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OF

LONDON.

VOLUME THE SEVENTIETH.

LONDON:
LONGMANS, GREEN, AND CO.,
PATERNOSTER ROW.

1887.

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VOLUME THE FIFTY-SECOND.



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OCTOBER, 1887.

Those marked thus (†) have paid the Composition Fee in lieu of further annual subscriptions.

Amongst the non-residents those marked thus (*) are entitled by composition to receive the Transactions.

Elected

1846 *Abercrombie, John, M.D.

1877 Abercrombie, John, M.D., Assistant Physician to, and Lecturer on Forensic Medicine at, Charing Cross Hospital; 23, Upper Wimpole street, Cavendish square. *Trans.* 1.

1885 Abraham, Phineas S., M.A., M.D., 29, Upper Montagu street, Montagu square.

1851 *Acland, Sir Henry Wentworth, K.C.B., M.D., LL.D., F.R.S., Honorary Physician to H.R.H. the Prince of Wales; Regius Professor of Medicine in the University of Oxford; Broad street, Oxford.

Elected

1885 ACLAND, THEODORE DYKE, M.D., Assistant Physician to St. Thomas's Hospital and to the Hospital for Consumption and Diseases of the Chest, Brompton; 7, Brook street, Hanover square.

1852 †ADAMS, WILLIAM, Surgeon to the Great Northern Hospital and to the National Hospital for the Paralysed and Epileptic; Consulting Surgeon to the National Orthopædic Hospital, Great Portland street; 5, Henrietta street, Cavendish square. C. 1873-4. *Trans.* 3.

1867 AIKIN, CHARLES ARTHUR, 7, Clifton place, Hyde Park.

1837 *AINSWORTH, RALPH FAWSETT, M.D., Consulting Physician to the Manchester Royal Infirmary; Cliff Point, Lower Broughton, Manchester.

1839 ALCOCK, SIR RUTHERFORD, K.C.B., K.C.T., K.T.S., D.C.L., late H.M.'s Envoy Extraordinary at the Court of Pekin. *Trans.* 1.

1866 ALLBUTT, THOMAS CLIFFORD, A.M., M.D., F.R.S., Physician to the Leeds General Infirmary; 35, Park square, Leeds. *Trans.* 3.

1879 ALLCHIN, WILLIAM HENRY, M.B., F.R.S. Ed., Physician to, and Lecturer on Medicine at, the Westminster Hospital; 5, Chandos street, Cavendish square, W.

1863 ALTHAUS, JULIUS, M.D., Senior Physician to the Hospital for Epilepsy and Paralysis, Regent's Park; 48, Harley street, Cavendish square. *Trans.* 2.

1884 ANDERSON, ALEXANDER RICHARD, Resident Surgeon, General Hospital, Nottingham.

1881 ANDERSON, JAMES, A.M., M.D., Assistant Physician to the London Hospital; 84, Wimpole street, Cavendish square.

1862 ANDREW, JAMES, M.D., Physician to, and Lecturer on Medicine at, St. Bartholomew's Hospital; 22, Harley street, Cavendish square. S. 1878-9. C. 1881-2. *Trans.* 1.

1880 *APPLETON, HENRY, M.D., Staines.

1874 AVELING, JAMES H., M.D., Physician to the Chelsea Hospital for Women; 1, Upper Wimpole street, Cavendish square.

Elected

1851 *Baker, Alfred, Consulting Surgeon to the Birmingham General Hospital; 3, Waterloo street, Birmingham.

1873 *Baker, J. Wright, Senior Surgeon to the Derbyshire General Infirmary; 102, Friargate, Derby.

1865 Baker, William Morrant, Surgeon to, and Lecturer on Physiology at, St. Bartholomew's Hospital; Consulting Surgeon to the Evelina Hospital for Sick Children; Examiner in Surgery at the University of London; 26, Wimpole street, Cavendish square. C. 1878-9. *Referee*, 1886-7. *Lib. Com.* 1876-7. *Trans.* 7.

1869 Bakewell, Robert Hall, M.D., Ross, Westland, New Zealand.

1839 †Balfour, Thomas Graham, M.D., F.R.S., Honorary Physician to H. M. the Queen; Surgeon General; Coombe Lodge, Wimbledon Park. C. 1852-3. V.P. 1860-1. T. 1872. *Lib. Com.* 1849. *Trans.* 2.

1887 Ball, James Barry, M.D., 29, Belgrave road.

1885 Ballance, Charles Alfred, M.S., 56, Harley street, Cavendish square. *Trans.* 1.

1848 †Ballard, Edward, M.D., Inspector, Medical Department, Local Government Board; 12, Highbury terrace, Islington. C. 1872. V.P. 1875-6. *Referee*, 1853-71. *Lib. Com.* 1855. *Trans.* 5.

1866 *Banks, John Thomas, M.D., Physician in Ordinary to the Queen in Ireland; Physician to Richmond, Whitworth, and Hardwicke Hospitals; Regius Professor of Physic in the University of Dublin; Member of the Senate of the Queen's University in Ireland; 45, Merrion square, Dublin.

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

1879 Barker, Arthur Edward James, Surgeon to University College Hospital, and Assistant Professor of Clinical Surgery and Teacher of Practical Surgery at University College, London; 87, Harley street, Cavendish square *Trans.* 5.

Elected

1882 BARKER, FREDERICK CHARLES, M.D., Surgeon-Major, Bombay Medical Service [care of ARTHUR E. J. BARKER, 87, Harley street].

1833 †BARKER, THOMAS ALFRED, M.D., Consulting Physician to St. Thomas's Hospital; 109, Gloucester place, Portman square. C. 1844-5. V.P. 1853-4. T. 1860-2. *Referee*, 1848-51. *Trans.* 6.

1876 BARLOW, THOMAS, M.D., B.S., Physician to University College Hospital; Physician to the Hospital for Sick Children, Great Ormond street, and Assistant Physician to the London Fever Hospital; 10, Wimpole street, Cavendish square. *Trans.* 2.

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

1861 BARNES, ROBERT, M.D., 15, Harley street, Cavendish square. C. 1877-8. *Referee*, 1867-76. *Lib. Com.* 1869-73. *Trans.* 4.

1864 BARRATT, JOSEPH GILLMAN, M.D.

1880 BARROW, A. BOYCE, Assistant Surgeon to King's College Hospital, to the Westminster Hospital, and to the West London Hospital; 17, Welbeck street, Cavendish square.

1840 BARROW, BENJAMIN, Surgeon to the Royal Isle of Wight Infirmary; Southlands, Ryde, Isle of Wight.

1859 BARWELL, RICHARD, Surgeon to, and Lecturer on Surgery at, the Charing Cross Hospital; 55, Wimpole street, C. 1876-77. V.P. 1883-4. *Referee*, 1868-75, 1879-82. *Trans.* 11.

1868 BASTIAN, HENRY CHARLTON, M.A., M.D., F.R.S., Professor of Medicine in University College, London; Physician to University College Hospital and to the National Hospital for the Paralysed and Epileptic; 20, Queen Anne street, Cavendish square. *Referee*, 1886-7. C. 1885. *Trans.* 1.

1875 BEACH, FLETCHER, M.B., Medical Superintendent, Metropolitan District Asylum, Darenth, near Dartford, Kent.

Elected

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

1862 BEALE, LIONEL SMITH, M.B., F.R.S., Professor of the Principles and Practice of Medicine in King's College, London, and Physician to King's College Hospital; 61, Grosvenor street. C. 1876-77. *Referee*, 1873-5. *Trans.* 1.

1860 *BEALEY, ADAM, M.D., M.A., Oak Lea, Harrogate.

1856 BEARDSLEY, AMOS, F.L.S., Bay villa, Grange-over-Sands, Lancashire.

1871 BECK, MARCUS, M.S., Professor of Surgery in University College, London, and Surgeon to University College Hospital; 30, Wimpole street, Cavendish square. C. 1886-7. *Referee*, 1882-5. *Lib. Com.* 1881-5.

1880 BEEVOR, CHARLES EDWARD, M.D., Assistant Physician to the National Hospital for the Paralysed and Epileptic; 33, Harley street, Cavendish square. *Trans.* 1.

1871 BELLAMY, EDWARD, Surgeon to, and Lecturer on Anatomy at, Charing Cross Hospital; Lecturer on Artistic Anatomy to the Science and Art Department, South Kensington; Examiner in Surgery in the Victoria University, Manchester; 17, Wimpole street, Cavendish square. C. 1886. *Referee*, 1882-5. *Lib. Com.* 1879-81. *Trans.* 1.

1847 BENNET, JAMES HENRY, M.D., Mentone, Alpes Maritimes, France.

1880 BENNETT, ALEX. HUGHES, M.D., Assistant Physician to the Westminster Hospital; 76, Wimpole street, Cavendish square. *Trans.* 1.

1883 BENNETT, STORER, Dental Surgeon to, and Lecturer on Dental Surgery at, the Middlesex Hospital; Dental Surgeon to the Dental Hospital of London; 17, George street, Hanover square.

Elected

1877 BENNETT, WILLIAM HENRY, Assistant Surgeon to, and Lecturer on Anatomy at, St. George's Hospital; 1, Chesterfield street, Mayfair. *Trans.* 1.

1845 †BERRY, EDWARD UNWIN, 17, Sherriff road, West Hampstead.

1885 BERRY, JAMES, Assistant Demonstrator of Anatomy, St. Bartholomew's Hospital; 60, Welbeck street, Cavendish square.

1820 BERTIN, STEPHEN, Paris.

1872 BEVERLEY, MICHAEL, M.D., Assistant Surgeon to the Norfolk and Norwich Hospital; 63, St. Giles's street, Norwich.

1865 *BICKERSTETH, EDWARD ROBERT, Surgeon to the Liverpool Royal Infirmary, and Lecturer on Clinical Surgery in the Liverpool Royal Infirmary School of Medicine; 2, Rodney street, Liverpool. *Trans.* 1.

1878 BINDON, WILLIAM JOHN VEREKER, M.D., 48, St. Ann's street, Manchester.

1854 BIRD, PETER HINCKES, F.L.S.

1856 †BIRD, WILLIAM, Consulting Surgeon to the West London Hospital; Bute House, Hammersmith.

1849 †BIRKETT, EDMUND LLOYD, M.D., Consulting Physician to the City of London Hospital for Diseases of the Chest; 48, Russell square. C. 1865-6. *Referee*, 1851-9.

1851 †BIRKETT, JOHN, F.L.S., Consulting Surgeon to Guy's Hospital; Corresponding Member of the "Société de Chirurgie" of Paris; Inspector of Anatomy for the Provinces in England and Wales; 62, Green street, Grosvenor square. L. 1856-7. S. 1863-5. C. 1867-3. T. 1870-78. V.P. 1879-80. *Referee*, 1851-5, 1866, 1869, *Sci. Com.* 1863. *Lib. Com.* 1852. *Trans.* 8.

1866 BISHOP, EDWARD, M.D.

1881 BISS, CECIL YATES, M.D., Assistant Physician to the Hospital for Consumption, Brompton, and to the Middlesex Hospital; 135, Harley street, Cavendish square. *Trans.* 1.

Elected

1865 Blanchet, Hilarion, Examiner to the College of Physicians and Surgeons, Lower Canada; 6, Palace street, Quebec, Canada east.

1865 Blandford, George Fielding, M.D., Lecturer on Psychological Medicine at St. George's Hospital; 71, Grosvenor street. C. 1883-4.

1846 †Bostock, John Ashton, C.B., Hon. Surgeon to H.M. the Queen; Surgeon-Major, Scots Fusilier Guards; 73, Onslow gardens, Brompton. C.1861-2. V.P. 1870-71. *Sci. Com.* 1867.

1869 Bourne, Walter, M.D. [care of the National Bank of India, 80, King William street, City]; Archaco, France.

1882 Bowlby, Anthony Alfred, Surgical Registrar to St. Bartholomew's Hospital; 75, Warrington crescent, Maida hill. *Trans.* 2.

1870 *Bowles, Robert Leamon, M.D., 8, West terrace, Folkestone.

1841 †Bowman, Sir William, Bart., LL.D., F.R.S., F.L.S., Consulting Surgeon to the Royal London Ophthalmic Hospital, Moorfields; 5, Clifford street, Bond street. C. 1852-3. V.P. 1862. *Referee*, 1845-50, 1854-6. *Lib. Com.* 1847. *Trans.* 3.

1886 Boxall, Robert, M.D., Physician to the General Lying-in Hospital; 6, Nottingham terrace, York Gate, Regent's Park.

1884 Boyd, Stanley, M.B., Assistant Surgeon to, and Demonstrator of Anatomy at, Charing Cross Hospital; 27, Gower street.

1862 Brace, William Henry, M.D., 7, Queen's Gate terrace, Kensington.

1874 Bradshaw, A. F., Surgeon-Major; Surgeon to the Rt. Hon. the Commander in Chief in India; Army Head Quarters, Bengal Presidency. [Agent: Vesey W. Holt, 17, Whitehall place.]

1883 Bradshaw, James Dixon, M.B., 30, George Street, Hanover square.

Elected

1867 *Brett, Alfred T., M.D., Watford, Herts.

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

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

1868 Broadbent, William Henry, M.D., Physician to, and Lecturer on Medicine at, St. Mary's Hospital; Consulting Physician to the London Fever Hospital; Examiner in Medicine at the University of London; 34, Seymour street, Portman square. C. 1885. *Referee*, 1881-4. *Trans.* 5.

1851 †Brodhurst, Bernard Edward, F.L.S., Surgeon to the Royal Orthopædic Hospital; 20, Grosvenor street. C. 1868-9. *Lib. Com.* 1862-3. *Trans.* 2. *Pro.* 1.

1872 Brodie, George Bernard, M.D., Consulting Physician-Accoucheur to Queen Charlotte's Hospital; 3, Chesterfield street, Mayfair. *Trans.* 1.

1860 Brown-Séquard, Charles Edouard, M.D., LL.D., F.R.S., Laureate of the Academy of Sciences of Paris; Professor of Medicine at the College of France; Professor of General Physiology at the Museum of Natural History; Paris. *Sci. Com.* 1862.

1878 Browne, Sir James Crichton, M.D., LL.D., F.R.S., Lord Chancellor's Visitor in Lunacy; 7, Cumberland terrace, Regent's Park.

1880 Browne, James William, M.B., 8, Norland place, Holland Park.

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

1881 Browne, Oswald Auchinleck, M.A., M.B., Casualty Physician to St. Bartholomew's Hospital and Physician to the Royal Hospital for Diseases of the Chest; 30A, George street, Hanover square.

Elected

1874 Bruce, John Mitchell, M.D., Physician to, and Lecturer on Materia Medica at, the Charing Cross Hospital; Assistant Physician to the Hospital for Consumption, Brompton; 70, Harley street. *Referee*, 1886-7. *Trans.* 1.

1871 Brunton, Thomas Lauder, M.D., F.R.S., Assistant Physician to, and Lecturer on Materia Medica and Therapeutics at, St. Bartholomew's Hospital; Examiner in Materia Medica in the University of London; 10, Stratford place, Oxford street, *Referee*, 1880-87. *Lib. Com.* 1882-7.

1860 Bryant, Thomas, Surgeon to, and Lecturer on Surgery at, Guy's Hospital; 65, Grosvenor street, Grosvenor square. C. 1873-4. V. P. 1885-6. *Sci. Com.* 1863. *Referee*, 1882-4. *Lib. Com.* 1868-71. *Trans.* 10. *Pro.* 1.

1855 †Bryant, Walter John, Consulting Physician to the Home for Incurable Children, Maida vale; 23A, Sussex square, Hyde Park gardens.

1864 Buchanan, George, M.D., F.R.S., Medical Officer of the Local Government Board; Member of the Senate of the University of London; 24, Nottingham place, Marylebone road.

1864 Buckle, Fleetwood, M.D.

1881 Buller, Audley Cecil, M.D., Oxford and Cambridge Club, Pall Mall.

1833 †Burrows, Sir George, Bart., M.D., D.C.L., LL.D., F.R.S., Physician in Ordinary to H.M. the Queen; Consulting Physician to St. Bartholomew's Hospital; Member of the Senate of the University of London; 18, Cavendish square. C. 1839-40, 1858-9. T. 1845-7. V. P. 1849-50. P. 1869-70. *Referee*, 1842-6, 1850-7, 1861-68, 1875-81. *Lib. Com.* 1836. *Trans.* 2.

1885 Butler-Smythe, Albert Charles, Senior Surgeon to the Grosvenor Hospital for Women and Children; 35, Brook street, Grosvenor square.

Elected

1873 BUTLIN, HENRY TRENTHAM, Assistant Surgeon to, and Demonstrator of Practical Surgery and of Diseases of the Larynx at, St. Bartholomew's Hospital; 82, Harley street, Cavendish square. C. 1887. *Trans.* 3.

1871 BUTT, WILLIAM F., 48, Park street, Park lane.

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

1868 BUZZARD, THOMAS, M.D., Physician to the National Hospital for the Paralysed and Epileptic; 56, Grosvenor street, Grosvenor square. C. 1885-6. *Referee.* 1887.

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

1885 CAHILL, JOHN, 12, Seville street, Lowndes square.

1875 CARTER, CHARLES HENRY, M.D., Physician to the Hospital for Women, Soho square; 45, Great Cumberland place, Hyde Park.

1853 CARTER, ROBERT BRUDENELL, Ophthalmic Surgeon to, and Lecturer on Ophthalmic Surgery at, St. George's Hospital; 27, Queen Anne street, Cavendish square. *Trans.* 1.

1845 †CARTWRIGHT, SAMUEL, late Professor of Dental Surgery at King's College, London, and Surgeon-Dentist to King's College Hospital; Consulting Surgeon to the Dental Hospital; 32, Old Burlington street. C. 1860-1. *Sci. Com.* 1863.

1879 CARTWRIGHT, S. HAMILTON, Professor of Dental Surgery at King's College, London, and Surgeon Dentist to King's College Hospital; 32, Old Burlington street.

1868 CAVAFY, JOHN, M.D., Physician to St. George's Hospital; 2, Upper Berkeley street, Portman square. C. 1887. *Trans.* 1.

Elected

1871 CAYLEY, WILLIAM, M.D., Physician to, and Lecturer on the Principles and Practice of Medicine at, the Middlesex Hospital; Physician to the London Fever Hospital and to the North-Eastern Hospital for Children; 27, Wimpole street, Cavendish square. *Referee*, 1886-7. *Lib. Com.* 1886-7. *Trans.* 2.

1884 CHAFFEY, WAYLAND CHARLES, M.B., Assistant Physician to the Royal Alexandra Hospital for Sick Children, Brighton.

1845 †CHALK, WILLIAM OLIVER, 3, Nottingham terrace, York Gate, Regent's Park. C. 1872-3.

1844 †CHAMBERS, THOMAS KING, M.D., Hon. Physician to H.R.H. the Prince of Wales; Consulting Physician to St. Mary's Hospital and to the Lock Hospital; Shrubs Hill House, Sunningdale. C. 1861. V.P. 1867. L. 1869-72. *Referee*, 1851-60, 1866. *Lib. Com.* 1852, 1868. *Trans.* 1.

1879 CHAMPNEYS, FRANCIS HENRY, M.A., M.B., Obstetric Physician to, and Lecturer on Midwifery at, St. George's Hospital; Examiner in Obstetric Medicine in the University of London; 60, Great Cumberland place. *Lib. Com.* 1885-7. *Trans.* 7.

1859 CHANCE, FRANK, M.D., Burleigh House, Sydenham Hill.

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

1877 CHARLES, T. CRANSTOUN, M.D., Lecturer on Practical Physiology at St. Thomas's Hospital; 9, Albert Mansions, Victoria street, Westminster.

1881 *CHAVASSE, THOMAS FREDERICK, M.D., C.M., Surgeon to the Birmingham General Hospital; 24, Temple row, Birmingham. *Trans.* 2.

1868 CHEADLE, WALTER BUTLER, M.D., *Secretary*, Physician to, and Lecturer on Medicine at, St. Mary's Hospital; Senior Physician to the Hospital for Sick Children; 19, Portman street, Portman square. S. 1886-7. *Referee*, 1885.

Elected

1879 Cheyne, William Watson, M.B., Assistant Surgeon to King's College Hospital, and Demonstrator of Surgery in King's College, London; 59, Welbeck street, Cavendish square. *Lib. Com.* 1886-7.

1873 *Chisholm, Edwin, M.D., Abergeldie, Ashfield, near Sydney New South Wales.

1865 Cholmeley, William, M.D., Physician to the Great Northern Hospital; 63, Grosvenor street, Grosvenor square. C. 1881-2. *Referee*, 1873-80.

1872 Christie, Thomas Beith, M.D., Medical Superintendent, Royal India Asylum, Ealing.

1866 Church, William Selby, M.D., Physician to, and Lecturer on Clinical Medicine at, St. Bartholomew's Hospital; 130, Harley street, Cavendish square. C. 1885-6. *Referee*, 1874-81.

1860 Clark, Sir Andrew, Bart., M.D., LL.D., F.R.S., Physician to, and Emeritus Professor of Clinical Medicine at, the London Hospital; 16, Cavendish square. C. 1875.

1879 Clark, Andrew, Assistant Surgeon to, and Lecturer on Practical Surgery at, the Middlesex Hospital; 19, Cavendish place, Cavendish square, W.

1839 †Clark, Frederick Le Gros, F.R.S., Consulting Surgeon to St. Thomas's Hospital; The Thorns, Sevenoaks. S. 1847-9. V.P. 1855-6. *Referee*, 1859-81. *Lib. Com.* 1847. *Trans.* 5.

1882 Clarke, Ernest, M.D., B.S., Surgeon to the Miller Hospital, and Senior Assistant Surgeon to the Central London Ophthalmic Hospital; 21, Lee terrace, Blackheath.

1848 †Clarke, John, M.D., 42, Hertford street, May Fair. C. 1866.

1881 Clarke, W. Bruce, M.B., Assistant Surgeon to, and Demonstrator of Anatomy at, St. Bartholomew's Hospital; 46, Harley street, Cavendish square.

1842 †Clayton, Sir Oscar Moore Passey, C.B., Extra Surgeon-in-Ordinary to H.R.H. the Prince of Wales, and Surgeon-in-Ordinary to H.R.H. the Duke of Edinburgh; 5, Harley street, Cavendish square. C. 1865.

Elected

1879 †Clutton, Henry Hugh, M.A., M.B., Assistant Surgeon to, and Lecturer on Forensic Medicine at, St. Thomas's Hospital; 2, Portland place.

1857 Coates, Charles, M.D., Consulting Physician to the Bath General and Royal United Hospitals; 10, Circus, Bath.

1868 Cockle, John, M.D., F.L.S., Physician to the Royal Free Hospital; 8, Suffolk street, Pall Mall. *Trans.* 2.

1885 Collins, William Maunsell, M.D., 10, Cadogan place.

1865 Cooper, Alfred, Consulting Surgeon to the West London Hospital; Surgeon to the Lock Hospital and to St. Mark's Hospital; 9, Henrietta street, Cavendish square.

1868 Cornish, William Robert, Surgeon-Major, Madras Army; Sanitary Commissioner for Madras; Secretary to the Inspector-General, Indian Medical Department.

1860 *Corry, Thomas Charles Steuart, M.D., Ormean Terrace, Belfast.

1864 Coulson, Walter John, Surgeon to the Lock Hospital, 17, Harley street, Cavendish square.

1860 †Couper, John, Surgeon to the London Hospital; Assistant Surgeon to the Royal London Ophthalmic Hospital; 80, Grosvenor street. C. 1876. *Referee* 1882-3.

1877 Coupland, Sidney, M.D., Physician to, and Lecturer on Practical Medicine at, the Middlesex Hospital; 14, Weymouth street, Portland place.

1862 Cowell, George, Surgeon to, and Lecturer on Surgery at, the Westminster Hospital; Surgeon to the Royal Westminster Ophthalmic Hospital; Surgeon to the Victoria Hospital for Children; 3, Cavendish place, Cavendish square. C. 1882-3.

1841 Crawford, Mervyn Archdall Nott, M.D., Millwood, Wilbury road, Brighton. C. 1853-4.

1868 Crawford, Sir Thomas, K.C.B., M.D., Hon. Surgeon to H.M. the Queen; Director General, Army Medical Department; 2, Victoria street, Westminster, and 5, St. John's Park, Blackheath. C. 1887.

Elected

1873 CREIGHTON, CHARLES, M.D., 11, New Cavendish street. *Referee*, 1882-7. *Trans.* 1.

1869 *CRESSWELL, PEARSON R., Dowlais, Merthyr Tydvil.

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

1882 CROCKER, HENRY RADCLIFFE, M.D., Physician to the Skin Department, University College Hospital; Physician to the East London Hospital for Children; 28, Welbeck street, Cavendish square. *Trans.* 2.

1868 CROFT, JOHN, Surgeon to, and Lecturer on Clinical Surgery at, St. Thomas's Hospital; 48, Brook street, Grosvenor square. C. 1884. *Referee*, 1885-87. *Lib. Com.* 1877-8. *Trans.* 1.

1862 CROMPTON, SAMUEL, M.D., Brookmead, Cranleigh, Surrey.

1837 CROOKES, JOHN FARRAR, 45, Augusta gardens, Folkestone.

1872 CROSSE, THOMAS WILLIAM, Surgeon to the Norfolk and Norwich Hospital; 22, St. Giles's street, Norwich.

1879 CUMBERBATCH, A. ELKIN, Aural Surgeon to St. Bartholomew's Hospital; Aural Surgeon to the Great Northern Hospital; 17, Queen Anne street.

1837 †CURLING, THOMAS BLIZARD, F.R.S., Consulting-Surgeon to the London Hospital; 27, Brunswick square, Brighton. S. 1845-6. C. 1850. T. 1854-7. V.P. 1859. P. 1871-2. *Referee*, 1844-6, 1851-3, 1858, 1865-70, 1875-9. *Sci. Com.* 1863. *Lib. Com.* 1839. *Trans.* 13. *Pro.* 1.

1873 CURNOW, JOHN, M.D., Professor of Anatomy in King's College, London, and Physician to King's College Hospital; 3, George street, Hanover square. *Referee*, 1884-7.

1847 CURREY, JOHN EDMUND, M.D., Lismore, County Waterford.

1886 DAKIN, WILLIAM RADFORD, M.D., 57, Welbeck street, Cavendish square.

1872 DALBY, SIR WILLIAM BARTLETT, M.B., Aural Surgeon to, and Lecturer on Aural Surgery at, St. George's Hospital; 18, Savile row. *Trans.* 3.

Elected

1884 DALLAWAY, DENNIS.

1877 DARBISHIRE, SAMUEL DUKINFIELD, M.D., Physician to the Radcliffe Infirmary, Oxford; Beaumont street, Oxford.

1879 DARWIN, FRANCIS, M.B., F.R.S., The Grove, Huntingdon road, Cambridge.

1874 DAVIDSON, ALEXANDER, M.D., Physician to the Liverpool Northern Hospital; 2, Gambier terrace, Liverpool.

1853 DAVIES, ROBERT COKER NASH, Rye, Sussex.

1852 DAVIES, WILLIAM, M.D., 2, Marlborough buildings, Bath.

1876 DAVIES-COLLEY, J. NEVILLE C., M.C., Surgeon to, and Lecturer on Anatomy at, Guy's Hospital; 36, Harley street, Cavendish square. *Trans.* 2.

1878 DAVY, RICHARD, F.R.S. Ed., Surgeon to, and Lecturer on Surgery at, the Westminster Hospital; 33, Welbeck street, Cavendish square. *Trans.* 1.

1882 *DAWSON, YELVERTON, M.D., Heathlands, Southbourne-on-Sea, Hants.

1867 DAY, WILLIAM HENRY, M.D., Physician to the Samaritan Free Hospital for Women and Children; 10, Manchester square.

1878 DENT, CLINTON THOMAS, Assistant Surgeon to, and Lecturer on Practical Surgery at, St. George's Hospital; 61, Brook street. *Trans.* 2.

1859 †DICKINSON, WILLIAM HOWSHIP, M.D., *Vice-President*, Physician to, and Lecturer on Medicine at, St. George's Hospital, and Consulting Physician to the Hospital for Sick Children; Honorary Fellow of Caius College, Cambridge; 9, Chesterfield street, Mayfair. C. 1874-5. V.P. 1887. *Referee*, 1869-73, 1882-6. *Sci. Com.* 1867-79. *Trans.* 13.

1839 †DIXON, JAMES, Consulting Surgeon to the Royal London Ophthalmic Hospital, Moorfields; Harrow Lands, Dorking. L. 1849-55. V.P. 1857-8. T. 1863-4. C. 1866-7. *Referee*, 1865. *Lib. Com.* 1845-8. *Trans.* 4.

Elected

1862 Dobell, Horace B., M.D., Consulting Physician to the Royal Hospital for Diseases of the Chest, City road; Streate place, Bournemouth. *Trans.* 2.

1845 Dodd, John.

1879 Donkin, Horatio, M.B., Physician to the Westminster Hospital; Physician to the East London Hospital for Children; 60, Upper Berkeley street, Portman square.

1877 Doran, Alban Henry Griffiths, Assistant Surgeon to the Samaritan Free Hospital; 9, Granville place, Portman square. *Trans.* 1.

1863 Down, John Langdon Haydon, M.D., Physician to, and Lecturer on Clinical Medicine at, the London Hospital; 81, Harley street, Cavendish square. C. 1880. *Trans.* 2.

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

1884 Drage, Lovell, M.B., B.S., The Small House, Hatfield, Herts.

1879 Drewitt, F. G. Dawtrey, M.D., Physician to the West London Hospital and to the Victoria Hospital for Children; 52, Brook street, Grosvenor square.

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

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

1865 Drysdale, Charles Robert, M.D., Physician to the Farringdon Dispensary; Assistant-Physician to the Metropolitan Free Hospital; 23, Sackville street, Piccadilly.

1865 †Duckworth, Sir Dyce, M.D., Physician to, and Lecturer on Clinical Medicine at, St. Bartholomew's Hospital; 11, Grafton street, Bond street. C. 1883-4. *Referee* 1885-7. *Trans.* 1.

1876 Dudley, William Lewis, M.D., Physician to the City Dispensary; 149, Cromwell road, South Kensington.

1845 Duff, George, M.D., High street, Elgin.

1874 Duffin, Alfred Baynard, M.D., Professor of Pathological Anatomy in King's College, London, and Physician to King's College Hospital; 18, Devonshire street, Portland place.

Elected

1871 Duke, Benjamin, Windmill House, Clapham Common.

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, M. Charles, M.D., Wellesley Villa, Wellesley road, Croydon.

1880 Dunbar, James John Macwhirter, M.D., Hedingham House, Clapham Common.

1877 Duncan, James Matthews, M.D., LL.D., F.R.S., Obstetric Physician to, and Lecturer on Midwifery and Diseases of Women at, St. Bartholomew's Hospital; 71, Brook street, Grosvenor square. *Referee*, 1881-7. *Trans.* 1.

1884 Duncan, William, M.D., Assistant Obstetric Physician and Teacher of Operative Midwifery, Middlesex Hospital; 6, Harley street, Cavendish square.

1887 Dunn, Hugh Percy, 29, Upper Montagu street, Montagu square.

1863 Durham, Arthur Edward, F.L.S., *Vice-President*, Surgeon to, and Lecturer on Surgery at, Guy's Hospital; 82, Brook street, Grosvenor square. C. 1876-7. V.P. 1887. *Referee*, 1880-1. *Sci. Com.* 1867. *Lib. Com.* 1872-5. *Trans.* 5.

1874 Durham, Frederic, M.B., 82, Brook street, Grosvenor square.

1843 Durrant, Christopher Mercer, M.D., Consulting Physician to the East Suffolk and Ipswich Hospital; Northgate street, Ipswich, Suffolk.

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.

1868 Eastes, George, M.B.Lond., 69, Connaught street, Hyde Park square.

1883 Edmunds, Walter, M.C., 79, Lambeth Palace road, Albert Embankment. *Trans.* 2.

Elected

1883 Edwardes, Edward Joshua, M.D., 16, Acacia road, St. John's Wood.

1884 Edwards, Frederick Swinford, Surgeon to the West London Hospital, and to St. Peter's Hospital for Stone; 93, Wimpole street, Cavendish square.

1824 Edwards, George.

1869 Elam, Charles, M.D., 75, Harley street, Cavendish square. *Lib. Com.* 1886-7.

1887 Elliott, John, Resident Surgical Officer, The General Hospital, Birmingham.

1848 Ellis, George Viner, late Professor of Anatomy in University College, London; Minsterworth, Gloucester. C. 1863-4. *Trans.* 2.

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

1854 *Ellison, James, M.D., Surgeon-in-Ordinary to the Royal Household, Windsor; 14, High street, Windsor.

1842 †Erichsen, John Eric, LL.D.,F.R.S., Surgeon Extraordinary to H.M. the Queen; Emeritus Professor of Surgery in University College, London, and Consulting Surgeon to University College Hospital; 6, Cavendish place, Cavendish sq. C. 1855-6. V.P. 1868. P. 1879-80. *Referee*, 1866-7, 1884-7. *Lib. Com.* 1844-7, 1854. *Trans.* 2.

1879 Eve, Frederic S., Pathological Curator of the Museum, Royal College of Surgeons; Assistant Surgeon to the London Hospital; 15, Finsbury circus. *Trans.* 2.

1877 Ewart, William, M.D., Assistant Physician to, and Lecturer on Physiology at, St. George's Hospital; 33, Curzon street, Mayfair. *Trans.* 1.

1875 *Fagan, John, Surgeon to, and Lecturer on Clinical Surgery at, the Belfast Royal Hospital; 1, Glengall place, Belfast.

1869 Fairbank, Frederick Royston, M.D., 46, Hallgate, Doncaster.

Elected

1862 FARQUHARSON, ROBERT, M.D., LL.D., M.P., Migvie Lodge, Porchester gardens, Hyde park; Finzean, Aboyne, Aberdeenshire, and the Reform Club, Pall Mall. *Lib. Com.* 1876-80.

1844 †FARRE, ARTHUR, M.D., F.R.S., Physician Extraordinary to H.M. the Queen; Physician-Accoucheur to H.R.H. the Princess of Wales; 18, Albert Mansions, Victoria street, Westminster. C. 1857. V.P. 1864. *Referee*, 1848-54, 1861-3, 1865-6. *Sci. Com.* 1863. *Lib. Com.* 1847.

1872 FAYRER, SIR JOSEPH, K.C.S.I., M.D., F.R.S., Honorary Physician to H.M. the Queen, and to H.R.H. the Prince of Wales, and Physician to H.R.H. the Duke of Edinburgh; late Surgeon-General Bengal Medical Service; Examining Medical Officer to the Secretary of State for India in Council; President of the Indian Medical Board; 53, Wimpole street, Cavendish square. *Referee*, 1881-7.

1872 *FENWICK, JOHN C. J., M.D., Physician to the Durham County Hospital; 25, North road, Durham.

1863 FENWICK, SAMUEL, M.D., Physician to the London Hospital; 29, Harley street, Cavendish square. C. 1880. *Referee*, 1882-7. *Trans.* 4.

1880 FERRIER, DAVID, M.D., LL.D., F.R.S., Professor of Forensic Medicine in King's College, London, and Physician to King's College Hospital; Physician for Out-patients to the National Hospital for the Paralysed and Epileptic; 34, Cavendish square. *Trans.* 2.

1852 *FIELD, ALFRED GEORGE.

1849 †FINCHAM, GEORGE TUPMAN, M.D., Consulting Physician to the Westminster Hospital; 13, Belgrave road, Pimlico. C. 1871.

1879 FINLAY, DAVID WHITE, M.D., Physician to, and Lecturer on Forensic Medicine at, the Middlesex Hospital; Physician to the Royal Hospital for Diseases of the Chest; 9, Lower Berkeley street, Portman square. *Trans.* 2.

Elected

1866 Fish, John Crockett, B.A., M.D., 92, Wimpole street, Cavendish square.

1866 Fitz-Patrick, Thomas, M.D., M.A., 30, Sussex gardens, Hyde Park.

1842 Fletcher, Thomas Bell Elcock, M.D., Consulting Physician to the Birmingham General Hospital; 8, Clarendon crescent, Leamington. *Trans.* 1.

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

1877 de Fonmartin, Henry, M.D., Parkhurst, Isle of Wight.

1848 †Forbes, John Gregory, Egerton House, Egerton, Ashford, Kent. C. 1868-9. *Lib. Com.* 1855. *Trans.* 2.

1865 Foster, Sir Balthazar Walter, M.D., M.P., Professor of Medicine at the Queen's College, Birmingham, and Physician to the Birmingham General Hospital; 14, Temple row, Birmingham.

1883 Fowler, James Kingston, M.A., M.D., Assistant Physician to, and Lecturer on Pathological Anatomy at, the Middlesex Hospital, and Assistant Physician to the Hospital for Consumption, Brompton; 35, Clarges street, Piccadilly.

1859 Fox, Edward Long, M.D., Consulting Physician to the Bristol Royal Infirmary; Church House, Clifton, Gloucestershire.

1887 Fox, Richard Hingston, M.D., 43, Finsbury circus.

1880 Fox, Thomas Colcott, B.A., M.B., Physician to the Skin Department of the Paddington Green Hospital for Children, and Assistant Physician to the Victoria Hospital for Children; 14, Harley street, Cavendish square. *Trans.* 1.

1871 Frank, Philip, M.D., Cannes, France.

1884 *Franks, Kendal, M.D., Surgeon to the Adelaide Hospital and to the Throat and Ear Hospital, Dublin; 69, Fitzwilliam square, Dublin.

1843 Fraser, Patrick, M.D., C. 1866.

Elected

1868 FREEMAN, WILLIAM HENRY, 21, St. George's square, South Belgravia.

1836 †FRENCH, JOHN GEORGE, 10, Cunningham place, St. John's Wood road. C. 1852-3.

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

1883 FULLER, HENRY ROXBURGH, M.D., 45, Curzon street, May Fair.

1876 FURNER, WILLOUGHBY, Assistant Surgeon to the Sussex County Hospital; 2, Brunswick place, Brighton.

1864 *GAIRDNER, WILLIAM TENNANT, M.D., LL.D., Physician in Ordinary to H.M. the Queen in Scotland; Professor of the Practice of Medicine in the University of Glasgow; Physician to the Western Infirmary, Glasgow; 225, St. Vincent street, Glasgow.

1874 GALABIN, ALFRED LEWIS, M.A., M.D., Obstetric Physician to, and Lecturer on Midwifery and the Diseases of Women at, Guy's Hospital; Assistant Physician to the Hospital for Sick Children; 49, Wimpole street, Cavendish square. *Referee*, 1882-7. *Lib. Com.* 1883-4. *Trans.* 2.

1883 GALTON, JOHN CHARLES, M.A., F.L.S., 45, Great Marlborough street.

1885 GAMGEE, ARTHUR, M.D., F.R.S., Fullerian Professor of Physiology in the Royal Institution of Great Britain; 11, Warrior square, St. Leonard's-on-Sea.

1865 GANT, FREDERICK JAMES, Senior Surgeon to the Royal Free Hospital; 16, Connaught square, Hyde Park. C. 1880-81. *Referee*, 1886-7. *Lib. Com.* 1882-5. *Trans.* 3.

1867 GARLAND, EDWARD CHARLES, Yeovil, Somerset.

1867 GARLIKE, THOMAS W., Malvern Cottage, Churchfield road, Ealing.

1854 †GARROD, SIR ALFRED BARING, M.D., F.R.S., Consulting Physician to King's College Hospital; 10, Harley street, Cavendish square. C. 1867. V.P. 1880-81. *Referee*, 1855-65. *Trans.* 8.

Elected

1886 Garrod, Archibald Edward, M.A., M.D., Casualty Physician to St. Bartholomew's Hospital, and Physician to the Marylebone Dispensary; 9, Chandos street, Cavendish square.

1879 Garstang, Thomas Walter Harropp, The Heath, Knutsford, Cheshire.

1819 Gaulter, Henry.

1866 Gee, Samuel Jones, M.D., *Librarian*, Physician to, and Lecturer on Medicine at, St. Bartholomew's Hospital; Consulting Physician to the Hospital for Sick Children; 31, Upper Brook street, Grosvenor square. C. 1883-4. L. 1887. *Sci. Com.* 1879. *Referee*, 1885-7. *Lib. Com.* 1871-6. *Trans.* 1.

1885 Gell, Henry Willingham, M.B., 43, Albion street, Hyde Park.

1878 Gervis, Henry, M.D., Obstetric Physician to, and Lecturer on Obstetric Medicine at, St. Thomas's Hospital; 40, Harley street, Cavendish square. *Referee*, 1884-7. *Trans.* 1.

1884 Gibbes, Heneage, M.D., Physician to the Metropolitan Dispensary; Lecturer on Morbid Histology, Westminster Hospital; Park Cottage, Hampton Wick.

1880 Gibbons, Robert Alexander, M.D., Physician to the Grosvenor Hospital for Women and Children; 29, Cadogan place.

1877 Godlee, Rickman John, Surgeon to University College Hospital, and Teacher of Operative Surgery in University College, London; Surgeon to the North-Eastern Hospital for Children, and to the Hospital for Consumption, Brompton; 81, Wimpole street, Cavendish square. *Referee*, 1886-7. *Trans.* 3.

1870 Godson, Clement, M.D., Assistant-Physician-Accoucheur to St. Bartholomew's Hospital; Consulting Physician to the City of London Lying-in Hospital; 9, Grosvenor street, Grosvenor square.

1886 Golding-Bird, Cuthbert Hilton, M.B., Assistant Surgeon and Lecturer on Physiology at Guy's Hospital; 13, St. Thomas street, Southwark.

Elected

1851 GOODFELLOW, STEPHEN JENNINGS, M.D., Consulting Physician to the Middlesex Hospital; Swinnerton Lodge, near Dartmouth, Devon. C. 1864-5. *Referee*, 1860-3. *Lib. Com.* 1863. *Trans.* 2.

1883 GOODHART, JAMES FREDERIC, M.D., Physician to, and Lecturer on Pathology at, Guy's Hospital; Physician to the Evelina Hospital for Sick Children; 25, Weymouth street, Portland place.

1877 GOULD, ALFRED PEARCE, M.S., Assistant Surgeon to the Middlesex Hospital; 16, Queen Anne street, Cavendish square. *Trans.* 2.

1873 GOWERS, WILLIAM RICHARD, M.D., F.R.S., Professor of Clinical Medicine in University College, London, and Physician to University College Hospital; Physician for Out-patients to the National Hospital for the Paralysed and Epileptic; 50, Queen Anne street, Cavendish square. *Lib. Com.* 1884-6. *Trans.* 6.

