke, M.B.

[January 8th, 1907.]

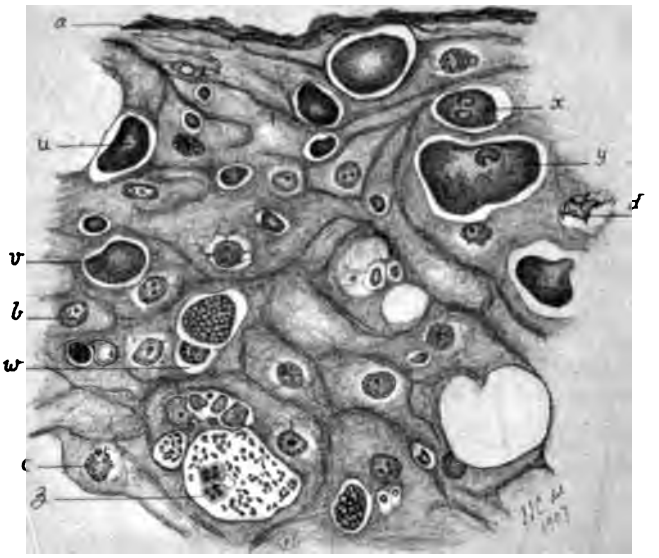
The object of this communication is to call your attention to what I believe to be the connection between the *Spirochæta pallida*, described by Schaudinn in 1905, and the Protozoa in syphilitic lesions which I described ten years earlier, and some of which are shown in the section under the microscope now before the Society; anyone who is practically acquainted with the commoner parasitic protozoa will recognise the striking resemblance of these bodies to such protozoa.

I. In Fig. 1 they are indicated as the bodies *u*, *v*, *w*, *x*, and *y*. Another (*z*) has subdivided, and each of the resulting minute bodies contains a chromatic portion, and represents in all probability a potential Spirochæta.

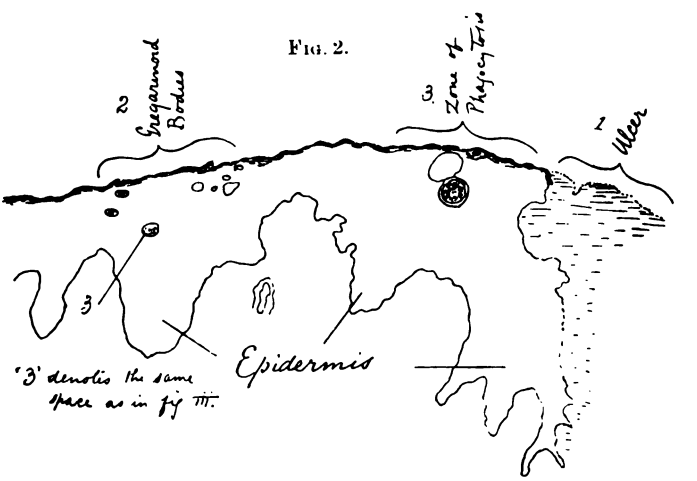
II. In Fig. 2 are shown the broad features of the same section; one of a piece of skin at the margin of a secondary syphilitic ulcer from a man's abdomen; (1) The ulcerated surface, (2) the part already shown on a larger scale in Fig. 1, and (3) a part where similar bodies to those of part (2) appear to be undergoing destruction by phagocytosis.

III. In addition to fixed and stained preparations it is important to study fresh preparations. In *Molluscum contagiosum*, as described by me in 1895, oscillating movements were associated with the origin of flagellated protozoa. In 1901 I examined on the warm stage a scraping of a chronic warty tertiary lesion of the hard palate of a man, who was subsequently cured by intra-muscular injections of mercury, and I made a graphic note of what I saw. This note is reproduced in Fig. 3.

Fig. 1.