1851 †GOWLLAND, PETER YEAMES, Surgeon to St. Mark's Hospital; Surgeon-Major Hon. Artillery Company; 34, Finsbury square.

1846 GREAM, GEORGE THOMPSON, M.D., Physician-Accoucheur to H.R.H. the Princess of Wales; Mixbury, Eastbourne, Sussex. C. 1863.

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

1875 *GREENFIELD, WILLIAM SMITH, M.D., Professor of General Pathology in the University of Edinburgh; 7, Heriot row, Edinburgh. *Sci. Com.* 1879. *Referee*, 1881.

1843 †GREENHALGH, ROBERT, M.D., Consulting Physician to the Samaritan Free Hospital for Women and Children, and to the City of London Lying-in Hospital; 35, Cavendish square. C. 1871-2. *Referee*, 1876-7. *Trans.* 1.

Elected

1860 GREENHOW, EDWARD HEADLAM, M.D., F.R.S., Consulting Physician to the Middlesex Hospital; and Consulting Physician to the Western General Dispensary; Castle Lodge, Reigate. C. 1876-7. *Referee*, 1870-5. *Trans.* 3.

1882 GRESSWELL, DAN ASTLEY, M.B., 87, Queen's crescent, Haverstock hill.

1885 GRIFFITH, WALTER SPENCER ANDERSON, M.B., Physician to the Samaritan Free Hospital for Women and Children; 114, Harley street, Cavendish square.

1884 GRIFFITHS, HERBERT TYRRELL, M.D., 57, Brook street.

1868 GRIGG, WILLIAM CHAPMAN, M.D., Assistant Obstetric Physician to the Westminster Hospital; Physician to the In-Patients, Queen Charlotte's Lying-in-Hospital; 27, Curzon street, Mayfair.

1852 GROVE, JOHN, Fyning, Austen road, Guildford.

1860 GUENEAU DE MUSSY, HENRI, M.D.; 15, Rue du Cirque, Paris. *Lib. Com.* 1863-5.

1849 †GULL, SIR WILLIAM WITHEY, Bart., M.D., D.C.L., LL.D., F.R.S., Physician-Extraordinary to H.M. the Queen; and Physician in Ordinary to H.R.H. the Prince of Wales; Member of the Senate of the University of London; Consulting Physician to Guy's Hospital; 74, Brook street, Grosvenor square. C. 1864. V.P. 1874. *Referee*, 1855-63. *Trans.* 4.

1885 GULLIVER, GEORGE, M.B., Assistant Physician to, and Lecturer on Comparative Anatomy at, St. Thomas's Hospital; 16, Welbeck street.

1883 GUNN, ROBERT MARCUS, M.B., Assistant Surgeon to the Royal London Ophthalmic Hospital, Moorfields; 54, Queen Anne street, Cavendish square.

1886 HABERSHON, SAMUEL HERBERT, M.D., 2, Upper Wimpole street, Cavendish square.

1854 †HABERSHON, SAMUEL OSBORNE, M.D., 70, Brook street, Grosvenor square. S. 1867. C. 1869-70. V.P. 1881-2. *Referee*, 1862-6, 1868, 1871-80. *Trans.* 3.

Elected

1885 HAIG, ALEXANDER, M.B., Casualty Physician to St. Bartholomew's Hospital; 30, Welbeck street, Cavendish square. *Trans.* 1.

1881 HALL, FRANCIS DE HAVILLAND, M.D., Assistant Physician, and Physician to the Throat Department, and Lecturer on Forensic Medicine at the Westminster Hospital; Physician to St. Mark's Hospital; 47, Wimpole street, Cavendish square.

1885 HALLIBURTON, WILLIAM DOBINSON, M.D., Assistant Professor of Physiology, University College, London; 25, Maitland Park Villas, Haverstock Hill.

1870 HAMILTON, ROBERT, Surgeon to the Royal Southern Hospital, Liverpool; 1 Prince's road, Liverpool.

1874 HARDIE, GORDON KENMURE, M.D., Deputy Inspector General of Hospitals; Florence road, Ealing, and Duff House, Banff, N.B.

1856 †HARE, CHARLES JOHN, M.D., *Treasurer*, late Professor of Clinical Medicine in University College, London, and Consulting Physician to University College Hospital; Berkeley House, 15, Manchester square. C. 1873-4. T. 1887.

1857 HARLEY, GEORGE, M.D., F.R.S. 25, Harley street, Cavendish square. C. 1871-2. *Referee*, 1865-70, 1873-6. *Sci. Com.* 1862-3. *Trans.* 1.

1864 HARLEY, JOHN, M.D., F.L.S., Physician to, and Lecturer on General Anatomy and Physiology at, St. Thomas's Hospital; 9, Stratford place, Oxford street. S. 1875-7. C. 1879-80. *Referee*, 1871-4, 1882-7. *Sci. Com.* 1879. *Trans.* 10.

1880 HARRIS, VINCENT DORMER, M.D., Physician to the Victoria Park Hospital; Demonstrator of Physiology at St. Bartholomew's Hospital; 31, Wimpole street, Cavendish square.

1870 HARRISON, REGINALD, Surgeon to the Liverpool Royal Infirmary, and Lecturer on Clinical Surgery in the Victoria University; 41, Rodney street, Liverpool. *Trans.* 1.

Elected

1854 HAVILAND, ALFRED.

1870 HAWARD, J. WARRINGTON, Surgeon to, and Lecturer on Clinical Surgery at, St. George's Hospital; 16, Savile row, Burlington Gardens. C. 1885. *Lib. Com.* 1881-4. *Trans.* 1.

1838 †HAWKINS, CHARLES, Inspector of Anatomical Schools in London; 9, Duke street, Portland place. C. 1846-7. S. 1850. V.P. 1858. T. 1861-2. *Referee*, 1859-60. *Lib. Com.* 1843. *Trans.* 2.

1885 HAWKINS, FRANCIS HENRY, M.B., Physician to St. George's and St. James's Dispensary; 22, Henrietta street, Cavendish square.

1848 †HAWKSLEY, THOMAS, M.D., 11, Albert Mansions, Victoria street, and Beomands, Chertsey, Surrey.

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

1860 HAYWARD, HENRY HOWARD, Surgeon Dentist to, and Lecturer on Dental Surgery at, St. Mary's Hospital; 38, Harley street, Cavendish square. C. 1878-9.

1861 HAYWARD, WILLIAM HENRY.

1848 *HEALE, JAMES NEWTON, M.D.

1865 HEATH, CHRISTOPHER, Holme Professor of Clinical Surgery in University College, London; and Surgeon to University College Hospital; 36, Cavendish square. C. 1880. *Lib. Com.* 1870-3. *Trans.* 3.

1850 HEATON, GEORGE, M.D., Boston, U.S.

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

1877 HERMAN, GEORGE ERNEST, M.B., Obstetric Physician to, and Lecturer on Midwifery at, the London Hospital; 20, Harley street, Cavendish square. *Trans.* 1.

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.

Elected

1883 Herringham, Wilmot Parker, M.B., 22, Bedford square.

1843 †Hewett, Sir Prescott Gardner, Bart., F.R.S., Serjeant-Surgeon to H.M. the Queen; Surgeon in Ordinary to H.R.H. the Prince of Wales; Consulting Surgeon to St. George's Hospital; Corresponding Member of the "Académie de Médecine," and of the "Société de Chirurgie," Paris; Chesnut Lodge, Horsham, Sussex. C. 1859. V.P. 1866-7. *Referee*, 1850-8, 1860-5, 1868-83. *Sci. Com.* 1863. *Lib. Com.* 1846-7. *Trans.* 7.

1887 Hewitt, Frederic William, M.D., 10, George street, Hanover square.

1855 †Hewitt, W. M. Graily, M.D., Emeritus Professor of Midwifery in University College, London, and Consulting Obstetric Physician to University College Hospital; 36, Berkeley square. C. 1876. *Referee*, 1868-75, 1877-87. *Lib. Com.* 1868, 1874.

1880 Hicks, Charles Cyril, M.D., Wokingham, Berks.

1873 Higgens, Charles, Assistant Ophthalmic Surgeon to, and Lecturer on Ophthalmic Surgery at, Guy's Hospital; 38, Brook street, Grosvenor square. *Trans.* 2.

1862 Hill, M. Berkeley, M.B., Professor of Clinical Surgery in University College, London, and Surgeon to University College Hospital; Surgeon to the Lock Hospital; 66, Wimpole street, Cavendish square. C. 1878-9. S. 1881-4. V.P. 1885-6. *Trans.* 1.

1867 Hill, Samuel, M.D., 22, Mecklenburgh square.

1861 *Hoffmeister, Sir William Carter, M.D., Surgeon to H.M. the Queen in the Isle of Wight; Clifton House, Cowes, Isle of Wight.

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

1879 Holland, Philip Alexander, M.A.

Elected

1868 Hollis, William Ainslie, M.A., M.D., Assistant-Physician to the Sussex County Hospital; 8, Cambridge road, Brighton.

1861 Holman, William Henry, M.B., 68, Adelaide road, South Hampstead.

1856 †Holmes, Timothy, M.A., *Treasurer*, Surgeon to St. George's Hospital; Corresponding Member of the "Société de Chirurgie," Paris; 18, Great Cumberland place, Hyde Park. C. 1869-70. L. 1873-7. S. 1878-80. V.P. 1881-2. T. 1885-7. *Referee*, 1866-8, 1872, 1883-4. *Sci. Com.* 1867. *Lib. Com.* 1863-5. *Trans.* 8.

1846 †Holt, Barnard Wight, Consulting Surgeon to the Westminster Hospital; Medical Officer of Health for Westminster; 14, Savile row, Burlington Gardens. C. 1862-3. V.P. 1879-80.

1846 †Holthouse, Carsten, 35, Essex street, Strand. C. 1863. *Referee* 1870-6. *Lib. Com.* 1859-60.

1878 Hood, Donald William Charles, M.D., Senior Physician to the North-West London Hospital; Physician to the West London Hospital; 43, Green street, Park lane.

1883 Horsley, Victor Alexander Haden, F.R.S., Assistant Surgeon to University College Hospital, Surgeon to the National Hospital for the Paralysed and Epileptic; Professor of Pathological Anatomy in University College, London; Superintendent of the Brown Institution, Wandsworth road; 80, Park street, Grosvenor Square.

1878 Houghton, Walter B., M.D., Church Villa, Warrior square, St. Leonards-on-Sea.

1865 Howard, Benjamin, M.D., New York, U.S.

1881 Howard, Henry, M.B., abroad [6, The Terrace, Mount Pleasant, Cambridge].

1874 Howse, Henry Greenway, M.S., Surgeon to, and Lecturer on Anatomy at, Guy's Hospital; Surgeon to the Evelina Hospital for Sick Children; 59, Brook street, Grosvenor square. *Sci. Com.* 1879. *Referee*, 1887. *Trans.* 2.

1886 Hudson, Charles Leopold, Pathologist and Curator of the Museum, Middlesex Hospital; 34, Welbeck street, Cavendish square.

Elected

1884 Huggard, William R., M.D. [Place de la Synagogue, 2, Genève.]

1857 Hulke, John Whitaker, F.R.S., *Librarian*, Surgeon to the Middlesex Hospital; Surgeon to the Royal London Ophthalmic Hospital, Moorfields; 10, Old Burlington street. C. 1871-2. S. 1876-7. L. 1879-87. *Sci. Com.* 1867. *Lib. Com.* 1864-8. *Trans.* 9.

1844 †Humby, Edwin, M.D., 83, Hamilton terrace, St. John's wood. C. 1866-7.

1855 Humphry, George Murray, M.D., F.R.S., Surgeon to Addenbrooke's Hospital; Professor of Surgery in the University of Cambridge. *Trans.* 6.

1882 Humphry, Laurence, M.B., 3, Trinity street, Cambridge.

1873 Hunter, Sir W. Guyer, M.D., M.P., Hon. Surgeon to H.M. the Queen; late Principal of, and Professor of Medicine in, Grant Medical College, Bombay; Surgeon-General Bombay Army; 21, Norfolk crescent, Hyde park.

1849 Hussey, Edward Law, Consulting Surgeon to the County Lunatic Asylum and the Warneford Asylum; 24, Winchester Road, Oxford. *Trans.* 1.

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 Surgeon to the Hospital for Diseases of the Skin; 15, Cavendish square. C. 1870. V.P. 1882. *Referee*, 1876-81, 1883-7. *Lib. Com.* 1864-5. *Trans.* 14. *Pro.* 2.

1820 Hutchinson, William, M.D.

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

1856 Inglis, Cornelius, M.D., Cairo. [Athenæum Club, Pall Mall.]

1871 Jackson, J. Hughlings, M.D., F.R.S., Physician to the London Hospital; Physician to the National Hospital for the Paralysed and Epileptic; 3, Manchester square.

Elected

1841 †Jackson, Paul, 51, Wellington road, St. John's Wood. C. 1862.

1863 Jackson, Thomas Vincent, Senior Surgeon to the Wolverhampton and Staffordshire General Hospital; 47, Waterloo road, south, Wolverhampton.

1883 Jacobson, Walter Hamilton Acland, B.A., M.B., M.S., Assistant Surgeon to Guy's Hospital; Surgeon to the Royal Hospital for Children and Women; 41, Finsbury square. *Trans.* 1.

1825 James, John B., M.D.

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

1851 †Jenner, Sir William, Bart., M.D., K.C.B., D.C.L., LL.D., F.R.S., Physician in Ordinary to H.M. the Queen, and to H.R.H. the Prince of Wales; Emeritus Professor of Clinical Medicine in University College, London; and Consulting Physician to University College Hospital; Member of the Senate of the University of London; 63, Brook street, Grosvenor square. C. 1864. V.P. 1875. *Referee*, 1855, 1859-63. *Trans.* 3.

1884 Jennings, Charles Egerton, M.S., M.B., 15, Upper Brook street, Grosvenor square.

1881 Jennings, William Oscar, M.D., 8, Rue Roy, Paris.

1884 Jessett, Frederic Bowreman, Surgeon to the Royal General Dispensary; 16, Upper Wimpole street.

1883 Jessop, Walter H. H., M.B., Demonstrator of Anatomy at St. Bartholomew's Hospital; 73, Harley street.

1851 Johnson, Edmund Charles, Corresponding Member of the Medical and Philosophical Society of Florence, and of "l'Institut Génevois."

1847 †Johnson, George, M.D., F.R.S., Consulting Physician to King's College Hospital; Member of the Senate of the University of London; 11, Savile row, Burlington gardens. C. 1862-3. V.P. 1870. P. 1884-5. L. 1878-80. *Referee*, 1853-61, 1864-9. *Lib.Com.* 1860-1. *Trans.* 10. *Pro.* 1.

Elected

1881 JOHNSON, GEORGE LINDSAY, M.A., M.D., Cortina, Netherhall terrace, South Hampstead, and 14, Stratford place, Oxford street.

1884 JOHNSTON, JAMES, M.D., 11, Chester place, Hyde Park square, and Oriental Club, Hanover square.

1848 JOHNSTONE, ATHOL ARCHIBALD WOOD, Consulting Surgeon to the Royal Alexandra Hospital for Sick Children, St. Moritz House, 61, Dyke road, Brighton. *Lib. Com.* 1860. *Trans.* 1.

1887 JONES, HENRY LEWIS, M.D., Casualty Physician to St. Bartholomew's Hospital, 5, Barnard's Inn, Holborn.

1876 JONES, LESLIE HUDSON, M.D., Limefield House, Cheetham hill, Manchester.

1875 *JONES, PHILIP SYDNEY, M.D., Consulting Surgeon to the Sydney Infirmary; Examiner in Medicine, Sydney University; 10, College street, Sydney, New South Wales. [Agents: Messrs. D. Jones & Co., 1, Gresham buildings, Basinghall street.]

1859 JONES, WILLIAM PRICE, M.D., Claremont road, Surbiton, Kingston.

1865 JORDAN, FURNEAUX, Consulting Surgeon to the Queen's Hospital, Birmingham; Selly Hill, Birmingham.

1881 JULER, HENRY EDWARD, Assistant Surgeon Royal Westminster Ophthalmic Hospital; Junior Ophthalmic Surgeon to St. Mary's Hospital; 77, Wimpole street, Cavendish square.

1816 *KAUFFMANN, GEORGE HERMANN, M.D., Hanover.

1882 KEETLEY, CHARLES R. B., Senior Surgeon to the West London Hospital; Surgeon to the Surgical Aid Society; 10, George street, Hanover square.

1872 KELLY, CHARLES, M.D., Professor of Hygiene in King's College, London, and Medical Officer of Health for the West Sussex Combined Sanitary District; Broadwater road, Worthing, Sussex.

1848 *KENDELL, DANIEL BURTON, M.D., Heath House, Wakefield, Yorkshire.

Elected

1884 KESER, JEAN SAMUEL, M.D., Surgeon to the French Hospital, Leicester place; 60, Queen Anne street.

1877 *KHORY, RUSTONJEE NASERWANJEE, M.D., Physician to the Parell Dispensary, Bombay; Girgaum road, Bombay.

1857 KIALLMARK, HENRY WALTER, 5, Pembridge gardens, Bayswater.

1881 KIDD, PERCY, M.A., M.D., Assistant Physician to the Hospital for Consumption, Brompton; 60, Brook street, Grosvenor square. *Trans.* 3.

1851 †KINGDON, JOHN ABERNETHY, Surgeon to the City of London Truss Society, and Consulting Surgeon to the City Dispensary; 2, New Bank buildings, Lothbury. C. 1866-7. V.P. 1872-3. *Sci. Com.* 1867. *Trans.* 1.

1885 KLEIN, EDWARD EMANUEL, M.D., F.R.S., Lecturer on Physiology, St. Bartholomew's Hospital; 94, Philbeach gardens, Earl's Court.

1883 KNAPTON, GEORGE, 11, Hoghton street, Southport.

1840 †LANE, SAMUEL ARMSTRONG, Consulting Surgeon to St. Mary's Hospital and to the Lock Hospital; 49, Norfolk square, Hyde park. C. 1849-50. V.P. 1865. *Referee*, 1850.

1884 LANE, WILLIAM ARBUTHNOT, M.S., Assistant Surgeon to the Hospital for Sick Children; 14, St. Thomas's street, Southwark. *Trans.* 1.

1882 LANG, WILLIAM, Ophthalmic Surgeon to, and Lecturer on Ophthalmic Surgery at, the Middlesex Hospital; Assistant Surgeon to the Royal London Ophthalmic Hospital, Moorfields; 26, Upper Wimpole street, Cavendish square.

1865 LANGTON, JOHN, Surgeon to, and Lecturer on Anatomy at, St. Bartholomew's Hospital; Surgeon to the City of London Truss Society; 2, Harley street, Cavendish square. C. 1881-2. *Referee*, 1885-7. *Lib. Com.* 1879-80. *Trans.* 1.

Elected

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

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

1816 Lawrence, G. E.

1884 Lawson, George, Surgeon-Oculist to H.M. the Queen; Surgeon to the Royal London Ophthalmic Hospital and to the Middlesex Hospital; 12, Harley street, Cavendish square.

1880 Laycock, George Lockwood, M.B., Physician to the Paddington Green Children's Hospital; 12, Upper Berkeley street, Portman square.

1886 *Lediard, Henry Ambrose, M.D., Surgeon to the Cumberland Infirmary; 41, Lowther street, Carlisle.

1882 Ledwich, Edward l'Estrange, Lecturer on Surgical and Descriptive Anatomy in the Ledwich School of Medicine, Dublin; 23, Upper Leeson Street, Dublin.

1843 †Lee, Henry, Consulting Surgeon to St. George's Hospital; 9, Savile row, Burlington gardens. C. 1856-7. L. 1863-4. V.P. 1868-9. *Referee*, 1855, 1866-8. *Sci. Com.* 1867. *Trans.* 14. *Pro.* 2.

1884 Lee, Robert James, M.D., 6, Savile row.

1883 Leeson, John Rudd, M.D., C.M., 6, Clifden road, Twickenham.

1869 Legg, John Wickham, M.D., 47, Green street, Park lane. C. 1886. *Referee*, 1882-5. *Lib. Com.* 1878-85. *Trans.* 2.

1836 Leighton, Frederick, M.D.

1886 Lewers, Arthur Hamilton Nicholson, M.D., 60, Wimpole street, Cavendish square.

1872 Liebreich, Richard, Consulting Ophthalmic Surgeon to St. Thomas's Hospital; Paris.

Elected

1878 LISTER, SIR JOSEPH, Bart., D.C.L., LL.D., F.R.S., Surgeon Extraordinary to H.M. the Queen; Professor of Clinical Surgery at King's College, London; and Surgeon to King's College Hospital; 12, Park crescent, Regent's Park.

1872 *LITTLE, DAVID, M.D., Senior Surgeon to the Royal Eye Hospital, Manchester; 21, St. John street, Manchester.

1871 LITTLE, LOUIS STROMEYER, Shanghai, China.

1819 LLOYD, ROBERT, M.D.

1820 LOCHER, J. G., M.C.D., Town Physician of Zurich. *Trans.* 2.

1881 LOCKWOOD, CHARLES BARRETT, Surgeon to the Great Northern Central Hospital, and Demonstrator of Anatomy and Operative Surgery at St. Bartholomew's Hospital; 19, Upper Berkeley street. *Trans.* 1.

1860 LONGMORE, SIR THOMAS, C.B., Hon. Surgeon to H.M. the Queen; Surgeon-General, Army Medical Staff, and Professor of Military Surgery, Army Medical School, Netley, Southampton; Woolston Lawn, Woolston, Hants. *Trans.* 2.

1836 LÖWENFELD, JOSEPH S., M.D., Berbice.

1871 LOWNDS, THOMAS MACKFORD, M.D., late Professor of Anatomy and Physiology at Grant Medical College, Bombay; Egham Hill, Surrey.

1881 LUCAS, RICHARD CLEMENT, Senior Assistant Surgeon to, and Demonstrator of Operative and Practical Surgery at, Guy's Hospital; Surgeon to the Evelina Hospital for Sick Children; 18, Finsbury square.

1883 LUND, EDWARD, Professor of Surgery, and Member of Senate, Victoria University, Manchester; Consulting Surgeon to the Manchester Royal Infirmary; 22, St. John street, Manchester.

1857 LYON, FELIX WILLIAM, M.D., 7, South Charlotte street, Edinburgh.

1867 MABERLY, GEORGE FREDERICK, Mailai Valley, Nelson, New Zealand.

Elected

1873 MacCarthy, Jeremiah, M.A., Surgeon to the London Hospital and Lecturer on Physiology at the London Hospital Medical College; 15, Finsbury square. C. 1886-7. *Lib. Com.* 1882-5.

1867 Mac Cormac, Sir William, M.A., Surgeon to, and Lecturer on Surgery at, St. Thomas's Hospital; 13, Harley street. C. 1884-5. *Trans.* 1.

1887 Macdonald, George Childs, M.D.

1862 *M'Donnell, Robert, M.D., F.R.S., Surgeon to Steevens' Jervis street Hospitals; 89, Merrion square west, Dublin. *Trans.* 2.

1880 *Macfarlane, Alexander William, M.D., Examiner in Medicine and Clinical Medicine, University of Glasgow; 6, Manchester Square.

1866 Macgowan, Alexander Thorburn, M.D., Vyvyan House, Clifton, near Bristol.

1880 McHardy, Malcolm Macdonald, Ophthalmic Surgeon to King's College Hospital, and Professor of Ophthalmic Surgery in King's College, London; Surgeon to the Royal South London Ophthalmic Hospital; 5, Savile row.

1822 Macintosh, Richard, M.D.

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

1873 MacKellar, Alexander Oberlin, M.S.I., Surgeon to St. Thomas's Hospital; Surgeon-in-Chief to the Metropolitan Police Force; 22, George street, Hanover square.

1881 Mackenzie, Stephen, M.D., Physician to the London Hospital, and Lecturer on the Principles and Practice of Medicine at the London Hospital Medical College; Physician to the Royal London Ophthalmic Hospital; 26, Finsbury square. *Trans.* 1.

1885 Mackern, John, M.D., Assistant Physician to the Chelsea Hospital for Women; 30, Cambridge street, Hyde park.

Elected

1876 MACKEY, EDWARD, M.D., Assistant Physician to the Sussex County Hospital; 1, Brunswick road, Hove, Brighton.

1854 *MACKINDER, DRAPER, M.D., Consulting Surgeon to the Dispensary, Gainsborough, Lincolnshire.

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

1876 MACNAMARA, CHARLES N., Surgeon to, and Lecturer on Surgery at, the Westminster Hospital; Surgeon to the Royal Westminster Ophthalmic Hospital; Surgeon-Major Bengal Medical Service; Fellow of the Calcutta University; 13, Grosvenor street. *Referee*, 1884-7. *Lib. Com.* 1886-7.

1881 MACREADY, JONATHAN FORSTER CHRISTIAN HORACE, Surgeon to the Great Northern Hospital; 51, Queen Anne street, Cavendish square.

1880 MADDICK, EDMUND DISTIN, 2, Chandos street, Cavendish square.

1886 MAGUIRE, ROBERT, M.D., Warden of St. Mary's Hospital Residential College; 33, Westbourne Terrace.

1880 MAKINS, GEORGE HENRY, Assistant Surgeon to St. Thomas's Hospital and to the Evelina Hospital for Children; 2, Queen street, May Fair.

1885 MALCOLM, JOHN DAVID, M.B., Surgeon in charge of Out-Patients, Samaritan Free Hospital; 24, Bryanston street, Portman square.

1876 MALLAM, BENJAMIN, Rose Bank, Blackall road, Exeter.

1855 MARCET, WILLIAM, M.D., F.R.S., Flowermead, Wimbledon Park, and Athenæum Club, Pall Mall. C. 1871. *Referee*, 1866-70, 1883-6. *Sci. Com.* 1863. *Lib. Com.* 1866-8. *Trans.* 3.

1867 MARSH, F. HOWARD, *Secretary*, Assistant Surgeon to, and Lecturer on Anatomy at, St. Bartholomew's Hospital; Surgeon to the Hospital for Sick Children, Great Ormond street; 30, Bruton street, Berkeley square. C. 1882-3. S. 1885-7. *Lib. Com.* 1880-1. *Trans.* 4.

Elected

1851 †Marshall, John, F.R.S., Professor of Anatomy to the Royal Academy of Arts; Emeritus Professor of Surgery in University College, London, and Consulting Surgeon to University College Hospital; 10, Savile row, Burlington gardens. C. 1866. V.P. 1875-6. P. 1882-3. *Referee*, 1867, 1871-4, 1877-81. *Trans.* 3.

1884 Martin, Sidney Harris Cox, M.D.; 60, Gower street.

1883 Maudsley, Henry Carr, M.D., Resident Medical Officer, University College Hospital, Gower street.

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

1865 Medwin, Aaron George, M.D., Consulting Dental Surgeon to the Royal Kent Dispensary, 34, Bruton street, Berkeley square, and 11, Montpellier row, Blackheath.

1880 Meredith, William Appleton, M.B., C.M., Surgeon to the Samaritan Free Hospital for Women and Children; 6, Queen Anne street, Cavendish square.

1874 Merriman, John J., 45, Kensington square.

1815 Meyer, Augustus, M.D., St. Petersburg.

1840 Middlemore, Richard, Consulting Surgeon to the Birmingham Eye Hospital; The Limes, Bristol road, Edgbaston, Birmingham.

1854 Middleship, Edward Archibald.

1885 Millican, Kenneth William, B.A., 58, Welbeck street.

1882 Mills, Joseph, 15, Henrietta street, Cavendish square.

1873 Milner, Edward, Surgeon to the Lock Hospital; 32, New Cavendish street, Portland place.

1883 Money, Angel, M.D., Assistant Physician to University College Hospital, to the Hospital for Sick Children, Great Ormond Street, and to the City of London Hospital for Diseases of the Chest, Victoria Park; 24 Harley street, Cavendish square. *Trans.* 4.

Elected

1873 MOORE, NORMAN, M.D., Assistant Physician and Warden of the College, and Demonstrator of Morbid Anatomy, St. Bartholomew's Hospital; The Warden's House, St. Bartholomew's Hospital. *Referee*, 1886-7.

1857 MORGAN, JOHN, 3, Sussex place, Hyde park gardens. C. 1880-1. *Lib. Com.* 1862-3. *Trans.* 1.

1861 MORGAN, JOHN EDWARD, M.D., Physician to the Manchester Royal Infirmary, and Professor of Medicine in the Victoria University, Manchester; 1, St. Peter's square, Manchester.

1878 MORGAN, JOHN HAMMOND, M.A., Assistant Surgeon to the Charing Cross Hospital, and to the Hospital for Sick Children, Great Ormond street; 68, Grosvenor street. *Trans.* 1.

1874 MORRIS, HENRY, M.A., Surgeon to, and Lecturer on Surgery at, the Middlesex Hospital; 2, Mansfield street, Portland place. *Referee*, 1882-7. *Trans.* 10.

1879 MORRIS, MALCOLM ALEXANDER, Surgeon to the Skin Department of, and Lecturer on Dermatology at, St. Mary's Hospital; 8, Harley street, Cavendish square.

1885 MOTT, FREDERICK WALKER, M.D., Lecturer on Physiology, Charing Cross Hospital; Meadowlead, Gayton road, Harrow.

1879 MUNK, WILLIAM, M.D., Harveian Librarian, Royal College of Physicians; Consulting Physician to the Royal Hospital for Incurables; 40, Finsbury square.

1873 MURRAY, J. IVOR, M.D., F.R.S.Ed. 24, Huntriss row, Scarborough.

1880 MURRELL, WILLIAM, M.D., Assistant Physician to the Royal Hospital for Diseases of the Chest; Assistant Physician to, and Lecturer on Materia Medica and Therapeutics at, the Westminster Hospital; 38, Weymouth street, Portland place. *Trans.* 1.

Elected

1863 MYERS, ARTHUR BOWEN RICHARDS, Surgeon to the 1st Battalion, Coldstream Guards; 3, Park Terrace, Windsor. C. 1878-9. *Lib. Com.* 1877.

1882 MYERS, ARTHUR THOMAS, M.D., Medical Registrar, St. George's Hospital; 9, Lower Berkeley street, Portman square.

1881 NALL, SAMUEL, M.B., Disley, Stockport, Cheshire.

1870 NEILD, JAMES EDWARD, M.D., Lecturer on Forensic Medicine in the University of Melbourne; 166, Collins street east, Melbourne, Victoria.

1835 †NELSON, THOMAS ANDREW, M.D., 10, Nottingham terrace, York Gate, Regent's Park. *Lib. Com.* 1841.

1877 NETTLESHIP, EDWARD, Ophthalmic Surgeon to, and Lecturer on Ophthalmology at, St. Thomas's Hospital; Assistant Surgeon to the Royal London Ophthalmic Hospital; Ophthalmic Surgeon to the Hospital for Sick Children; 5, Wimpole street, Cavendish square.

1843 †NEWTON, EDWARD, 85, Gloucester terrace, Hyde Park. C. 1863-4.

1868 NICHOLLS, JAMES, M.D., Senior Medical Officer, Essex and Chelmsford Infirmary and Dispensary; the Old Infirmary, Chelmsford, Essex.

1849 NORMAN, HENRY BURFORD, Portland Lodge, Southsea, Hants. *Lib. Com.* 1857.

1847 *NOURSE, WILLIAM EDWARD CHARLES, Bouverie House, Exeter.

1864 NUNN, THOMAS WILLIAM, Consulting Surgeon to the Middlesex Hospital; 8, Stratford place, Oxford street.

1870 NUNNELEY, FREDERICK BARHAM, M.D. *Trans.* 2.

1884 OAKES, ARTHUR, M.D., 99, Priory road, West Hampstead.

1880 O'CONNOR, BERNARD, A.B., M.D., Physician to the North London Hospital for Consumption; 17, St. James's place.

1847 O'CONNOR, THOMAS, March, Cambridgeshire.

Elected

1880 OGILVIE, GEORGE, M.B., Lecturer on Experimental Physics at the Westminster Hospital; Physician to the Hospital for Epilepsy and Paralysis, Regent's Park; 13, Welbeck street, Cavendish square.

1880 OGILVIE, LESLIE, M.B., Lecturer on Comparative Anatomy at the Westminster Hospital; 46, Welbeck street, Cavendish square.

1858 OGLE, JOHN WILLIAM, M.D., Consulting Physician to St. George's Hospital; 30, Cavendish square. C. 1873. V.P. 1886. *Referee*, 1864-72. *Trans.* 4.

1855 *OGLE, WILLIAM, M.A., M.D., Physician to the Derbyshire Infirmary; The Elms, Duffield road, Derby.

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

1870 OLDHAM, CHARLES FREDERIC, India [Agents: Messrs. Grindlay and Co., 55, Parliament street].

1883 *OLIVER, THOMAS, M.D., Lecturer on Practical Physiology, University of Durham; and Physician to the Newcastle-upon-Tyne Infirmary; 12, Eldon square, Newcastle-upon-Tyne.

1871 *O'NEILL, WILLIAM, M.D., Physician to the Lincoln Lunatic Hospital, Silver street, Lincoln.

1873 ORD, WILLIAM MILLER, M.D., Physician to, and Lecturer on Medicine at, St. Thomas's Hospital; 37, Upper Brook street, Grosvenor square. *Referee*, 1884-7. *Trans.* 6.

1877 ORMEROD, JOSEPH ARDERNE, M.D., Assistant Physician to the National Hospital for the Paralysed and Epileptic, Queen square, and to the City of London Hospital for Diseases of the Chest, Victoria Park; 25, Upper Wimpole street. *Trans.* 1.

Elected

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

1879 Owen, Edmund, Surgeon to St. Mary's Hospital; Surgeon to the Hospital for Sick Children; 49, Seymour street, Portman square. *Trans.* 1.

1882 Owen, Herbert Isambard, M.D., Assistant Physician to, and Lecturer on Forensic Medicine at, St. George's Hospital; 5, Hertford street, May Fair.

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. *Referee*, 1884-7. *Lib. Com.* 1886-7. *Trans.* 2.

1887 Paget, Charles Edward, Kendal, Westmoreland.

1840 †Paget, Sir James, Bart., D.C.L., LL.D., F.R.S., Sergeant-Surgeon to H.M. the Queen; Surgeon-in-Ordinary to H.R.H. the Prince of Wales; Consulting Surgeon to St. Bartholomew's Hospital; Vice-Chancellor of the University of London; Foreign Associate of the 'Académie de Médecine,' Paris; 1, Harewood place, Hanover square. C. 1848-9. V.P. 1861. T. 1867. P. 1875-6. *Referee*, 1844-6, 1848, 1851-60, 1862-6, 1868-74. *Sci. Com.* 1863. *Lib. Com.* 1846-7. *Trans.* 12.

1886 Paget, Stephen, 57, Wimpole street, Cavendish square.

1858 *Paley, William, M.D., Physician to the Ripon Dispensary; The Old Residence, Ripon, Yorkshire.

1847 Parker, Nicholas, M.D., Paris.

1873 Parker, Robert William, Surgeon to the East London Hospital for Children; 8, Old Cavendish street. *Lib. Com.* 1885-7. *Trans.* 4.

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

Elected

1883 PASTEUR, WILLIAM, M.D., Medical Registrar to the Middlesex Hospital; Physician to the North-Eastern Hospital for Children; 19, Queen street, May Fair.

1865 PAVY, FREDERICK WILLIAM, M.D., F.R.S., Physician to Guy's Hospital; 35, Grosvenor street. C. 1883-4. *Referee*, 1871-82. *Trans.* 1.

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

1879 PEEL, ROBERT, 120, Collins street east, Melbourne, Victoria.

1856 PEIRCE, RICHARD KING, Woodside, Windsor forest, Berks.

1830 PELECHIN, CHARLES P., M.D., St. Petersburg.

1855 *PEMBERTON, OLIVER, Senior Surgeon to the Birmingham General Hospital, and Professor of Surgery at the Queen's College, Birmingham; 12, Temple row, Birmingham. *Trans.* 1.

1874 PENHALL, JOHN THOMAS, 5, Eversfield place, St. Leonard's, Sussex.

1887 PENROSE, FRANCIS GEORGE, M.D., 50, Torrington square.

1879 *PESIKAKA, HORMASJI DOSABHAI, Marine Lines, Bombay.

1878 *PHILIPSON, GEORGE HARE, M.D., M.A., D.C.L., Professor of Medicine at Durham University; Senior Physician to the Newcastle-upon-Tyne Infirmary; 7, Eldon square, Newcastle-upon-Tyne.

1883 PHILLIPS, CHARLES DOUGLAS F., M.D., F.R.S.Ed., 10, Henrietta street, Cavendish square, W.

1884 PHILLIPS, GEORGE RICHARD TURNER, 24, Leinster square, Bayswater.

1867 PICK, THOMAS PICKERING, Surgeon to, and Lecturer on Surgery at, St. George's Hospital; 18, Portman street, Portman square. C. 1884-5. *Referee*, 1882-3. *Sci. Com.* 1870. *Lib. Com.* 1879-81.

Elected

1841 †Pitman, Sir Henry Alfred, M.D., Consulting Physician to St. George's Hospital; 28, Gordon square. 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., Assistant Physician to, and Pathologist at, Guy's Hospital; 9, St. Thomas's street, Southwark.

1885 Poland, John, Demonstrator of Anatomy, Guy's Hospital; 16, St. Thomas's street, Southwark.

1884 Pollard, Bilton, Assistant Surgeon and Surgical Reigstrar to University College Hospital, Surgeon to the North Eastern Hospital for Children; 24, Harley street, Cavendish square.

1871 Pollock, Arthur Julius, M.D., Senior Physician to, and Lecturer on the Principles and Practice of Medicine at, Charing Cross Hospital; Physician to the Foundling Hospital; 85, Harley street, Cavendish square.

1845 †Pollock, George David, *President*, Surgeon-in-Ordinary to H.R.H. the Prince of Wales; Consulting Surgeon to St. George's Hospital; 36, Grosvenor street. C. 1856-7. L. 1859-62. V.P. 1870-1. P. 1886-7. *Referee*, 1858, 1864-9, 1877-85. *Trans.* 5.

1865 Pollock, James Edward, M.D., Consulting Physician to the Hospital for Consumption, Brompton; 52, Upper Brook street, Grosvenor square. C. 1882-3. *Referee*, 1872-81.

1871 Poore, George Vivian, M.D., Professor of Medical Jurisprudence in University College, London; Physician to University College Hospital; Consulting Physician to the Royal Infirmary for Children and Women, Waterloo road; 30, Wimpole street. *Referee*, 1887. *Trans.* 2.

1885 Port, Heinrich, M.D., Physician to the German Hospital; 48, Finsbury square.

1846 Potter, Jephson, M.D., F.L.S.

1842 Powell, James, M.D.

Elected

1867 POWELL, RICHARD DOUGLAS, M.D., Physician to, and Lecturer on Practical Medicine at, the Middlesex Hospital; Physician to the Hospital for Consumption and Diseases of the Chest, Brompton; 62, Wimpole st., Cavendish square. S. (Oct.), 1883-5. C. 1887. *Referee*, 1879-83, 1886. *Trans.* 2.

1867 POWER, HENRY, Senior Ophthalmic Surgeon to, and Lecturer on Ophthalmic Surgery at, St. Bartholomew's Hospital; 37A, Great Cumberland place, Hyde park. C. 1882-3. *Referee*, 1870-81. *Sci. Com.* 1870. *Lib. Com.* 1872-8.

1857 PRIESTLEY, WILLIAM OVEREND, M.D., LL.D., Consulting Physician to King's College Hospital, and to the St. Marylebone Infirmary; 17, Hertford street, Mayfair. C. 1874-5. V.P. 1884-5. *Referee*, 1867-73, 1877-83. *Sci. Com.* 1863.

1883 PRINGLE, JOHN JAMES, M.B., C.M., Assistant Physician to the Middlesex Hospital, and Physician to the Royal Hospital for Diseases of the Chest; 35, Bruton street, Berkeley square. *Trans.* 1.

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

1878 PYE, WALTER, Surgeon (with charge of out-patients) to St. Mary's Hospital and to the Victoria Hospital for Children; 4, Sackville street, Piccadilly.

1877 PYE-SMITH, PHILIP HENRY, M.D., F.R.S., Physician to, and Lecturer on Medicine at, Guy's Hospital; Member of the Senate of the University of London; 54, Harley street, Cavendish square. *Lib. Com.* 1887.

1850 †QUAIN, RICHARD, M.D., F.R.S., Consulting Physician to the Hospital for Consumption, Brompton; Member of the Senate of the University of London; 67, Harley street, Cavendish square. C. 1866-7. V.P. 1878-9. *Sci. Com.* 1863. *Trans.* 1.

Elected

1852 †Radcliffe, Charles Bland, M.D., Consulting Physician to the Westminster Hospital; Physician to the National Hospital for the Paralysed and Epileptic; 25, Cavendish square. C. 1867-8. V.P. 1879-80. T. 1881-6. *Referee*, 1862-6, 1870-8.

1871 Ralfe, Charles Henry, M.D., M.A., Assistant Physician to the London Hospital, and late Physician to the Seamen's Hospital, Greenwich; 26, Queen Anne street, Cavendish square. *Referee*, 1885-7.

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

1854 Ransom, William Henry, M.D., F.R.S., Physician to the Nottingham General Hospital, Nottingham.

1869 Read, Thomas Laurence, 11, Petersham terrace, Queen's gate.

1858 Reed, Frederick George, M.D., 46, Hertford street, Mayfair. *Trans.* 1.

1821 Reeder, Henry, M.D., Varick, Seneca County, New York, United States.

1857 Rees, George Owen, M.D., F.R.S., Physician Extraordinary to H.M. the Queen, Consulting Physician to Guy's Hospital. C. 1873. *Referee*, 1860-72, 1875-81. *Trans.* 1.

1882 Reid, James, M.D., Resident Physician to H.M. the Queen, Windsor Castle.

1884 Reid, Thomas Whitehead, Surgeon to the Kent and Canterbury Hospital; 34, St. George's place, Canterbury.

1855 †Reynolds, John Russell, M.D., F.R.S., Physician-in-Ordinary to H.M.'s Household; Consulting-Physician to University College Hospital; 38, Grosvenor street. C. 1870. V.P. 1883. *Referee*, 1867-9.

1865 Rhodes, George Winter, Surgeon to the Huddersfield Infirmary; Queen street south, Huddersfield.

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

1852 Richardson, Christopher Thomas, M.B., 13, Nelson crescent, Ramsgate.

Elected

1887 RICHARDSON, GILBERT, M.D., Thornton, Upper Richmond road, Putney.

1845 †RIDGE, BENJAMIN, M.D., 8, Mount street, Grosvenor square.