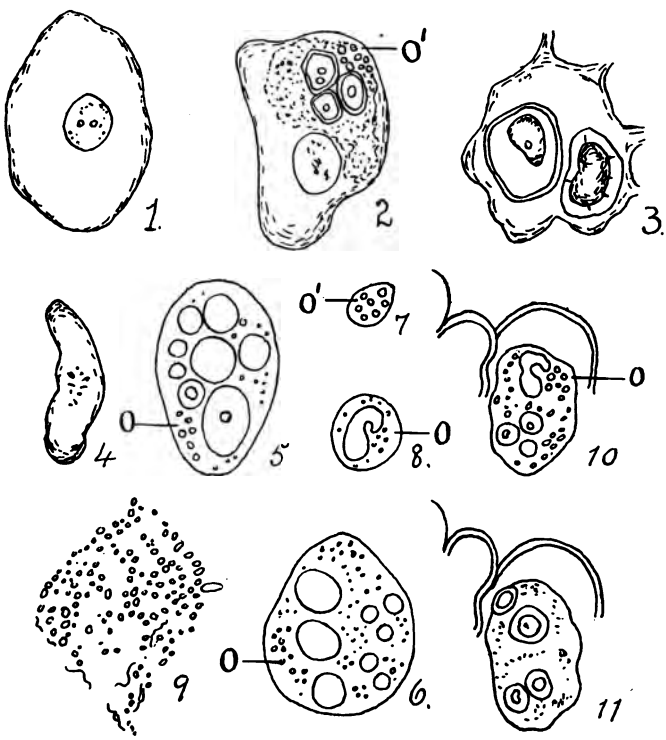
Part of the epidermis in a section of a spreading secondary syphilitic ulcer. *a*. Horny layer. *b*. Normal nucleus of epidermal cell. *c* and *d*. Nuclei of epidermal cells that are breaking up. *u*, *v*, *w*, *x*, *y*, *z*. Various stages of the bodies described as protozoa by the author in 1895 ('Trans. Path. Soc.'). Two bodies resembling leucocytes are present among the minute bodies at "*z*." Reduced from a camera drawing made with Leitz's drawing eyepiece and $\frac{1}{12}$ oil immersion lens.



Outline drawing of the section from which Fig. 1 was taken, to show the general relations of the parts.

(1) Is a normal epidermal cell devoid of movement; (2) a similar cell invaded by a parasitic mass, which includes three "bird's-eye" bodies and a cluster of granules in lively oscillation. I think these moving granules indicated a fourth bird's-eye body in course of formation by a process equivalent to

FIG. 3.



Bodies seen in a scraping of a tertiary warty lesion of the hard palate examined on Stricker's warm-stage under a $\frac{1}{12}$ in. oil-immersion lens. All the bodies except No. 11 were observed about 7 p.m. No. 11 is the same body as No. 10 but drawn as seen over two hours later when the stage had cooled down. Further description is given in the text. "*o*" signifies oscillation, ond "*o'*" lively oscillation.

internal budding. Among the remaining ones (5) and (6) are bodies which contain oscillating particles and resemble other bodies described by myself in sarcoma, and by Calkins and Bosc in variola; (9) is, I believe, probably a formation of Spiro-

chætæ in involution forms. I believe that the bodies I described in 1895 are homologous with the "gregarinoid" phases of the *Spirochæta ziemanni*, as described by Schaudinn. The gregarine-like bodies occur in such great numbers, and are distributed in the lesions of syphilis in such a way that the causal relationship of the mobile spiral forms (Spirochæta, or Spironema) to the disease must remain doubtful unless they can be shown to be members of the same cycle that also includes these gregarine-like forms.

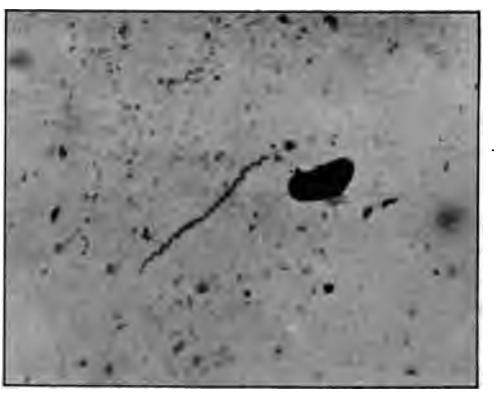
Demonstration of the variations of the *Spirochæta pallida*, etc. (Epidiascope and Microscope).