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

1871 RIVINGTON, WALTER, M.S., Surgeon to, and Lecturer on Surgery at, the London Hospital; 22, Finsbury square. C. 1885-6. *Trans.* 4.

1871 *ROBERTS, DAVID LLOYD, M.D., Obstetric Physician to the Manchester Royal Infirmary, Physician to St. Mary's Hospital, Manchester; 11, St. John street, Manchester.

1878 ROBERTS, FREDERICK THOMAS, M.D., Professor of Materia Medica and Therapeutics in University College, London; and Physician to University College Hospital; Physician to the Hospital for Consumption, Brompton; 102, Harley street, Cavendish square.

1857 ROBERTSON, JOHN CHARLES GEORGE, Medical Superintendent of the Cavan District Lunatic Asylum; Monaghan, Ireland.

1873 ROBERTSON, WILLIAM HENRY, M.D., Consulting Physician to the Buxton Bath Charity and Devonshire Hospital; Buxton, Derbyshire.

1885 ROCKWOOD, WILLIAM GABRIEL, M.D., Colombo, Ceylon.

1850 ROPER, GEORGE, M.D., Consulting Physician to the Eastern Division of the Royal Maternity Charity; Physician to the Royal Infirmary for Children and Women, Waterloo Bridge road; 19, Ovington Gardens. C. 1879-80.

1857 ROSE, HENRY COOPER, M.D., F.L.S., Consulting Surgeon to the Hampstead Dispensary; Penrose House, Hampstead. C. 1886-7. *Trans.* 1.

Elected

1883 Rose, William, M.B., Surgeon to King's College Hospital and to the Royal Free Hospital; 50, Harley street, Cavendish square.

1882 Routh, Amand Jules McConnel, M.D., B.S., Physician to the Samaritan Free Hospital for Women; Assistant Obstetric Physician to the Charing Cross Hospital; Obstetric Physician to the St. Marylebone General Dispensary; 6, Upper Montagu street, Montagu square.

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

1863 Rowe, Thomas Smith, M.D., Senior Visiting Surgeon to the Royal Sea-Bathing Infirmary; Cecil street, Margate, Kent.

1882 Roy, Charles Smart, M.D., F.R.S., Professor of Pathology in the University of Cambridge; Trinity College, Cambridge.

1871 Rutherford, William, M.D., F.R.S., Professor of the Institutes of Medicine in the University of Edinburgh; 14, Douglas crescent, Edinburgh.

1886 Sainsbury, Harrington, M.D., Assistant Physician and Pathologist to the Royal Free Hospital; 63, Welbeck street, Cavendish square. *Trans.* 1.

1856 Salter, S. James A., M.B., F.R.S., F.L.S., Basingfield, near Basingstoke, Hants. C. 1871. *Lib. Com.* 1878. *Trans.* 2.

1849 †Sanderson, Hugh James, M.D., 26, Upper Berkeley street, Portman square. C. 1872-3. *Lib. Com.* 1862-3.

1855 †Sanderson, John Burdon, M.D., LL.D., F.R.S., Waynflete Professor of Physiology in the University of Oxford; 50, Banbury road, Oxford. C. 1869-70. V.P. 1882. *Referee*, 1867-8, 1876-81. *Sci. Com.* 1862, 1870. *Lib. Com.* 1876-81. *Trans.* 2.

1867 Sandford, Folliott James, M.D., Market Drayton, Shropshire.

Elected

1879 SANGSTER, ALFRED, B.A., M.B., Physician to the Skin Department, and Demonstrator of Skin Diseases at the Charing Cross Hospital; 6, Savile row. *Trans.* 1.

1847 †SANKEY, WILLIAM HENRY OCTAVIUS, M.D., Boreatton park, Baschurch, near Shrewsbury.

1869 SANSOM, ARTHUR ERNEST, M.D., Senior Physician to the North-Eastern Hospital for Children; Physician (with charge of out-patients) to the London Hospital; 84, Harley street, Cavendish square. C. 1887. *Trans.* 2.

1886 SAUNDBY, ROBERT, M.D., Physician to the General Hospital, and Consulting Physician to the Hospital for Women, and to the Eye Hospital, Birmingham; 83A, Edmund street, Birmingham.

1845 †SAUNDERS, SIR EDWIN, Surgeon-Dentist to H.M. the Queen, and to their R.H. the Prince and Princess of Wales; 13A, George street, Hanover square. C. 1872-3.

1834 SAUVAN, LUDWIG V., M.D., Warsaw.

1879 SAVAGE, GEORGE HENRY, M.D., Medical Superintendent and Resident Physician to the Bethlem Royal Hospital, St. George's road, Southwark.

1859 SAVORY, WILLIAM SCOVELL, F.R.S., Surgeon to, and Lecturer on Surgery at, St. Bartholomew's Hospital; Surgeon to Christ's Hospital; 66, Brook street, Grosvenor square. C. 1871-2. L. 1878. V.P. 1883-4. *Referee*, 1865-70, 1873-77, 1879-82. *Sci. Com.* 1862, 1867, 1870. *Lib. Com.* 1866-8. *Trans.* 8.

1883 SCHÄFER, EDWARD ALBERT, F.R.S., Jodrell Professor of Physiology, University College, London; University College, Gower street.

1887 SCOTT, HARRY, M.B., 8, Queen Anne's Gate.

1861 *SCOTT, WILLIAM, M.D., Senior Physician to the Huddersfield Infirmary; Waverley House, Huddersfield.

1882 SCRIVEN, JOHN BARCLAY, Brigade Surgeon, Bengal (retired), late Professor of Anatomy, Surgery, and Ophthalmic Surgery at the Lahore Medical School; 95, Oxford gardens, Notting hill.

Elected

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

1877 SEMON, FELIX, M.D., Assistant Physician for Diseases of the Throat to St. Thomas's Hospital; 39, Wimpole street, Cavendish square. *Trans.* 1.

1875 SEMPLE, ROBERT HUNTER, M.D., Physician to the Bloomsbury Dispensary; 8, Torrington square. *Sci. Com.* 1879.

1873 *SHAPTER, LEWIS, B.A., M.B., Physician to the Devon and Exeter Hospital; the Barnfield, Exeter.

1882 SHARKEY, SEYMOUR JOHN, M.B., Assistant Physician, Joint Lecturer on Pathology, and Demonstrator of Morbid Anatomy, to St. Thomas's Hospital; 2, Portland place. *Trans.* 2.

1840 SHARP, WILLIAM, M.D., F.R.S., Horton House, Rugby. *Trans.* 1.

1836 †SHAW, ALEXANDER, Consulting Surgeon to the Middlesex Hospital; 136, Abbey road, Kilburn. C. 1842. S. 1843-4. V.P. 1851-2. T. 1858-60. *Referee*, 1842-3, 1846-50, 1855-7, 1865. *Lib. Com.* 1843. *Trans.* 4.

1886 SHAW, LAURISTON ELGIE, M.D., Medical Registrar and Demonstrator of Practical Medicine, Guy's Hospital; Assistant Physician to the City of London Hospital for Diseases of the Chest, Victoria Park; 15, St. Thomas's street, Southwark.

1884 SHEILD, ARTHUR MARMADUKE, M.B., B.S., Assistant Surgeon, Charing Cross Hospital; 20, Stratford place, Oxford street.

1859 SIBLEY, SEPTIMUS WILLIAM, 7, Harley street, Cavendish square. C. 1882-3. *Sci. Com.* 1863. *Trans.* 4.

1848 †SIEVEKING, SIR EDWARD HENRY, M.D., Physician-Extraordinary to H.M. the Queen; Physician-in-Ordinary to H.R.H. the Prince of Wales; Consulting Physician to St. Mary's Hospital; 17, Manchester square. C. 1859-60. S. 1861-3. V.P. 1873-4. L. 1881-2. *Referee*, 1855-8, 1864-72, 1875-80. *Sci. Com.* 1862. *Trans.* 2.

Elected

1886 SILCOCK, ARTHUR QUARRY, M.D., B.S., Surgeon in charge of out-patients, St. Mary's Hospital; Assistant Surgeon, Royal London Ophthalmic Hospital; 101, Harley street, Cavendish square.

1842 †SIMON, Sir JOHN, K.C.B., D.C.L., LL.D., F.R.S., Consulting Surgeon to St. Thomas's Hospital; 40, Kensington square. C. 1854-5. V.P. 1865. *Referee* 1851-3, 1866-81. *Trans.* 1.

1857 SIORDET, JAMES LEWIS, M.B., Villa Preti, Mentone, Alpes Maritimes, France.

1882 SMITH, CHARLES JOHN, 27, Selborne road, Brighton, and 75, Park street, Grosvenor square.

1879 SMITH, E. NOBLE, Senior Surgeon and Surgeon to the Orthopædic Department of the Farringdon Dispensary; Orthopædic Surgeon to the British Home for Incurables; 24, Queen Anne street, Cavendish square.

1881 SMITH, EUSTACE, M.D., Physician to H.M. the King of the Belgians; Physician to the East London Children's Hospital, and to the Victoria Park Hospital for Diseases of the Chest; 5, George street, Hanover square.

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

1886 SMITH, HOWARD LYON, 80, Tollington Park.

1885 SMITH, JAMES GREIG, M.B., C.M., F.R.S.Ed., Surgeon to the Bristol Royal Infirmary; 16, Victoria square, Clifton.

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

1838 †SMITH, SPENCER, Consulting Surgeon to St. Mary's Hospital; 92, Oxford terrace, Hyde Park. C. 1854. S. 1855-8. V.P. 1859-60. T. 1865. *Referee*, 1851-3, 1862-4, 1866-78. *Lib. Com.* 1847.

Elected

1863 SMITH, THOMAS, *Vice-President*, Surgeon to, and Lecturer on Clinical Surgery at, St. Bartholomew's Hospital; 5, Stratford place, Oxford street. S. 1870-2. C. 1875-6. V.P. 1887. *Referee*, 1873-4, 1880-6. *Sci. Com.* 1867. *Trans.* 3.

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

1874 *SMITH, WILLIAM ROBERT, M.D., D.Sc., F.R.S.Ed., 74, Great Russell Street, Bloomsbury.

1868 SOLLY, SAMUEL EDWIN, Colorado Springs, Colorado, U.S.

1865 SOUTHEY, REGINALD, M.D., Commissioner in Lunacy; 32, Grosvenor road, Westminster. C. 1881-2. S. 1883. *Referee*, 1873-80. *Trans.* 1.

1844 SPACKMAN, FREDERICK ROBERT, M.D., Consulting Physician to St. Alban's Hospital, Harpenden, St. Alban's.

1887 SPENCER, WALTER GEORGE, M.B., Assistant Surgeon to the Westminster Hospital; 94, Wimpole street, Cavendish square.

1875 SPITTA, EDMUND JOHNSON, Ivy House, Clapham Common, Surrey.

1851 †SPITTA, ROBERT JOHN, M.D., East Side, Clapham Common, Surrey. C. 1878-9. *Trans.* 1.

1885 SQUIRE, JOHN EDWARD, M.D., Assistant Physician to the North London Hospital for Consumption; 23, Seymour street, Portman square. *Trans.* 1.

1882 STEAVENSON, WILLIAM EDWARD, M.D., Electrician to St. Bartholomew's Hospital; Physician to the Alexandra Hospital for Children; 39, Welbeck street, Cavendish square.

1854 STEVENS, HENRY, M.D., Inspector, Medical Department, Local Government Board, Whitehall; Mitcham House, Mitcham, Surrey.

1884 STEWART, EDWARD, M.D., 16, Harley street, Cavendish square.

1859 STEWART, WILLIAM EDWARD, 16, Harley street, Cavendish square.

Elected

1879 *Stirling, Edward Charles, late Assistant Surgeon and Lecturer on Physiology at St. George's Hospital; Adelaide, South Australia [care of T. Gemmell, Esq., 11, Essex street, Strand].

1856 †Stocker, Alonzo Henry, M.D., Peckham House, Peckham.

1865 Stokes, Sir William, M.D., M.C., Surgeon to the Richmond Surgical Hospital; 5, Merrion square north, Dublin. *Trans.* 1.

1884 Stonham, Charles, Assistant Surgeon to the Westminster Hospital, and to the Cancer Hospital, Brompton; 37, Welbeck street, Cavendish square.

1843 Storks, Robert Reeve, Paris.

1871 Strong, Henry John, M.D., Surgeon to the Croydon General Hospital; Whitgift House, George street, Croydon.

1863 †Sturges, Octavius, M.D., Physician to, and Lecturer on Medicine at, the Westminster Hospital; Physician to the Hospital for Sick Children; 85, Wimpole street, Cavendish square. C. 1878-9. *Referee,* 1882-7.

1871 †Sutherland, Henry, M.D., Lecturer on Insanity at the Westminster Hospital; 6, Richmond terrace, Whitehall.

1871 Sutton, Henry Gawen, M.B., Physician to, and Lecturer on Pathology at, the London Hospital; 9, Finsbury square. *Trans.* 1.

1883 Sutton, John Bland, Assistant Surgeon, Lecturer on Comparative Anatomy, and Senior Demonstrator of Anatomy to the Middlesex Hospital; 22, Gordon street, Gordon square. *Trans.* 3.

1886 Symonds, Charters James, M.S., Assistant Surgeon to Guy's Hospital; 26, Weymouth street, Portland place.

1878 *Sympson, Thomas, Surgeon to the Lincoln County Hospital; 3, James street, Lincoln.

1870 Tait, Lawson, Surgeon to the Birmingham and Midland Hospital for Women; 7, The Crescent, Birmingham. *Trans.* 4.

Elected

1864 TAUSSIG, GABRIEL, M.D., 70, Piazza Barberini, Rome.

1875 TAY, WAREN, Surgeon to the London Hospital, to the Royal London Ophthalmic Hospital, to the North Eastern Hospital for Children, and to the Hospital for Skin Diseases, Blackfriars; 4, Finsbury square.

1873 TAYLOR, FREDERICK, M.D., Physician to, and Lecturer on Medicine at, Guy's Hospital; Physician to the Evelina Hospital for Sick Children; 11, St. Thomas's street, Southwark. *Referee*, 1887. *Trans.* 1.

1845 †TAYLOR, THOMAS, Warwick House, 1, Warwick place, Grove End road, St. John's Wood.

1886 TEALE, THOMAS PRIDGIN, M.B., Consulting Surgeon to the Leeds General Infirmary; 38, Cookridge street, Leeds.

1859 TEGART, EDWARD, 49, Jermyn street, St. James's.

1874 THIN, GEORGE, M.D., 22, Queen Anne street, Cavendish square. *Trans.* 9.

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

1852 †THOMPSON, SIR HENRY, Surgeon-Extraordinary to H.M. the King of the Belgians; Emeritus Professor of Clinical Surgery in University College, London; and Consulting Surgeon to University College Hospital; Member of the "Société de Chirurgie," Paris; 35, Wimpole street, Cavendish square. C. 1869. *Trans.* 7.

1862 THOMPSON, REGINALD EDWARD, M.D., Physician to the Hospital for Consumption, Brompton; 47, Park street, Grosvenor square. C. 1879. S. 1880-82. V.P. 1883-4. *Referee*, 1873-8. *Sci. Com.* 1867. *Trans.* 2.

1881 THOMSON, WILLIAM SINCLAIR, M.D., 40, Ladbroke grove, Kensington Park Gardens.

1876 THORNTON, JOHN KNOWSLEY, M.B., C.M., Surgeon to the Samaritan Free Hospital for Women and Children; 22, Portman street, Portman square. *Lib. Com.* 1886-7 *Trans.* 4.

Elected

1883 THURSFIELD, THOMAS WILLIAM, M.D., Physician to the Warneford and South Warwickshire General Hospital; 26, The Parade, Leamington.

1848 †TILT, EDWARD JOHN, M.D., Consulting Physician to the Farringdon General Dispensary and Lying-in Charity; 27, Seymour street, Portman square. *Referee*, 1874-81.

1880 TIVY, WILLIAM JAMES, 8, Lansdowne place, Clifton, Bristol.

1872 TOMES, CHARLES SISSMORE, M.A., F.R.S., 37, Cavendish square. C. 1887. *Lib. Com.* 1879.

1867 TONGE, MORRIS, M.D., Harrow-on-the-Hill, Middlesex.

1882 TOOTH, HOWARD HENRY, M.D., Assistant Medical Tutor St. Bartholomew's Hospital; 34, Harley street, Cavendish square.

1871 *TREND, THEOPHILUS W., M.D., Raeberry Lodge, Southampton.

1879 TREVES, FREDERICK, Surgeon to, and Lecturer on Anatomy at, the London Hospital; 6, Wimpole street, Cavendish square. *Trans.* 4.

1881 *TREVES, WILLIAM KNIGHT, Surgeon to the National Hospital for Scrofula; 31, Dalby square, Cliftonville, Margate.

1867 TROTTER, JOHN WILLIAM, late Surgeon-Major, Coldstream Guards; 4, St. Peter's terrace, York.

1859 TRUMAN, EDWIN THOMAS, Surgeon-Dentist in Ordinary to Her Majesty's Household; 23, Old Burlington street.

1862 TUKE, THOMAS HARRINGTON, M.D., Manor House, Chiswick, and 37, Albemarle street, Piccadilly.

1875 TURNER, FRANCIS CHARLEWOOD, M.A., M.D., Physician to the North-Eastern Hospital for Children, and to the London Hospital; 15, Finsbury square.

1873 TURNER, GEORGE BROWN, M.D., Vernon House, Ryde, Isle of Wight.

1882 TURNER, GEORGE ROBERTSON, Visiting Surgeon to the Seamen's Hospital, Greenwich; Demonstrator of Anatomy and Joint Lecturer on Practical Surgery at St. George's Hospital; 49, Green street, Park lane.

Elected.

1881 TYSON, WILLIAM JOSEPH, M.D., Medical Officer of the Folkestone Infirmary; 10, Langhorne Gardens, Folkestone.

1876 VENN, ALBERT JOHN, M.D., Obstetric Physician to the Metropolitan Free Hospital; Physician to the Victoria Hospital for Children, Chelsea; and Assistant Physician for the Diseases of Women, West London Hospital; 27, George street, Hanover square.

1870 VENNING, EDGCOMBE, 30, Cadogan place.

1865 VERNON, BOWATER JOHN, Ophthalmic Surgeon to St. Bartholomew's Hospital and to the West London Hospital; 14, Clarges street, Piccadilly.

1867 VINTRAS, ACHILLE, M.D., Physician to the French Embassy, and to the French Hospital, Leicester place; 19A, Hanover square.

1828 VULPES, BENEDETTO, M.D., Physician to the Hospital of Aversa, and the Hospital of Incurables, Naples.

1854 WADDINGTON, EDWARD, Hamilton, Auckland, New Zealand.

1870 WADHAM, WILLIAM, M.D., Physician to St. George's Hospital; 14, Park lane.

1886 WAINEWRIGHT, BENJAMIN, M.B., C.M., 6, Harley street, Cavendish square.

1864 WAITE, CHARLES DERBY, M.B., Consulting Physician to the Westminster General Dispensary; 3, Old Burlington street.

1884 WAKLEY, Thomas, jun., 96, Redcliffe Gardens.

1868 *WALKER, ROBERT, Honorary Surgeon to the Carlisle Dispensary; 2, Portland square, Carlisle.

1887 WALLACE, EDWARD JAMES, M.D., Holmbush, Grove road, Southsea.

1883 WALLER, AUGUSTUS, M.D., Lecturer on Physiology, St. Mary's Hospital; Weston Lodge, 16, Grove End road.

1867 *WALLIS, GEORGE, Surgeon to Addenbrooke's Hospital, Corpus Buildings, Cambridge.

Elected

1873 WALSHAM, WILLIAM JOHNSON, C.M., Assistant Surgeon to, and Demonstrator of Practical and Orthopædic Surgery at, St. Bartholomew's Hospital; Surgeon to the Metropolitan Free Hospital; 27, Weymouth street, Portland place. *Lib. Com.* 1882-5. *Trans.* 3.

1852 †WALSHE, WALTER HAYLE, M.D., Emeritus Professor of the Principles and Practice of Medicine, University College, London; Consulting Physician to the Hospital for Consumption and to University College Hospital; 41, Hyde Park square. C. 1872. *Trans.* 1.

1883 *WALTERS, JAMES HOPKINS, 15, Friar street, Reading.

1851 †WALTON, HAYNES, Consulting Surgeon to St. Mary's Hospital, 1, Brook street, Grosvenor square. *Trans.* 1. *Pro.* 1.

1886 WARD, ALLAN OGIER, M.D., Casita, Northumberland Park, Tottenham.

1821 WARD, WILLIAM TILLEARD, Tilleards, Stanhope, Canada.

1846 WARE, JAMES THOMAS, Tilford House, near Farnham, Surrey.

1866 WARING, EDWARD JOHN, C.I.E., M.D., 49, Clifton Gardens, Maida vale. *Referee*, 1881-5.

1877 WARNER, FRANCIS, M.D., Assistant Physician and Lecturer on Botany to the London Hospital; 24, Harley street, Cavendish square. *Trans.* 1.

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

1879 WATERS, JOHN HENRY, M.D., C.M., 101, Jermyn street.

1861 †WATSON, WILLIAM SPENCER, M.B., Surgeon to the Great Northern Hospital; Surgeon to the Royal South London Ophthalmic Hospital; 7, Henrietta street, Cavendish square. C. 1883-4. *Trans.* 1.

1879 DE WATTEVILLE, ARMAND, M.A., M.D., B.Sc., Physician in Charge of the Electro-therapeutical Department at St. Mary's Hospital; 30, Welbeck street, Cavendish square.

Elected

1854 WEBB, WILLIAM, M.D., Gilkin View House, Wirksworth, Derbyshire.

1840 WEBB, WILLIAM WOODHAM, M.D., Neuilly-sur-Seine, France.

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

1844 †WEGG, WILLIAM, M.D., 15, Hertford street, Mayfair. L. 1854-8. C. 1861-2. T. 1873-80. *Lib. Com.* 1851-3.

1878 WEISS, HUBERT FOVEAUX, Assistant Surgeon to the West London Hospital; 11, Hanover square.

1874 WELLS, HARRY, M.D., San Ysidro, Buenos Ayres, S. America.

1854 †WELLS, SIR THOMAS SPENCER, Bart., Surgeon-in-Ordinary to H.M.'s Household; Consulting Surgeon to the Samaritan Free Hospital for Women and Children; Corresponding Member, "Académie de Médecine," Paris; 3, Upper Grosvenor street. C. 1870. V.P. 1881. *Trans.* 13. *Pro.* 1.

1842 †WEST, CHARLES, M.D., Corresponding Member of the Academy of Medicine of Paris; 55, Harley street, Cavendish square. C. 1855-6. V.P. 1863. P. 1877-8. *Referee*, 1848-54, 1857-62, 1864-76, 1880. *Sci. Com.* 1863. *Lib. Com.* 1844-7, 1851. *Trans.* 2.

1877 WEST, SAMUEL, M.D., Assistant Physician to St. Bartholomew's Hospital; Physician to the City of London Hospital for Diseases of the Chest, Victoria Park, and to the Royal Free Hospital; 15, Wimpole street, Cavendish square. *Trans.* 3.

1882 WHARRY, CHARLES JOHN, M.D., Resident Superintendent, Government Civil Hospital, Hong Kong.

1881 WHARRY, ROBERT, M.D., Physician to the Westminster Dispensary; 6, Gordon square.

1878 WHARTON, HENRY THORNTON, M.A., Honorary Surgeon to the Kilburn Dispensary; 39, St. George's road, Kilburn.

Elected

1828 WHATLEY, JOHN, M.D.

1875 WHIPHAM, THOMAS TILLYER, M.B., Physician to, and Lecturer on Pathology and Practical Medicine at, St. George's Hospital; 11, Grosvenor street, Grosvenor square.

1849 WHITE, JOHN.

1881 WHITE, WILLIAM HALE, M.D., Senior Assistant Physician to, and Lecturer on Materia Medica at, Guy's Hospital; 65, Harley street, Cavendish square. *Trans.* 1.

1881 *WHITEHEAD, WALTER, F.R.S. Ed., Surgeon to the Manchester Royal Infirmary; Senior Surgeon to the Manchester and Salford Lock and Skin Hospital; 24, St. Ann's square, Manchester. *Trans.* 1.

1885 *WHITLA, WILLIAM, M.D., Physician to, and Lecturer in Medicine at, the Belfast Royal Hospital; Consulting Physician to the Ulster Hospital for Women and Children; 8, College square north, Belfast.

1877 WHITMORE, WILLIAM TICKLE, Surgeon to the Westminster General Dispensary; 7, Arlington street, Piccadilly.

1852 WIBLIN, JOHN, M.D., Medical Inspector of Emigrants and Recruits; Southampton. *Trans.* 1.

1870 *WILKIN, JOHN F., M.D., M.C., The Warren, Beckenham park, Kent.

1883 *WILKINSON, THOMAS MARSHALL, Surgeon to the Lincoln County Hospital and to the Lincoln General Dispensary; 7, Lindum road, Lincoln.

1837 WILKS, GEORGE AUGUSTUS FREDERICK, M.D., Stanbury, Torquay.

1863 WILKS, SAMUEL, M.D., LL.D., F.R.S., Physician in Ordinary to their Royal Highnesses the Duke and Duchess of Connaught, and to H.R.H. the Duke of Edinburgh; Consulting Physician to Guy's Hospital, and Member of the Senate of the University of London; 72, Grosvenor street, Grosvenor square. *Referee*, 1872-81. *Sci. Com.* 1.

Elected

1883 *WILLANS, WILLIAM BLUNDELL, Great Hadham, Herts.

1865 †WILLETT, ALFRED, Surgeon to St. Bartholomew's Hospital; Surgeon to St. Luke's Hospital; 36, Wimpole street, Cavendish square. C. 1880-81. *Referee*, 1882-7. *Trans.* 2.

1887 WILLETT, EDGAR WILLIAM, M.B., 60, Welbeck street, Cavendish square.

1864 WILLETT, EDMUND SPARSHALL, M.D., Resident Physician, Wyke House, Isleworth, Middlesex.

1840 †WILLIAMS, CHARLES JAMES BLASIUS, M.D., F.R.S., Physician-Extraordinary to H.M. the Queen; Consulting Physician to the Hospital for Consumption, Brompton [47, Upper Brook street, Grosvenor square]; Villa de Rocher, Cannes. C. 1849-50. V.P. 1860-1. P. 1873-4. *Referee*, 1843-4. *Sci. Com.* 1862. *Trans.* 1.

1859 *WILLIAMS, CHARLES, Surgeon to the Norfolk and Norwich Hospital; 48, Prince of Wales road, Norwich.

1866 WILLIAMS, CHARLES THEODORE, M.A., M.D., Physician to the Hospital for Consumption and Diseases of the Chest, Brompton; 47, Upper Brook street, Grosvenor square. C. 1884-5. *Lib. Com.* 1880-3. *Trans.* 4.

1881 WILLIAMS, DAWSON, M.D., Assistant Physician to the East London Hospital for Children; Elstree, Kent.

1872 WILLIAMS, JOHN, M.D., Professor of Midwifery, University College, London; Obstetric Physician to University College Hospital; Examiner in Obstetric Medicine at the University of London; 11, Queen Anne street, Cavendish square. *Referee*, 1878-87. *Lib. Com.* 1876-82.

1868 WILLIAMS, WILLIAM RHYS, M.D., Commissioner in Lunacy; 13, Gloucester street, Warwick square.

1887 WILSON, ARTHUR HERVEY, M.D., 504, Broadway, Boston, U.S.A.

1863 WILSON, ROBERT JAMES, 7, Warrior square, St. Leonard's-on-Sea, Sussex.

Elected

1850 *Wise, Robert Stanton, M.D., Consulting Physician to the Southam Eye and Ear Infirmary; Beech Lawn, Banbury.

1825 Wise, Thomas Alexander, M.D., Thornton, Beulah Hill, Upper Norwood.

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

1885 Wolfenden, Richard Norris, M.D., Assistant Physician to the North-West London Hospital; 19, Upper Wimpole street.

1851 †Wood, John, F.R.S., Professor of Clinical Surgery in King's College, London, and Senior Surgeon to King's College Hospital; 61, Wimpole street, Cavendish square. C. 1867-8. V.P. 1877-8. *Referee*, 1871-6, 1880-87. *Lib. Com.* 1866. *Trans.* 3.

1887 Wood, Thomas Outterson, M.D., 40, Margaret street, Cavendish square.

1848 †Wood, William, M.D., Physician to St. Luke's Hospital for Lunatics; 99, Harley street, Cavendish square. C. 1867-8. V.P. 1877-8.

1883 Wood, William Edward Ramsden, M.A., M.D., Rockhampton, Queensland.

1879 Woodward, G. P. M., M.D., Deputy Surgeon-General; Sydney, New South Wales.

1878 Yeo, Gerald Francis, M.D., M.C., Professor of Physiology in King's College, London; Examiner in Physiology, University of London; Thorncote, Staines.

[It is particularly requested that any change of Title, Appointment, or Residence, may be communicated to the Secretaries before the 1st of October in each year, in order that the List may be made as correct as possible.]

HONORARY FELLOWS.

(Limited to Twelve.)

Elected

1847 CHADWICK, EDWIN, C.B., Corresponding Member of the Academy of Moral and Political Sciences of the Institute of France; Park Cottage, East Sheen.

1887 FLOWER, WILLIAM HENRY, C.B., LL.D., F.R.S., Director of the Natural History Department, British Museum, Cromwell road.

1887 FOSTER, MICHAEL, LL.D., F.R.S., Professor of Physiology in the University of Cambridge.

1883 FRANKLAND, EDWARD, M.D., D.C.L., Ph.D., F.R.S., Corresponding Member of the French Institute; The Yews, Reigate Hill, Reigate.

1868 HOOKER, SIR JOSEPH DALTON, C.B., M.D., K.C.S.I., D.C.L., LL.D., F.R.S., Member of the Senate of the University of London, Director of the Royal Botanic Gardens, Kew; Corresponding Member of the Academy of Sciences of the Institute of France; The Camp, Sunningdale.

1868 HUXLEY, THOMAS HENRY, LL.D., D.C.L., F.R.S., late Professor of Natural History in the Royal School of Mines; Corresponding Member of the Academies of Sciences of St. Petersburg, Berlin, Dresden, &c.; 4, Marlborough place, St. John's wood.

1878 LUBBOCK, SIR JOHN, Bart., M.P., D.C.L., LL.D., F.R.S., High Elms, Hayes, Kent.

1847 OWEN, SIR RICHARD, K.C.B., D.C.L., LL.D., F.R.S., late Superintendent of the Natural History Departments in the British Museum; Foreign Associate of the Academy of Sciences of the Institute of France; Sheen Lodge, East Sheen, Mortlake.

Elected

1883 PARKER, WILLIAM KITCHEN, F.R.S., Crowland, Trinity road, Upper Tooting.

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

1887 TURNER, SIR WILLIAM, LL.D., F.R.S., Professor of Anatomy in the University of Edinburgh.

1868 TYNDALL, JOHN, D.C.L., LL.D., F.R.S., Honorary Professor of Natural Philosophy in the Royal Institution; Corresponding Member of the Academies and Societies of Sciences of Göttingen, Haarlem, Geneva, &c.; Hind Head House, Shotter Mill, near Petersfield.

FOREIGN HONORARY FELLOWS.

(Limited to Twenty.)

Elected

1878 Baccelli, Guido, M.D., Professor of Medicine at Rome.

1883 Bigelow, Henry J., M.D., Professor of Surgery at Harvard University, and Surgeon to the Massachusetts General Hospital.

1887 Billings, John S., M.D., Surgeon U.S. Army; Librarian, Surgeon-General's Office, Washington.

1876 Billroth, Theodor, M.D., Professor of Surgery in the University of Vienna; 20, Alger Strasse, Vienna.

1883 Charcot, J. M., M.D., Physician to the Hôpital de la Salpêtrière, and Professor at the Faculty of Medicine of Paris; Member of the Academy of Medicine; Quai Malaquais 17, Paris.

1864 Donders, Franz Cornelius, M.D., LL.D., Professor of Physiology and Ophthalmology at the University of Utrecht.

1883 DuBois Reymond, Emil, M.D., Professor in Berlin; N. W. Neue Wilhelmstrasse 15, Berlin.

1887 Esmarch, Friedrich, M.D., Professor of Surgery in the University of Kiel.

1866 Hannover, Adolph, M.D., Professor at Copenhagen.

1873 von Helmholtz, Hermann Ludwig Ferdinand, Professor of Physics and Physiological Optics; Berlin.

1873 Hofmann, A. W., LL.D., Ph.D., Professor of Chemistry, Berlin.

1868 Kölliker, Albert, Professor of Anatomy in the University of Würzburg.

Elected

1856 VON LANGENBECK, BERNHARD, M.D., late Professor of Surgery in the University of Berlin ; Wiesbaden.

1868 LARREY, HIPPOLYTE BARON, Member of the Institute of France ; Inspector of the "Service de Santé Militaire," and Member of the "Conseil de Santé des Armées ;" Commander of the Legion of Honour, &c. ; Rue de Lille, 91, Paris.

1883 PASTEUR, LOUIS, LL.D., Member of the Institute of France (Academy of Sciences).

1878 SCANZONI, FRIEDREICH WILHELM VON, Royal Bavarian Privy Councillor, and Professor of Medicine in the University of Würzburg.

1856 VIRCHOW, RUDOLPH, M.D., LL.D., Professor of Pathological Anatomy in the University of Berlin ; Corresponding Member of the Academy of Sciences of the Institute of France ; 10, Schellingstrasse, Berlin.

1887 VON VOLKMANN, RICHARD, Professor in Halle.

LIST OF RESIDENT FELLOWS

ARRANGED ACCORDING TO

DATE OF ELECTION.

1833 Sir George Burrows, Bt., M.D., F.R.S.
Thomas A. Barker, M.D.
1835 Thomas A. Nelson, M.D.
1836 Alexander Shaw.
J. George French.
1837 Thomas Blizard Curling, F.R.S.
1838 Charles Hawkins.
Henry Spencer Smith.
1839 T. Graham Balfour, M.D., F.R.S.
Fred. Le Gros Clark, F.R.S.
James Dixon.
1840 Chas. J. B. Williams, M.D., F.R.S.
Samuel A. Lane.
Sir James Paget, Bt., F.R.S.
1841 Sir Henry A. Pitman, M.D.
Sir William Bowman, Bart., F.R.S.
Paul Jackson.
1842 Charles West, M.D.
John Simon, C.B., F.R.S.
John Erichsen, F.R.S.
Sir Oscar M. P. Clayton, C.B.
1843 Robert Greenhalgh, M.D.
Sir Prescott G. Hewett, Bt., F.R.S.
Henry Lee.
Luther Holden.
Edward Newton.
1844 Arthur Farre, M.D., F.R.S.
William Wegg, M.D.
1844 Thomas King Chambers, M.D.
Edwin Humby.
1845 Samuel Cartwright.
George D. Pollock.
Thomas Taylor.
Sir Edwin Saunders.
1845 William Oliver Chalk.
Edward U. Berry.
Benjamin Ridge, M.D.
1846 John A. Bostock.
Barnard Wight Holt.
Carsten Holthouse.
1847 W. H. O. Sankey, M.D.
George Johnson, M.D., F.R.S.
1848 Sir Edward H. Sieveking, M.D.
Edward Ballard, M.D.
William Wood, M.D.
Thomas Hawksley, M.D.
Edward John Tilt, M.D.
John Clarke, M.D.
John Gregory Forbes.
1849 Hugh J. Sanderson, M.D.
C. H. F. Routh, M.D.
Edmund L. Birkett, M.D.
George T. Fincham, M.D.
Sir William W. Gull, Bt., M.D., F.R.S.
1850 Richard Quain, M.D., F.R.S
George Roper, M.D.
1851 Sir Wm Jenner, Bt., M.D., F.R.S.
H. Haynes Walton.
John Birkett.
John A. Kingdon.
Peter Y. Gowlland.
John Marshall, F.R.S.
John Wood, F.R.S.
Bernard E. Brodhurst.
Robert J. Spitta, M.D.
1852 C. Bland Radcliffe, M.D.
Walter H. Walshe, M.D.
William Adams.

1852 Sir Henry Thompson.
1853 Robert Brudenell Carter.
1854 Alfred Baring Garrod, M.D., F.R.S.
Samuel O. Habershon, M.D.
Sir Thomas Spencer Wells, Bt.
1855 W. M. Graily Hewitt, M.D.
J. Burdon Sanderson, M.D., F.R.S.
J. Russell Reynolds, M.D., F.R.S.
Walter John Bryant, M.D.
1856 Charles J. Hare, M.D.
William Bird.
Jonathan Hutchinson, F.R.S.
Timothy Holmes.
Alonzo H. Stocker, M.D.
1857 William Overend Priestley, M.D.
George Harley, M.D., F.R.S.
Hermann Weber, M.D.
George Owen Rees, M.D., F.R.S.
John Whitaker Hulke, F.R.S.
John Morgan.
Henry Cooper Rose, M.D.
Henry Walter Kiallmark.
1858 Fred. George Reed, M.D.
John William Ogle, M.D.
1859 Wm. Howship Dickinson, M.D.
William Scovell Savory, F.R.S.
Edwin Thomas Truman.
Richard Barwell.
Edward Tegart.
Septimus William Sibley.
William E. Stewart.
1860 Sir Andrew Clark, Bt., M.D., F.R.S.
William Ogle, M.D.
Thomas Bryant.
John Couper.
Henry Howard Hayward.
1861 Robert Barnes, M.D.
William Spencer Watson.
William Henry Holman, M.B.
1862 James Andrew, M.D.
Lionel Smith Beale, M.B., F.R.S.
Thomas H. Tuke, M.D.
Edmund Symes Thompson, M.D.
Reginald Edward Thompson, M.D.
William Henry Brace, M.D.
George Cowell.
Robert Farquharson, M.D., M.P.
M. Berkeley Hill.
1863 Octavius Sturges, M.D.
John Langdon H. Down, M.D.
Samuel Wilks, M.D., F.R.S.
Samuel Fenwick, M.D.
Julius Althaus, M.D.
Sydney Ringer, M.D., F.R.S.
1863 Thomas Smith.
Arthur B. R. Myers.
Arthur E. Durham.
William Sedgwick.
1864 George Buchanan, M.D., F.R.S.
Charles Derby Waite, M.B.
John Harley M.D.
Walter John Coulson.
Thomas William Nunn.
1865 Charles Robert Drysdale, M.D.
James Edward Pollock, M.D.
William Cholmeley, M.D.
Reginald Southey, M.D.
George Fielding Blandford, M.D.
Sir Dyce Duckworth, M.D.
Frederick W. Pavy, M.D., F.R.S.
William Morrant Baker.
John Langton.
Frederick James Gant.
Alfred Willett.
Bowater John Vernon.
Alfred Cooper.
Christopher Heath.
1866 Thomas Fitz-Patrick, M.D.
Samuel Jones Gee, M.D.
Charles Theodore Williams, M.D.
Heywood Smith, M.D.
John Crockett Fish, M.D.
William Selby Church, M.D.
Edward John Waring, M.D.
1867 William Henry Day, M.D.
Achille Vintras, M.D.
Richard Douglas Powell, M.D.
F. Howard Marsh.
Henry Power.
Sir William MacCormac.
Thomas Pickering Pick.
Charles Arthur Aikin.
Samuel Hill, M.D.
1868 H. Charlton Bastian, M.D., F.R.S.
William Henry Broadbent, M.D.
Thomas Buzzard, M.D.
John Cavafy, M.D.
Walter Butler Cheadle, M.D.
John Cockle, M.D.
Sir Thos. Crawford, K.C.B., M.D.
T. Henry Green, M.D.
William Rhys Williams, M.D.
William Chapman Grigg, M.D.
John Croft.
George Eastes.
William Henry Freeman.
1869 Joseph Frank Payne, M.D.
Arthur E. Sansom, M.D.

1869 John Wickham Legg, M.D.
Charles Elam, M.D.
Thomas Laurence Read.
1870 William Wadham, M.D.
J. Warrington Haward.
Edgcombe Venning.
Clement Godson, M.D.
1871 William Cayley, M.D.
Charles Henry Ralfe, M.D.
Arthur Julius Pollock, M.D.
Thomas L. Brunton, M.D., F.R.S.
Henry Gawen Sutton, M.D.
J. Hughlings Jackson, M.D.,F.R.S.
Henry Sutherland, M.D.
George Vivian Poore, M.D.
Walter Rivington.
Marcus Beck.
Edward Bellamy.
William F. Butt.
Benjamin Duke.
1872 Gilbart Smith, M.D.
Thomas B. Christie, M.D.
George B. Brodie, M.D.
John Williams, M.D.
Sir J. Fayrer, M.D., F.R.S.
Charles S. Tomes, B.A., F.R.S.
Sir William Bartlett Dalby.
1873 William Miller Ord, M.D.
Frederick Taylor, M.D.
Norman Moore, M.D.
John Curnow, M.D.
William R. Gowers, M.D., F.R.S.
Sir Wm. Guyer Hunter, M.D., M.P.
Charles Creighton, M.D.
Jeremiah McCarthy.
Wm. Johnson Smith.
Robert William Parker.
Alex. O. McKellar.
Henry T. Butlin.
Charles Higgens.
William J. Walsham.
Edward Milner.
1874 Alfred Lewis Galabin, M.D.
George Thin, M.D.
Alfred B. Duffin, M.D.
James H. Aveling, M.D.
John Mitchell Bruce, M.D.
Henry Morris.
William Laidlaw Purves.
William Harrison Cripps.
Henry G. Howse.
Herbert William Page.
Frederic Durham.
John J. Merriman.
1874 William Robert Smith, M.D.
1875 Thomas T. Whipham, M.B.
Francis Charlewood Turner, M.D.
Robert Hunter Semple, M.D.
Thomas Crawford Hayes, M.D.
Charles Henry Carter, M.D
Fletcher Beach, M.B
Waren Tay.
Edmund J. Spitta.
1876 Thomas Barlow, M.D.
Wm. Lewis Dudley, M.D.
Albert J. Venn, M.D.
John Knowsley Thornton.
Charles Macnamara.
John N. C. Davies-Colley.
1877 Felix Semon, M.D.
Sidney Coupland, M.D.
Francis Warner, M.D.
William Ewart, M.D.
Alfred Pearce Gould.
Rickman J. Godlee.
Alban H. G. Doran.
George Ernest Herman, M.B.
Samuel West, M.D.
John Abercrombie, M.D.
J. Matthews Duncan, M.D., F.R.S.
Henry de Fonmartin, M.D.
George Allan Heron, M.D.
Joseph A. Ormerod, M.D.
P. Henry Pye-Smith, M.D., F.R.S.
Edward Nettleship.
William Henry Bennett.
William T. Whitmore.
1878 Sir Jas. Crichton Browne, M.D.
Fred. T. Roberts, M.D.
Sir Joseph Lister, Bart., F.R.S.
Clinton T. Dent.
John H. Morgan.
Walter Pye.
Gerald F. Yeo, M.D.
Donald W. Charles Hood, M.B.
Henry Gervis, M.D.
Richard Davy.
Hubert Foveaux Weiss.
Henry Thornton Wharton.
1879 Alfred Sangster, M.B.
Edward Woakes, M.D.
Armand de Watteville, M.D.
Malcolm A. Morris.
A. E. Cumberbatch.
Edmund Owen.
Arthur E. J. Barker.
Frederick Treves.
Horatio Donkin, M.B.