By Alex. Maclennan, M.B., C.M., Surgeon to Out-patients, Western Infirmary; Extra Honorary Surgeon, Royal Hospital for Sick Children, Glasgow.

[January 8th, 1907.]

I have to thank the Society for affording me the privilege of giving this demonstration. The specimens under the microscopes, and more especially the lantern slides to be shown, form a pictorial synopsis of what I wish to convey to the Society. I should like to make a few preliminary remarks.

Fig. 1.



Photomicrograph of typical *Spirochæta pallida* with straighter part about the middle. Distorted red cell at side, ring and tailed bodies scattered over the field. (×1800, taken by Dr. Buchanan.)

The *Spirochæta pallida* has taken the scientific world by storm. It may be that its resemblance to a corkscrew has given to it a fictitious personality. Be it what it may this organism is an object highly prized—on a glass slide. Whether it be the infective agent in syphilis or not cannot be definitely settled till the organism has been successfully cultivated and inoculated.

Fig. 2.

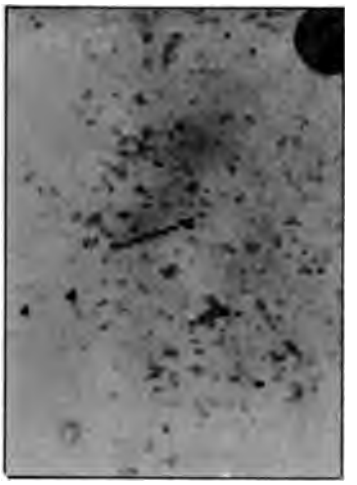


Fig. 3.

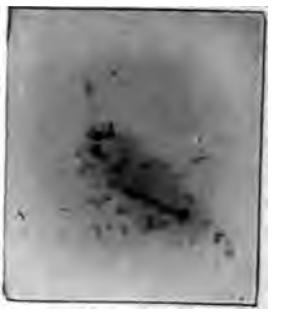


Fig. 2.—Photomicrograph (× 1800), taken by Dr. Buchanan, of spirochæte and tailed body.

Fig. 3.—Photomicrograph (× 1800), taken by Dr. Buchanan, of spirochæte.

Fig. 4.

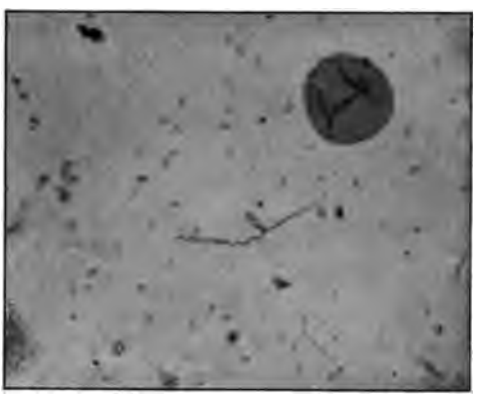


Fig. 5.

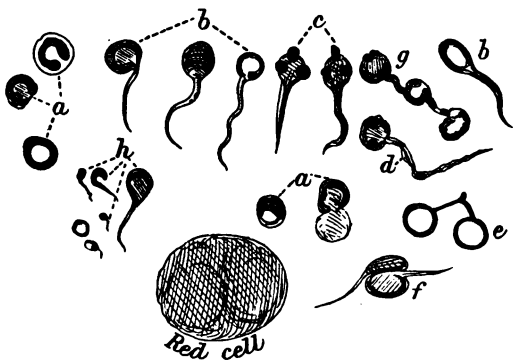
Fig. 4. - Photomicrograph of *Spirochæta pallida* with head attached, developed (?) from an excrescence. Ring body at side of one end. (× 1800, taken by Dr. Buchanan.)

Fig. 5.—Photomicrograph of spirochæte dividing longitudinally, also orming spores.

In the meantime I should like to accentuate certain facts which bear upon this point.

1. The organism has not been found in experimentally infective material. 2. It is admittedly scarce in the surface discharge in primary and secondary lesions (possibly because the organism is anærobic). 3. Congenital syphilis is considered (wrongly perhaps) to have a low infective power while its lesions teem with spirochætes. On the other hand the infectivity of the tertiary is more in conformity with the numbers of spirochætes present. 4. Smaller forms which I think are certainly related to the spirochætes are invariably present in preparations from syphilitic lesions.

FIG. 6.



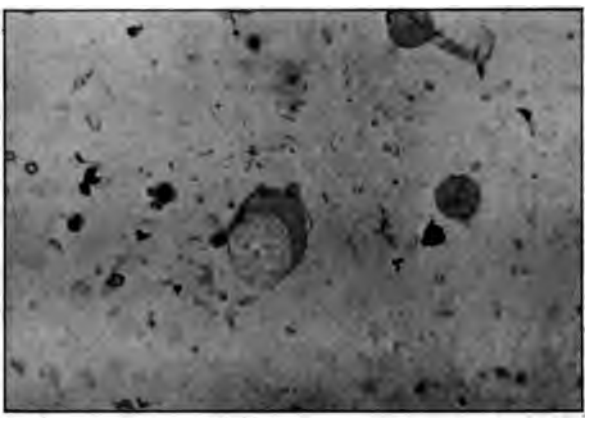
a Types of the ring and horseshoe bodies; *b*, the same with addition of tails; *c*, type found more or less universally, glands, blood, etc.; *d*, form showing the formation of a daughter body; *e*, unusual form, indicating fusion or fission; *f*, commoner configuration, indicating fusion or fission (probably the latter); *g*, chain of bodies approaching to spirochæte form, a further development of *d*; *h*, apparent uncurling of horseshoe body, with minute examples of same. A red cell is drawn in about the same proportion.

Personally, I do not consider that we have enough data definitely to accept the *Spirochæta pallida* as specific, but I accept its presence as diagnostic. This may seem rather paradoxical, but the explanation is that the *Spirochæta pallida* is only one phase in the life cycle of the infecting organism. Part at least of that life history I hope to show to-night.

The following lantern slides were shown:

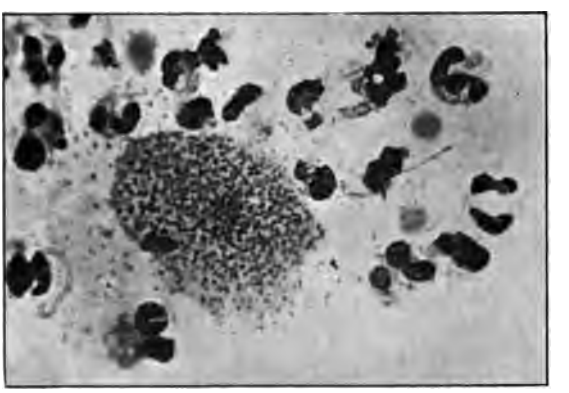
1. A typical *Spirochætes pallida* with a straight part in the centre. This form is explained by supposing the ends of the organism as the film dries to become fixed sooner than its

FIG. 7.



Photomicrograph (× 900) taken by Dr. Buchanan from smear from small, cleaned, primary sore. Stained with gentian violet. Shows a red corpuscle distended by small bodies, some similar to those shown in Fig. 6, *c*. Intermediate stages between normal cell and such a one or one ruptured were visible on the same film. Note also the numerous dots in the same field, identical with those contained in the red cell.

FIG. 8.

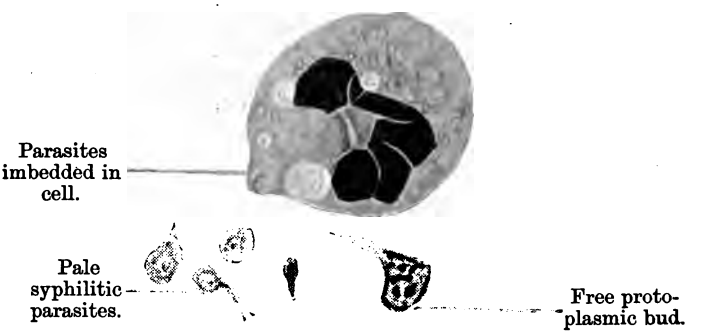


Photomicrograph (× 900) taken by Dr. Leslie Buchanan from preparation from vulvar sore, stained with Giemsa. Distended pus cell ruptured, freeing small bodies.

middle, and as the shrinkage takes place the still moist part becomes straightened. Possibly also the straight central part might be the preparation for division.