1879 Thomas John Maclagan, M.D.
David White Finlay, M.D.
Andrew Clark.
S. Hamilton Cartwright.
John H. Waters, M.D.
Francis Henry Champneys, M.B.
William Watson Cheyne.
William Munk, M.D.
George Henry Savage, M.D.
H. H. Clutton, M.A.
Frederic S. Eve.
E. Noble Smith.
William Henry Allchin, M.B
F. G. Dawtrey Drewitt, M.D.

1880 Robert Alex. Gibbons, M.D.
David Ferrier, M.D., F.R.S.
Vincent Dormer Harris, M.D.
Edmund Distin Maddick.
Jas. John MacWhirter Dunbar, M.B
James William Browne, M.B.
William Appleton Meredith, M.B.
Alexander Hughes Bennett, M.D.
Malcolm Macdonald McHardy.
Alexander Wm. Macfarlane, M.D.
A. Boyce Barrow.
William Murrell, M.D.
Bernard O'Connor, A.B., M.D.
Leslie Ogilvie, M.B.
George Lockwood Laycock, M.B.
George Ogilvie, M.B.
Charles Edward Beevor, M.D.
Thomas Colcott Fox, M.B.
George Henry Makins.

1881 Francis de Havilland Hall, M.D.
Robert Wharry, M.D.
Cecil Yates Biss, M.D.
Richard Clement Lucas.
Stephen Mackenzie, M.D.
James Anderson, M.D..
William Hale White, M.D.
Eustace Smith, M.D.
William Sinclair Thomson, M.D.
Percy Kidd, M.D.
Oswald A. Browne, M.A.
W. Bruce Clarke, M.B.
Dawson Williams, M.D.
George Lindsay Johnson, M.A., M.D.
Henry Edward Juler.
Jonathan F. C. H. Macready.
C. B. Lockwood.

1882 Philip J. Hensley, M.D.
Ernest Clarke, M.D.
John Barclay Scriven.
George Robertson Turner.
Howard Henry Tooth, M.D.
Herbert Isambard Owen, M.D.
Charles R. B. Keetley.
Joseph Mills.
A. T. Myers, M.D.
Anthony A. Bowlby.
Amand J. McC. Routh, M.D.
Seymour J. Sharkey, M.B.
William Lang.
Henry Radcliffe Crocker, M.D.
William Edward Steavenson, M.D.
Dan Astley Gresswell, M.B.

1883 Edwin Clifford Beale, M.A., M.B.
James Kingston Fowler, M.D.
James Frederic Goodhart, M.D.
John Charles Galton, M.A.
Walter Hamilton Acland Jacobson.
Edward Joshua Edwardes, M.D.
Walter H. Jessop, M.B.
Walter Edmunds, M.C.
Victor A. Horsley, F.R.S.
Dudley Wilmot Buxton, M.D.
Charles Douglas F. Phillips, M.D.
Angel Money, M.D.
John James Pringle, M.B.
Henry Roxburgh Fuller, M.D.
Wilmot Parker Herringham, M.B.
Augustus Waller, M.D.
William Pasteur, M.D.
Edward Albert Schäfer, F.R.S.
John Bland Sutton.
William Rose, M.B.
Storer Bennett.
Henry Carr Maudsley, M.B.
Robert Marcus Gunn, M.B.
James Dixon Bradshaw, M.B.

1884 George Newton Pitt, M.D.
Charles Stonham.
Stanley Boyd, M.B.
William Arbuthnot Lane, M.S.
Dennis Dallaway.
Thomas Whitehead Reid.
Arthur Marmaduke Sheild, M.B.
Frederic Bowreman Jessett.
Sidney Harris Cox Martin, M.B.
Wayland Charles Chaffey, M.B.
George Lawson.
Heneage Gibbes, M.D.
Thomas Wakley, Jun.
Robert James Lee, M.D.
F. Swinford Edwards.
Herbert Tyrrell Griffiths, M.D.
James Johnston, M.D.

1884 Arthur Oakes, M.D.
Edward Stewart, M.D.
William Duncan, M.D.
Charles Chinner Fuller.
Lovell Drage, M.B., M.S.
Jean Samuel Keser, M.D.
Charles Egerton Jennings, M.S.
George Richard Turner Phillips.
Bilton Pollard.

1885 Alexander Haig, M.B.
Wm. Dobinson Halliburton, M.D.
Theodore Dyke Acland, M.D.
Kenneth William Millican.
Frederick Walker Mott, M.D.
William Maunsell Collins, M.D.
James Berry.
John Cahill.
Francis Henry Hawkins, M.B.
John Poland.
James Greig Smith.
John Mackern, M.D.
George Gulliver, M.B.
Heinrich Port, M.D.
Edward Emanuel Klein, M.D., F.R.S.
R. Norris Wolfenden, M.D.
A. C. Butler-Smythe.
Arthur Gamgee, M.D., F.R.S.
Charles Alfred Ballance, M.S.
Walter Spencer Anderson Griffith, M.B.
John Edward Squire, M.D.

1885 John D. Malcolm, M.B., C.M.
Phineas S. Abraham.
Henry Willingham Gell, M.B.

1886 Robert Maguire, M.D.
Harrington Sainsbury, M.D.
Cuthbert Hilton Golding-Bird, M.S.
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Charles Leopold Hudson.
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.
Howard Lyon Smith.
William Radford Dakin, M.D.
Samuel Herbert Habershon, M.D.
Arthur Quarry Silcock.
Arthur Hamilton Nicholson Lewers, M.D.

1887 Walter George Spencer.
Thomas Outterson Wood, M.D.
Richard Hingston Fox, M.D.
Edgar William Willett, M.B.
Henry Lewis Jones, M.D.
Francis George Penrose, M.D.
Hugh Percy Dunn.
Frederic William Hewitt, M.D.
Harry Scott, M.B.
James Barry Ball, M.D.
Gilbert Richardson, M.D.
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Regulations relative to the publication of the 'Proceedings of the Society.'

That, as a general rule, the 'Proceedings' will be issued every two months, subject to variations dependent on the extent of matter to be printed.

That a Copy of the 'Proceedings' will be sent, postage free, to every Fellow of the Society resident in the United Kingdom.

That the 'Proceedings of the Society' may be obtained by non-members at the Society's House, 53, Berners Street, on pre-payment of an annual subscription of five shillings, which may be transmitted either by post-office order or in postage-stamps; —this will include the expense of conveyance by post to any part of the United Kingdom; to other places they will be sent, carriage free, through a bookseller, or by post, the receiver paying the foreign charges.

That a notice of every paper will appear in the 'Proceedings.' Authors will be at liberty, on sending their communications, to intimate to the Secretary whether they wish them to appear in the 'Proceedings' *only*, or in the 'Proceedings' and 'Transactions;' and in all cases they will be expected to furnish an Abstract of the communication.

The Abstracts of the papers read will be furnished to the Journals as heretofore.

ADVERTISEMENT.

THE Council of the Royal Medical and Chirurgical Society deems 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 its 'Transactions.'

ADDRESS

OF

GEORGE DAVID POLLOCK, F.R.C.S.,

PRESIDENT,

AT THE

ANNUAL MEETING, MARCH 1ST, 1887.

GENTLEMEN,—The year that has passed has removed by death no less than fifteen of our Fellows; and I fear that even the short account of their lives and work which I have to lay before you this evening, will sufficiently exhaust your patience, without reference on my part to other matters connected with our Society. I must, however, mention that the names of two of our late Fellows were omitted in the address of our President at the Annual General Meeting of last year through their deaths not having been publicly announced. It is therefore incumbent on me to speak of them on this occasion before I allude to those removed from our list during the past twelve months.

Dr. William Roden was elected a Fellow of this Society in 1843. He was a native of Knowle, in Warwickshire. At the early age of fourteen he was articled to a general practitioner at Kidderminster. At the expiration of his articles his medical education was continued, first at

Queen's College, Birmingham, and subsequently at University College, London.

He became a Licentiate of the Society of Apothecaries in 1836, and M.D. of St. Andrew's in 1844. In 1857 he became a Fellow of the Royal College of Surgeons of England, and in 1860 a Licentiate of the Royal College of Physicians of Edinburgh. He appears to have entered upon the active duties of his profession at a very early age, and was equally precocious in his devotion to public matters. He settled in practice in Kidderminster, and there remained in active professional work till his death, which took place on the 19th of October, 1884, in the seventieth year of his age.

From an early period of his connection with the town of Kidderminster, Dr. Roden took an active part in both its municipal and political affairs. He was a member of its Corporation, and on four occasions was elected Mayor. He was also a Justice of the Peace of the Borough. A few weeks prior to his death he presided at a meeting of the medical profession, held at Kidderminster, to consider the cause of the severe epidemic of enteric fever then raging in the town. He was a frequent contributor to the horticultural journals of the day, and was extremely devoted to all that related to horticultural pursuits.

Some three or four years prior to his death, Dr. Roden was disabled for some little time by an attack of paralysis.[1] From this he so far recovered as to be enabled to resume practice. It was on the 16th October, while making his morning round of visits, that he became conscious of a seizure similar to that from which he had previously suffered. This induced him to return home. He now found himself unable to partake of his mid-day meal; nor was he able to take the medicine he had at once prepared for himself. He spent the afternoon in moving from room to room, and in visiting his much-loved greenhouse and garden, as if taking mentally a final farewell of all his earthly surroundings. Towards the evening he retired to his bed; but on

[1] Letter from Dr. Jotham.

the arrival of his son soon after, he was unable to converse with him. Shortly afterwards he became insensible, and so continued till early the following day, when he died.

Throughout his active life he appeared to find sufficient relaxation in change of occupation; he was satisfied to vary his medical and public duties by indulgence in the pleasures of horticulture. His habits of scientific experiment and observation enabled him to make many suggestions of improvement in this, his favourite pursuit.

Mr. John Ware died at Clifton, on the 27th of February, 1885, in the ninetieth year of his age. He was born in London on the 15th of June, 1795. His father, Mr. James Ware, of Lawrence Lane, in the City, was a well-known oculist of his day, and a Fellow of the Royal Society. He was the founder of the School for the Indigent Blind. He was also one of the founders of the Society for the Relief of Widows and Orphans of Medical Men; and founder, by donation, in 1788, of the Fund, the interest of which is paid to the Directors for attendance at the meetings of the Society. He left £200 to the Society, of which he was President from the year 1809 to his death in 1815.

After a short University career at Cambridge, Mr. John Ware commenced his medical education at St. Bartholomew's Hospital, where he was a pupil of Mr. John Abernethy. He became a Member of the Royal College of Surgeons in 1817, and subsequently commenced to practise in New Bridge Street, in partnership with his brother, Mr. Martin Ware. He there devoted himself very successfully to ophthalmic practice. His professional career was, however, cut short by failing health, and in 1829 he was compelled to relinquish practice. He left London for Clifton, near Bristol, and there he resided till his death.

During his long residence at Clifton, Mr. Ware never actively resumed the practice of his profession. He chiefly devoted himself to works of philanthropy. He was ever interested in all that related to medical science, and to the last continued his membership of the different Medical Societies to which he had subscribed in earlier life.

He was one of the founders of the Clifton Dispensary, the Clifton Friendly Society, and the District Visiting Society; foremost in starting and supporting all the various national schools of Clifton and the Hot Wells, the Bristol Ragged School and Blind Asylum, also the Clergy Daughters' School, of which he was secretary for some time. He aided Bishop Monk in starting the Diocesan Training College, and Bishop Baring in his new Diocesan Society.

For the last fifteen years of his life failing strength and increasing deafness compelled him to withdraw entirely from all active work. But there was no failure in his mental powers. To the last he retained a lively interest in the works in which he could no longer take part. For many years he was confined to his room, where, with a cheerfulness and humility very touching to see, in peace and hope he waited with patience the end, which came within a few months of his ninetieth year.[1]

Great was the regret expressed by all who knew *John Cooper Forster* when told of his sudden and severe illness. While our late distinguished President was addressing us at the last Annual General Meeting poor Cooper Forster was fast sinking into his grave.

He had but a short time previously vacated the seat of President of the Royal College of Surgeons of England. He was well known to many Fellows of our Society, in which he had not infrequently held office, so that this sad news came as a shock, and spread much sorrow among us. He expired the following morning.

Cooper Forster, as he was familiarly known, had appeared amongst us but a few days previously, vigorous, active, and hearty; in such apparent health and strength that a long happy repose from professional labours, and continued enjoyment of a prolonged life in the midst of his family, promised to be the outcome of the uncertain future, if we had ventured to forecast what that future might bring; for

[1] Letters from Mr. James T. Ware, Dr. Henry Marshall, and Mr. Charles Hawkins.

Cooper Forster had retired with ample means from professional work.

John Cooper Forster was born in Lambeth on the 13th November, 1823. He was a son of Mr. John Forster, who practised for many years in that neighbourhood. He was educated at King's College School, and subsequently entered at Guy's Hospital. He passed through a distinguished career as a student; graduated as Bachelor of Medicine in 1847 at the University of London, and was placed second in honours, with gold medal, in Surgery, and also second in honours in Midwifery. He was one of the first among consulting surgeons who availed himself of the University of London Degree.

He became a Fellow of the Royal College of Surgeons of England in 1849. In 1855 he was appointed Assistant Surgeon to Guy's Hospital, and continued so to act till 1870, when he became Surgeon on the retirement of Mr. Hilton.

An opportunity soon offered itself to test his judgment and qualities as an operator, and he proved himself equal to the occasion.[1] The facts are of so much importance as bearing on the advance in this country of the operative surgery of the abdomen, that I make no apology for referring to them.

A patient came under the care of Dr. Habershon, in Guy's Hospital, with symptoms of obstruction of the œsophagus, which in the course of some few weeks became so complete that it was manifest the patient would be starved, unless some measure of relief could be afforded. Consequently Mr. Cooper Forster was consulted, and he at once decided to open the stomach. With regard to the operation and operator Dr. Habershon's words are the best commentary: "The skill with which the operation was executed, the scientific coolness and care displayed, and the manner in which it was brought to a successful termination, all who witnessed the operation can confirm."[2] Mr. Forster

[1] Letter from Dr. Wilks.

[2] 'Guy's Hospital Reports,' 3rd series, vol. iv, 1858.

had no precedent to guide him, yet he brought the operation to a satisfactory termination, though the patient died in the course of two days from exhaustion.

It is not desirable to discuss here the circumstances connected with Guy's Hospital which immediately preceded Mr. Cooper Forster's resignation of the office of Surgeon; but, as a result of what took place, he, in conjunction with Dr. Habershon, finding their position more than uncomfortable, tendered their resignations, and thus was severed the long official connection with Guy's Hospital, not, however, without an expression of regret on the part of the Staff and his numerous pupils. A handsome testimonial was also presented to him—satisfactory evidence of the opinions of those who knew him best, and were able to judge of his conduct.

As a Surgeon Cooper Forster was considered prompt and decided. Ever alive to test any suggested or supposed improvement in surgical practice, those he approved of he carried into effect with an enthusiasm quite characteristic of the man.

A paper on 'Acupressure,' published by him in the 'Guy's Hospital Reports,' relates how he journeyed to Aberdeen to see for himself this method of arresting hæmorrhage, practically illustrated by Dr. Pirrie and Dr. Keith; and how, on his return home, he at once adopted it. The following year, however, produced another article from his pen, on the comparative merits of acupressure and torsion of arteries, and he soon learnt to prefer the latter to the exclusion of every other method of arresting arterial bleeding after operations. When the use of antiseptics in the surgical treatment of wounds was first being advocated, Cooper Forster was one of the earliest at Guy's Hospital to test the worth of their employment. Some years past, when the temperance movement began to make an impression on the general public, Cooper Forster adopted the treatment to the strict letter with patients admitted to his wards. This for a time only; for as a result of his investigations he came to the conclusion that the judicious use of

stimulants was not only advantageous, but often necessary, under many surgical conditions.

Besides the publication of numerous papers in 'Guy's Hospital Reports,' two were contributed to our Society, and were printed in the 'Proceedings.'[1] Mr. Cooper Forster was also the author of a work on the 'Surgical Diseases of Children,' a work founded on his large experience at the Hospital for Children and Women in the Waterloo Road, a charity to which he was Surgeon for many years.

He lectured on Anatomy at Guy's Hospital School for some time, and on the death of Mr. Poland he succeeded to the Chair of Surgery, and resigned it on the retirement of Mr. Birkett from the Hospital.

Cooper Forster served this Society in many offices, and in all faithfully and well. He acted as Surgical Secretary in 1873, 74, and 75. As Treasurer from 1879 to 1884. He served on the Council in 1868-69; was Vice-President in 1877-78, and Referee for many years. He had long been a member of the Council of the College of Surgeons, for two years was one of the Vice-Presidents, and in 1884 was elected President of the College. When his period of office terminated in 1885, his seat in the Council also became vacant by lapse of time. Nor did he seek re-election. He now decided to relinquish practice, and his private means enabled him to look forward to the indulgence of those tastes which were congenial to his feelings, without the drawbacks of professional work.

He left England in January of last year for the South of France, exchanging the comforts of an English home for the doubtful atmosphere of Continental hotels. While at Nice he began to lose appetite, and as a feeling of weakness continued, he decided to return to London without delay. He travelled without rest. The exposure and the privations of such a journey must have seriously added to the gravity of his case. He arrived at his house in Upper Grosvenor Street on the evening of Wednesday, the 24th of February, in a state of extreme collapse. He was at once seen by his

[1] 'Proceedings,' vol. ii, p. 43, and vol. v, p. 319.

old friends Dr. Habershon and Dr. Wilks, but he daily became worse and died the following Tuesday, within a week of his return home, in the sixty-third year of his age.

Dr. Frederic Weber was born at Trieste in 1808. He died in the seventy-eighth year of his age, on the 10th of March, 1886.

He was a Fellow of the Royal College of Physicians of London, had served on the Council, and also occupied the office of Vice-President in 1865.

At the age of eighteen Frederic Weber went from Trieste to Heidelberg University, and from thence to Pavia, where he obtained his Doctor of Medicine degree in 1836. He subsequently travelled for two years to attend the hospitals of Parma, Leipzig, Berlin, and Paris, and lastly came to London.

To the latter place he came for the first time in 1838, intending eventually to settle in his native town. But this was not to be. The late Sir Thomas Watson, Robert Ferguson, and other physicians already in good practice in London, were greatly taken by his charm of manner and excellent qualities, and persuaded him to remain here to practise. He had made an especial study of the properties of the mineral waters and baths of Europe, while he had secured the friendship of a large number of medical men at the different watering places which he had visited in his travels on the Continent. In addition to all this good foundation for a successful start in practice, he had made many friends—attracted by his courteous manners and medical acumen—and consequently commenced practice in London with many professional advantages.

Dr. Weber worked for some time at the Middlesex Hospital, and was connected for many years as Physician with the St. George's and St. James's Dispensary in King Street. He took great interest in, and devoted much time to, the Harley Street Institute for Invalid Ladies, and was attached to it from its first establishment until within a few years of his death, a period of forty-six years' service. He also held at different times the appointment of Physician to several Foreign Embassies.

All who were acquainted with Dr. Frederic Weber learnt to appreciate his gentle nature. A more kindly, courteous character it would be difficult to meet with. I had the privilege of knowing him for many years and frequently met him in consultation. I cannot express myself too highly in his favour to do justice to my own estimate of his character. I have seldom met with one so diffident, more clear-headed, more charitable, and yet so firm of purpose. He was, too, a man of much deep feeling. His son, a pupil at St. George's while I was still on the active staff of the Hospital, was taken seriously ill and died soon after. I cannot forget the tenderness of expression and the gratitude with which he received my sympathy on that occasion; and I was especially struck by the truly patient and gentle submission with which he met his severe affliction. Dr. Weber possessed great musical talent. His voice would have enabled him to realise a fortune had his tastes led him in that direction, rather than to lean on the merits of his professional acquirements. He invariably declined the society of the most fashionable, when satisfied that he was only invited to make himself of use; Vanity Fair had no attractions for one of his worth.

Mr. John Fremlyn Streatfeild was the seventh son of the Rev. Thomas Streatfeild, of Charts Edge, Kent. He was born on the 14th of October, 1828, and died on the 18th of March, 1886, in the fifty-eighth year of his age.

The Streatfeilds, with whom the subject of this memoir was connected, are a very old Kentish family, and have been settled at Chiddingstone, near Penshurst, in the County of Kent, for more than 350 years. John Fremlyn Streatfeild himself took a great interest in the antiquities of his native county, and spared no pains to collect all that related in literature and other ways to the history and antiquities of that part of Kent. He was a good artist, especially in architectural subjects, and was at the time of his death preparing a work on the Antiquities of Kent, with illustrations by himself.[1]

[1] Letter from Mr. Marcus Beck.

Mr. Streatfeild was a pupil at the London Hospital. He became a Member of the Royal College of Surgeons in 1852, and a Fellow by examination in 1862.

Soon after the commencement of the war in the Crimea, he volunteered for service in the East. His services were accepted, and on his arrival there he was appointed Assistant Surgeon to the British Civil Hospital at Smyrna. There he remained until peace was proclaimed between England and Russia. His services were then no longer in requisition, and he returned home.

Soon after settling himself in practice he was elected Assistant Surgeon to the Royal London Ophthalmic Hospital in Moorfields, and Assistant Ophthalmic Surgeon to University College Hospital. He was Senior Surgeon to the former Institution, Professor of Clinical Ophthalmic Surgery in University College, and Surgeon to the Hospital at the time of his death.

Mr. Streatfeild had been in a somewhat delicate state of health for some few years previous to his death. In the bitter cold weather of last March he became the subject of an acute attack of pneumonia which proved fatal in the course of a very few days, at his house in Upper Brook Street.

He was the first Editor of the 'Ophthalmic Hospital Reports;' and many of the contributions to its numbers were the work of his pen. He was also author of the chapters on Ophthalmic Surgery in Erichsen's 'Science and Art of Surgery,' also of articles on ophthalmic subjects in 'Quain's Dictionary of Medicine.'[1]

As an operator on the eye, Mr. Streatfeild, in the opinion of those best capable of judging, appears to have excelled, with much-deserved distinction. He was dexterous in manipulation, accurate and collected, but rapid in execution.

The subject of adhesions of the iris had been for some time one of great interest to him, and he had given much time to it. To correct the evil results of the various forms of iritis, he had introduced an operation for liberating adhesions of that membrane to the capsule of the lens, and

[1] 'British Medical Journal,' March 27th, 1886.

of late years had conducted a series of experiments in the treatment of synechiæ by electricity, though I am informed without satisfactory results.

At University College Hospital he was very much liked by those who knew him intimately; but he was somewhat shy and retiring in disposition, and frequently failed to make that impression on strangers, and acquaintances generally, that a more genial, though less sincere, man would more frequently secure with advantage to himself and his well-doing.

Mr. Streatfeild was elected a Fellow of this Society in 1858. He was a Member of the Council in 1874-75, and served on the Library Committee in 1867-68.

Dr. John Parkin died at Brighton on the 18th of March, 1886. He was born on the 10th of May, 1801. His father was one of the principal officers of Her Majesty's Dockyards at Sheerness and Chatham. He was educated by Dr. Griffiths, one of the canons of Rochester, and studied under Abernethy at St. Bartholomew's Hospital. He was a Fellow of the Royal College of Physicians of Edinburgh, a Fellow of the Royal College of Surgeons of England, and a Corresponding Fellow of the Royal Academies of Medicine and Surgery of Madrid, Barcelona, and Cadiz.

Dr. Parkin commenced practice in London, in Dover Street, Piccadilly, and from all I can learn his practice was chiefly confined to the treatment of mental disorders. During his residence in London he had, first at Chelsea, and subsequently at Battersea, private accommodation for the reception and treatment of the insane. In the study of this special subject he is reported to have taken the deepest interest, though I do not discover that he has left behind him the results of his observations and experience. This is remarkable, as he was a most industrious and thoughtful writer on subjects he took up for examination or investigation.

Prior to his settling in London he had spent much time in different parts of Spain, that he might have the opportunity of investigating the subject of cholera.

When that disease broke out in the West India Islands, he, at great pecuniary sacrifice, at once severed his connection with York House, at Battersea, and went out at his own expense. He remained there for many months, during which time he was continuously engaged in attending and prescribing for all who came within his province. Such was his experience, and such the estimate of his services, that on a second visitation of cholera in the West Indies he was sent out by the Government as Her Majesty's Medical Inspector.

Dr. Parkin visited Calcutta after his return from the West Indies, for the special purpose of studying the phenomena of cholera, under the different circumstances of soil, climate, and race.

From his long observation of this disease under various conditions and in various climates, he became strongly impressed with the conviction that cholera and other epidemics are in some measure due to those atmospheric conditions which attend or follow volcanic disturbances. He was a strong opponent of all measures of quarantine, and although his statements may not be considered sufficient to prove the soundness of his views, still he has brought a large amount of evidence to bear upon this question; and much that is worthy the consideration of all interested in the subject.

Dr. Dill attended Dr. Parkin in his last illness at Brighton, and has furnished me with the following notes. He writes: "The intense earnestness of his character and indomitable pursuit after knowledge in the investigation of any subject which laid hold of his mind, was shown by the fact that all his life he laboured under the physical infirmity of spina bifida. My intercourse with him having been always professional, I had little opportunity of knowing his views on subjects generally, but I saw enough of him to reach his mind on the one subject which regulated his daily life: he was a humble and sincere Christian."

It is interesting to be able to record that one suffering from spina bifida should, notwithstanding the general ten-

dency of this affection to shorten life, have gone through so much exposure and active exertion, as did the subject of this memoir, and yet have lived to the ripe age of eighty-five. He died in the full possession of every faculty, and to the last took a most vivid interest in everything relating, not only to his own profession, but to every topic of the day.

Mr. Isidore Isaac Lyons was born in 1843, and after receiving his education at Dover, entered as a student at St. Bartholomew's Hospital in 1866, and was elected a Fellow of this Society in 1882.

Having become a member of the Royal College of Surgeons, he turned his attention to Dental Surgery, and studied this branch of the profession with great industry, both at St. Bartholomew's and the Dental Hospital.

Having long assisted, unofficially, in the dental department at St. Bartholomew's Hospital, and rendered services that were highly appreciated by the Staff and students, as well as by the patients, he was appointed Assistant Dental Surgeon to that Institution in 1879. He also became Dental Surgeon to the Evelina Hospital in Southwark.

Mr. Lyons was rapidly gaining practice when, in 1882, he was attacked by disease of the nervous system, and he died in May, 1886.

He was not only able and successful in his profession, but he had won the sincere regard of all who knew him by the modesty and kindness of his disposition, and the conscientious and devoted manner in which he performed his duties.[1]

He was buried in the Jewish Cemetery at Willesden.

Mr. William White Cooper died on the 1st of June, 1886. He was born on the 17th November, 1816, so that he had lived to the age of threescore years and ten.

I was well acquainted with him. As pupils we had worked together at the Ophthalmic Hospital in Moorfields, in the days of Tyrrell, Scott, and Dalrymple. As colleagues we were associated for a few years in connection

[1] Letter from Mr. Howard Marsh.

with a small ophthalmic institution, which he and some few friends established in Charlotte Street, Portland Place.

Mr. White Cooper came of an old Wiltshire family. His grandfather held the Rectory of Hambledon in Surrey, and was also Vicar of Yetminster. The eldest son of the Rector was the father of White Cooper, and being a man of independent means had no occasion to follow a profession. Mr. White Cooper came to London about the year 1834 to study medicine. He entered at St. Batholomew's Hospital, and at the same time became a house pupil of the late Mr. Stanley. While a pupil he gave much attention to comparative anatomy, a subject on which Richard Owen then lectured at St. Bartholomew's.

It was in consequence of White Cooper's love for comparative anatomy that an intimacy was established between him and Richard Owen, which lasted through life. In a letter received from the latter, he says, "My scientific relations with my dear and deeply regretted friend were as follows. In a course of lectures on comparative anatomy at Bartholomew's Hospital, Cooper, then a student, gained my prize for his diligent attendance, and the fulness and accuracy of his notes. These after revision I consented to his publishing, and they appeared in successive 'numbers,' making an octavo volume on the Anatomy and Physiology of the Invertebrate Animals, published in 1843. Mr. Cooper accompanied me to the 'meeting of the German Association of Science,' under the Presidency of Professor Oken, at Freiburg. On the journey there and back, we visited the notable museums of natural history and anatomy in both Germany and Holland; and he added much to my profiting thereby, by taking notes of every object, or series, that seemed worthy of attention. These notes have done good service in subsequent editions (1853 and 1855) of my 'Anatomy of Invertebrates,' and in my 'Anatomy of Vertebrates.'"[1]

Having secured the membership of the College of Surgeons in 1838, White Cooper visited Madeira before settling

[1] Letter from Sir Richard Owen.

in practice, not apparently on a matter of health, but rather one of pleasure, and change after his hospital life. On his return he published a guide for the use of those who wished to seek a more genial climate in winter than England can offer. Subsequently he travelled for a short time on the Continent, and then settled in London.

He continued to be connected with the Institution in Charlotte Street, already referred to, until elected Ophthalmic Surgeon to St. Mary's Hospital in 1851, when the duties of this appointment, and his increasing private work, obliged him to sever his connection with that which might be justly called the offspring of his own influence and exertions. In 1859 White Cooper was gazetted Surgeon Oculist to the Queen, an appointment which was due entirely to the high professional estimate in which he was held by the late Sir James Clark and Sir Benjamin Brodie. This appointment only terminated with his death.

As an ophthalmic surgeon he always appeared careful and discriminating in the examination of cases; judicious in treatment, and most kind and attentive to all under his care. As an operator he was skilful and neat, and simple in his management of cases after operation. He resigned his appointment to St. Mary's Hospital in 1862, and was then appointed Consulting Ophthalmic Surgeon to that charity.

White Cooper published in 1847 his 'Practical Remarks on Near Sight, Aged Sight, and Impaired Vision.' He also published a short account of "Conical Cornea," a work on 'Wounds and Injuries of the Eye,' and was also author of the article "Vision" in the 'Cyclopædia of Anatomy and Physiology.'

In May of last year, after a slight chill, he was attacked by acute inflammation of the lungs, which, notwithstanding the attention of Sir William Jenner and Dr. Wilson Fox, proved fatal in the course of four days. After many years of professional attendance on the Queen, Her Majesty intimated to Mr. White Cooper her intention of conferring upon him the honour of knighthood. He was naturally

extremely gratified at this evidence of a just appreciation of his services. But "man walketh in a vain shadow." The offer came too late. Before his name could be published in the Gazette, or he could appear before his Royal Mistress, to receive from Her Majesty the Badge of Honour, his body was carried to its last resting place. He died on the 1st of June. On the 2nd of June there appeared in the 'Court Circular,' by command of the Queen, the following kindly allusion to his death and to his long services to Her Majesty:—"The Queen has received this morning, with great regret, the news of the death of Mr. White Cooper, who attended Her Majesty for upwards of thirty years as oculist, and for whom Her Majesty had a sincere regard." Nor did Her Majesty intimate her regard and esteem for White Cooper in words only. While he was confined to his bed at his country residence, Her Majesty, on her return from Balmoral to Windsor in the autumn of 1885, drove to his house, sat by his bedside for some time, and evinced great feeling and interest in his illness and recovery. The world is familiar with Her Majesty's generous sympathy and kindness to those who have met with affliction or are in sorrow; but it is almost exceptional to have to record the visit of a Queen to the sick chamber of one of her trusted medical attendants, a visit intended to cheer the suffering invalid and to brighten his spirits in his convalescence. Such sympathy, like the quality of mercy, "is twice blessed, it blesseth him that gives and him that takes," and it did for White Cooper, to use his own words, "more good in recovery than any other thing."

White Cooper married, in 1845, the eldest daughter of Mr. Samuel Poyser, of Derby, who survives him. He was connected by marriage with the families of the first Sir Benjamin Brodie and the late Sir George Staunton.

Mr. Francis Mason was well known to a large number of the Fellows of this Society, and was most esteemed where most intimately known.

He was, at the time of his death, one of the surgeons of St. Thomas's Hospital, and a most active and industrious

member of the Surgical Staff. He was born in 1837; the youngest son of Mr. Nicholas Mason, of Wood Street, Cheapside. His early education was commenced at the Islington Preparatory School, at the time when Dr. Jackson, the late Bishop of London was head-master there. Subsequently he became a student at King's College Medical School and Hospital. In the wards of the latter he appears to have worked, and to have discharged the duties of dresser, with such interest and zeal, as to secure the notice of those under whom he acted. He also carried off the prize in Surgery of his year.

Sir William Fergusson early learnt to appreciate his excellent qualities, and constantly called on him for assistance at operations in private and for other professional purposes. To the day of Sir William's death, Francis Mason was his intimate and trusted friend.

Mr. Mason became a member of the Royal College of Surgeons in 1858. He officiated, as House Surgeon, at King's College Hospital in the years 1859–60. He became a Fellow of the Royal College of Surgeons by examination in 1862. His first public appointment was that of Surgeon to the St. Pancras and Northern Dispensary. He was then appointed Assistant Surgeon to King's College Hospital. But as promotion here appeared remote and uncertain, he took advantage of an opening in the Surgical Staff of Westminster Hospital, and passed over to it as Surgeon. He was also appointed Lecturer on Anatomy at the Medical School.

In 1871, when the new buildings of St. Thomas's Hospital were ready for the reception of patients, the Governing Body decided to reorganise and increase the Medical and Surgical Staff. It deservedly fell to the lot of Francis Mason to be selected as one of the Assistant Surgeons, whereupon he resigned the Westminster Hospital. He was also appointed one of the Lecturers on Anatomy. He succeeded Mr. Simon as Surgeon in 1876.[1]

Mr. Mason delivered the oration at the Medical Society

[1] 'Lancet,' June 12th, 1886.

in 1882, and the Lettsomian Lectures in 1878. The subject of these Lectures was "The Surgery of the Face," and included a very complete review of the whole question. These lectures were published in the 'Lancet,' and were subsequently re-issued in a separate form. Mr. Mason was also the author of a work, entitled, "Harelip and Cleft Palate," and although nothing in the shape of novelty was added to this already well-considered portion of surgery, still it must be said that all points connected with it were very clearly brought to the notice of the profession, and were illustrated by several plates of original cases.

Mr. Mason was a man of much genial character, generous, and hospitable—a character which, combined with the high musical talents with which he was gifted, his kind and cheerful disposition, and his friendly and social habits, not only endeared him to all who knew him, but secured him many sincere friends amongst his professional acquaintances and his patients.

Erysipelas of the upper part of the larynx, with diffuse inflammation of the soft tissues of the neck, attended by great constitutional disturbance, were the marked symptoms of his last and fatal illness. These conditions ran a rapid course, and notwithstanding the constant and careful attention of several colleagues and professional friends, he sank within four days of the commencement of his attack.

Mr. Hutchinson Royes Bell died on the 15th June, 1886. He was born at Sydney in 1843, but as his family returned to this country when he was quite young, his education was conducted partly in Jersey, and partly at King's College School in London. As he subsequently decided on Medicine as his future profession, he became, in 1860, a private pupil of Mr. Henry Smith, Surgeon to King's College Hospital. Shortly afterwards he entered the Medical School of King's College, where, it is reported, he was remarkable for his diligence, and especially for his devotion to the study of surgery.

After going through the course of lectures and attendance on hospital practice, he obtained his diploma as

member of the Royal College of Surgeons of England. In 1864 he was appointed House Surgeon to King's College Hospital; and subsequently held, consecutively, the offices of Surgical Registrar and Administrator of Anæsthetics. After a tour of study at Vienna and Paris, he returned to London, and set himself down to hospital work. For some short time he held the appointment of Prosector at the College of Surgeons; and in addition acted as Assistant Demonstrator of Anatomy in his own school at King's College. He became a Fellow of the College of Surgeons by examination in 1868, and was also an Honorary Fellow of King's College, London. When a vacancy occurred in the Surgical Staff of King's College Hospital, at no great distance of time subsequent to his having held the office of House Surgeon, Mr. Bell was elected Assistant Surgeon. In this position he worked diligently for eight years, at the expiration of which time he was appointed Surgeon to the hospital. With this appointment a certain number of beds were allotted to him for his especial use.

In the out-patients' department he was distinguished for the zeal and assiduity with which he endeavoured to impart instruction to the pupils of his class, and was consequently much appreciated by them. Notwithstanding a certain shyness and indecision of manner he became a great favourite with the students, and was justly looked upon as a thoroughly good teacher. Mr. Bell was very much interested at all times in all that related to plastic surgery. His operations for the remedy of various deformities, and the measures taken to overcome congenital defects, are reported to me to have been very successful.

His own contributions to surgical literature consisted of the Lettsomian Lectures, delivered to the Medical Society of London, on "Diseases of the Testis," and not long antecedent to his death he had completed an article on "Diseases and Injuries of the Male Genital Organs" for Ashhurst's 'Encyclopædia of Surgery.' This article evinces a great amount of careful research and a considerable general knowledge of the subjects therein considered.

Royes Bell was never a strong man. He appeared to many, who knew him but slightly, what may be denominated as "prematurely old," and although very energetic in the performance of duty he felt much the effects of work. He had gone to Folkestone for change in the month of June. On the morning of the 14th of that month he was seized with complete paralysis of one side, and became insensible. So sudden and severe was the attack of cerebral hæmorrhage that he did not recover consciousness. He expired the following day, as related, in the forty-fourth year of his age.

Hutchinson Royes Bell came of a worthy family, which had long been established near Leaconfield, in the East Riding of Yorkshire, and who formerly possessed a considerable estate in that neighbourhood. He retained himself a small freehold property in that county.

He is said to have been grave in manner and of a very modest and retiring disposition—so much so, that in his intercourse with strangers, or those not intimate with him, he failed to do himself justice; but by those who knew him intimately he was much appreciated as a most agreeable companion, and as one on whose judgment they could rely whenever it became necessary or desirable to solicit his opinion or advice.[1]

Dr. Walter Moxon died on the 21st of July, 1886, having but just completed his fiftieth year. His death was sudden, and not only its suddenness, but its coming at a comparatively early period of life, threw a sad gloom over those with whom he was associated at Guy's Hospital, as well as those by whom he was beloved and all who knew him well. His father was in the Civil Service of the Government, and became Chief Accountant of Inland Revenue. In the course of his duties he had to serve some time in Ireland, and it was during this time that Walter Moxon was born. This was on the 27th June, 1836, at Middleton, in the County of Cork. He entered as a student

[1] Letter from Mr. Henry Smith and 'British Medical Journal,' June 26th, 1886.

at Guy's Hospital in 1854. At his first M.B. examination at the University of London he took many prizes, and when he graduated as Bachelor of Medicine in 1859 he took honours in every subject. In 1864 he took his Doctor of Medicine degree.

Dr. Moxon was appointed one of the Assistant Demonstrators of Anatomy at Guy's Hospital Medical School in 1857, and in 1864 was elected Assistant Physician to the Hospital. He succeeded Dr. Wilks in the Chair of Pathology, and here he appears to have been in his element, showing great power of work and accuracy in detail, with extreme aptitude of conveying information in pithy and expressive language. His demonstrations are described as deliberate, elaborate, and emphatic. Disease was illustrated by finished drawings, many of which may be referred to in the 'Transactions of the Pathological Society.' Everything from his pen was clear, terse, and the result of honest work, honest conviction, and much original thought, "never slight or trivial in substance, and always brought up to the requirements of scientific accuracy." After being engaged for several years in teaching pathology, he was appointed to the Joint Lectureship of Medicine, and he continued his work at that post to the time of his death.

In 1868 Dr. Moxon became a Fellow of the Royal College of Physicians, and subsequently took an active part in its affairs. He had of late years resided at Highgate, but retained his consulting rooms in the City.

He had not been in good health for some time; his look was delicate, his temperament nervous and energetic. He had had several attacks of hæmaturia, and suffered on occasions from headache, and often from want of sleep. It is reported that on the day of his death he had suffered in this manner, with the addition of persistent vomiting and some hemiplegic weakness. And thus passed away one of the most honorable of men, one of the most careful, courteous, and conscientious of physicians, a warm-hearted and generous friend.

Dr. Moxon was elected a Fellow of this Society in 1868,

and was a Member of the Council at the time of his death. He contributed one paper to the 'Transactions.'

To 'Guy's Hospital Reports' his communications were numerous and important, and conspicuous among others, articles on Chronic Arteritis, Atheroma, Aneurysm and Insular Sclerosis may be mentioned. He also delivered the Croonian Lectures before the College of Physicians, which were illustrative of his accuracy of observation and minute investigation of all that related to the subject under consideration.

It is impossible for me to do justice to the character and work of Walter Moxon. I did not know much of him till of late, and knew nothing of his hospital work. I do not attempt therefore to speak of him from my own knowledge.

Walter Moxon was a man of much original mind, of great energy, of great capacity for work, of great honesty of purpose, seeking truth in every subject he took up, and working always for a good purpose.

As a lecturer he was clear, impressive, and entertaining; as a teacher, exact, accurate, and convincing. His powers were very considerable, whether as a worker, a teacher, or as a companion. He was a warm-hearted man, generous, and hospitable.

A true lover of nature he delighted in all that woods and pastures and running streams could offer for admiration or study. He not only cultivated in his garden every flower or plant that soil and climate would permit, but his daily care was to supply food for the wild birds which frequented his grounds, and which soon learnt to recognise the hand that fed them.

One of the most interesting papers from his pen was published in the 'Medico-Chirurgical Review,' under the title of "The Explanation of the Association of Aphasia with Right Hemiplegia." In this paper he put forth the view that the left hemisphere takes the initiative in right-handed persons, whilst the right acts in unison with it, and so under ordinary conditions the left hemisphere controls the organs of speech.

Dr. Moxon was not only a writer and thinker, and a man of action in his profession, but occasionally he contributed to the non-professional literature of the day.

He was a devoted and loving husband, a kind and affectionate father, and a true friend. Generous, warm-hearted, and benevolent, he was ever ready to lend a helping hand to those in distress. Many a poor student has been assisted through his difficulties and many an orphan has been temporarily supported by his open purse. The amount he gave away in private charity would be scarcely credited. Sensitive and tender-hearted he was deeply affected by the signs of pain, and he rebelled against anything akin to oppression.[1]

Mr. George Busk died at his house in Harley Street on the 10th of August, 1886, after a long and painful illness.

He was born in 1807 at St. Petersburgh. At an early age he gave promise of those tastes and of that aptitude for research which, developing with his years, gained for him the high position which he was destined to hold among the scientific workers of his time.

He was a student at St. Thomas's Hospital, but attended the practice of St. Bartholomew's Hospital for one year from October, 1828.

After being admitted a Member of the College Mr. Busk was appointed in 1832 Assistant Surgeon to the Seamen's Hospital Ship at Greenwich, when the "Grampus" was used for this purpose. Subsequently the "Dreadnought" was given over by the Government for the accommodation of a larger number of patients, and Mr. Busk became in time Surgeon to that floating hospital.

Mr. Busk held the appointment to the Hospital Ship for about twenty-five years; and it was this twenty-five years of hospital work which may be said to have embraced the strictly professional period of his life. In 1855 he resigned his appointment to the "Dreadnought," and at the same time came the determination to withdraw from the practice of his profession. He settled in London, but while refusing

[1] 'Lancet,' August 7th, 1886.

to be hampered by such calls on his time as the attendance on patients would necessitate, he wisely decided not to dissociate himself from all public professional occupation. He was nominated a Fellow of the College when the Fellowship was first established, and was elected a Member of the Council in 1863. In 1868, he was elected a Member of the Board of Examiners. Having held the office of Vice-President of the College for two years, he succeeded to that of President in 1871.

Mr. Busk was one of the Trustees of the Hunterian Museum, and for three years held the Hunterian Professorship in Comparative Anatomy. He was a Member of the Senate of the University of London; and for many years acted as Treasurer of the Royal Institution of Great Britain.

By refusing the demands of private practice he secured the leisure requisite for the cultivation of those studies which were congenial to one of his scientific turn of mind, and he warmly threw himself into biological work. About this time he became one of the Editors of the 'Microscopical Journal;' and the numerous communications which appeared from his pen in the pages of that periodical contributed largely to its popularity and success.

In 1856 his article "Polyzoa" was published in the 'English Cyclopædia.' In it we have an exhaustive account of the structure, and the first satisfactory attempt at a scientific arrangement of that group. Soon after this he undertook to draw up an illustrated descriptive Catalogue of the Polyzoa contained in the collection of the British Museum, and brought to bear on the descriptions and systematic arrrangement of the species those principles whose soundness he had already established. He also reported on the new species of Polyzoa and Hydroids collected during the voyage of the "Rattlesnake" in the Australian Seas. His report is published in the narrative of the voyage, and forms an important addition to our knowledge of these animals.

In company with Dr. Falconer he visited Gibraltar in

1864, for the purpose of investigating the ancient fauna which had been discovered in the caves of that region.

To attempt on this occasion to do anything like justice to the work and character of George Busk would be to trespass far too long on the time allotted to us this evening. I must not, however, omit a reference to his last labours. These were devoted to the preparation of a Report on the Polyzoa collected during the Voyage of the "Challenger." The first part of this work was completed in 1884, while the second and concluding portion he left behind in a condition nearly ready for the press. This under the judicious supervision of the proofs by his eldest daughter, through whose loving care during his last months of suffering he was enabled to carry on his work to completion, is now ready for publication.

Early elected a Fellow of the Royal Society he served on the Council and was four times nominated as a Vice-President. He was, more than once, President of the Microscopical Society, and of the Anthropological Society. He was for a time Zoological Secretary of the Linnean Society, and would have been made President had he not felt himself unequal to the duties of the office.

For his researches in Zoology and Comparative Anatomy, the Royal Society in 1871 awarded him the Royal Medal, while for his palæontological researches he received from the Geological Society the Lyell and Wollaston Medals. On the passing of the Cruelty to Animals Act, he was appointed inspector of the various medical schools and physiological laboratories registered under the Act, and the judgment and skill with which he performed the difficult duties of the office bear ample testimony to the wisdom of his appointment.[1]

George Busk was a genuine lover of nature. Generous and liberal to his fellow-workers, he was ever ready to place his rich store of material at their service, and never envious of whatever success might accrue to them as a result of his liberality.

[1] Letter from Dr. Allman.

When it was decided by Government to throw open to public competition the medical services of the Army, Navy, and India, it became requisite to appoint examiners to test the relative merits of the candidates, and Mr. Busk was selected for the office of Examiner in Physiology and Anatomy, an appointment which he held to within a few years of his death.

His latter days were days of much suffering, but he bore all with patient fortitude. His health broke down as the result of long-continued neuralgic suffering, consequent on an attack of herpes. But notwithstanding these and other physical troubles incident to advanced life, severe drawbacks to his ease and comfort, he was engaged to within a few days of his death on the work already alluded to.

I am permitted to add some few lines, forwarded to me by one who knew him long and intimately, and who was well capable of judging of his worth. He says that no one in our time has more remarkably combined the possession of complete skill in surgery with an accurate and far-extending knowledge of comparative anatomy and anthropology. He wrote but little on surgery, and it was only by report that I knew of his work on board the Dreadnought Hospital Ship. His operations were admirable, and his dexterity in the many difficult things that are classed in minor surgery was greater than his colleagues had ever seen. It was on board the Dreadnought that he studied the pathology of cholera, on which he and George Budd published their celebrated paper in our 'Transactions,'[1] and there he studied scurvy, and showed many interesting and novel facts concerning the changes in the effused blood. In his time the Dreadnought was a place of very active pathological study.

As an Examiner, he was careful, patient, and very just, though always, I think, with an inclination to mercy. Very few, I believe, have been members of the councils of as many learned and scientific societies as he was; and in all these he was singularly punctual, business-like, studious of

[1] Vol. xxi, p. 152.

the questions to be settled, and very fair and courteous in discussion. He was altogether one of the best men I have ever known; full of knowledge, but without one shade of personal vanity; laborious, but always as if with enjoyment of his work; a patient and industrious collector of facts, and of their illustrations in specimens and drawings, and very cautious in drawing his conclusions from them. He had a remarkable knowledge of languages. He could read nearly every one in which scientific works are published, and he continued to the last a habit of reading some of the best classics. And it is but right to tell that, with all this rare knowledge and laborious devotion to science, those who knew him in his home might often have thought that domestic love determined his whole course in life.[1] He died in his seventy-ninth year, beloved and regretted by all who knew him well.

Dr. Samuel Woodman died at Ramsgate on the 13th September, 1886, in his forty-third year. His death was the result of an attack of typhoid fever, which he was supposed to have contracted while absent from home on a short holiday.

He was born in Exeter, in which town his father practised as a surgeon. He was educated at the Exeter Grammar School, and subsequently entered as a pupil at St. Mary's Hospital in London in 1861. After having officiated as House Surgeon at this hospital for the usual time he settled at Ramsgate, in partnership with the late Dr. Webster of that town.

Dr. Woodman was a Fellow by examination of the Royal College of Surgeons of England, and a Doctor of Medicine of Durham University, the Doctorate given to practitioners of fifteen years' standing. He was in a large and a good practice. He was much liked, and was very active in other matters besides those connected with the practice of his profession. His bent was towards surgery; he was a good and bold operator, especially in cases of lithotrity. He was a man of genial manners and a

[1] Letter from Sir J. Paget.

thorough optimist, believing in his friends as well as in himself, and taking the cheery view of things in general, in which must also be included the prospects of his patients.[1] He was Consulting Surgeon to the Ramsgate and St. Lawrence Royal Dispensary, and Surgeon to the Board of Trade for Ramsgate Harbour.

He was the trusted adviser of the late Sir Moses Montefiore; and he had established for himself locally a considerable professional reputation. He was also a Justice of the Peace for the Cinque Ports' division of Kent.

One who knew him well has favoured me with the following account of him: "Dr. Woodman was a very able man, honest, straightforward, and manly, and at the same time modest and unselfish; altogether a most attractive character."[2]

As medical adviser to the late Sir Moses Montefiore he may be said to have gained a well-deserved credit. His constant attendance during the last few days of his patient's life was the admiration of both relatives and friends, and was undoubtedly a severe strain on himself. But during the recent illnesses of his well-known patient, when a large public daily looked for bulletins as to the condition of the good old man, it was Dr. Woodman's constant endeavour not to have any allusion made to his name in the hourly telegrams and reports which were most widely solicited and dispatched; a high sense of honour and a refined feeling in regard to professional propriety which might not be inappropriately taken as a guide under all similar circumstances.

About a month previous to his death Dr. Woodman proceeded to the Continent with the intention of visiting Switzerland, but at Nuremberg he was not well and at once returned to Ramsgate with the commencing symptoms of the attack of typhoid to which allusion has already been made. On reaching home the nature of his malady was at once detected by his brother-in-law, Mr. Raven, and under his

[1] Letter from Mr. Raven, Dr. Woodman's brother-in-law.

[2] Letter from Dr. Broadbent.

advice Dr. Woodman took to his bed. Hæmorrhage with symptoms of perforation of the bowels manifested themselves some two days before his death, and then fatal collapse.

"He met death," writes his friend, "not bravely merely but cheerfully, trying in this way to help his poor wife; his mind was clear to the last." And thus passed away one who was beloved and respected by all who knew him; one to whom life seemed to be bestowed for all that was right in work, kindliness, and friendship.

Mr. Frederick Chapman, of Old Friars, Richmond Green, was well-known as a sound practitioner, and highly esteemed by a large number of his professional brethren. He had practised in the town for many years, and his death occurred there on the 26th of October, 1886, after a painful illness, in his seventy-third year.

He was born at Richmond in 1813. He commenced his professional career by being apprenticed to Mr. Taylor, of Kingston; after this he entered the Medical School at University College, and I am informed on excellent authority that he was one of the most distinguished pupils of his year. He commenced practice in Richmond as a partner of the late Mr. James Smith, and continued in the active performance of professional duties for upwards of forty-five years.

He took a most active part in the foundation of the Richmond Hospital, of which he held the office of Treasurer and Chairman of Committee, besides taking his place on the Hospital Staff. The latter appointment he resigned in 1882, when he was nominated Consulting Surgeon to the Hospital. This hospital now contains thirty-six beds. Mr. Chapman had always taken the warmest interest in its well-doing, and worked hard for its maintenance; and it was largely due to his exertions that this charity has been placed in its present prominent and useful position.

A subscription has been started since Mr. Chapman's death in order to establish a memorial to him in connection with his work for the hospital, and a sum of about £260 has been subscribed for this object. It has been decided that the interest of this money shall be appropriated as a

"Convalescent and Surgical Aid Fund" to be called "The Chapman Fund."

Mr. Chapman was Medical Officer to the Richmond Union Workhouse for upwards of forty years, and the long time he occupied this office was ample evidence of the efficiency and punctuality with which he performed his duties.

Mr. Chapman was a member of an old and much respected family, long resident in Richmond. His father had been in practice there for many years as an architect and surveyor. He was himself one of a large family, which included a brother, who became the senior partner of that well-known and highly-respected firm of publishers, Messrs. Chapman and Hall.

Towards his latter days he suffered much from pain and local discomfort, which seemed to point to some malignant complication about the bladder or lower bowel. He gradually became emaciated and sank after great and prolonged suffering. He was buried at Petersham, amidst every mark of respect and regret. No one in the place was more loved and respected. His memory will long be cherished as an active and useful member of society, as well as gratefully treasured by all who can testify practically to the value of his professional tact and experience.[1]

Dr. Edwyn Andrew, of Shrewsbury, became a Fellow of this Society in 1862. He was a student at University College Medical School, and became a Member of the College of Surgeons and Licentiate of the Society of Apothecaries in 1855. He took his M.B. degree in 1856, and became M.D. London, in 1859. In 1866 he became a Master in Surgery and was Gold Medallist in Surgery of that year.

He was Resident Medical Officer and House Surgeon at University College Hospital, and also Physician's Assistant. For a short time he held the office of Assistant Surgeon to St. Pancras Infirmary. He was a Member of the Pathological and Ophthalmological Societies, President of the Shropshire and Mid-Wales Branch of the British Medical

[1] Letter from his son, Mr. H. F. Chapman.

Association, and Honorary Local Secretary and Treasurer to the Royal Medical Benevolent College.

Dr. Andrew settled in Shrewsbury, and soon appears to have devoted himself especially to the treatment of diseases of the eye. He became attached to the institution known as the Shropshire and North Wales Eye and Throat Hospital, a small and very inadequate building, when he was first appointed Surgeon to it, but under his exertions, and with the aid of others, he lived to see a new hospital erected, and completed in 1881, replete with every comfort and with ample accommodation within its walls for the reception of patients.

Dr. Andrew died in January of this year. He had been confined to his bed for about nine weeks, his illness arising in the first place from a cold, but ultimately developing into a serious attack; and although he rallied at intervals very slight hopes of his recovery appear to have been entertained. He contributed to the journals occasionally, and among his writings may be found some "Observations on Extirpation of the Lachrymal Gland in Obstruction of the Nasal Duct," "A new method of Extraction of Cataract," and "the Use of the Cautery in Eye Diseases."

Dr. William Daubeny was elected a Fellow of this Society in 1848. He was born on the 18th of June, 1820, at Wraxhall Lodge, Wiltshire, and was educated at Rugby. He subsequently studied at St. Thomas's Hospital, became a Member of the College of Surgeons in 1843, and a Doctor of Medicine of St. Andrew in 1845. He commenced practice in London, in partnership with the late Mr. Pope, of Manchester Square, but the differences of opinion and character which mark men did not apparently blend harmoniously in this professional alliance. Dr. Daubeny found it desirable for his future prospects and general comfort that he should be able to act independently in his practice, and so severed this professional connection. He then practised for some short time in the neighbourhood of Portman Square, and soon secured a large and good class of patients, with all of whom he was ever a personal

favourite, and with most of whom he became the trusted and honoured friend.

In the midst of this fair promise of a successful future Dr. Daubeny became the subject of an attack of acute rheumatism, the effects of which he apparently was never able to shake off. His health broke down, and he was crippled to such an extent that he was compelled to give up practice in London, in order to seek a climate more congenial to his constitutional condition than any place in England is able to afford. After travelling abroad for some two years, he decided to settle at San Remo. This was twenty-five years ago, when San Remo was but little known to the English public, and when it could have afforded but little professional employment to an English physician. But twenty-five years have worked a great change in many of what were then but small villages on the Mediterranean coast of France and Italy, and San Remo has in no small measure participated in the improvement and growth. To some extent this change may justly be attributed to Dr. Daubeny's advocacy of its attractions, and his personal merits and good qualities as a physician. He might justly be looked upon as one of the founders of this popular and desirable winter residence for those who seek protection from the cold winds and frost, of more northern latitudes; only one English resident having preceded him in making San Remo a home.

Dr. Daubeny was an extremely shrewd and clever practitioner; a keen obsever, with great knowledge of human nature; very witty and clever in repartee; a most genial companion and firm friend.

He was greatly beloved by all who knew him, and highly esteemed by all the residents of San Remo, English and foreign. He was a most straightforward, honorable member of our profession, a man respected by patients and friends.[1]

Dr. Daubeny died at San Remo on the 26th of January of this year, after an illness of some thirty days, due to

[1] Letters from Dr. Freeman, Dr. Kay Shuttleworth, Dr. Turner, and Mr. Le Gros Clark.

congested lungs and cardiac complications. He was assiduously attended by his friend Dr. Freeman, in consultation by Dr. Kay Shuttleworth. He died in his sixty-seventh year.

Mr. George Gaskoin died on the 5th of February, in the seventieth year of his age. He commenced his professional education as hospital apprentice at St. George's Hospital, and after passing through the usual period of studentship, served as House Surgeon in 1839. It was during this time that I acted under him as a dresser to the late Mr. Robert Keate, then Senior Surgeon to St. George's Hospital and Serjeant Surgeon to the Queen.

Mr. Gaskoin became a Member of the College of Surgeons in 1838, and a Licentiate of the Society of Apothecaries in 1841.

He settled in London, and his first intention was to occupy himself with general practice, but subsequently he confined himself in some measure to the treatment of diseases of the skin. In this department of practice his uncle, Mr. John Samuel Gaskoin, had obtained a well-deserved reputation, and for forty years was looked upon as an excellent authority on diseases of the skin, and might truly be said to have been a most successful and popular practitioner. Mr. George Gaskoin became connected as Surgeon with the British Hospital for Diseases of the Skin, an appointment which he held to within a short time of his death. He also held the appointment of Surgeon to "The Artists' Benevolent Fund."[1]

He gained the Wyatt Edgell Prize of £200 for his essay on 'The Range of Hereditary Tendencies in Health and Disease.' He also contributed some interesting papers to the medical journals at different periods on the subjects of cholera, hereditary tendencies in health and disease, and the history of syphilis. He published a translation of the medical writings of Francisco de Villalobos in 1870, and so highly was his work in this translation estimated, both in Spain and Portugal, that he was created a Knight Com-

[1] Letter from Mr. Charles Hawkins.

mander of the Royal Military Order of Christ of Portugal, and Knight Commander of the Order of Isabella la Catholica of Spain.

He was a man of much general information, was well read, and of good conversational powers; of a retiring disposition, but a courteous gentleman, and professionally most honorable and straightforward. He might truly be said to have been a man without guile, so simple, true, and honest was he in all his views and actions in life.

Mr. George Gaskoin was elected a Fellow of this Society in 1851. He communicated two papers to our 'Transactions.'

He was never married, and had retired from practice a short period prior to his death. About two years ago symptoms of serious cerebral disease manifested themselves, obviously of an incurable character, and although intelligence remained intact almost to the last days of his life, he gradually sank, and died last month at the residence of his brother in Wales.

Gentlemen, in conclusion, allow me before I sit down to offer my best thanks to the Vice-Presidents and other members of the Council for the kindness and support I have received from them during the past year of office. My thanks are also greatly due to my friends on my right and my left, Dr. Cheadle and Mr. Howard Marsh, for the ease with which my work has been accomplished, both in Council and in this chair. I beg also to express here my best thanks to our active Resident Librarian, Mr. Bailey, for the assistance he has afforded me in collecting information about those who have been taken from us; and, lastly, to you, gentlemen, I beg to offer my sincere thanks for the kindness with which you have received me on this and all occasions.

A CASE

OF

EXTRA-UTERINE GESTATION.

BY

HENRY GERVIS, M.D.

OBSTETRIC PHYSICIAN TO ST. THOMAS'S HOSPITAL.

Received March 26th—Read October 26th, 1886.

THE following case is, I think, of sufficient rarity to merit a permanent record. For the notes, as well as for much care given to the case during its progress, I am indebted to Mr. C. Green, M.B., who was at that time acting as resident accoucheur at St. Thomas's Hospital.

Previous to her admission to St. Thomas's, I had seen the patient at her own residence by request of her husband's employer, and had been told it was a case of cancer of the uterus. My examination, however, led me to believe that the condition of the pelvic contents was not malignant but inflammatory, and probably connected with some error of gestation, and I advised her removal to the hospital. She was admitted on November 19th, 1885. Her family history was bad, but her own previous history good. She was thirty-five years of age; was married at thirty, and had had two children, both born at the eighth month, the youngest in December, 1883. She had had one early miscarriage between the two confinements. The catamenia, when not pregnant, had always been regular and normal

and her general health good. The history of her present illness was as follows. She ceased to menstruate on the 9th of May and soon after considered herself pregnant, and her pregnancy proceeded without incident, except that she thought she was larger than at corresponding periods in former pregnancies, until the beginning of July, when she had an attack of what she was told was "inflammation" in the right lower abdomen, and since then she had never been free from pain. In the first week in September, after an unusually severe attack of pain in the right iliac region, she suddenly passed *per vaginam* more than a pint of what she described as a thick greenish fluid, and for a fortnight subsequently she was much more comfortable. Then the pain again became severe, and was shortly followed by a fresh discharge of a similar nature, but more offensive in character; and this sequence of events, viz. pain followed by discharge, recurred from time to time up to her admission. On one occasion there came away in the discharge what she thought was a piece of "skin," but her nurse told her it was a piece of afterbirth. There had been no uterine hæmorrhage at any time, but on two or three occasions a discharge which resembled coffee-grounds. The enlargement of the abdomen, which had progressively increased until the first appearance of the discharge in September, had since then steadily diminished.

On her admission she was somewhat emaciated and very weak. Pulse 114, temp. 100·4°. In the hypogastric region, and extending towards the right of the median line, was an ill-defined swelling, tender on pressure. On examination *per vaginam* the lower segment of the uterus appeared expanded and fixed; the external os was patulous, and admitted the finger as far as the inner os, which was tightly closed. The sound, however, passed easily, and apparently in the normal axis to a distance of about three inches. Bimanual examination was difficult both on account of the condition of the vagina, to be described, and the fixation of the pelvic contents, and added but little to the information gained by abdominal palpation. There was a large

quantity of offensive discharge issuing from the vagina, and owing to its irritating character the labia and nates were much excoriated and inflamed. The discharge was yellow in colour, with a slightly fæcal odour, and resembled in character the contents of the small intestine. Examined microscopically it showed broken-down bits of muscular fibre, and broken-down vegetable tissue, and differed entirely in character from the contents of the rectum, which contained ordinary lumpy fæces. Examination with the speculum was difficult on account of the vaginitis present, but it showed that this yellow discharge issued through the os uteri, and that there was no recto-vaginal fistula present. The bladder contained about twelve ounces of high-coloured urine, which was free from albumen.

As a preliminary to further treatment, I directed the vagina to be regularly washed out with warm Condy and water, and kept plugged with pledgets of iodoform wool, and the external eczematous parts to be dressed with iodoform and vaseline. As a medicine she had quinine and such sedatives as appeared necessary. Under this treatment she became much more comfortable and the inflamed condition of the external genitals subsided.

On December 14th she passed *per vaginam* the ossified shaft of a fœtal tibia, which when compared with specimens in the museum appeared to correspond with that of a fœtus between five and six months of uterine age. I then decided upon dilating the cervix, and exploring the uterine cavity; and on December 18th this was effected, the patient being anæsthetized. The cervix was dilated with Hegar's dilators up to No. 20. The cervical canal was unusually long; on passing the finger through the inner os it reached the body of a small fœtus in a decomposing condition, lying transversely as it were in the uterine cavity, with the head towards the left side. I made several efforts to extract it, firstly by attempts at version with traction, but fragments only of the limbs came away, including one entire femur with remnants of soft parts attached, the main portion of the body appearing to be tightly fixed in

its position. I then tried traction with ovum forceps, but with no greater success. Some hæmorrhage, but not much, occurred during the operation; but the patient's pulse beginning to flag, it was decided to defer further attempts at extraction, and after irrigation of the cervix and vagina with warm antiseptic solution she was removed to bed. In the evening symptoms of collapse set in, and she died in the course of the following day.

Autopsy twenty-four hours after death. Body somewhat emaciated. Some old adhesions in the right pleura. Heart and pericardium normal. The peritoneal cavity contained some dark brown material looking like intestinal contents but having no distinctly fæcal odour. The intestines were matted together by fairly firm but not fibrous adhesions, and there were also several patches of recent inflammatory lymph both visceral and parietal. The peritoneum was much discoloured. The great omentum and the intestines were adherent to the brim of the pelvis. On lifting up this adherent mass the partially disintegrated remains of a fœtus were seen lying transversely in the posterior half of the pelvis, the parts surrounding it being of a dark slate colour, and almost sloughy in appearance. In the pouch with the fœtus were several grape stones. The fœtus itself was more or less decomposed. Its relation to the uterus was not at first clear, but on removing the pelvic contents and carefully examining them, it was found that almost the whole of the posterior wall of the uterus had sloughed away, from close to the inner os below to within half an inch of the fundus above. The left Fallopian tube and ovary were fairly healthy. The right tube and ovary could not be traced. No trace of placenta was found. The small intestine was ulcerated in several places, and two perforations were found in it leading into the cyst-like pouch in which the fœtus was contained.

Remarks.—My impression of this case at the first was, as I have indicated at the beginning of the history just given, that it was connected with some error of gestation, and neither malignant on the one hand nor simply inflammatory

on the other. I thought it probably a case where the gestation was extra-uterine and the fœtus dead, and this view was strengthened, after her admission to the hospital, by the observation made as to the intestinal character of the uterine discharge, and the passage of the sound to little more than its normal length. The intestinal character of the discharge might, I thought, be accounted for by some entero-uterine fistula resulting from one or other of the abscesses which had successively occurred in connection with the progress of the case. But while we were waiting for the eczematous condition of the genital surfaces to improve, and also, if possible, her general health, the fœtal tibia was passed in the discharges, and the idea then occured to me that it might possibly be a case of missed abortion. Anyway the indication now seemed clear to dilate the cervix and explore the uterine interior. When this was done the second view appeared to be verified, for on dilating the inner os the finger at once came upon the body of the fœtus, and it was not until the post-mortem examination that we ascertained that the fœtus was essentially *extra*-uterine in position, and occupying a sac of which the anterior wall was the anterior wall of the uterus, and the remaining walls formed by the inflammatory adhesions already described, and with the posterior wall of the uterus destroyed by ulceration. The age of the fœtus, as determined by comparison with others of known age, seemed to be close upon six months, which does not entirely agree with the time the patient believed herself pregnant; but it is not uncommon in cases of extra-uterine gestation for a quasi menstrual discharge to appear once or twice after conception has occurred, and it is quite probable that the pregnancy began earlier than May. Had the case been diagnosed at its commencement, or had the interior of the uterus been explored when the discharge first appeared, it is of course possible that abdominal section might have been successfully adopted; but at the time when she was admitted into the hospital I do not think any treatment could have saved her. So far as I have ascertained by reference to authori-

ties and specimens, there is no case on record such as this of an extra-uterine gestation occupying Douglas's pouch communicating both with the uterine cavity and the small intestines.

(For report of the discussion on this paper, see 'Proceedings of the Royal Medical and Chirurgical Society,' New Series, vol. ii, p. 137.)

THREE HUNDRED ADDITIONAL

CASES OF COMPLETE OVARIOTOMY

AND

TWENTY CASES OF EXPLORATORY OPERATION.

BY

J. KNOWSLEY THORNTON, M.B., C.M.,

SURGEON TO THE SAMARITAN FREE HOSPITAL.

Received April 13th—Read October 26th, 1886.

On January 11th, 1881, I had the honour to bring before the Royal Medical and Chirurgical Society[1] a record of 150 cases of complete ovariotomy, with twenty-two other cases of abdominal section. I had previously published thirty-eight complete ovariotomies, twenty-five of them having been presented to this Society in a paper read on May 8th, 1877.[2]

I now present a farther table of 300 cases of complete ovariotomy, with twenty miscellaneous cases.

In my previous tables I followed closely the plan of those of Sir Spencer Wells, but in the present series I have thought it well to introduce, in addition to the details in former tables, two columns showing the highest temperatures and quickest pulses recorded after operation, with a note of any special treatment required. In order to do this without making the tables too cumbersome, I have omitted the

[1] 'Med. Chir. Trans.,' vol. lxiv, p. 141.

[2] Ib., vol. lx, p. 297.

column which contained the names of the private medical attendants of the patients. With such full details as are given in the present tables, anyone wishing to identify a case can have no difficulty in doing so, without his name being attached, and the temperatures, pulses, and special treatment are of great practical value, as showing not merely the result in each case, but the amount of disturbance following the operation, and the treatment by which it was met.

As the greater number of the cases required no special treatment, I will briefly describe the general method followed in the absence of special complications.

Each patient is nursed by a single nurse, who takes entire charge night and day. The patient is placed upon her back in bed with the head and shoulders well supported by an inclined plane of pillows, and the knees raised and supported by a firm pillow placed under them. This position is maintained for a fortnight, *i.e.* until the patient gets up on to the couch; after this she is allowed to lie on her side, or in any position which she prefers. The temperature is taken in the vagina for the first twenty-four hours at frequent intervals, afterwards at gradually lengthened intervals in the axilla; the pulse is also noted from time to time by the nurse, and by myself at the visits. As soon as the patient is sufficiently recovered from the chloroform to feel pain, twenty minims of laudanum, in an ounce of tepid water, are injected into the rectum. If two or three such doses are necessary they are given at short intervals. Clear beef-tea, made without salt, is injected into the rectum, three ounces every three hours, till sufficient nourishment can be taken by the mouth; and to every other of these injections twenty minims of laudanum are added for the first few days; quinine, wine, or other medicines being given in the same way if required. A little cracked ice, sips of warm water, or of plain soda-water, according to the fancy of the patient, are allowed during the first twenty-four hours, or until any sickness which may be present has ceased, and then milk and soda-

water, small quantities of bread and milk, or of clear beef-tea, at frequent intervals, till the patient can take more solid food, such as boiled sole, chicken, &c.

The abdominal wound, when the drainage-tube is not used, is first dressed at the end of the week, and the sutures removed; a small narrow dressing of dry carbolic gauze is then applied, and the abdomen, as immediately after the operation, is again well supported by adhesive straps. If drainage be necessary the glass tube is cleared under the spray every twelve hours, till it is removed, the india-rubber sheet and sponge dressing introduced by Keith being used under the gauze dressing.

In my last paper I gave tables and particulars of a number of other abdominal sections, and I give in the present paper a detailed table of twenty cases of exploratory operations in which the diagnosis was "probably ovarian tumour;" but the number of abdominal operations has so increased that it would only cause confusion if one attempted to deal in the same paper with all the different cases, and hysterectomy, oöphorectomy in its various applications, nephrectomy, hepatotomy, splenectomy, &c., will form the subject of separate papers, to be brought before this or some of the sister societies.

The tables contain such a mass of facts that it will not be either necessary or possible to refer to all or most of them, but I shall follow, as nearly as I think advisable, the lines of my previous papers, and dwell especially upon the fatal cases, as we learn, or should learn, more from our failures than from our successes; and it is too much the fashion to make it appear that failures are so rare that they are not worth special record.

All these operations have been performed with full antiseptic precautions, and I am not prepared to alter my method; nor have I as yet seen any full and trustworthy evidence that I should benefit my patients by so doing. I cannot claim to have banished septic fatalities in the whole series, and probably no system will ever quite attain such perfection. How much care will do in this direction

is, however, shown by the fact that my work at the Samaritan Hospital has yielded one consecutive series of one hundred complete ovariotomies without a death from septicæmia, and with the more healthy surroundings of the new hospital, which we hope soon to build, such pleasant experience will doubtless recur.

There are twenty-one deaths in the 300 ovariotomies, or a mortality of 7 per cent., not quite what I had hoped to reach, but still a result of which I shall not pretend to be ashamed, especially when I remember that in a similar series of 300 ovariotomies published in the 'Transactions' of this Society by Sir Spencer Wells in 1877, the mortality was 25·66 per cent., and that in 1881, when the 'Transactions' contain tables of 512 ovariotomies performed by Sir Spencer Wells, Dr. Bantock and myself, the average mortality of the whole series is still 13·28 per cent., or nearly double that of my present series.

The most recent publication of any large number of cases is that of Mr. Lawson Tait.[1] He gives 405 ovariotomies with thirty-three deaths, a mortality of 8·15 per cent.

In my 150 cases published in 1881, and included in the 512 cases just alluded to, the mortality was 10 per cent.

In the first 150 of the present series, it is 7·33 per cent., and in the second 150 it is 6·66 per cent.

The decrease in the mortality becomes smaller with each 150 cases, and it must be so until the minimum is reached. I only trust that in each future 150 cases a fractional improvement may still be found.

In 106 of the 300 cases there were no adhesions, and in eighteen others the adhesions were only slight; but in the large proportion of 176 cases the adhesions were extensive, and in these all the mortality occurs.

Forty-two were operated upon in private houses, forty-eight in nursing homes, and the remaining 210 in the Samaritan Hospital.

In the former series, all the cases operated upon at their

[1] 'Medical Press and Circular,' vol. i, 1885, pp. 65, 89.

own homes, or in nursing homes, recovered, but in this series the mortality is relatively high in the cases operated upon at homes, for reasons which will become apparent when I deal with the details of the fatal cases. There are certain advantages and certain disadvantages in operating in private houses, and these, in a long series of cases, seem nearly to balance the more obvious disadvantages of operating in the hospital. In the whole of my practice the mortality is very nearly the same in private houses, private nursing homes, and hospitals.

A glance down the columns headed respectively "age" and "condition of the patient," will show that these have little or no influence on the mortality, but I retain them in the tables because of their other very obvious interest. An additional column, which I did not mention when speaking of the alterations, is that which contains the record of the side upon which the tumour grew. When both ovaries were removed the letter L. or R. in brackets denotes the side which appeared to be first diseased.

In my previous paper the removal of both ovaries seemed a far more fatal procedure than the removal of one, but here again we see how necessary it is to have large numbers of cases to deal with before attempting to settle questions of this kind, for the mortality in the present cases is nearly equal.

The weight of the tumour does not, in the majority of cases, influence the result, but with the very large tumours the mortality is high, chiefly, I believe, because they have been long carried by the patient; and as a consequence important organs, especially the kidneys, have suffered, and are less able to perform their functions after operation.

In examining the column which records the highest point of reactionary temperature it must be remembered that in nearly all the cases this is a mere single record, so that, if seen on a chart, it would represent the highest peak of the mountain range, the lines sloping away gradually on each side of it; not forming, as in some other diseases, a succession of peaks on succeeding evenings, all

of, or about, the same height. It must also be remembered that the temperatures on the evening of operation are taken in the vagina, and that at this time the vaginal temperature is nearly always, in these cases, a degree or more above the axillary temperature. This must be borne in mind, in comparing the highest temperatures reached on the operation evening with those on succeeding evenings. Allowing for this, I find that the highest point is reached on the evening of the day after operation in 129 cases, on the operation evening in 93 cases; on the evening of the second day in 35 cases; on the evening of the third day in 9 cases; on the evenings of the fourth and fifth days in 4 cases each, and in single cases it is not reached till the sixth, seventh, eighth, thirteenth, and fifteenth evenings. Thus, excluding two or three exceptional cases, all those that recover have established convalescence within the week, and considerably over two thirds within forty-eight hours of the operation. While pointing to these remarkable facts, I must add the caution, that it is unwise to let the patient or the nurse think that convalescence is established; liberties taken under such circumstances are the chief cause of the few exceptional delays named above.

Wunderlich, in his standard work on 'Medical Thermometry,' has given the following as the limits of the states of health, fever, hyperpyrexia, &c.

B. Normal or almost normal temperatures.

a. Subnormal, 96·8° to 97·7°.

b. Really normal, 97·88° to 99·12°.

c. Sub-febrile, 99·5° to 100·4°.

One hundred and thirty-six of the two hundred and seventy-nine recoveries fall within these limits, and recover without fever.

C. Febrile temperatures.

a. Slight febrile action, 100·4° to 101·12°.

b. Moderate degree of fever, 101·12° to 103·1°.

c. Considerable fever, 103·1° to 104°.

d. High fever, up to 105°.

Forty-five cases then have slight febrile reaction; eighty-

three cases have a moderate degree of fever; ten have considerable fever; four have high fever; and only one hyperpyrexia.

I have appended to the tables a careful abstract of the temperature columns, which shows at a glance the number of cases reaching their highest at each two tenths of a degree.

Passing now to the column for special treatment, and specially interesting features in the cases, we find that in about twenty-five cases some cooling method was adopted; either pounded ice tied up in a piece of mackintosh and applied to the head, or the coil ice-water cap, which I had the honour to bring to the notice of the Society with the record of my first twenty ovariotomies;[1] or a partial or complete cold pack. There is one case of abnormally low temperature with complete recovery; and there are a few cases of abnormally rapid pulse, *i. e.* of a pulse altogether out of proportion to the temperature. There are also a few cases in which chest complications, such as bronchitis, broncho-pneumonia, &c., are responsible for unusual elevations of temperature.

By far the commonest causes of any rise above the subfebrile temperatures are the on-coming of metrostaxis or menstruation, and the curious eruption of sudamina known as "the sweat rash;" this is directly responsible for the only case of hyperpyrexia in the series, No. 196 in the Tables. Either condition will often give rise to decided elevation of temperature, and if they happen to be combined in the same patient the fever may run high, then the metrostaxis is checked, and more serious complications may arise.

I have used the drainage-tube very little, but am certain that in some cases it is valuable, and I am inclined to think, from recent experience, that had I used it a little oftener the temperatures would have been lower in some

[1] "Twenty-five Cases of Ovariotomy, with remarks on the Causes and Treatment of the Fever so frequently following the operation," 'Med. Chir. Trans.,' vol. lx, 1877.

of the severe cases. It is a most difficult matter to decide when to use it and when not to do so. I shall refer to this matter again in speaking of the fatal cases.

Passing now to the consideration of the column which deals with the after-history of the patients, one is at once struck with some very important facts.

Thus, all but two of the patients from whom I have removed sarcomatous ovarian tumours have died within or near the year, with recurrence, or are now in a hopeless condition. The two exceptions are both married women, have both borne children since, and have both survived the operation long enough to make me hope that they are not going to have any recurrence.

The saddest fact brought out in this column is the number of patients that sooner or later succumb to malignant disease, in one form or another, or with some mysterious undiagnosed ailment which makes one suspect malignant disease.

I believe that the vast majority of these recurrent growths are sarcomata. I have only seen one undoubted case of solid cancer of the ovary, No. 229. Some of the papillomata very closely resemble the epitheliomata and cylindrical cancers, but the recurrent tumours seem generally to be sarcomata. With the abundant material at hand there is a fine field open to the pathologist in the study of these ovarian tumours and the recurrent growths which follow their removal, especially if it be worked side by side with the development of the ovary and its contained follicles and ova. I have myself worked in it sufficiently, when I had more leisure, to make me quite sure that no one has as yet got upon the right track for thoroughly mastering the histology and pathology of the ovary.

Anyone looking down the columns of "subsequent history," will see how many cases have died of recurrent sarcoma, and how many in which the tumour removed has been to all appearance of the ordinary innocent type have died of peritoneal cancer. I have used this term because

it is the one in common use for describing these cases, but I do it under protest, and with the full belief that it means peritoneal sarcoma.

The question naturally arises, can nothing be done to lessen this terrible after mortality from malignant disease? I reply, much can be done when the profession is made thoroughly alive to the dangers of tapping ovarian and parovarian or broad ligament cysts. As long as this pernicious and unscientific practice receives encouragement from authority, and in high places, so long will the most brilliant successes of the ovariotomist be clouded by this sad subsequent death-roll.

Tapping is directly responsible for most of the recurrences, and not infrequently the tapping puncture is one of the earliest seats of the new disease, while the ovariotomy scar remains healthy. The reason is not far to seek. It is impossible for anyone, however skilful, to tap without some escape of the small infective cells into the peritoneum, or among the tissues of the tapping puncture. On the other hand, an operator of average skill can, in the great majority of cases, remove an ovarian tumour without any fouling of peritoneum or wound, and if any small escape does take place it is at once sponged away, while after tapping it remains absolutely undisturbed.

Another fruitful cause of infection is the rupture of cysts into the peritoneum. This accident may arise in many different ways. Papilloma will grow through the cyst wall and cause an opening. Vessels may block from inflammation, and the starved part of the cyst wall will then give way; direct violence may cause a split in the cyst wall; twisting of the pedicle may cause extreme distension and rupture, &c.; but in whatever way the accident happens it is a serious disaster for the patient, for from that moment we cannot say, however successful may be a subsequent ovariotomy, that she will remain free from recurrent disease. The only way to render these accidents less common is to make early ovariotomy the rule.

I would sum up this matter by saying, never tap, and, given an ovarian tumour distinctly to be diagnosed, perform ovariotomy without delay.

After histories also give very important information, as to the effect of the removal of both ovaries on menstruation; but this is a subject too large to treat here, and will require a special paper to deal adequately with it.

I have given a very full table of the incomplete and exploratory operations, and explanatory remarks at the end of the table, so that it will not be necessary for me to refer to them separately; but I would call attention to the fact that all but one of the incomplete ovariotomies were rendered so by the infiltrations of malignant disease; that all the immediate deaths were in these malignant cases; that the subsequent deaths were due to the progressive advance of such disease; and that the non-malignant cases all did well.

I will now briefly allude to the causes of death in the fatal cases, not taking them quite in numerical order, but grouping them together under the following heads.

Septicæmia.

Case 191.—The septic mischief was, I believe, derived from a dilated and cystic Fallopian tube, the symptoms came on early, as they generally do in well-marked cases of septicæmia (*i. e.* they are not well-doing cases from the first). She seemed, however, to have fought through it, and to be on the way to recovery, when suddenly her right parotid gland swelled and became very painful, her temperature rose again, and in twelve hours she was dead. This is one of the cases in which it is possible that drainage might have been useful, but I think it is very doubtful; my notion being that the ligatures were infected by passing through the cystic tube, and that the pedicle sloughed from the putridity spreading into the distal part.

Case 471.—The last death in the series I will take next, because it has the common feature of suppuration of parotid glands. It was a case of ruptured, semi-solid tumour, with an immense quantity of ascitic fluid and chronic peritonitis. The patient had septicæmic symptoms before I operated, dark vomit having been troublesome, with a feeble-running pulse. She did badly from the first, and died with suppuration of both parotid glands, death coming rather suddenly at last, with rapid rise of temperature as in the previous case. I doubt if anything but earlier operation would have altered the result.

Case 204 was another one due to rupture of a degenerating semi-solid tumour into the peritoneum, in a feeble elderly woman; and I think, from watching her condition, that the use of the drainage-tube might possibly have saved her.

Case 428 was a clear case of septicæmia, and I am equally clear that I ought to have used the drainage-tube. There was a quantity of broken-down lymph and clot in the pelvis, and it was very fœtid. I did not put in a tube because everything seemed so dry and clean after sponging out. I am certain, however, that in every case in which fœtor is present, the wise course is to use the tube.

These septicæmic cases must be considered as among the preventible fatalities, and I will take next another group belonging to the same class.

Hæmorrhage.

Case 304.—A simple ovariotomy, complicated by the removal of a small, unhealthy, adherent ovary from the opposite side. This had to be regularly scraped out, and there was free oozing at the time from a largish surface. I did not see the patient after the operation, but she died in twenty-nine hours with symptoms which would have led me to reopen the wound and look for hæmorrhage, had I been sent for.

Case 396 died from exhaustion some days after a very severe hæmorrhage. I opened the abdomen and found that the inner loop of the transfixing ligatures had slipped (the only time I have ever seen this accident happen, it being always the outer loop which is the danger); the hæmorrhage was easily stopped, but she had lost an enormous quantity of blood and never rallied.