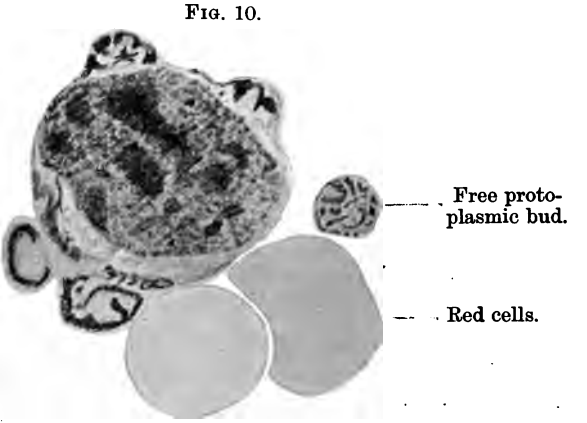
2. Spirochætes similar to the above, but less typical in form.

FIG. 9.



Multinuclear cell (pus) from primary sore. Stained thionin, Giemsa, gentian violet. Containing clear bodies.

FIG. 10.



Mononuclear cell from syphilitic gland stained by Giemsa's stain. Nucleus purple, protoplasm blue. Shows formation of protoplasmic buds.

3. *Spirochæta pallida* with a "head" (Fig. 2). The head has a peg which is thicker than the rest of the organism. Near the other end is a separate "head," with a spine from one side and a fine flagellum from the other. The similarity of these two shows conclusively that the head in either is not due to a complete

curl of the spirochæte. Such heads either with or without tails are the progenitors or the progeny of the spirochætes, probably both, but they are the infective agents.

4. Another spirochæte with a head having a peg at its side, a hollow sphere about its middle, and a solid head at the other end. This is not merely a partial longitudinal division, but a real breaking up of the spirochæte (Fig. 3).

5. Spirochæte with a hollow excrescence attached to its side, and with a ring lying free beside it (Fig. 4).

6. A spirochæte having sphere formations at both ends, and in the centre—probable longitudinal fission.

Fig. 11.

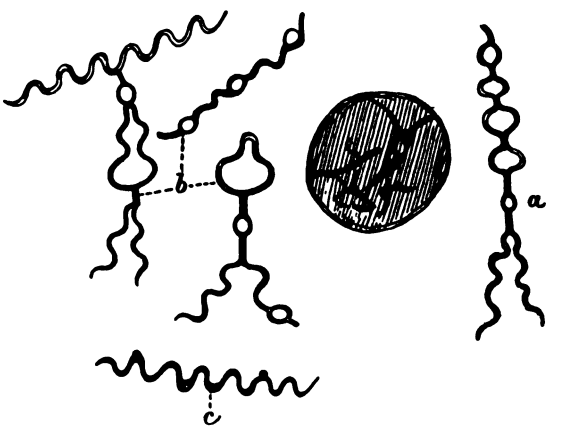


Diagram of types of spirochætes (*a*) indicating longitudinal division and also (*b*) formation of round bodies (a combined method); (*c*) *Spirochæta pallida* with dots or thickenings which tend to become hollow. A red cell for comparison in size.

7. Spirochæte dividing longitudinally and also forming spheres (Fig. 5).

8. *Spirochæta pallida* and *refringens* for comparison.

9. Small flagellated bodies from the juice of a lymphatic gland.

10. Similar bodies in a red cell.

11. Similar bodies in a pus cell.

12. Lymph gland mononuclear cell giving off buds. Such seen in all enlarged glands, producing forms very difficult to distinguish from parasites.

13. *Spirochæta refringens* "sporing."

FIG. 12.

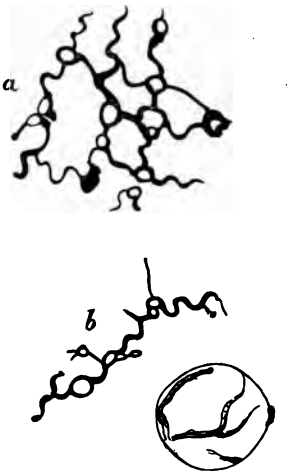


FIG. 13.