Case 401 was also a new experience in the matter of hæmorrhage. It was a case in which a very difficult double enucleation was performed, and the oozing was very free and difficult to check at the time; but when I closed the abdomen I thought that all was safe. Symptoms of hæmorrhage, however, appeared, and I re-opened the wound, and then found it absolutely impossible to check the hæmorrhage that was going on from a very large surface.

Diarrhœa and exhaustion.

I think the only fatal case which I should consider really preventible, was one which died from the exhaustion of diarrhœa.

Case 438 was practically convalescent, but had slight looseness of the bowels, which I did not consider sufficiently, and she was moved into the convalescent ward at the week's end as usual. In the night diarrhœa became very violent, and the nurse, a new hand, did not attempt to check it, or seek any help, and at the morning visit I found the patient blue, cold, and collapsed, and she died in a few hours.

Shock.

Cases 200 and 353 were both due to shock, both were ruptured dermoids, and both were, I think, hopeless under any conceivable management. The former was barely placed in bed alive, and the latter only lived a few hours.

Exhaustion from malignant disease.

Cases 229 and 283 were both due to cancer. The former, the only case of true cancer of the ovary I have ever seen, was really an incomplete operation, for I had regularly to carve the sigmoid flexure, and a coil of small intestine, out of the mass, and of course the disease was not completely removed. The patient, a child of fifteen, never rallied, though she lingered on for a few days. The latter was a simple enough ovariotomy, but the patient had also extensive cancer of both stomach and sigmoid flexure, and could not assimilate food either by the stomach or the rectum, and literally died of starvation.

Exhaustion.

Cases 427 and 455 also died of exhaustion, the latter after the enucleation of two malignant papillomatous cysts from the pelvis, one of which had been tapped and drained before I operated, and the patient had been long bedridden. The former was a feeble, unhealthy woman, with a ruptured cyst and chronic peritonitis, and she never showed any rallying powers.

Suppression of urine (Chronic Bright's disease).

Case 217 had an enormous tumour weighing eighty-eight pounds, which had been slow in growing, and had contracted very extensive and firm adhesions. Suppression of urine followed quickly upon the operation, and the post-mortem revealed very advanced granular disease of both kidneys.

Tetanus.

Case 231 had a ruptured cyst, and the peritoneum was tapped before I saw her. After this tapping she had a

slight threatening of tetanus. I went into the country several weeks later, and performed ovariotomy, and tetanus carried her off on the sixth day after operation. This is the only case of the disease I have ever had in my practice, and it is worthy of note that it occurred in cold, windy weather in the spring, when tetanus was apparently rife, for there were notices of several cases in the journals at the time. The paroxysms in my patient were first started by a violent gust of wind, and recurred every time the house was shaken by the wind.

Abscess in pedicle stump.

Case 254 was that of a young woman with a solid sarcoma of the left ovary, the disease penetrated so deeply into the broad ligament, that I had to tie my ligatures in sarcomatous tissue. She did well at first, then got very high temperature, for which we could find no cause, and literally died of fever without any other bad symptoms. The post-mortem revealed a small slough and confined abscess in the pedicle. She was probably saved the worse fate of a lingering death from recurrent sarcoma.

Obstruction of intestines.

Cases 276 and 371 died of obstruction, the former acute from infective peritonitis, and the latter worn out by chronic obstruction, which never seemed bad enough to justify reopening the abdomen, till she was too exhausted to give one any chance of doing so with success. It is always a subject of wonder to me that more cases do not die of obstruction while carrying these great adherent tumours, and after their removal.

Peritonitis.

Case 291 was one of acute torsion of the pedicle, with general effusion of blood into the tumour and broad ligament. At the time I was allowed to operate, acute peritonitis had set in, and she was very feeble from the hæmorrhage; the operation was a forlorn hope, and she never rallied.

Embolism.

Case 370 was a case of sudden death from embolism. The patient was so well, and had been so well all through that I had allowed her husband to leave town; she woke in the night, chatted and laughed with the nurse, and fell back dead. No post-mortem was allowed, but it was a clear case of sudden movement of clot.

I am afraid that these brief notes of failure may seem tedious, but when read with the other details of the cases given in the tables, they are full of useful suggestions as to the possibility and impossibility of avoiding like misfortunes in the future. I can truly say, that I never go over my case-books without getting useful hints for practice, and food for thought. Frequently a direct reference to my tables, when in difficulty and doubt, helps the treatment or prognosis of a similar case.

All the tables have been compiled as my operations were done, and all have been very frequently revised, but it is possible that there may be some errors. If any of those who have sent me cases can find any errors I shall be grateful if they will point them out.

An analysis of the temperature columns gives the following results:

Temp.	1st 100.	2nd 100.	3rd 100.	Totals.	Cases.
98·6	1	0	1	2	99·0 or under 11.
99·0	3	2	4	9	
99·2	4	3	7	14	100·0 „ 81.
99·4	4	6	0	10	
99·6	4	4	9	17	
99·8	5	5	6	16	
100·0	9	4	11	24	
100·2	7	9	6	22	101·0 or under 89.
100·4	5	14	3	22	
100·6	3	8	9	20	
100·8	4	2	1	7	
101·0	7	5	6	18	
101·2	1	5	5	11	102·0 „ 52.
101·4	4	2	4	10	
101·6	3	5	5	13	
101·8	4	3	3	10	
102·0	2	4	2	8	
102·2	3	2	4	9	103·0 „ 31.
102·4	4	1	3	8	
102·6	1	4	0	5	
102·8	2	0	2	4	
103·0	2	3	0	5	
103·2	0	0	0	0	104·0 „ 10.
103·4	1	0	1	2	
103·6	1	1	1	3	
103·8	0	1	0	1	
104·0	4	0	0	4	
104·2	0	1	0	1	105·0 „ 5.
104·4	1	0	0	1	
104·6	1	0	0	1	
104·8	0	1	0	1	
105·0	0	0	0	0	
106·2	1	0	0	1	Exceptional.
Totals	91	95	93	279	Recoveries.

Day after operation at which highest temperature is reached.

Operation evening	93 cases.
1st „	129 „
2nd „	35 „
3rd „	9 „
4th and 5th evening, each	4 „
6th, 7th, and 8th „ „	1 „
13th and 15th „ „	1 „

(For report of the discussion on this paper, see 'Proceedings of the Royal Medical and Chirurgical Society,' New Series, vol. ii, p. 139.)

Three Hundred additional Complete Ovariotomies.

No.	Place of operation.	Date of operation.	Age.	Condition.	Length of incision.	Adhesions.	Ovary removed.	Treatment of pedicle.	Weight of tumour.	Highest temperature.	Quickest pulse.	Special treatment, or special features of case.	Immediate result.	Subsequent history, and general remarks on case.	No. in antiseptic series.
		1880			Ins.				lbs.						
189	Samar. Hosp.	June	28	M.	6	Parietal, omental, and vesical	Left	Ligatures	42	102·0 O. E.	140	Pulse was 112 before operation and remained over 120 for some days. Digitalis	Recovered	Girl born May, 1882. Nat. labour	151
190	Ditto	June	27	M.	4	Extensive parietal, intestinal, and pelvic	Right	Enuclen. and lig.	8	104·0 E. 1	128	Ice in bladder on head for 3 days	Recovered	—	152
191	Ditto	June	50	M.	4	None.....	Both	Enuclen. cautery and lig.	9	104·0 M. 4	120	Ice-water cap on till hydro-salpinx of left tube	Died 10th day—pyæmia	These were the broadest pedicles I ever saw, and I have no doubt the mischief arose from the ligatures passing through a cystic tube	153
192	Ditto	June	27	M.	3½	To opposite tube and uterus	Right	Ligatures	11	101·0 E. 2	100	None	Recovered	Continues well	154
193	Ditto	July	70	M.	3½	None.....	Right	Enuclen. and lig.	13	101·4 E. 1	84	None	Recovered	—	155
194	Ditto	July	25	S.	3½	None.....	Both	Ligatures	17	100·8 E. 1	108	None	Recovered	Continues well. Has not menstruated	156
195	Ditto	July	18	S.	3½	Intestinal.....	Left	Enuclen. and lig.	10	102·6 M. 1	124	Ice-water cap 4 days	Recovered	Continues well	157
196	Ditto	July	38	M.	4	Parietal and omental	Both (R.)	Ligatures	30	106·2 M. 3	120	Ice-pack arms and legs for 4 hours	Recovered	Hyperpyrexia was very sudden, and was I believe due to immense crop of sudamina coming out suddenly in very fat patient. Well	158
197	Nursing Home	July	40	S.	4	General from twisted pedicle	Left	Ligatures	7	100·2 E. 1	96	None	Recovered	Continues well	159
198	Private house	July	—	M.	4	Omental	Left	Ligatures	15	100·0 O. E.	96	None	Recovered	Child born. Nat. labour	160

No.	Place of operation.	Date of operation.	Age.	Condition.	Length of incision.	Adhesions.	Ovary removed	Treatment of pedicle.	Weight of tumour.	Highest temperature.	Quickest pulse.	Special treatment, or special features of case.	Immediate result.	Subsequent history, and general remarks on case.	No. in antiseptic series.
		1880			Ins.				lbs.						
199	Samar. Hosp.	July	28	M.	4	Ruptured cyst, omental, chronic peritonitis	Left	Ligatures	11	101·0 O. E.	104	None	Recovered	Boy born January 12th, 1882. Nat. labour	161
200	Private house	July	61	W.	6	Universal adhesions from twisted pedicle, general purulent peritonitis	Left dermoid	Ligatures	10	—	—	None	Died	Shock soon after being placed in bed	162
201	Samar. Hosp.	July	21	S.	4	None.....	Left dermoid	Ligatures	30	100·4 E. 1	80	None	Recovered	Just going to be married	163
202	Ditto	Oct.	63	M.	4	Extensive parietal and omental	Left	Ligatures	28	102·8 M. 5	104	None	Recovered	Rise of temperature due to sweat rash. Continues well	164
203	Ditto	Oct.	39	M.	3	None.....	Left	Ligatures	16	102·4 M. 3	120	None	Recovered	The temperature in this case also coincided with eruption of sudamina and at once subsided. Has not menstruated since operation	165
204	Ditto	Oct.	61	M.	6	Extensive parietal and omental	Left	Ligatures	33	103·6 E. 4	120	Quinine freely used	Died 5th day	Septicæmia. Cyst ruptured into peritoneum during removal	166
205	Nursing Home	Oct.	28	S.	3	None.....	Right	Ligatures	7	100·0 E. 1	100	None	Recovered	Continues well	167
206	Samar. Hosp.	Oct.	64	S.	3	Parietal.....	Right	Ligatures	14	103·4 E. 1	108	Had broncho-pneumonia for which Dr. Day treated her	Recovered	—	168
207	Ditto	Oct.	31	M.	4	None.....	Right	Ligatures	18	100·2 E. 1	100	None	Recovered	Continues well	169
208	Ditto	Oct.	52	M.	5	None.....	Left	Ligatures	27	100·8 O. E.	106	None	Recovered	Continues well	170
209	Ditto	Oct.	35	M.	4	None; ruptured cyst with chronic peritonitis	Both	Ligatures	18½	99·6 E. 1	98	None	Recovered	Continues well	171
210	Ditto	Nov.	27	S.	4	None.....	Both (L.)	Ligatures	29	100·6 E. 1	108	None	Recovered	Continues well and has not menstruated	172
211	Ditto	Nov.	39	M.	5	Vascular parietal	Right	Ligatures	28	104·0 M. 4	112	Partial cold pack ...	Recovered	Case read before Clinical Society. Remains in perfect health	173
212	Nursing Home	Nov.	24	S.	4	None.....	Left	Ligatures	11	100·4 E. 1	104	None	Recovered	Continues well	174
213	Samar. Hosp.	Nov.	35	S.	4	None.....	Left	Ligatures	32	101·4 E. 3	80	None	Recovered	Died of cancer in abdominal scar, Dec., 1881. Had been tapped before operation, fluid draining away for some time from	175

215	Ditto	Nov.	20	M.	4	and pelvic None.....	Left	and ligs. Ligatures	17	100·8 M. 1	120	None	Recovered	Boy, Jan. 24, 1882, natural labour. Had no children before operation	177
216	Nursing Home	Nov.	54	M.	5	Parietal, omental, and intestinal	Left	Ligatures	30	102·0 O. E	96	None	Recovered	Continues well	178
217	Samar. Hosp.	Nov.	35	S.	7	Parietal, intestinal, and omental	Left	Ligatures	88	106·6 E. 1	132	Partial pack	Died 73 hours	Chronic Bright's disease. Complete suppression of urine	179
218	Ditto	Dec.	19	S.	4	Parietal and omental	Right	Ligatures	19	102·0 E. 2	112	None	Recovered	Continues well	180
219	Ditto	Dec.	48	S.	4	Parietal.....	Left dermoid	Ligatures	70	104·0 M. 2	120	Ice-water cap 54 hours	Recovered	Continues well	181
220	Ditto	1881 Jan.	55	M.	4	Opposite ovary and tube	Left	Ligatures	12	100·4 O. E	84	None	Recovered	Continues well	182
221	Ditto	Jan.	49	M.	4	Extensive parietal	Right	Ligatures	19	104·0 E. 1	136	Ice-water cap 68 hours	Recovered	There was a nasty suppurating tapping puncture open into peritoneum at time of operation	183
222	Ditto	Feb.	41	M.	5½	Ruptured cyst, intestinal, intense chronic peritonitis	Left	Ligatures	35	103·6 M. 3	124	Ice-water cap 39 hours	Recovered	Has a girl a year and a half old. Well	184
223	Ditto	Feb.	49	M.	5½	Parietal, omental, and intestinal	Left	Ligatures	27	100·6 O. E.	96	None	Recovered	Continues well	185
224	Ditto	Feb.	57	W.	4	Parietal and omental	Right	Ligatures	80	100·4 E. 1	108	None	Recovered	Continues well	186
225	Ditto	March	37	M.	7	Parietal, omental, intestinal, uterine, ovarian, and vesical	Right	Ligatures	50	102·8 E. 2	104	Ice-water cap 60 hours	Recovered	Has a tumour of other ovary	187
226	Ditto	March	49	M.	3½	None.....	Both (L.)	Ligatures	12	99·8 E. 1	104	None	Recovered	Continues well. Menopause at 46	188
227	Ditto	March	48	W.	6	Parietal, omental, and mesenteric	Left	Ligatures	37	102·0 O. E.	112	None	Recovered	Continues well	189
228	Ditto	Mar.	43	M.	3	None.....	Both (L.)	Ligatures	27	100·8 O. E.	116	None	Recovered	Continues well. Has not menstruated since operation	190
229	Ditto	Mar.	15	S.	7	Universal cancerous adhesions	Both	Ligatures and cautery	7	106·8 dying	168 dying	Ice-water cap, quinine, jacket poultices, &c.	Died 48 hours	Exhaustion. Choked with phlegm which she could not expectorate	191
230	Ditto	Mar.	36	M.	3½	None.....	Left	Ligatures	20	100·2 O. E.	92	None	Recovered	Continues well	192
231	Country House	Mar.	53	S.	6	Omental and intestinal, ruptured semi-solid tumour	Left	Ligatures	25	100·0 E. 5	112	None	Died 6th day	Tetanus. There had been a slight threatening of it after tapping	193
232	Samar. Hosp.	April	33	M.	4	Omental, transplanted tumour	Both dermoid	Ligatures	—	100·8 O. E.	98	None	Recovered	Continues well. Menstruated twice in first nine months, but not since	194
233	Nursing Home	April	64	M.	4	Ruptured cyst.....	Left	Enuclen. and ligs.	19	101·4 E. 2	100	None	Recovered	Continues well	195

No.	Place of operation.	Date of operation.	Age.	Condition.	Length of incision.	Adhesions.	Ovary removed.	Treatment of pedicle.	Weight of tumour.	Highest temperature.	Quickest pulse.	Special treatment, or special features of case.	Immediate result.	Subsequent history, and general remarks on case.	No. in antiseptic series.
		1881			Ins.				lbs.						
234	Samar. Hosp.	April	25	S.	4	Ruptured cyst with chronic peritonitis	Both (L.)	Ligatures	17	100·6 O. E.	104	None	Recovered	Has not menstruated since. Married fifteen months	196
235	Private House	April	7	S.	6	Parietal, omental, and intestinal	Left dermoid	Ligatures	4½	102·4 E. 1	140	None	Recovered	Continues well	197
236	Sam Hosp.	April	47	M.	5	Parietal, omental, intestinal, and pelvic	Left	Ligs., enuclen., and cautery	19	102·2 E. 1	114	None	Recovered	Cysto-sarcoma and sarcoma also removed from omentum and cæcum. Has now recurrence	198
237	Ditto	April	40	S.	4	None.....	Both (R.)	Enuclen. and ligs.	27	100·2 O. E.	96	None	Recovered	Has not menstruated since. Is quite well	199
238	Ditto	May	51	M.	5	Extensive parietal	—	Ligatures	48	101·2 O. E	98	None	Recovered	Continues well	200
239	Ditto	May	39	W.	4	Parietal and omental	Both (L.)	Ligatures	38	100 8 O. E.	92	None	Recovered	Has not menstruated since operation. Quite well	201
240	Nursing Home	May	64	S.	—	None.....	Right	Ligatures	15	102·4 E. 1	96	None	Recovered	Is now suffering from scirrhus of breast	202
241	Samar. Hosp.	May	51	M.	4	Ruptured colloid, omental	Right	Ligatures	22	104·6 E. 1	130	Ice-water cap some days	Recovered	Died in December, 1885. Obstruction from malignant disease in pelvis	203
242	Ditto	June	31	M.	3½	Twisted pedicle. None	Right dermoid	Ligatures	10	101·6 E. 1	124	None	Recovered	Continues well	204
243	Ditto	June	48	M.	4	None.....	Both (R.)	Ligatures	16	100·6 O. E	92	None	Recovered	Continues well. Has not menstruated	205
244	Ditto	June	41	S.	4	None	Both (L.)	Ligatures	15	101·6 M. 1	82	None	Recovered	Continues well	206
245	Ditto	June	30	M.	3½	None.....	Left	Ligatures	7	100·6 E. 1	120	None	Recovered	Two children since operation; first was stillborn, second a girl	207
246	Nursing Home	June	27	S.	4	Ruptured colloid, parietal	Left	Ligatures	14	101·0 O. E.	108	None	Recovered	Continues well	208
247	Samar. Hosp.	June	39	M.	4	Omental and intestinal (malignant)	Left dermoid	Ligatures	12	100·0 M. 3	92	None	Recovered	Died of peritoneal cancer, May, 1882	209
248	Ditto	July	26	S.	4	None.....	Right	Ligatures	44	100·2 E. 1	100	None	Recovered	Married. Girl born February, 1886; natural labour. Continues well	210
249	Ditto	July	19	S.	7	Solid sarcoma	Right	Ligatures	8	103·4 O. E.	116	None	Recovered	Died with sarcomata all over body, April, 1882	211
250	Ditto	July	48	M.	5	None.....	Left	Ligatures	45	101·0 O. E.	96	None	Recovered	Died of jaundice (? hepatic cancer), March, 1882	212
251	Ditto	July	40	M.	4	Rectal and uterine	Right	Ligs. and enuclen.	14½	100·2 E. 1	100	None	Recovered	Continues well	213
252	Ditto	July	40	W.	3	None.....	Left	Ligatures	5	99·6 O. E.	96	None	Recovered	—	214

														1883, of apoplexy	
254	Ditto	Oct.	26	S.	7	Universal recent; solid sarcoma	Left	Ligatures	8	107·4 E. 9	168	Ice-water cap and cold pack	Died 15th day	Abscess of pedicle	216
255	Ditto	Oct.	31	S.	5	Parietal, omental, intestinal, and pelvic	Right	Ligatures	7½	100·2 M. 1	120	None	Recovered	Died of cancer of cervix uteri, May, 1882	217
256	Ditto	Oct.	46	S.	5	Parietal and omental	Right	Ligatures	27	101·0 O. E	136	None	Recovered	—	218
257	Private House	Nov.	63	M.	4	Cæcal	Right	Enuclen. and ligs.	8	100·0 O. E.	84	None	Recovered	Continues well	219
258	Samar. Hosp.	Nov.	43	S.	4	Parietal and pelvic	Both (R.)	Enuclen. and ligs.	14	101·8 E. 4	120	None	Recovered	Continues well. Menstruated regularly for twelve months	220
259	Ditto	Nov.	26	M.	3½	None.....	Left	Ligatures	6	100·4 E. 6	110	None	Recovered	Continues well	221
260	Ditto	Nov.	48	M.	5	Ruptured colloid; none	Right	Ligatures	28	102·0 bronchitis	108	Poultices, &c., for bronchitis	Recovered	Continues well	222
261	Nursing Home	Nov.	53	M.	4	Parietal.....	Both (R.)	Ligatures	18	101·0 E. 1	94	None	Recovered	Continues well	223
262	Ditto	Nov.	41	M.	4	None.....	Left	Ligatures	7	101·6 E. 1	88	None	Recovered	Continues well	224
263	Ditto	Nov.	30	S.	4	None.....	Right	Ligatures	10	100·0 M. 1	92	None	Recovered	Continues well	225
264	Samar. Hosp.	Nov.	28	S.	4	Extensive parietal and omental	Both (R.)	Ligatures	46	100·4 O. E.	108	None	Recovered	Continues well and has not menstruated	226
265	Ditto	Nov.	40	M.	4	Left entirely twisted off and transplanted on to omentum	Both (L.)	Ligatures	5	101·0 E. 1	104	None	Recovered	Continues well and does not menstruate	227
266	Nursing Home	Nov.	40	S.	3½	None.....	Right	Ligatures	14	103·0 E. 2	112	Great crop of sudamina on evening of 2nd day	Recovered	Continues well	228
267	Samar. Hosp.	Dec.	21	S.	6	Parietal, omental, and intestinal	Right dermoid	Ligatures	5	100·6 E. 1	96	None	Recovered	—	229
268	Ditto	Dec.	39	M.	5	Uterine	Right	Ligatures	12	101·4 E. 2	96	Uterus covered with little sub-peritoneal outgrowths	Recovered	Address lost	230
269	Nursing Home	Dec.	28	S.	3½	Fungating melanotic sarcoma, adherent in pelvis, both ruptured	Both	Ligatures	16	101·2 O. E.	134	None	Recovered	Died with sarcomata all over body, June, 1882	231
270	Samar. Hosp.	Dec.	45	M.	6	Bladder and right ovary. Solid sarcomata	Both (L.)	Ligatures	10	101·8 M. 1	128	None	Recovered	Died with recurrence in pelvic glands, March, 1882	232
271	Ditto	Dec.	32	W.	6	Omental	Right	Enuclen. and ligs.	34	100·4 O. E.	108	None	Recovered	Recurrent growths in peritoneum, 1885	233
		1882													
272	Private House	Jan.	65	W.	4	None.....	Both (R.)	Ligatures	14	101·8 M. 2	88	None	Recovered	Continues well	234
273	Ditto	Jan.	34	S.	3½	None.....	Right	Ligatures	6	100·4 E. 1	108	None	Recovered	Had chronic Bright's disease after scarlet fever at time of operation. Continues well	235

No.	Place of operation.	Date of operation.	Age.	Condition.	Length of incision.	Adhesions.	Ovary removed.	Treatment of pedicle.	Weight of tumour.	Highest temperature.	Quickest pulse.	Special treatment, or special features of case.	Immediate result.	Subsequent history, and general remarks on cases.	No. in antiseptic series.
		1882			Ins.				lbs.						
274	Samar. Hosp.	Jan.	62	M.	6	Parietal, omental, and pelvic	Both (L.)	Ligatures	37	100·2 O. E.	92	Hydro-salpinx of right tube	Recovered	Continues well	236
275	Ditto	Jan.	49	S.	6	Extensive pelvic and omental. Sarcomata	Both	Ligatures	7	100·0 O. E	100	None	Recovered	Died in King's College Hospital, Nov., 1882. Sarcoma of skull implicating brain	237
276	Ditto	Jan.	49	S.	6	Pelvic	Right	Ligatures	11½	102·0 ante mortem	132	—	Died 5th day	Obstruction of intestine due to infective peritonitis	238
277	Ditto	Jan.	51	M.	4	None.....	Right	Ligatures	25	100·2 O. E.	104	None	Recovered	Continues well	239
278	Ditto	Jan.	43	M.	7	Parietal, omental, and pelvic	Both (L.)	Ligatures	41	100·8 E. 3	96	Double hydro-salpinx	Recovered	Has not menstruated since operation. Is quite well	240
279	Ditto	Feb.	47	M.	4	Extensive pelvic...	Both (L.)	Enuclen. and ligs.	5	102·2 E. 1	96	Right hydro-salpinx	Recovered	Died in following August of obstruction	241
280	Ditto	Feb.	49	M.	4	Parietal.....	Left	Ligatures	20	103·0 E. 3	108	Ice-water cap 36 hours	Recovered	Continues well	242
281	Ditto	Feb.	34	M.	4	Parietal, omental, and intestinal	Right	Ligatures	20	100·0 O. E.	100	None	Recovered	Continues well	243
282	Samar. Hosp.	Feb.	30	M.	6	Parietal, omental, and hepatic	Left	Ligatures	13	101·8 E. 1	112	None	Recovered	—	244
283	Ditto	Feb.	53	M.	4	Malignant adhesions to omentum, intestine, &c.	Both (R.)	Ligatures	17	105·6 ante mortem	128	Cancer of stomach, liver, and rectum	Died in 34 hours	Could not feed either by mouth or rectum	245
284	Ditto	Mar.	28	M	4	Omental and of the two tumours to one another	Both (L.)	Ligatures	17	100·4 E. 1	110	Right hydro-salpinx	Recovered	Continues well and does not menstruate	246
285	Nursing Home	Mar.	32	S.	4	None.....	Both (R.)	Ligatures	4	102·2 E. 2	96	None	Recovered	Had been tapped many times by vagina and drained, yet there were no adhesions. Has since married and is well	247
286	Ditto	Mar.	44	M.	4	None.....	Right	Ligatures	5	101·0 E. 2	88	None	Recovered	Continues well	248
287	Samar. Hosp.	Mar.	44	M.	4	Universal from twisted pedicle	Both (L.)	Ligatures	17½	100·8 E. 1	108	None	Recovered	Continues well. Menstruation ceased four years before operation and has not returned	249
288	Ditto	Mar.	41	M.	4½	Ditto.....	Both (R.)	Ligatures	10	101·0 O. E.	120	None	Recovered	Has not menstruated since. Is quite well	250
289	Private House	Mar.	27	S.	4	Ditto.....	Right dermoid	Ligatures	12	100·4 O. E.	100	None	Recovered	Continues well	251
290	Samar. Hosp.	Mar.	33	M.	5	Ruptured cyst.....	Both (L.)	Ligatures	35	100·2 E.	112	None	Recovered	Continues well and does not menstruate	252

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292	Samar. Hosp.	April	43	M.	6	Parietal and omental	Right	Ligatures	43	101·4 O. E.	120	None	Recovered	Continues well	254
293	Ditto	April	38	W.	6	Parietal, omental, and intestinal	Both (L.)	Ligatures	14½	101·6 O. E.	88	None	Recovered	Going to be married again	255
294	Country House	April	46	S.	4	Extensive papillomatous from rupture of cyst	Both	Ligatures	10	99·2 E. 1	—	Large omental tumour removed at the same time	Recovered	Died of peritoneal cancer, December, 1882	256
295	Nursing Home	April	31	S.	4	None.....	Both (L.)	Ligatures	3	100·4 E. 1	108	None	Recovered	Continues well. Large fibroid uterus at time of operation, is now atrophic	257
296	Samar. Hosp.	April	34	M.	5	Parietal and omental	Left	Ligatures	20	100·8 O. E.	94	None	Recovered	Continues well	258
297	Ditto	May	43	S.	5	Right derm. twisted off and transplanted	Both (R.)	Ligatures	5	100·4 E. 2	100	None	Recovered	Died of phthisis some months after return home	259
298	Nursing Home	May	49	M.	6	Universal adhesions from rupture of cyst	Left	Ligatures	13	101·4 E. 1	116	None	Recovered	Continues well	260
299	Samar. Hosp.	May	50	S.	4	Parietal and pelvic	Left	Ligatures	55	100·8 O. E.	120	None	Recovered	Fibroid also removed from fundus uteri. Continues well	261
300	Ditto	May	54	M.	6	None.....	Right	Ligatures	14	99·4	88	None	Recovered	I had previously removed her left ovary, see old tables, Case 68. Continues well	262
301	Private House	May	41	S.	4	None.....	Left	Ligatures	12	100·2 O. E.	88	None	Recovered	Continues well	263
302	Samar. Hosp.	May	51	S.	4	Large omental hernia dissected out and removed	Left	Ligatures	20	100·2 O. E.	92	None	Recovered	Continues well. Had tumour of right ovary removed by Sir Spencer Wells some years before	264
303	Ditto	June	35	M.	4	None.....	Left	Enuclen. and ligs.	7	99 4 E. 1	92	None	Recovered	Continues well	265
304	Private House	June	—	M.	5	Extensive pelvic (right)	Both (L.)	Ligatures	30	103·2 ante mortem	142	None	Died in 29 hours	I did not see the patient after the operation, but believe there was some internal hæmorrhage	266
305	Samar. Hosp.	June	66	W.	4	Parietal	Left	Ligatures	14	101·0 O. E.	96	None	Recovered	Continues well	267
306	Nursing Home	June	49	M.	4	None.....	Both (L.)	Ligatures	9	101·2 E. 1	88	None	Recovered	Continues well	268
307	Samar. Hosp.	June	47	M.	4	None.....	Left	Ligatures	14	100·4 O. E.	84	None	Recovered	Continues well	269
308	Ditto	June	52	S.	4	Pelvic	Both (R.)	Ligatures	6	100·2	92	None	Recovered	Continues well	270
309	Private House	June	29	M.	3½	None.....	Right	Ligatures	7	99·6	—	None	Recovered	Has had a girl since, natural labour	271
310	Samar. Hosp.	June	68	W	6	Extensive parietal and omental	Right	Ligatures	43½	103·0 E. 3	104	Ice-water cap 3 days	Recovered	Continues well	272

No.	Place of operation.	Date of operation.	Age.	Condition.	Length of incision.	Adhesions.	Ovary removed.	Treatment of pedicle.	Weight of tumour.	Highest temperature.	Quickest pulse.	Special treatment, or special features of case.	Immediate result.	Subsequent history, and general remarks on case.	No. in antiseptic series.
		1882			Ins.				lbs.						
311	Samar. Hosp.	July	42	M.	3½	Omental	Double dermoid (R.)	Ligatures	6	100·2 E. 1	96	None	Recovered	Once after birth of child cyst discharged through vagina for some weeks. Continues well and does not menstruate	273
312	Ditto	July	30	M.	3	None.....	Left	Ligatures	16	100·6 M. 1	100	None	Recovered	Continues well. Son in July, 1883, natural labour; son, Sept., 1885, natural labour	274
313	Ditto	July	31	S.	3	None.....	Right	Ligatures	10	99·8 E. 1	100	None	Recovered	Continues well	275
314	Ditto	July	29	S.	4	None.....	Both (R.)	Ligatures	9	101·6 O. E.	88	None	Recovered	Continues well. Has married and does not menstruate	276
315	Ditto	July	37	S.	4	Omental	Both (L.)	Ligatures	20	101·2 O. E.	96	None	Recovered	Continues well and does menstruate	277
316	Ditto	July	49	W.	4	None.....	Left	Ligatures	67	100·8 O. E.	100	None	Recovered	Continues well	278
317	Nursing Home	Aug.	41	M.	4	Parietal.....	Both (L.)	Ligatures	21	100·4 M. 1	112	Right hydro-salpinx	Recovered	Continues well. Has not menstruated	279
318	Ditto	Aug.	42	W.	4	Omental	Left	Ligatures	22	100·6 E. 1	100	None	Recovered	Continues well	280
319	Ditto	Aug.	41	S.	4	Omental	Both (R.)	Ligatures	8	101·0 O. E.	96	None	Recovered	Left ovary removed also because there was a large fibroid of uterus which has quite disappeared. Continues well	281
320	Samar. Hosp.	Oct.	27	S.	3½	None.....	Left	Ligatures	12	100·2 E. 2	108	None	Recovered	Continues well. Married	282
321	Ditto	Oct.	33	W.	5	Intestinal and pelvic	Left	Ligatures	10	101·2 O. E.	108	None	Recovered	I had previously (Case 52) removed a ruptured cyst of right ovary. Continues well. Has not menstruated since 2nd operation	283
322	Ditto	Oct.	17	S.	4	Parietal	Left	Ligatures	17	100·2 E. 1	116	None	Recovered	Continues well	284
323	Ditto	Nov.	38	M.	4	None.....	Both (L.)	Ligatures	8	100·4 E. 1	100	None	Recovered	Cannot get an answer as to menstruation; is quite well	285
324	Ditto	Nov.	45	M.	5	Universal from rupture of cyst	Both (L.)	Enuclen. and ligs.	20	100·4 E. 1	92	Right hydro-salpinx	Recovered	Continues well. Menstruates freely	286
325	Ditto	Nov.	39	M.	4	Pelvic	Left dermoid	Ligatures	3½	100·4 M. 2	100	None	Recovered	—	287
326	Ditto	Nov.	48	M.	5	Parietal and omental	Right	Ligatures	20	100·4 E. 1	92	None	Recovered	Continues well	288
327	Ditto	Nov.	25	S.	3½	None.....	Right	Ligatures	6	101·2 E. 1	120	None	Recovered	Continues well	289

330	Samar. Hosp.	Jan.	56	S.	5	Ruptured cyst, none	Left	Enucleat. and ligs.	17	101·2 E. 1	84	None	Recovered	Had also cancer of left breast and axillary glands. I removed these also a few weeks after ovariotomy. Died of cancer. Jan., 1884	292
331	Private House	Jan.	40	S.	4½	Ruptured colloid. None	Both (R.)	Ligatures	5½	101·0 O. E.	96	None	Recovered	Continues well and does not menstruate	293
332	Samar. Hosp.	Jan.	13	S.	6	Twisted pedicle, omental	Right	Ligatures	11	101·4 O. E.	124	None	Recovered	I had operated upon her father's sister for ovarian tumour and Baker Brown on another. I had also operated upon her second cousin on father's side	294
333	Private House	Jan.	28	M.	3½	None.....	Left	Ligatures	8½	103·6 E. 1	120	Severe feverish cold on at the time of operation	Recovered	Continues well. Has had two children	295
334	Samar. Hosp.	Jan.	34	W.	3½	None.....	Left	Ligatures	18	101·2 E. 2	108	None	Recovered	Continues well	226
335	Ditto	Feb.	42	M.	4	Universal.....	Left	Ligatures	42	102·6 E. 1	136	I cut the left ureter and afterwards performed nephrectomy	Recovered	Continues well	297
336	Ditto	Feb.	38	M.	4	Parietal.....	Both (R.)	Ligatures	40	100·6 E. 1	96	None	Recovered	Has not menstruated since operation. Is well	298
337	Ditto	Feb.	50	S.	3½	Small fibroid also removed from uterus	Left	Ligatures	6	101·6 E. 1	96	None	Recovered	Continues well	299
338	Ditto	Mar.	20	S.	7	Parietal and omental	Left	Ligatures	34	101·0 E. 1	112	Right hemiplegia from birth	Recovered	Continues well	300
339	Ditto	Mar.	62	M.	7	Extensive parietal	Right	Ligatures	38½	102·6 M. 2	96	Ice to head	Recovered	Continues well	301
340	Ditto	Mar.	24	M.	4	None.....	Both (R.)	Ligatures	22	102·0 E. 1	104	None	Recovered	Has not menstruated since operation	302
341	Ditto	April	56	M.	5	Ruptured cyst, extensive omental	Left	Ligatures	21½	99·4 E. 1	92	None	Recovered	Continues well	303
342	Ditto	April	33	M.	8	Twisted pedicle, extensive adhesions	Right	Ligatures	18½	102·2 E. 3	112	None	Recovered	Continues well	304
343	Ditto	April	24	M.	5	Twisted pedicle, universal adhesions	Right	Ligatures	5	101·8 E. 1	160	Pregnant and dangerously ill at time of operation. Girl born 36 hours after and lived 24 hours	Recovered	Is pregnant again	305
344	Nursing Home	April	44	M.	4	None	Right	Ligatures	9	100·6 E. 2	22	None	Recovered	Continues well	306
345	Samar. Hosp.	April	44	M.	4	Transplanted dermoid of left ovary from twisted pedicle	Both (R.)	Ligatures	—	100·4 E. 2	88	Also right hydrosalpinx	Recovered	Died 12 months after return home, probably of malignant disease	307

No.	Place of operation.	Date of operation.	Age.	Condition.	Length of incision.	Adhesions.	Ovary removed	Treatment of pedicle.	Weight of tumour.	Highest temperature.	Quickest pulse.	Special treatment, or special features of case.	Immediate result.	Subsequent history, and general remarks on case.	No. in antiseptic series.
		1883			Ins.				lbs.						
346	Samar. Hosp.	May	27	M.	4½	Twisted pedicle, pregnant 4 months	Left	Ligatures	8	101·0 M. 1	104	—	Recovered	Had a natural labour afterwards at full time	308
347	Ditto	May	43	M.	8	Twisted pedicle, extensive adhesions	Right	Ligatures	9	101·6 E. 1	140	When apparently well had sudden upset with head symptoms from mental worry, and was very ill for some weeks	Recovered	Continues well	309
348	Ditto	May	32	S.	4	Universal	? Both(L)	Ligatures	3	100·8 E. 2	132	—	Recovered	Menstruates regularly with much pain	310
349	Ditto	May	57	S.	6	Parietal, omental, intestinal and hepatic	Right	Ligatures	48	102·6 E. 2	120	Ice-water cap 4 days	Recovered	Continues well	311
350	Ditto	May	48	W.	7	Ruptured cyst.....	Left	Ligatures	34	101·0 E. 1	96	None	Recovered	Well when last seen	312
351	Ditto	May	29	S.	5	Chronic peritonitis from escape of ovarian fluid after tapping	Both (L.)	Enuclen. and ligs.	8	103·0 E. 5	116	Ice-water cap 48 hours. Right hydro-salpinx	Recovered	Menstruates without pain regularly. Is well	313
352	Nursing Home	June	43	M.	5	None	Left	Ligatures	10	102·0 E. 3	106	Suppuration of parotid gland	Recovered	Continues well	314
353	France	June	35	S.	8	Ruptured dermoid, universal adhesions	Left	Ligatures	58	103·4 ante mortem	144	—	Died in 24 hours	An utterly hopeless case, sank from exhaustion. Dermoid had ruptured into peritoneum some years previously	315
354	Samar. Hosp.	June	50	W.	5	Parietal, omental, intestinal and to gall-bladder	Right	Ligatures	28	101·8 E. 2	112	None	Recovered	—	316
355	Ditto	June	31	S.	3½	None	Right	Ligatures	28½	99·8	84	None	Recovered	—	317
356	Ditto	June	29	S.	4	Ruptured cyst, universal adhesions	Right	Ligatures	4	102·6 E. 5	108	None	Recovered	Continues well	318
357	Private House	June	60	W.	4	None	—	Ligatures	21	100·4	100	None	Recovered	Continues well	319
358	Samar. Hosp.	June	38	W.	4	Omental and to other ovary	Both (R.)	Enuclen. and ligs.	9½	102·4 E. 4	100	None	Recovered	Has not menstruated since operation. Is well	320
359	Private House	July	53	W.	5	Universal from twisted pedicle ...	Right	Ligatures	—	103·0 M. 1	120	None	Recovered	Continues well	321
360	Samar. Hosp.	July	28	S.	3½	Pelvic	Double dermoid (L.)	Enuclen. and ligs.	11	100·6 O. E.	112	None	Recovered	Has not menstruated since operation. Is well	322
361	Ditto	July	23	S.	3	None.....	Left	Ligatures	20	100·4 E. 1.	88	None	Recovered	Continues well	323
362	Nursing Home	July	39	M.	5	None.....	Right	Enuclen. and ligs.	—	—	—	None	Recovered	Continues well	324

						vic							ing convalescence		326
365	Samar. Hosp.	July	37	S.	3	None	Right	Ligatures	14	104·8 E. 2	104	Ice-water cap 44 hours	Recovered	Had septic abscess in abdominal parietes. Patient in opposite ward died of acute septicæmia	327
366	Nursing Home	Aug.	34	M.	5	Pelvic	Both (R.)	Enuclen. and ligs.	8	102·0 E. 7	110	Trouble with first action of bowels	Recovered	Continues well. Menstruated every 2 weeks at first after operation freely, now every month scanty	328
367	Samar. Hosp.	Oct.	32	S.	5	None	Both (R.)	Ligatures	26	104·2 E 15	100	Temp. had been normal for some days, but ran up directly she began to walk	Recovered	Continues well and does not menstruate	329
368	Ditto	Oct.	32	S.	3	None	Left dermoid	Ligatures	13½	101·6 E. 1	120	None	Recovered	—	330
369	Ditto	Oct.	34	M.	6	Extensive parietal	Right	Ligatures	69	101·6 E. 1	108	None	Recovered	Continues well	
370	Nursing Home	Oct.	23	M.	4	None	Both	Ligatures	11	102·0 E. 1	100	Two tumours about equal in size with broad bases	Died 11th day	Had been going on perfectly well and died suddenly while talking to her nurse. No P.M.	331 332
371	Private house	Oct.	35	M.	6	Universal with old pelvic inflammation	Right	Ligatures	5	100·6	96	Tried everything except reopening abdomen	Died 14th day	Chronic obstruction of intestine. Never complete, but wore her out. No P.M.	333
372	Samar. Hosp.	Oct.	45	S.	4	None	Left	Ligatures	14	100·2 E. 3	84	None	Recovered	Continues well	
373	Private house	Oct.	34	M.	3½	None	Right	Ligatures	6	100·6 E. 1	100	None	Recovered	Continues well	334
374	Samar. Hosp.	Oct.	38	M.	5	Omental, intestinal, and pelvic	Both (R.)	Ligatures	9	101·4 E. 1	120	Tumour had ruptured 5 or 6 times into peritoneum	Recovered	Has menstruated once. Is quite well	335 336
375	Nursing Home	Nov.	26	S.	3½	None	Left	Ligatures	11	101·8 M. 1	104	None	Recovered	Continues well	
376	Samar. Hosp.	Nov.	33	S.	5	None	Both der. (L.)	Ligatures	2½	101·2 E. 1	100	Double hydro-salpinx	Recovered	Menstruates	337 338
377	Samar. Hosp.	Nov.	62	S.	4	None	Double der. (R.)	Ligatures	6	101·6 bronchitis	96	None	Recovered	—	339
378	Ditto	Nov.	44	M.	4	Universal adhesions, probably malignant.	Left	Ligatures	5	100·8 E. 1	132	None	Recovered	Continues well, March, 1886	340
379	Ditto	Dec.	56	M.	4	Omental	Left	Ligatures	20	100·6 O. E.	88	None	Recovered	Continues well	341
380	Nursing Home	Dec.	33	S.	3½	None	Both (R.)	Ligatures	9½	102·0 E. 3	108	Was insane for a time after return home	Recovered	Continues well and has not menstruated	342
381	Private house	Dec.	63	M.	4	Omental, intestinal, and uterine	Left	Ligatures	30½	101·0	92	None	Recovered	Continues well	343
382	Samar. Hosp.	Dec.	26	M.	8	Cysto-sarcoma, chiefly solid. Omental	Right	Ligatures	21	102·2 E. 5	124	Ice-water cap 2 days	Recovered	Continues well	344