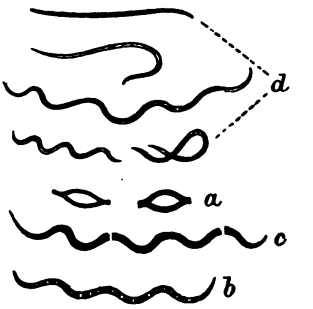
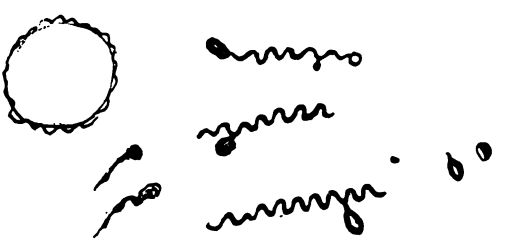


FIG. 12.—Drawing from composite types of spirochætes seen in smear stained with Giemsa and then with acetone gentian violet. There was a typical primary sore without secondaries. Nothwithstanding a full mercurial treatment the secondaries have since developed. *a* Shows three main spirochætes so attached as to form a colony, and fully establishing the view that the small-tailed bodies and the spirochætes are the same organism. *b* Is a single spirochæte showing the beginning of the colony formation or breaking up of the spirochæte.

FIG. 13.—Diagram of short *Spirochæta refringens*, showing: (*a*) longitudinal fission; (*b*) spore formation; (*c*) transverse division by attenuation of part; (*d*) various forms taken by the organism. Drawn from a film stained with acetone gentian violet from a primary commissural chancre. The film also contained the *Spirochæta pallida* in fewer numbers.

FIG. 14.



Types of spirochætes found in yaws with small bodies attached, as in the *Spirochæta pallida*. The same bodies are to be seen in countless numbers free in the field. Some of these are tailed, as are the similar bodies found in syphilis. A shrunken red cell is outlined for comparison. The smaller bodies are the infective agents, as are the corresponding ones in syphilis.

14. Drawing of spirochæte from yaws showing attached body and numbers of similar ones free.

15. Drawing of spirochæta from granuloma pudendi.

16. Slide to show the life cycle from the minute spheres to spirochætes, and both methods of multiplication (Fig. 17).

Fig. 15.

Spirochætes found in granuloma pudendi. These have very minute waves. They have also small bodies attached, and the same are to be found free in field. Their small size prohibits the same definite description as those occurring in yaws and syphilis. A red cell and a spirochæte of the refringens type.

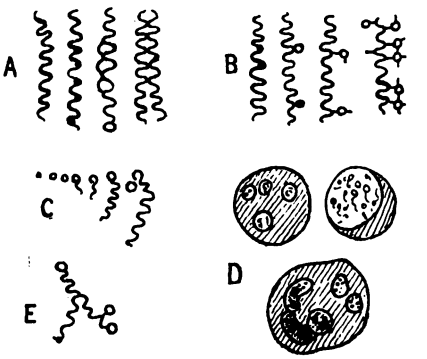
Fig. 16.

Spirochætes (Levaditi's method) from congenital syphilitic lung.

The following slides were exhibited under one twelfth oil immersion objective.

1. Pus-cell distended with sphere bodies.
2. *Spirochæta pallida* with head and sphere-tailed body.
3. *Spirochæta pallida* with sphere bodies near.
4. Spirochæte from yaws with sphere body attached, and numerous ones free in neighbourhood.

FIG. 17.



Life cycle. A. Longitudinal fission. B. Breaking up in small bodies. C. "Spores" into spirochætes. D. "Spores" in cells. E. Fission and breaking up.

5. Spirochætes from lung (congenital syphilis) stained by the method of Levaditi. Kindly given by Professor Doutrelepont, of Bonn (Fig. 16).

6. Cell from lymph gland showing formation of buds.

Demonstration of a Case of Recovery after Operation for simultaneous—(*a*) Acute Obstruction by Band formed of Vermiform Appendix and Omentum, and (*b*) General Septic Peritonitis.

By Jonathan Hutchinson, Jun.

[March 26th, 1907.