No.	Place of operation.	Date of operation.	Age.	Condition.	Length of incision.	Adhesions.	Ovary removed.	Treatment of pedicle.	Weight of tumour.	Highest temperature.	Quickest pulse.	Special treatment, or special features of case.	Immediate result.	Subsequent history, and general remarks on case.	No. in antiseptic series.
		1883			Ins.				lbs.						
383	Nursing Home	Dec.	53	M.	6	Parietal, omental, and intestinal ...	Right	Ligatures	39	100·4 E. 1	92	None	Recovered	Continues well	345
		1884													
384	Samar. Hosp.	Jan.	36	W.	5	Universal from twisted pedicle	Left	Enuclen. and ligs.	7	103·8 E. 1	140	A very sharp attack of bronchitis; ice-water cap 50 hours	Recovered	Continues well	346
385	Ditto	Jan.	39	M.	3½	Twisted pedicle, none	Left	Ligatures	3	101·0 E. 1	96	None	Recovered	Continues well	347
386	Ditto	Jan.	42	S.	3	Pelvic	Both (R.)	Ligatures	15½	100·6 O. E.	120	None	Recovered	Continues well	348
387	Ditto	Jan.	30	M.	3½	Omental and pelvic	Right	Ligatures	7	100·6 M. 1.	96	None	Recovered	Continues well	349
388	Ditto	Feb.	57	S.	6	Pelvic	Both (R.)	Ligatures	17	100·2 M. 1.	88	None	Recovered	Continues well	350
389	Nursing Home	Feb.	58	S.	4	Parietal and omental	Right	Ligatures	28	100·6 E. 1.	108	Large fibroid uterus	Recovered	Continues well	351
390	Samar. Hosp.	Feb.	44	M.	2½	Parietal and omental	Right	Ligatures	37	99·8	84	None	Recovered	Continues well	352
391	Nursing Home	Feb.	53	M.	4	Extensive parietal, right pedicle twisted	Both	Ligatures	18½	101·4 E. 1	104	Patient was insane for 3 weeks after operation	Recovered	Continues well	353
392	Private House	Feb.	27	S.	4	Ruptured cyst from twist of pedicle	Right	Ligatures	12	100·2 O. E.	80	None	Recovered	Continues well	354
393	Nursing Home	Feb.	—	M.	6	Twisted pedicle, omental, intestinal, &c.	Right	Ligatures	10	101·0 O. E.	100	None	Recovered	Had narrow escape from obstruction of intestines some months after operation. Second and fatal attack Nov., 1885	355
394	Samar. Hosp.	Mar.	47	M.	4	Twisted pedicle, parietal, omental, &c.	Right	Ligatures	7	100·6 E. 1	108	—	Recovered	Continues well	356
395	Ditto	Mar.	47	M.	4	Uterine, cæcal, &c.	Right	Ligatures	28	102·4 13th day	104	Rise of temp. due to trouble with action of bowels	Recovered	Continues well	357
396	Ditto	Mar.	46	M.	5	Extensive parietal	Right	Ligatures	12	103·2	144	Temp. and pulse after reopening wound	Died 5th day	Inner loop of ligatures slipped. Wound re-opened	358
397	Private House	Mar.	58	M.	6	Omental and intestinal	Right	Enuclen. and ligs.	31	101·0 O. E.	96	—	Recovered	Continues well	359
398	Samar. Hosp.	Mar.	46	W.	3½	None.....	Right	Ligatures	9	102·0 O. E.	76	—	Recovered	Continues well	360
399	Ditto	April	53	M.	6	Parietal, intestinal, and pelvic	Right	Ligatures	26	100·0 M. 1	92	Uterus enlarged and soft	Recovered	Continues well	361

												during transition by fine silk peritoneal suture. The only time this accident has ever happened to me		September 17th, 1885.	
401	Samar. Hosp.	April	25	M.	4	Omental, intestinal, and pelvic	Both	Enuclen. and ligs.	—	103·8 ante mortem	—	Could not stop general oozing in pelvis	Died 10½ hours	Opened abdomen 6 hours after operation, but entirely failed to stop the oozing	363
402	Ditto	April	30	S.	3½	None.....	Left	Ligatures	12½	100·6 O. E.	120	None	Recovered	Continues well	364
403	Ditto	April	25	M.	3	Parietal and pelvic	Left	Ligatures	11	100·6 O. E.	100	None	Recovered	Continues well	365
404	Nursing Home	May	71	S.	6	General papillomatous	Both	Enuclen. and ligs.	10	100·2 E. 1	80	Nodule in abdominal wall and papilloma of peritoneum	Recovered	Grew stout and well, but died 14 months afterwards of pleurisy (?) malignant	366
405	Private House	May	44	M.	—	Ruptured cyst. None	Both	Ligatures	11	100·6 E. 2	112	None	Recovered	—	367
406	Samar. Hosp.	May	35	M.	4	Parietal, omental, and pelvic	Both (L.)	Enuclen. and ligs.	12½	101·4 E. 1	116	Subacute peritonitis. Papilloma growing through cyst wall	Recovered	A large irregular fibroid uterus	368
407	Private House	June	57	S.	4	Ruptured papillomatous cysts with extensive adhesions	Both	Enuclen. and ligs.	11	100·6 O. E.	88	—	Recovered	Died after 3 days' illness within the year	369
408	Samar. Hosp.	June	38	M.	4	None.....	?	Ligatures	14	100·6 E. 2	112	Operated upon during menstruation	Recovered	Has not menstruated. Is quite well	370
409	Ditto	June	22	S.	3½	None.....	Right	Ligatures	14	101·8 E. 1	108	—	Recovered	Continues well	371
410	Samar. Hosp.	June	45	M.	5	Parietal, omental, and intestinal from twisted pedicle	Left	Ligatures	9	102·2 E. 2	96	—	Recovered	—	372
411	Nursing Home	June	43	S.	4	None.....	Both (L.)	Ligatures	12	101·2 O. E.	100	None	Recovered	Well and does not menstruate	373
412	Private House	June	57	S.	4	Extensive pelvic...	Left	Enuclen. and ligs.	—	99·6 M. 2	140	Abnormal pulse all through	Recovered	Continues well	374
413	Ditto	June	31	S.	3½	None...	Right	Ligatures	15	101·0 E. 2	84	—	Recovered	Continues well	375
414	Samar. Hosp.	June	46	M.	3½	None.....	Both (R.)	Ligatures	17	101·6 E. 2	112	—	Recovered	A large fibroid uterus	376
415	Nursing Home	June	52	S.	6	Parietal, omental, mesenteric, intestinal, and pelvic	Both (L.)	Ligatures	25	99·6 O. E.	100	Peritoneum full of ovarian fluid from tapping	Recovered	Continues well	377
416	Samar. Hosp.	July	17	S.	4	Omental	Left	Ligatures	16	102·0 O. E.	120	The cyst was lined with malignant papilloma	Recovered	Died in a few months with spindle-cell sarcomata in brain, pleura, lungs, &c.	378
417	Nursing Home	July	38	S.	8½	Omental, mesenteric, and pelvic	Left	Ligatures	8	102·8 M. 1	136	—	Recovered	The tumour was a soft, brain-like sarcoma	379
418	Samar. Hosp.	July	63	S.	3½	None.....	Left	Ligatures	15	100·2 O. E.	88	None	Recovered	Continues well	380
419	Ditto	July	20	M.	9	None.....	Left	Ligatures	12½	101·4 M. 1	100	—	Recovered	A solid tumour. Continues well	381

No.	Place of operation.	Date of operation.	Age.	Condition.	Length of incision.	Adhesions.	Ovary removed.	Treatment of pedicle.	Weight of tumour.	Highest temperature.	Quickest pulse.	Special treatment, or special features of case.	Immediate result.	Subsequent history, and general remarks on case.	No. in antiseptic series.
		1884			Ins.				lbs.						
420	Samar. Hosp.	July	58	M.	4½	Omental and intestinal	Right	Ligatures	7½	101·0 E. 1	96	Large fibroid uterus	Recovered	A solid tumour with much free fluid. Continues well	382
421	Nursing Home	July	65	W.	3	Parietal	Left	Ligatures	17½	101·0 E. 2	80	Parietal	Recovered	Continues well	383
422	Private House	July	26	M.	8	Parietal and omental	Both	Ligatures	7¼	102·2 E. 1	130	Ice to head for 6 days	Recovered	Solid sarcomata of both ovaries. Died in 9 months with recurrence	384
423	Samar. Hosp.	Oct.	40	S.	4	Ruptured cyst with chronic peritonitis. Parietal and omental	Both	Ligatures	7½	101·2 E. 1	104	None	Recovered	Has not menstruated, but has married	385
424	Ditto	Oct.	40	M.	4	Ruptured cyst with acute peritonitis. Omental	Left	Ligatures	13	102·8 E. 1	144	Ice to head for a week	Recovered	Died in 10 months with cancer of liver	386
425	Ditto	Oct.	43	S.	4½	Ruptured malignant cyst	Both	Ligatures	19	101·6 E. 1	106	—	Recovered	Died in 10 months with extensive disease of peritoneum, &c.	387
426	Ditto	Oct.	53	S.	3½	Pelvic	Right	Ligatures	16	100·2 E. 1	90	None	Recovered	Continues well	388
427	Ditto	Oct.	50	S.	5	Ruptured cyst with subacute peritonitis. Universal adhesions	Right	Ligatures	21	99·6 E. 1	104	Temp. and pulse both quiet up to time of death	Died 22 hours	Exhaustion	389
428	Ditto	Oct.	49	S.	5	Chronic peritonitis; universal adhesions	Right	Ligatures	10½	105·8 ante mortem	—	Fœtid stuff in pelvis when I operated	Died 4th day	Septic peritonitis. I certainly ought to have drained when I found the fœtid fluid in the pelvis	390
429	Private house	Nov.	47	M.	5	No pedicle; tumour attached to omentum and other parts	Left	Ligatures	26	100·0 E. 1	96	The right ovary had been previously removed by Dr. Tyler Smith	Recovered	I also removed left ovary with a small dermoid cyst in it. Continues well	391
430	Samar. Hosp.	Nov.	50	M.	5	Universal; dermoid	Right	Ligatures	8	102·0 E. 2	100	Could not find any left ovary	Recovered	Drained with glass tube for 48 hours	392
431	Ditto	Nov.	29	M.		Parietal, omental, cæcal and pelvic	Left	Ligatures	9	101·8 E. 1	116	—	Recovered	Continues well	393
432	Nursing Home	Nov.	46	M.	5	Extensive parietal, omental, mesenteric and pelvic	Both	Ligatures	10	101·6 E. 1	116	Malignant tumours of both ovaries	Recovered	I extirpated her sister's uterus successfully for cancer. This one is now dying of recurrence. Sister remains well	394
433	Samar. Hosp.	Nov.	31	S.	3½	None.....	Left	Ligatures	13	101·0 E. 1	100	—	Recovered	Continues well	395
434	Ditto	Dec.	52	W.	3½	None.....	Left	Ligatures	41½	100·2 E. 1	90	Uterus covered with small pediculate	Recovered	Continues well	396

437	Ditto	Dec. 1885	35	M.	3	None.....	Right	Ligatures	8	99·2 E. 1	88	Had been refused operation at one of large hospitals because she was phthisical	Recovered	Has phthisis Continues well	399
438	Ditto	Jan.	51	M.	6	Extensive parietal and omental	Right	Ligatures	36	101·6 O. E.	90	Uterus as large as a cocoa nut	Died 7th day	The bowels were a little relaxed on the 4th, 5th, and 6th days, but pulse and temp. were quiet, and she was moved into the convalescent. Violent diarrhœa set in after the move and carried her off	400
439	Private House	Jan.	20	S.	3½	Omental and pelvic	Left dermoid	Ligatures	6	101·2 M. 1	112	—	Recovered	Continues well	401
440	Samar. Hosp.	Jan.	32	M.	4	None.....	Left	Ligatures	4½	100·0 E. 1	104	Probably malignant. Suspicious disease of cervix	Recovered	—	402
441	Ditto	Feb.	22	M.	3½	None.....	Both dermoid	Ligatures	6	100·0 O. E.	92	Patient was pregnant three months	Recovered	Boy born June 23rd. Quick easy labour. See Report of Case, 'Obstet. Soc. Trans.,' 1886	403
442	Ditto	Feb.	24	S.	3½	None.....	Both	Ligatures	9	100·8 O. E.	104	—	Recovered	Continues well	404
443	Ditto	Feb.	61	W.	4	None.....	Right	Ligatures	9	99·6 E. 1	76	—	Recovered	—	405
444	Ditto	Feb.	49	S.	4	None.....	Right dermoid	Ligatures	15	99·6 E. 1	74	—	Recovered	Continues well	406
445	Nursing Home	Feb.	23	S.	5	Extensive pelvic...	Right	Enuclen. and ligs.	Small brain-like sarcoma	103·2 O. E.	108	Whole wound gaped down to peritoneum on removal of sutures and healed very slowly	Recovered	Drained with glass tube for 60 hours. Died of recurrent sarcomata, neck, &c., March 7th, 1886	407
446	Samar. Hosp.	March	46	M.	4	None.....	Right	Ligatures	20½	102·4 E. 1	108	Urine of very low sp. gr., but not albuminous to ordinary tests	Recovered	Died of Bright's disease soon after her return home	408
447	Nursing Home	March	43	S.	5	Omental and pelvic	Right	Enuclen. and ligs.	3	—	—	Also removed fibroid outgrowth from uterus	Recovered	—	409
448	Private House	March	55	S.	4	Omental	Left	Ligatures	10½	99·8 O. E.	78	—	Recovered	Continues well	410
449	Samar. Hosp.	March	40	M.	3½	None.....	Right	Ligatures	23	100·2 E. 2	94	A large fibroid uterus	Recovered	Continues well	411
450	Nursing Home	March	64	W.	5	Ruptured colloid, chronic peritonitis	Left	Ligatures	16	101·0 O. E.	96	—	Recovered	Continues well	412
451	Samar. Hosp.	March	39	S.	6	Ruptured colloid, omenta and cæcal	Right	Ligatures	10½	100·2 O. E.	76	I had removed a ruptured colloid of left ovary in May, 1879	Recovered	Continues well	413

No.	Place of operation.	Date of operation.	Age.	Condition.	Length of incision.	Adhesions.	Ovary removed	Treatment of pedicle.	Weight of tumour.	Highest temperature.	Quickest pulse.	Special treatment, or special features of case.	Immediate result.	Subsequent history, and general remarks on case.	No in antiseptic series.
		1885			Ins.				lbs.						
452	Nursing Home	May	34	S.	4	Parietal	Right	Ligatures	27	101·6 E.	196	—	Recovered	Continues well	414
453	Samar. Hosp.	May	59	M.	4	Ruptured cyst with extensive recent adhesions	Both (R.)	Ligatures	33	100·6 O. E.	100	Anterior and posterior cul-de-sacs covered with new growths	Recovered	Continues well	415
454	Nursing Home	May	43	M.	—	Ruptured cyst, parietal, omental, intestinal, and pelvic	Both (L.)	Ligatures	23	101·2 E. 1	124	Uterus as large as a cocoa nut	Recovered	Continues well	416
455	Private House	May	41	M.	6	Universal. Both tumours malignant and grown together	Both	Ligatures	8½	103·8 ante mortem		Glass drainage-tube till end	Died in 36 hours	An attempt had been made to drain one tumour after tapping, and it was suppurating	417
456	Samar. Hosp.	May	34	S.	6	Omental, intestinal, and pelvic	Both	Ligatures	11	100·8 E. 1	140	Sarmomata of both ovaries. Glass drainage-tube 142 hours	Recovered	Continues well	418
457	Ditto	May	53	S.	6	Omental, mesenteric, and intestinal	Left	Ligatures	26	100·6 M. 2	104	—	Recovered	Continues well	419
458	Ditto	May	37	M.	6	Omental, intestinal, and pelvic	Both (L.)	Ligatures	20½	101·6 E. 1	146	Temporary right hemiplegia with permanent aphasia followed the operation (day after)	Recovered	Glass drainage-tube in 42 hours. Well	420
459	Ditto	June	28	S.	5	Parietal, omental, and intestinal	Both (L.)	Ligatures	18	101·6 O. E.	120	Ruptured cyst	Recovered	Continues well	421
460	Ditto	June	30	M.	6	Parietal and omental	Both (R.)	Ligatures	19½	102·2 E. 1	124	Large irregularly fibroid uterus. Ice to head 88 hours	Recovered	Continues well	422
461	Ditto	June	32	S.	5	None	Right	Ligatures	7½	103·6 M. 8	130	High temp. and pulse caused by trouble with bowels which were not properly attended to	Recovered	Continues well	423
462	Ditto	June	16	S.	10	None	Both (L.) dermoid	Ligatures	9½	100·0 O. E.	100	Malignant dermoid removed without tapping	Recovered	Continues well	424
463	Ditto	July	27	M.	6	Omental and pelvic	Right	Ligatures	6	99·8 O. E.	92	Removed tumour without tapping, as it appeared to be malignant	Recovered	Continues well	425

465	Samar. Hosp.	July	33	M.	4	Capsule so adhe-	Right	Enuclen.	5	101·4 E. 1	124	Glass drainage-tube	Recovered	Fistula quite closed in 3	427
						cleated tumour without opening peritoneum						mia caught from neighbouring case. Sloughing of sac and fæcal fistula			
466	Ditto	July	31	S.	4	Omental	Right	Enuclen. and ligs.	5	100·0 E. 2	96	Left tube cystic and also removed	Recovered	—	428
467	Private House	July	22	S.	4	None	Right	Ligatures	14½	100·6 O. E.	112	—	Recovered	Continues well	429
468	Samar. Hosp.	July	28	M.	4	Universal. Recent from twist of pedicle	Right	Ligatures	6	101·0 O. E.	112	Whole tumour black and infiltrated with blood	Recovered	Continues well	430
469	Private House	Aug.	36	M.	6	Universal. Recent from twist of pedicle	Right	Ligatures	11½	101·4 O. E. and E. 2	106	Subacute perito-	Recovered	Continues well	431
470	Nursing Home	Aug.	40	S.	3½	None. Twisted pedicle	Left	Ligatures	3	100·8 O. E.	92	—	Recovered	Continues well	432
471	Private House	Oct.	37	M.	6	Ruptured cyst. Extensive omental	Right	Ligatures	44	103·8 E. 4 107·0 ante mortem	136	Low septic condition with dark vomit for some time before operation	Died 9th day	Septicæmia. Both parotids suppurated	433
472	Samar. Hosp.	Oct.	59	W.	4	Extensive intestinal and pelvic from twisted pedicle	Both (L.)	Ligatures	13	100·8 O. E.	116	—	Recovered	Continues well	434
473	Nursing Home	Oct.	43	S.	5	Parietal, omental, and intestinal from twisted pedicle	Both (L.)	Ligatures	2	103·4 M. 3	104	Surgical scarlatina	Recovered	Continues well	435
474	Private House	Oct.	21	S.	4	—	Both (R.)	Ligatures	9½	101·0 E. 1	100	No metrostaxis followed	Recovered	Continues well	436
475	Nursing Home	Oct.	58	M.	6	None. Twisted pedicle	Left	Ligatures	5½	101·2 O. E.	88	—	Recovered	Continues well	437
476	Samar. Hosp.	Oct.	26	M.	4	None	Left	Ligatures	20	100·6 M. 1	100	—	Recovered	Continues well	438
477	Private House	Nov.	39	S.	4	Ruptured cyst from twisted pedicle. Subacute peritonitis	Both (L.)	Ligatures	3½	100·2 O. E. and 1 E.	106	Glass tube for 24 hours. Malignant papilloma in cyst	Recovered	Continues well	439
478	Nursing Home	Nov.	25	S.	5	None.....	Both (L.)	Ligatures	12	102·4 E. 1	120	Rise of temp. and pulse for a few hours only with coming on of menstruation	Recovered	Continues well and has not menstruated	440
479	Ditto	Nov.	39	M.	6	Universal adhesions to left cyst from twisted pedicle. Right. None.	Right dermoid Both	Ligatures	6	100·0 O. E.	86	Glass drainage-tube 54 hours	Recovered	Continues well	441
480	Samar. Hosp.	Nov.	54	M.	5	Parietal, omental, and intestinal	Left	Ligatures	19	99·2 E. 1	106	Glass drainage-tube 60 hours	Recovered	Continues well	442

No.	Place of operation.	Date of operation.	Age.	Condition.	Length of incision.	Adhesions.	Ovary removed.	Treatment of pedicle.	Weight of tumour.	Highest temperature.	Quickest pulse.	Special treatment, or special features of case.	Immediate result.	Subsequent history, and general remarks on case.	No. in antiseptic series.
		1885			Ins.				lbs.						
481	Samar. Hosp.	Dec.	52	M.	3½	None.....	Left	Ligatures	26	101·0 O. E.	108	—	Recovered	Continues well	443
482	Ditto	Dec.	58	S.	3½	Omental	—	Ligatures	11	100·2 O. E.	96	—	Recovered	Continues well	444
483	Ditto	Dec.	31	S.	4	None.....	Both (L.)	Ligatures	20	101·2 E. 1	92	—	Recovered	Continues well	445
484	Ditto	Dec.	56	S.	5	Intestinal and pelvic	Left	Enuclen. and ligs.	11½	100·6 M. 1	100	Base of tumour sarcomatous and bedded in pelvis	Recovered	Continues well	446
		1886													
485	Ditto	Jan.	55	M.	4	None.....	Left	Ligatures	33	100·6 E. 1	88	—	Recovered	Is quite well	447
486	Nursing Home	Jan.	25	S.	3½	Omental	Right	Ligatures	19	100·6 E. 1	100	—	Recovered	Is quite well	448
487	Samar. Hosp.	Jan.	24	S.	5	Ruptured cyst from twisted pedicle. Chronic peritonitis	Left	Ligatures	21	101·8 M. 2	132	3½ pints of serum removed from right pleura 12 days before. Glass drainage-tube 53 hours	Recovered	Is quite well	449
488	Private house	Jan.	65	W.	3½	Much enucleation in pelvis	Right	Enuclen. and ligs.	20	100·4 E. 1	92	—	Recovered	Is quite strong and well	450

In the column headed "Condition," S., M., and W. signify respectively single, married, and widow. In that headed "Ovary removed," the capital letters R. and L. in brackets following the word "both," denote that so far as could be seen the Right or Left ovary was first affected. In the temperature column, the letters and figures after the Fahr. temperature denote the time of day, evening or morning, and the number of days after operation at which the highest temperature was recorded. It has not been found necessary to give these time references for the pulses, as they nearly always correspond with the highest temperatures.

The word "Enuclen." is the printer's abbreviation of Enucleation.

Incomplete Ovariotomies.

No.	Place.	Date.	Age.	Condition.	State of parts found after the abdominal cavity had been opened.	Nature of operative procedure.	Immediate result.	Ultimate result.
8	Nursing Home	Jan., 1881	29	M.	Suppurating dermoid cyst opening into bladder	Portions of cyst removed, remainder sutured to parietes and drained	Recovered	Cancerous infiltration of surrounding parts and death in two months
9	Ditto	April, 1882	70	W.	Cancer of both ovaries, of peritoneum, and of lumbar glands	After removing the main part of the left ovary, I found its base incorporated with tumour of the right ovary, which could not be got out	Died in 40 hours	Suppression of urine
10	Samar. Hosp.	May, 1882	27	S.	Ruptured papillomatous cysts of both ovaries with general infection of peritoneum	Prolonged attempt to dissect them out and parts removed	Died in 4 hours	Exhaustion
11	Private house	August, 1882	28	S.	Cysto-sarcoma involving both ovaries and uterus	Cysts broken up and cleared out. Removal impossible	Recovered	Only lived a month
12	Nursing Home	Nov., 1882	45	M.	Papillomatous cysts of both ovaries, universally adherent	Cysts cleared out and masses of papilloma removed	Recovered	—
13	Samar. Hosp.	March, 1883	34	M.	Thin-walled multilocular cysts on both sides, probably ovarian	Universally adherent, parts peeled away, remainder sponged out and drained	Recovered	Remains well
14	Nursing Home	Oct., 1883	4	S.	Suppurating dermoid cyst cleared out, sutured to parietes and drained	Firmly bedded among adherent intestines	Recovered	Remains well
15	Private house	Nov., 1883	42	S.	Multilocular ovarian cyst, which had ruptured many times during ten years into peritoneum	Density of adhesions rendered removal impossible. Parts removed, cysts cleared out, and incision closed	Recovered	—
16	Samar. Hosp.	June, 1884	27	M.	Similar case to No. 13	Broken up and partly removed, remains allowed to fall back into the pelvis. No drainage	Recovered	Remains well
17	Ditto	Dec., 1884	24	M.	Thin-walled multilocular tumour of right ovary	Ditto	Recovered	Remains well
18	Private house	Jan., 1885	50	S.	Ruptured papillomatous cyst of right ovary, left ovary and tube papillomatous on surface and peritoneum generally so	Portions of papilloma and left ovary and tube removed	Recovered	—
19	Samar. Hosp.	May, 1885	50	M.	Cancerous mass involving both ovaries, uterus, and bladder	Large portion removed and then the anterior wall of the bladder found to be extensively involved	Died in a few hours	Exhaustion
20	Ditto	July, 1885	33	M.	Thin walled, possibly broad ligament cyst, universally adherent	Opened, anterior part removed, remainder sponged out with tincture of iodine, sewn to parietes and drained	Recovered	Remains well.
21	Nursing Home	Oct., 1885	56	S.	Cysto-sarcoma of left ovary and body of the uterus	After removing greater part found that the base involved the uterus	Died in 12 hours	Exhaustion.

Simple Exploratory Incisions.

No.	Place.	Date.	Age.	Condition.	State of parts found after the abdominal cavity had been opened.	Nature of operative procedure.	Immediate result.	Ultimate result.
7	Samar. Hosp.	July, 1880	42	M.	Large cystic tumours of both ovaries, mass of cancer in omentum and nodules over peritoneum generally	Fluid sponged out and several cysts tapped so as to allow of closure of incision	Recovered	Died at home in 3 months
8	Ditto	April, 1882	25	M.	General papilloma of peritoneum and exterior of ovaries; it had apparently commenced from the right ovary	Fluid merely pressed out. No sponging. Incision closed. No drain	Recovered	Is now in perfect health.
9	Ditto	Oct., 1882	41	M.	Peritoneum covered with papilloma; large false cyst formed by adhesion of papillomatous omentum, intestines, ovaries, and uterus	Fluid sponged out and incision closed. No drain	Recovered	Was tapped again in 2 years, and is now very ill.
10	Ditto	June, 1884	49	S.	Malignant tumour involving both ovaries and uterus; peritoneum infiltrated and full of dark fluid	Fluid sponged out, causing hæmorrhage. Incision closed. No drain	Died 7th day	Bronchitis.
11	Nursing Home	Jan., 1886	39	M.	Multilocular ovarian tumour with intestinal and pelvic adhesions which could not be separated	Sacs cleaned out with iodine, some portions removed. Drained	Recovered	Remains well.

Enucleation of Peritoneal Cyst.

1	Samar. Hosp.	June, 1885	30	M.	A very vascular single cyst full of blood-clot	Enucleated cyst without opening general peritoneum. Drained capsule	Recovered	Remains well.

The classification of these incomplete and exploratory cases is very difficult, and possibly it would have been more correct to place No. 20 of the incomplete cases among the mere exploratory incisions, and No. 11 of the latter among the incomplete ovariotomies. No 8 of the exploratory incisions is a most remarkable case. The whole parietal peritoneum was thickened and covered with a fleshy papilloma, similar growth was scattered in nodules over the omentum, intestines, uterus, and ovaries, that about the right ovary appearing so much more fully developed than the rest, that I thought this was probably the starting-point of the disease. The growth was so vascular that I did not venture to sponge out the fluid, but merely pressed it out and closed the incision. In a few weeks she was filling rapidly and in great distress at finding that she was not cured; then I heard that she was getting smaller again, and in November of last year heard from Mr. Mallam, of Oxford, that she was quite well, had been for the last six months menstruating regularly, and often walking ten miles in the day. I have met with one similar case, No. 29 in my Ovariotomy Tables, of which a full report will be found in the 28th vol. of the 'Pathological Transactions.' In that case I removed the papillomatous ovarian tumours, leaving the peritoneum covered with growths. When I had an opportunity of examining the patient some years later she was in perfect health, and all trace of the peritoneal growths had disappeared. These growths seem to be like external warts.

The numbers given with these cases are continuous with numbers of the same class of cases, published with previous Ovariotomy Tables.

ON A CASE

OF

EARLY DISSEMINATED MYELITIS

OCCURRING IN THE EXANTHEM STAGE OF MEASLES AND FATAL ON THE ELEVENTH DAY OF THAT DISEASE.

BY

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REPORT ON THE MICROSCOPICAL EXAMINATION OF THE SPINAL CORD, BY

F. G. PENROSE, M.D.

Received April 13th—Read November 9th, 1886.

JOSEPH W—, æt. 23, a policeman, of previous good health, was admitted under my care at the London Fever Hospital on June 11th, 1885, suffering from measles.

He had felt ill first on the 4th of June. Between that day and the 10th he suffered from cough and soreness down the breast bone, and at the pit of the stomach; but continued at his duty till the 10th, when the rash appeared.

On admission on the 11th the patient had a well-marked measles rash. It was most abundant on the face, neck, and trunk; but was also present on the limbs. There was some crescentic arrangement on the arms; but on the

trunk many patches were confluent, and some areas were as big as a shilling.

The superficial lymphatic glands were enlarged.

There was injection of the conjunctiva, and the nostrils were red and swollen, but there was no actual coryza. The tongue was coated with a thin and creamy fur, and there was slight punctate injection of the palate and fauces. There was no audible rhonchus, but the patient was hoarse, and complained of soreness down the breast-bone, and at the epigastrium. He had no backache, and had had no vomiting.

His pulse was 92, and his temperature 100·8°.

He was kept in bed on spoon diet and ordered some colocynth and hyoscyamus pills, and a linctus for his cough.

His bowels acted freely next evening, and he passed his urine naturally. His temperature on the 12th was 100° F. and 100·2° F. He still complained of pain down the sternum, and at the ensiform cartilage, and was ordered some benzoin inhalations, with a view to relieve it.

On the 13th the rash was fading, but was still very marked, and there was considerable intermediate staining of the skin of the trunk.

There was a slight apex systolic murmur to be heard. The pulse was 76, soft and regular. There were a few râles in the chest. Again the patient's only complaint was of the pain down the sternum and at the epigastrium.

At midnight it was ascertained that he had been several hours unable to pass his urine, it was therefore withdrawn by catheter and found to be quite clear and free from albumen.

His temperature this morning was 100, but rose in the evening to 103·8°.

On the 14th the patient's morning temperature was 103·2°. He was very drowsy. He could be roused, and then answered questions with a little irritability, but presently relapsed into his former drowsy state. He swallowed without difficulty, but required to be persuaded to take food.

His pupils were equal, of medium size, and they reacted somewhat sluggishly to light. There was no ocular or facial paralysis. His tongue, which was dry, brown, and fissured, was protruded in a straight line. The grasp of the hands was distinctly weak, but equal on the two sides. The patient declared he was unable to move his legs. The right leg was semiflexed by Mr. Arkle, the house physician, without giving rise to any pain, rigidity, or spasm, and was found in exactly the same position some hours subsequently.

The patient felt and localised a pinch on the two legs. The plantar-reflex was absent, and no knee-jerk could be obtained. There was no ankle clonus. There was still retention of urine. The urine was withdrawn by catheter and contained a very faint trace of albumen.

On examination of the chest, the systolic murmur before noted was found still present, and it could be traced into the axilla.

There was some deficient resonance at the left base, and weak breathing. In the afternoon I found the condition as Mr. Arkle had noted it. Taking the above signs in conjunction with the entire absence of head retraction, of rigidity of back muscles, and of anything like spasm of limbs, the diagnosis appeared to me against meningitis, and in favour of acute myelitis, with its focus in the anterior grey cornua.

I was the more inclined to this view because, although I had never before seen a similar case in the acute stage of one of the exanthemata, some examples of infantile paralysis had come under my observation, in which the history of onset pointed to their being sequels of measles or typhoid, and I thought that this might be the early stage of such a condition.

Taking that view I did not at this visit regard the prognosis as necessarily grave, so far as life was concerned, but I hoped the general symptoms had arrived at their climax, and looked forward to the subsequent localization of the paralysis in some group of muscles. There appeared no

immediate indication for therapeutic measures except the application of ice-bags to the spine, in addition to the one on the head which Mr. Arkle had already ordered.

During the evening the temperature rose to 104·2°, and thirty grains of antipyrin were given.

In one hour's time there was subsidence to 101·2°, but there was no improvement in the general state. The patient retched at times, but there was no actual vomiting. At midnight he was not quite so drowsy and answered questions slowly but quite intelligently. His temperature was then 99°.

On the morning of the 15th the patient continued less drowsy. His mental state was indeed quite clear. He had had no delirium throughout. He recognised his father, and evidently understood questions, and answered them intelligibly. Otherwise he was much worse. He could only speak in a whisper, and his cough was quite ineffectual, and half suppressed. When questioned as to headache he said he had aching at the backs of his eyes, but his chief trouble now, as from the beginning, was of fixed pain down the breast bone and at the epigastrium, and he said he had a weight on his chest. The paralysis had evidently increased; for in addition to the paralysis of the lower limbs, the absence of reflexes and the retention of the urine, the weakness in the grasp of the hands was more marked, and there was now paralysis of the intercostals so that the upper part of the chest was quite immovable, and the respiration was entirely diaphragmatic.

The tongue when protruded deviated slightly to the left, and there was a suspicion of slight flattening of the right naso-labial fold, but not enough to justify any importance being attached to it.

The pulse was 76, soft and regular. Respirations 28.

The temperature ranged during the day between 100·2° and 100·8°.

The urine was withdrawn, and found free from albumen. The rectum was emptied by enema.

The patient gradually became cyanosed with rapid respiration, clammy sweat, and tracheal râles, and died at 9 p.m. I regret that we had no opportunity of making an electrical examination.

The date of appearance of symptoms may be summarised as follows:

June 4th (first day of illness).—First felt ill.

Between 4th and 10th complained of cough, pain, and tightness referred to breast bone.

10th (sixth day of illness).—Rash.

13th (ninth day of illness).—Retention of urine.

14th (tenth day of illness).—Paralysis of lower limbs and loss of superficial and deep reflexes. Paresis of upper limbs.

15th (eleventh day of illness).—Paralysis affecting intercostals. Death.

The post-mortem examination was made twenty hours after death. Rigor mortis was well marked. There was much staining of the skin, the vestige of the measles rash. The heart was free from valvular disease, the right cavities were greatly distended with coagulum. There was consolidation of the lower lobe of the left lung, and intense congestion with ecchymosis of the trachea and bronchi. The liver showed some pallid areas, and there was a small calcareous nodule in the right lobe of uncertain origin. No syphilitic lesions were to be found in any part of the body. There was no scar on the penis, and no enlargement of glands in the groins, and the tibiæ were natural. The spleen was enlarged and rather soft. The kidneys appeared healthy. The intestines were natural. The calvaria was natural; there was much venous distension of the meninges, but no lymph. The grey matter of the convolutions was darker than natural. The brain-substance generally was unduly soft. The ependyma of the lateral ventricles was very easily detached.

In the spinal canal there was much venous engorgement. On slitting up the membranes no lymph was found. The



FIG. 1.—Portion of left vagus nucleus, showing congested vessel and escaped leucocytes. × 180. L = Leucocytes. N = Nerve-ganglion cell. V = Distended vessel.

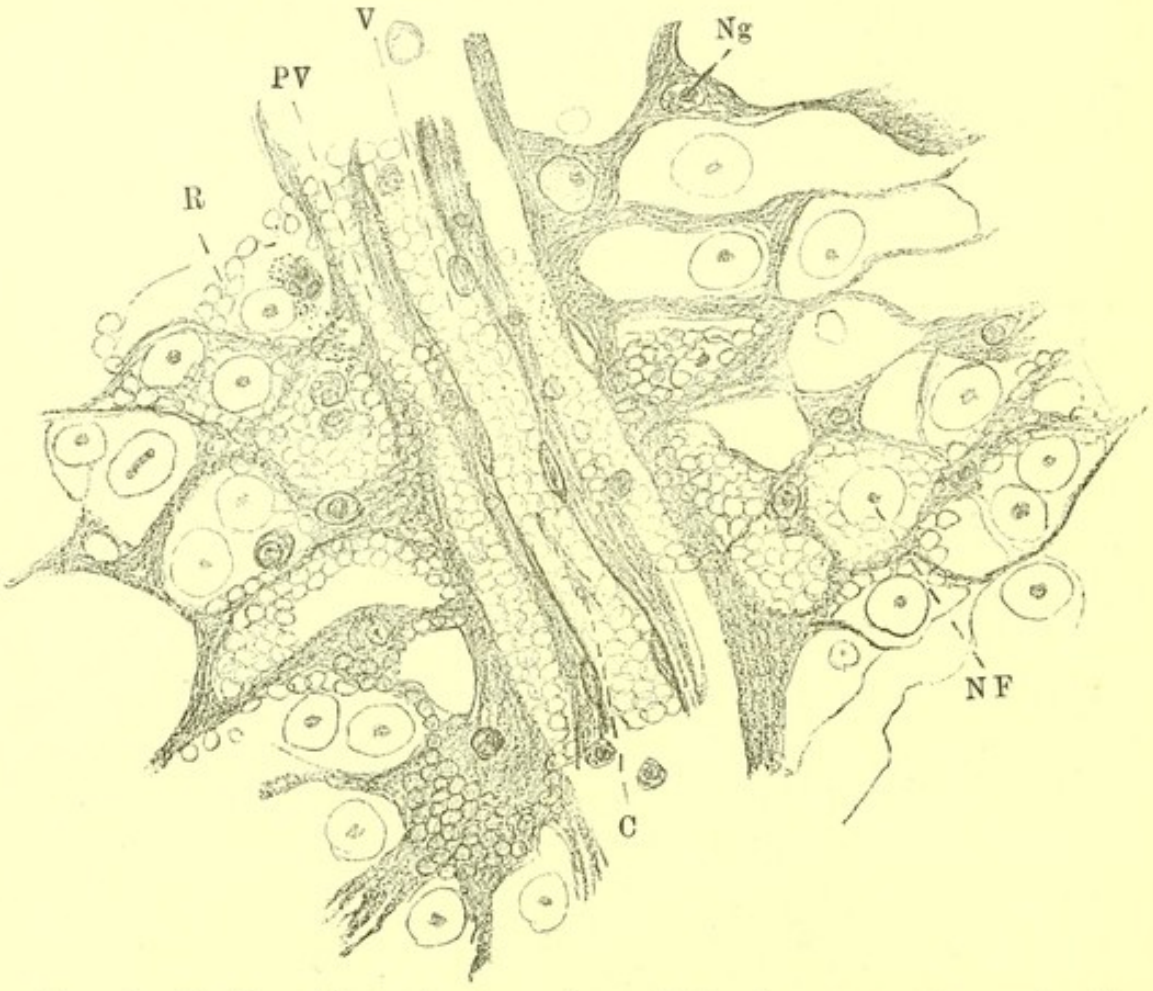


FIG. 2.—Portion of lateral column in cervical enlargement, showing longitudinal section of a small vessel; perivascular space distended and full of red blood-corpuscles; hæmorrhage into surrounding tissues. × 500. C = Clot in centre of vessel. N F = Trans. section of nerve-fibre. Ng = Neuroglia cell. R = Red blood-disc. P V = Perivascular space. V = Distended vessel.

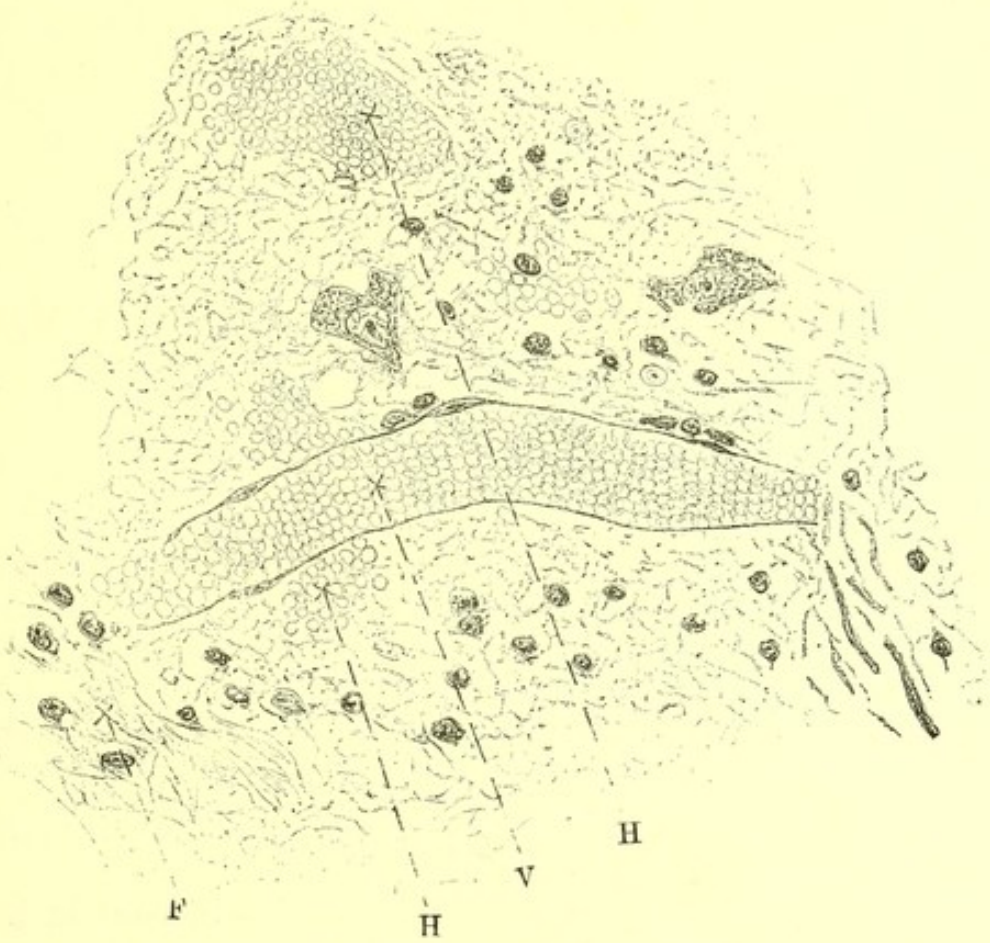


FIG. 3.—Portion of anterior cornu in lower dorsal region of cord, showing small hæmorrhages into grey substance. × 225. F = Fibrinous exudation. H = Hæmorrhage. V = Distended vessel.