Mr. Hutchinson demonstrated the case of a young girl in whom acute intestinal obstruction by strangulation co-existed with general septic peritonitis from appendicitis.

The patient, four months before admission, had an attack of appendicitis which subsided without operation.

She was admitted on the fourth day of her second attack with severe symptoms of peritonitis—chiefly abdominal distension and rigidity, with constipation and very frequent vomiting. Besides constant pain and tenderness there was a notable symptom in severe spasmodic attacks of pain.

Abdominal section was performed on the day of admission. The inflamed vermiform appendix was found to be buried in adherent omentum, which together formed a vertical band. Between this band and the ascending colon a loop of small intestine was tightly strangulated, and in an almost gangrenous condition.

The pelvis, etc., contained free stinking pus.

The omental band and appendix were excised, and the abdominal cavity cleansed as far as possible, and drained in each flank and above the pubis. Large flakes of lymph were left on the intestine.

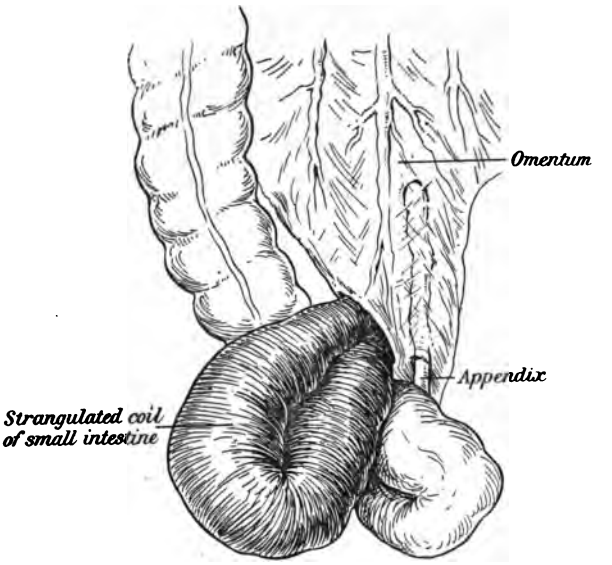
For some time the discharge was copious and offensive through the tubes, the last of which was removed at the end of four weeks from the operation.

Good recovery followed.

During the first twenty-four hours after the operation much benefit was obtained from the gradual infusion of saline solution into the rectum.

Mr. Hutchinson pointed out that, whilst intestinal obstruction by band was an occasional sequence of appendicitis, and that obstruction from inflammatory paralysis of gut was an im-

portant item of danger during an acute attack of appendicitis, yet that the coincidence of purulent peritonitis due to the appendix with true obstruction by band was certainly rare.



In the present case the fortunate recovery was largely due to the patient's youth and vigour, her condition at the time of operation having been as grave as it was possible to be.

Demonstration of Series of Drawings and Radiographs illustrating Dislocations and Fractures of Thumb and Great Toe.

By JONATHAN HUTCHINSON, Jun.

[March 26th, 1907.]

Mr. HUTCHINSON demonstrated, by means of the epidiascope, a series of radiographs relating to fractures and dislocations of the thumb and great toe. Amongst the latter were three cases of

dorsal dislocation of the first phalanx, together with its sesamoid bones and glenoid ligament. The resemblance to the comparatively common dorsal dislocation of the thumb was exact, though the deformity produced was not so conspicuous.

In one of the three cases Mr. Hutchinson succeeded in reducing the dislocated toe by hyperextension and downward traction. In both the others he had to resort to vertical division with a tenotome of the glenoid ligament, which bound the two sesamoid bones together. Traction after this was successful in both cases.

He pointed out the importance, in the case of the dislocation of the thumb, of using the tenotome in the right direction, namely, cutting downwards towards the base of the phalanx, so as to divide the ligament completely, and to allow the sesamoid bones to separate well.

A number of slides were shown illustrating fracture at the base of the metacarpal bone of the thumb (the fracture described by Bennett of Dublin), and its varieties and treatment were discussed.

A similar form of fracture was proved to occur in the case of the great toe.

INDEX

A General Index to the whole series (Vols. I—XC) of the 'Transactions' is kept in the Library, where it may always be consulted.

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