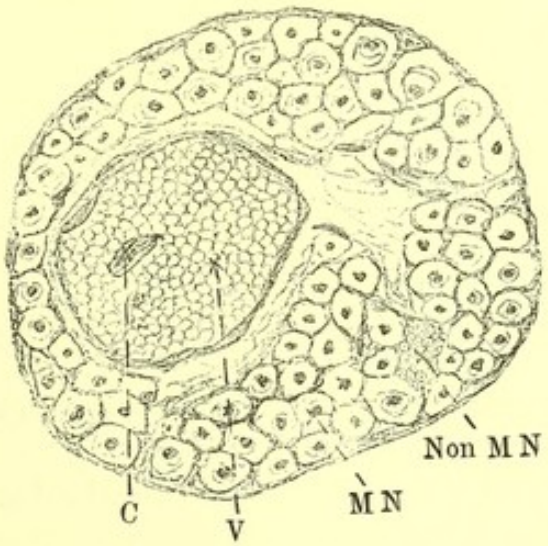


FIG. 4.—Transverse section of anterior root of a spinal nerve from dorsal region of cord, showing distended vessel. × 380. C = Clot in vessel. V = Distended vessel. M N = Medullated nerve-fibres. Non-M N = Non-medullated nerve-fibres.

DESCRIPTION OF PLATES I AND II.

(Case of Early Disseminated Myelitis, THOMAS BARLOW, M.D.)

PLATE I.

FIG. 1.—Transverse section of medulla oblongata. There are several congested vessels in the grey matter of the floor of the fourth ventricle. They are especially noticeable in the region of the left hypoglossal and vagus nuclei. × 9.

Hy = Hypoglossal nucleus.
Va = Vagus nucleus.
V = Distended vessel.

FIG. 2.—Semi-diagrammatic sketch of section of cord in lower cervical region. × 13.

H = Hæmorrhage.
V = Distended vessel.

Showing in both grey and white substance distension of vessels and exudation around many of them. There is a small hæmorrhage in the left anterior cornu.

PLATE II.

Portion of transverse section of lower part of dorsal region of cord (from a photograph), showing—

H = Hæmorrhage into grey matter.
V = Distended vessel.

The distended vessel marked V in the posterior column is surrounded by exudation. × 30.

For this photograph I am indebted to the kindness of Edgar Crookshank, M.B.

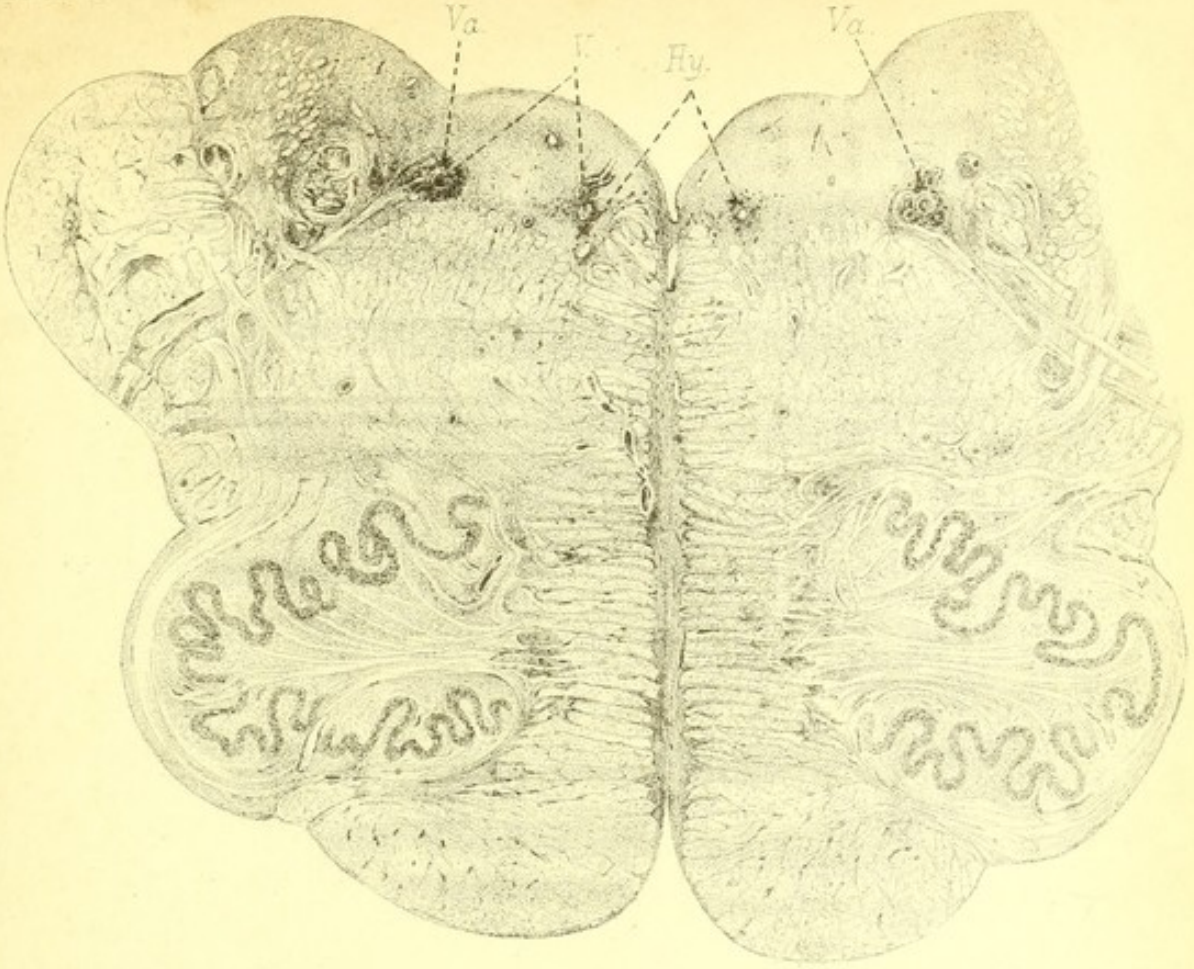


Fig. 1.

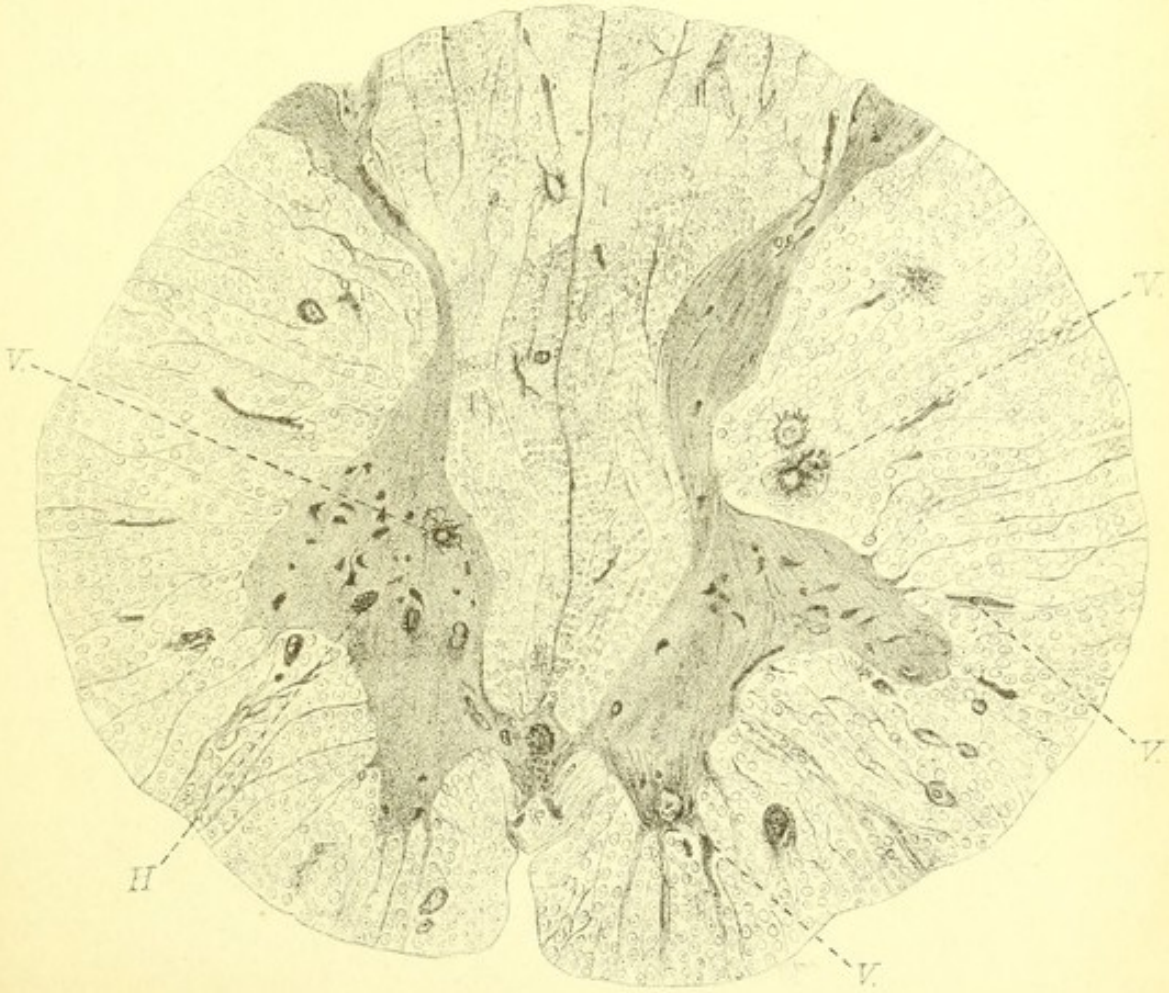
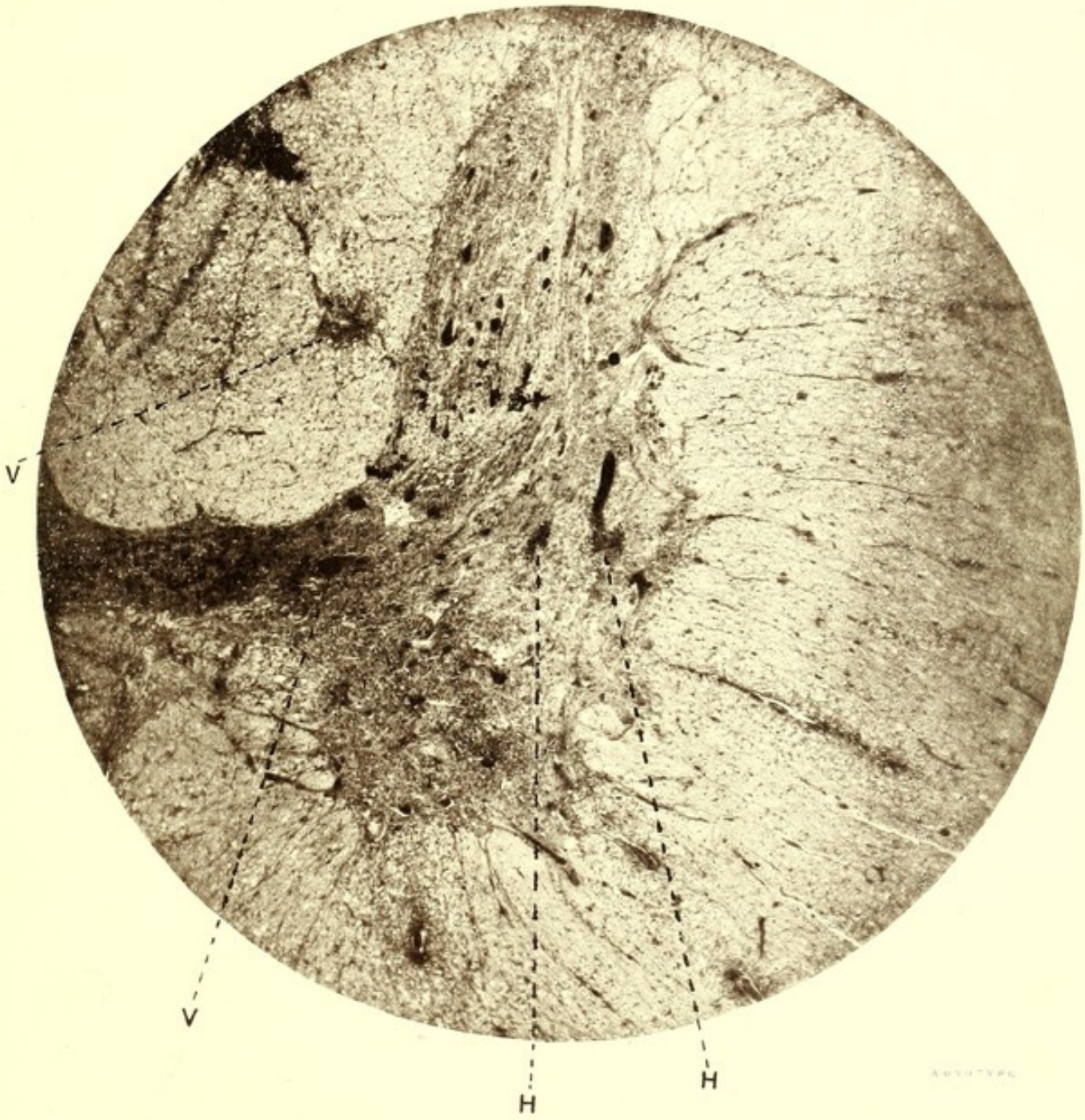


Fig. 2.

Danielsson & Co, lith.


V'
V
H
H

whole of the cervical enlargement was swollen and softer than natural.

At a situation corresponding with about the fifth dorsal vertebra, the whole of the cord was diffluent, and it could not be removed intact.

The lumbar enlargement, although it felt tolerably firm externally, was found on section to present some softening in the lateral and posterior columns, and the grey cornua seemed to have been the seat of small hæmorrhages, being discoloured, and in places tunnelled out, and the substance very pulpy.

The cord was hardened in a 2 per cent. solution of bichromate of ammonium, and I have to thank Dr. Penrose for the microscopic examination of it.

His report is as follows :

Examination of the cord shows that the changes are entirely vascular in character, that they are universally distributed, but of greater intensity in some regions than in others. There is great engorgement of the vessels, most of the veins being many times their normal calibre and crammed with corpuscles. Many vessels are surrounded by a zone of coagulated exudation, and beyond this for a considerable distance the surrounding tissues are infiltrated with leucocytes, giving the appearance in transverse section of a series of concentric rings.

Beyond extensive diapedesis there is further evidence of the severity of the congestion, shown by rupture of vessels with subsequent slight interstitial hæmorrhage.

The portion of the cord which had suffered most was the upper dorsal region, which was so soft that it was impossible to make sections of it. From this region the damage seems to have radiated upwards and downwards through the whole extent from the medulla to the lower part of the lumbar portion.

Throughout, the anterior cornua have been most affected, as it is chiefly in them that the hæmorrhages are to be found, but vascular changes in the other parts are nearly as great, and, moreover, are not confined to the

cord, for in both anterior and posterior nerve-roots the vessels are much engorged.

No changes have been found in the nerve-cells, fibres, or supporting tissues of the cord, and staining by Gram's, and the ordinary methods, has failed to demonstrate the presence of micro-organisms.

In the medulla oblongata the changes are very similar to those in the cord, except that no hæmorrhages have been seen. The stress seems to have fallen chiefly on the hypoglossal and vagus nuclei, and, from the sections which have been made, would appear to have been greater on the left than on the right side.

Commentary.

There could be no doubt during life that this patient was suffering from measles, and within a fortnight of his admission a comrade of his who slept in the same room at the police station was admitted to the London Fever Hospital also suffering from measles, which he passed through without complication.

Also the post-mortem appearances and the microscopic examination establish, I think, without doubt the existence of early myelitis.

The changes were, as Dr. Penrose has demonstrated, vascular in character, and they had led to such damage to the cord that in one part it was diffluent and in other parts there was red softening. The myelitis was of the disseminated form, for the lesions found were not along systematic lines but varied in intensity and distribution at different levels and in different regions of the cord. The retention of urine, the paralysis of lower limbs, and paresis of upper limbs, the paralysis of intercostals, the absence of knee-jerks and of plantar reflexes, were all spinal symptoms; and it seems probable that the pain down the breast bone and at the epigastrium, which was complained of by the patient even before the rash appeared,

was also a spinal symptom. The fatal issue may, I think, be referred in part to the involvement of the medulla. The loss of voice, the ineffectual paralytic cough, perhaps even the rapidly developed pneumonia, deserve to be thought of in connection with the damage to the vagus nuclei; and it is possible that the deviation of the tongue to the left which came on under observation may have had some relation to the damage to the hypoglossal nucleus. But although it be granted that the patient was the subject of measles and of myelitis, is it certain that there was any necessary connection between these two affections?

It is fair to state that in reply to inquiries made after the patient's death the father informs me by letter that his son's previous health had been good, but that for a month before his death he had complained of his chest. The patient himself, however, told me positively that his illness dated from June 4th, and only referred to the pain down the breast bone as having troubled him from that time forward.

Further, he had kept to his police duty until the 10th, on which date the rash appeared. I think, therefore, that there is no sufficient reason to suppose that the myelitis dated from a period anterior to the invasion of the measles.

The question now arises, Is there anything in the records of measles and its complications which bears on the case under discussion?

The clinical facts, though not abundant, are more to our purpose than the pathological, which are very scanty indeed. The earliest case in point that I have been able to find is recorded by Mr. James Lucas in 1790.[1] A woman, aged twenty-three, was feverish on the first day of her illness, developed the rash of measles on the third day, the rash was declining on the eighth day, and on the ninth day she became paralysed in both lower limbs. On the tenth day she had retention of urine, obstinate constipation, and a feeling of fulness of the abdomen. The

[1] 'London Medical Journal,' 1790, p. 325.

retention continued and for ten days the catheter was needed. The paraplegia was complete for one week, and two days after that time entirely disappeared. It was ascertained that the same patient, when attacked by smallpox nine years before, had been similarly affected with regard to her limbs and had completely recovered.

M. Liégard[1] relates the case of a child, aged two, who at the beginning of convalescence from measles developed an ascending paralysis of lower limbs, upper limbs, back and neck muscles, with difficulty of mastication and deglutition. There was loss of sensation in the limbs but no affection of sphincters and no convulsions, tremblings, or contractures. The child recovered completely in three weeks.

M. Bergeron[2] records a fatal case of paralysis after measles. A child, aged three, was admitted to hospital with measles, and in eight days afterwards developed superficial gangrene of one ear. About one month afterwards, when cicatrisation was nearly complete and the child's condition was satisfactory but for a certain amount of languor, she suddenly became paralysed in all four limbs and deglutition became difficult and the voice nasal. Ultimately the diaphragm and respiratory muscles became paralysed and the sphincters likewise, and the child died with asphyxia eleven days after the onset of the paralysis and about sixty-two days after the onset of the measles.

Post-mortem examination showed extensive pneumonia. The brain was congested, but no microscopic change could be detected by Ranvier. The cord is said to have presented very marked congestion, and this was attributed by M. Bergeron to the asphyxial condition, but no record is given of any microscopical examination.

The question may now be asked, Can any light be thrown on the present case by records of nervous complication in any of the other exanthemata?

A most satisfactory parallel is forthcoming in the first

[1] 'Gazette des hôpitaux,' 1859, p. 562.

[2] 'Gazette des hôpitaux, 1868, p. 5.

fatal case recorded by Westphal[1] in his paper on "Disseminated Myelitis."

A man, aged thirty-two, on the eleventh day after the first appearance of the rash of smallpox, had incontinence of urine. On the twelfth day he had retention of urine and paralysis of the left lower limb. On the thirteenth day both lower limbs were paralysed. Within the next few days he had involuntary defæcation. On the seventeenth day a sacral bedsore developed. The motor paralysis was complete, but sensation was only diminished. There was a feeling of constriction round the abdomen from the navel outwards. The muscles of the lower extremities responded to a strong induced current.

Catheterism was necessary and cystitis developed. The bedsore increased. Rigors and pyrexia set in. The sensorium became blunted and the patient died four weeks after the onset of the paraplegia. At the autopsy the bedsore was found to have extended down to the sacrum and the trochanters.

The pia mater of the brain was œdematous and moderately opaque in the sulci. There was no focus of disease in the hemispheres or medulla. The membranes of the cord were soft. There was marked congestion of the grey substance. In the lumbar region considerable difference was observed between the grey substance of the right side and that of the left.

Whilst the right anterior and posterior cornua were of the usual grey colour the left horns were of a dark reddishbrown. This condition extended up to the lower dorsal region. Above this the dark colour was manifest in the grey cornua of both sides. Higher still the normal colour reappeared. In the upper dorsal region there was a considerable extent of softening, so that the left anterior horn, with a little of the left posterior horn, was actually hollowed out. There was also slight hollowing out, consequent on softening in the right anterior horn.

[1] 'Archiv für Psychiatrie,' Bd. iv, 1873, p. 338.

The white substance was very pale but showed no other naked-eye change. On microscopic examination the blood-vessels were found remarkably dilated, filled with blood-corpuscles, and invested with sheaths of granular corpuscles. There was extensive infiltration of these granular corpuscles, especially in the grey matter, which throughout was prominently and often exclusively affected. In the irregular walls of the hollowed-out regions there was a great heaping-up of granular cells.

No hæmatoidin crystals were to be seen, and the dark brown colour in the softened regions was found to depend on the enormously distended vessels sheathed with granular cells.

The ganglion-cells were natural.

It will be observed how very remarkable is the resemblance in many respects, both clinical and pathological, between the above case and the one under discussion—the differences being rather of degree than of kind, and corresponding with the more acute and rapid course and the more extensive involvement of the nerve-centres in my case as compared with Westphal's. There is a second case, recorded by Westphal, of paraplegia commencing about three days after the beginning of the rash of small-pox and in which, post mortem, myelitis was found especially affecting the lateral columns, but as this case diverges considerably, both clinically and anatomically, from the one under discussion it need not further be considered.

I think enough has been said to justify the view that the myelitis in my case was not a mere asphyxial effect but an integral part of the acute specific disease from which the patient suffered. Whether it was produced by micro-organisms which we assume to be concerned in the other phenomena of measles I will not presume to say, but, at all events, its evolution was very nearly parallel with the skin and respiratory mucous membrane affections of the disease.

There only remains for me to ask the question (which has been partly broached by others) whether cases like

the one under discussion may not be regarded as a middle term between two clinically well-recognised spinal sequelæ of acute specific diseases.

The first group, originally described by Westphal,[1] comprises cases which present symptoms very like those of multiple sclerosis, the said symptoms being definite sequelæ of smallpox and typhoid.

To this group there have been added by subsequent observers more or less similar cases accompanying or following measles, scarlet fever, pneumonia, ague.[2]

It has been cautiously suggested by Westphal in one of his papers that a slighter degree of mischief than that found in his cases of fatal disseminated myelitis might have given rise to changes which eventuate in something resembling multiple sclerosis.

The second group of spinal sequelæ of fevers to which I refer comprises cases resembling infantile paralysis. Westphal is very careful to emphasize that in his first case of disseminated myelitis there was no damage to the great ganglion-cells, and the same fact obtains with regard to the case under discussion.

But it is easy to understand how such a vascular storm as we have pictured before us, if a little more localized in the anterior horns, might have swept away a number of ganglion-cells and thus induced atrophy of separate groups of muscles in relation to them.

Of such atrophies, in every respect resembling infantile paralysis, we have a fair number of cases on record as sequels of the acute specific diseases.

[1] 'Archiv für Psychiatrie,' Bd. iii, 1872, p. 376.

[2] For further cases and for bibliography of this group of nervous sequelæ (in which symptoms resemble the clinical type of multiple sclerosis), vide Whipham and Myers, 'Clin. Trans.,' vol. xix, 1886, p. 164.

(For report of the discussion on this paper, see 'Proceedings of the Royal Medical and Chirurgical Society,' New Series, vol. ii, p. 146.)

ON THREE CASES

OF

ACUTE TUBERCULAR ULCERATION OF THE FAUCES.

BY

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We propose briefly to narrate the cases upon which this paper is based, and then to offer a few remarks upon the disease.

Case 1.—Henry A—, æt. 7, was brought to the out-patient room at the Hospital for Sick Children, Great Ormond Street, in February of this year. When first seen his mother only complained of his wasting and of the enlarged glands in his neck; at that time the latter were very large. Nothing amiss was detected in his fauces. He was a delicate, thin child of a somewhat tubercular aspect and had been under treatment during the preceding

summer for whooping-cough. He had not had any other ailment.

Three weeks later, on March 8th, we learnt that he had been complaining of sore-throat and difficulty in swallowing for one week, and the soft palate and uvula were found to present numerous minute yellowish superficial points; the tonsils were not visible; the glands in his neck were certainly smaller than when first seen.

The mother further told us that since the birth of this child she had had one other but that it died shortly after birth. The child's father was said to be healthy, but there was a history of phthisis in his family, and one of his brothers, who was considered consumptive, had died at the age of twenty-eight with some throat affection, loss of voice having been a prominent symptom, and it was owing to this fact that on this occasion the mother asked us whether her child's throat was "consumptive."

Next week the child was too ill to be brought, so we saw him at his parents' home; he was restless and feverish at night, was taking his food badly, and rapidly losing flesh. The soft palate and uvula presented the same yellowish speckled appearance that they had on the previous occasion except that this was now much more marked; the specks were each about the size of a pin's head and in places were so numerous as almost to be confluent, the intervening mucous membrane being pale. There was much tenacious secretion about the fauces; the glands in his neck had still further decreased in size. Abundant sharp râles could be heard all over the chest, but owing to his feeble state no thorough examination could be made.

Beyond rapid emaciation and steadily increasing debility there was nothing in the after-progress of the case calling for comment, and he died a fortnight after the throat affection was first recognised and three weeks after the first complaint of sore-throat. The night before his death he was delirious. The body was examined nine hours after death.

The glands in the neck proved to be hardly, if at all, enlarged, and none were caseous. On examining the fauces and larynx very extensive, but superficial, ulceration was found affecting the posterior or nasal aspect of the soft palate, spreading down and around the pharynx; the anterior aspect of the uvula and soft palate was in a similar state. The tonsils were not enlarged or in any way affected; the ulceration did not extend into the œsophagus.

The epiglottis was greatly thickened, the lining membrane on its inner aspect as well as that of the whole larynx showing a very acute, but quite superficial, punctiform ulceration; the lining membrane of the trachea was very deeply injected. The drawing on Plate III shows the situation of the lesions well. In the specimen itself the effects of the spirit render it difficult to recognise the changes which we have endeavoured to describe.

There were general pleural adhesions over both lungs, those on the left side appearing to be of the longer standing. Both lungs were almost solid throughout from abundant deposits of closely aggregated tubercles, the lower lobes being the least affected; the upper lobe on the left side had already broken down in places into small cavities. The heart and pericardium were natural. The tracheal and bronchial glands were enlarged but not caseous. A few very early tubercles were found in the capsule of the spleen; the liver and kidneys were normal. The ileum was opened just above the cæcum and found to be ulcerated. Dr. F. W. Mott has been kind enough to examine a portion of the ulcerated pharynx for us and has given us the subjoined report: "Under a low power (100 diameters) the sections, stained with logwood and mounted in oil of cloves and Canada balsam, presented the following appearances: A round-celled infiltration throughout the mucous and submucous tissues, and involving the superficial layers of the muscle to a slight degree. The round cells are especially abundant near the surface, and are collected into little aggregations

forming granulations. Nowhere have these granulations undergone caseation. The mucous glands present the following appearances: The epithelial cells are everywhere destroyed and replaced by granular disintegrated matter. Around the alveoli outside the basement membrane are great numbers of small round cells. A few giant cells are seen. With high power ($\frac{1}{16}$) in oil immersion (sections stained with the Weigert-Erlich method and mounted in oil of cloves and Canada balsam) numerous bacilli tuberculosis are seen, especially abundant in the collections of small round cells, many being contained within the cells, others lying between them. Upon the surface no epithelial cells are seen, only round cells, disintegrated matter, and micro-organisms, for the most part not tubercular."

The tubercular nature of the ulceration is thus fully established.

Case 2.—George A. S—, æt. 3 years 5 months, was admitted into the Hospital for Sick Children on August 27th, 1880, under the care of Dr. D. B. Lees, who has kindly permitted us to report the case.

The mother gave the following history. The child has been ailing since he had whooping-cough eighteen months ago, cough being the chief trouble. He has had diarrhœa for four months, and been passing blood in his motions; and also for the last two months the motions have contained matter. He has had lumps in his neck for six months, these have been worse for the last month. About six weeks ago he complained of sore throat, and for three weeks drinks have returned through his nose, and his speech has been indistinct and nasal. There are three other children, healthy. The parents are healthy, but there is a history of "consumption" on the mother's side.

On admission the child was found to be pale and wasted. The tongue was clean, the tonsils a little swollen, and the glands in the neck decidedly enlarged. The child's speech was so indistinct that it was quite unintelligible; fluids did not return through his nose, nor did drinking cause

him to cough, but it was noticed that he drank slowly and with great caution. His chest was pigeon-breasted, and his ribs beaded; there was no dulness; there was a fair entry of air all over, and no adventitious sounds were heard. The abdomen was big, not very tense, tympanitic everywhere, more resistant on the right than the left side; no fluctuation; the spleen could not be felt. Limbs somewhat rickety.

The after-progress of the case may be briefly reported. He got steadily weaker, there was marked but irregular fever, some sharp moist râles became audible in his chest, and he had diarrhœa. A few days before his death it was noticed that there was superficial ulceration of the soft palate on the left side close to the base of the uvula, and this ulceration spread and became deeper, the edges of the ulcerated area presenting a ragged appearance. The condition was recognised as being probably of a tubercular nature. Death occurred on September 9th.

At the post-mortem examination there was found a superficial ulceration of the soft palate, extending along its free margin on both sides of the uvula, the edges of the ulcerated portion being irregular. The pharyngeal aspect of the soft palate and the posterior wall of the pharynx presented a superficial honeycomb appearance, formed by numerous small, irregularly-shaped bosses of substance of the mucous membrane alone. There was some doubt as to whether there was any thickening of the soft palate where it was not ulcerated; the epiglottis was thickened, the larynx healthy.

General tuberculosis of both lungs was found, the signs during life having no doubt been in some degree masked by the rather unusual amount of emphysema which was present. There was extensive ulceration of the small intestines, ulcers being also found in the ascending colon, the sigmoid flexure, and even in the rectum. The mesenteric glands were enlarged and caseous. The liver and spleen contained caseous nodules. A large caseous mass was found in the left lobe of the cerebellum, and some

transparent granulations in the left Sylvian fissure. There was pus in the internal ear, and some caries of bone. There were no tubercles in the choroids.

Case 3. John R. M—, æt. 6 years 3 months, was admitted into the Hospital for Sick Children on July 1st, 1881, under the care of Dr. Cheadle, by whose kind permission we are enabled to report the case. His history was that, two months previously a lump had appeared under his chin, and that this had been opened at one of the general hospitals, and had been discharging ever since; other lumps had appeared in his neck and under the jaw soon afterwards, and had continued to increase in size. For two weeks previous to his admission he had been complaining of a sore throat and pain on swallowing, but there had been no return of fluids through his nose, and no alteration in his voice. He had been losing flesh, was feverish, and at times had a cough. His appetite was stated to be good; his bowels confined. The parents were healthy, and denied any consumptive taint in their families; they had two other children in good health and had lost five, all, however, under the age of two years.

On admission the patient was thin and febrile, his tongue was furred; the tonsils and uvula were swollen, but not red; both here and on the soft palate some minute white specks could be seen; the glands in his neck were greatly enlarged, and there was a discharging abscess under his chin. Nothing was found in the chest except some coarse rhonchus all over, the abdomen appeared natural, and his urine was normal.

A few days after his admission it was noticed that there was distinct irregular abrasion of the mucous membrane on the posterior wall of the pharynx, but the examination of the fauces was always attended with great difficulty, owing to the large amount of viscid secretion that seemed to be constantly there, and to the patient's inability to open his mouth widely. There was pain on swallowing, and pain in his left ear after drinking quickly.

There had been a considerable degree of fever, and he perspired much during sleep.

After this there was nothing further calling for notice; the fever and emaciation continued, and no satisfactory examination of his throat or chest was possible; his voice was reduced to a mere whisper. He died on July 29th.

At the post-mortem examination, the whole of the pharynx was found to be extensively but superficially ulcerated, the ulceration extending to both aspects of the soft palate, and into the thyroid pouches; below it ceased at the œsophagus. The base of the tongue showed superficial erosion. The epiglottis was very thin, and the mucous membrane of the larynx above the cords was extensively ulcerated; the cords were healthy. A large abscess-cavity was found beneath the jaw on the left side extending round to the back of the pharynx.

Both lungs were studded throughout with tubercles, the tracheal and bronchial glands were enlarged and caseous. From the middle of the jejunum to the ileo-cæcal valve the small intestine showed most extensive ulceration, some of the ulcers completely encircling the gut; the large intestine was healthy. The mesenteric glands were enlarged and caseous, and several of them had broken down. No tubercles were found in the other viscera; the liver was somewhat fatty.

Although in these two latter cases no microscopical examination of the pharynx was made, we do not think there can be the slightest doubt that the ulceration was tubercular.

We have ventured to bring these cases before the Society, not so much in respect of their rarity, which we believe to be more apparent than real, but because the subject of acute pharyngeal tuberculosis has received scant notice in this country. The only cases we have met with in English medical literature are those described by Dr. Gee in the 7th and 11th volumes of the 'St. Bartholomew's Hospital Reports,' and those mentioned by Dr.

Mackenzie in the second volume of his work on 'Diseases of the Throat.' Our medical writers ignore the subject or pass it over with the remark, either that tuberculosis of the pharynx is rare, or that it is always consecutive to tuberculosis of the larynx.

On the Continent, however, and especially in France, the subject has met with far more attention. In Paris, cases have repeatedly been brought before the Société Médicale des Hôpitaux, the record of which will be found in almost every volume of the 'Bulletins' of that Society for the past fifteen years, whilst the subject has been specially treated of in more than one thesis, and notably by M. Barth[1] in 1880. The name of M. Isambert, too, ought to be mentioned as one of the earliest, if not actually the first, in that country, to recognise the nature of the affection, and give a concise description of the disease. In Germany, Wendt, Krause, O. Weber, E. and B. Wagner, and Fränkel have published cases, the last mentioned having made important contributions to the literature of the subject.

As to the relative frequency of this affection the following particulars may be of interest: Fränkel found pharyngeal tuberculosis only once out of 150 necropsies conducted at the Pathological Institute of Berlin during 1865-6,[2] but later, in 1881,[3] he got a much higher percentage, as he found 10 cases of pharyngeal tuberculosis out of a total of 50 cases examined. Navratil[4] found that in 20 out of 246 cases of laryngeal tuberculosis the disease had spread to the pharynx. Boecker and Schoetz stated to the Medical Society of Berlin, during a discussion on a case described by Krause, that they had met with 12 cases of pharyngeal tuberculosis in a total of 2950 cases of throat disease seen by them in two years, and Boecker mentioned

[1] 'De la Tuberculose du Pharynx, et de l'Angine Tuberculeuse.' Paris, 1880.

[2] 'Berliner Klin. Woch.,' 1876, pp. 657, 678.

[3] 'Zeitschrift für Ohrenh.,' x, 2, p. 113.

[4] 'Études Laryngoscop.,' Leipzig, 1871, p. 28.

having seen 8 cases in addition to these. Gurovitch[1] mentions 11 cases that had come under his own observation, and thinks that a certain number of cases must occur in the practice of most laryngologists. Unfortunately, in regard to the above statistics, it is impossible to tell which were instances of acute, and which of chronic, pharyngeal tuberculosis, a point to which we shall refer again directly. The post-mortem records of the Hospital for Sick Children, for the twenty-five years ending in 1884, yield 8 cases of acute tubercular ulceration of the fauces out of a total of 380 cases of tuberculosis; two of these are given in this paper (Nos. 2 and 3) and two have already been referred to as published by Dr. Gee.

We believe that a sharp distinction can, and ought, to be drawn between chronic and acute tubercular ulceration of the fauces. The former is essentially a local tuberculosis commencing in the pharynx or spreading there from the larynx, the latter is only a part of general tuberculosis.

Krishaber[2] and Barth[3] have shown that the affection may commence in the soft palate as an acute tuberculosis of the small adenoid glands. Microscopically, they found the follicles swollen and infiltrated with lymphoid cells, more numerous and more closely packed than in the normal state. In the centre the cells degenerated, the reticulum disappeared, and an abscess was formed, discharging externally, and causing a small rounded ulceration. In the two cases recorded by these authors, very acute general tuberculosis accompanied the condition, and death occurred in a few days. Wendt[4] describes a destructive follicular pharyngitis, which he terms pharyngitis scrofulosa, occurring in tuberculous subjects and also in the course of scrofula, in congenital and acquired syphilis, and even in healthy people. The follicles are attacked in

[1] 'St. Petersburgh Dissertation.'

[2] 'Thèse de Paris,' 1877.

[3] Loc. cit.

[4] 'Ziemssen's Cyclopædia,' vol. vii, p. 74.

great numbers in the lower part of the pharynx, ulceration ensues, not only in the follicles, but also in the intervening tissue, leaving a large ulcer on the posterior wall. Some of these ulcers, he says, showed nothing characteristic as to their origin, whilst others were follicular. It would seem probable that some of his cases were purely tubercular, whilst others were purely syphilitic, and some may have been, as Wendt seems inclined to think is frequently the case, the result of the combined effects of the two diatheses.

In the acute form (to which we consider our cases belong), the first sign is the appearance of small discrete papules on the soft palate, pillars of the fauces, and, still more rarely, on the posterior wall of the pharynx. In colour they are white, grey, or yellowish, and seem firmly embedded in the mucous membrane; they are indeed deposits of tubercle, and present the usual microscopical characters of that neoplasm. They soon attain a larger size, caseate, and undergo ulceration, rapidly implicating the surrounding parts, and ultimately causing great destruction of the mucous membrane lining the soft palate and adjoining parts. The mucous membrane in the immediate neighbourhood of the ulcers is swollen and congested, with tubercles scattered through it, and is often covered with a viscid, whitish secretion or exudation presenting some resemblance, at first sight, to diphtheritic membrane, but less organised and more friable. The retro-pharyngeal and cervical lymphatic glands become enlarged, and the latter sometimes suppurate; sometimes, and this happened in our Case No. 1, the enlargement of the glands entirely subsides before the termination of the case. The uvula is sometimes greatly thickened and elongated.

Of the ætiology of the acute affection we know nothing; whilst in regard to age, we find that it is most liable to occur at the period when tuberculosis is most frequent. Only four cases of its occurrence in children have been hitherto recorded, so far as we know, viz. two by Dr. Gee,

the patients being six and eight years old respectively, one by Wendt, who found a small tubercular ulcer on the pharyngeal tonsil of an infant nine months old (we are doubtful whether this case ought really to be included), and one by Isambert[1] in a child of four and a half years.

The first symptom, and often a very distressing one, is pain in the throat accompanied by more or less of dysphagia. On inspection of the fauces the greyish-white or yellowish punctiform speckled appearance, which we have endeavoured to describe above, will be seen, and is highly characteristic of the disease, though, owing to the large quantity of viscid secretion which so often covers the fauces in these cases, it is frequently difficult to establish its existence. In the course of a very few days these spots enlarge, coalesce, and break down to form small rounded ulcers, with the mucous membrane surrounding them swollen and sometimes congested. The edges of the ulcers are irregular, the bases have a caseous aspect, and they are surrounded by nodules in various stages of development preparatory to breaking down into ulcers. Eventually the ulcers coalesce to form one extensive raw surface, still covered with the viscid secretion already referred to. The disease appears not to spread into the œsophagus, but in one of the unrecorded Great Ormond Street cases mention is made of an ulcer in the œsophagus. The larynx does not often escape, and when it does, this is probably owing to the rapidity with which the disease has proved fatal. In our own cases and in others the onset of the laryngeal affection was marked by aphonia. It is not usual for the larynx to be extensively involved, and Fränkel notices that in none of his cases was any perichrondritis present. A nasal quality of voice from the impaired action of some of the palatal muscles is almost the rule, and pain in the ear from implication of the Eustachian tube is very common, as has been pointed out by Traube and Barth, but Fränkel having failed to recognise any lesion of the Eustachian tube with the

[1] 'Bull. de la Soc. Méd. des Hôp. de Paris,' 1876.

rhinoscope surmises that the pain is reflex, travelling along the glosso-pharyngeal and Jacobson's nerves. Deafness has sometimes been found.

Well-marked physical signs in the lungs will generally be found, but Isambert not being able to detect any in some of his cases concluded, as we think somewhat hastily, that the disease might be primary in the pharynx. The inability to detect any physical signs in the lungs in a case of suspected tuberculosis does not, in our opinion, go far to establish that such tuberculosis does not exist. Intestinal ulceration would appear to be almost constant, though during life there is frequently no proof of it. There is always fever, and death is often ushered in by delirium, being due to exhaustion consequent upon the dysphagia as much as anything else.

As regards the diagnosis it is only in the earlier stages that there should be much difficulty. Follicular tonsillitis and diphtheria are the conditions which would first have to be eliminated, and this may be a matter of some difficulty. The tubercular affection is much more likely to begin on the soft palate than the tonsils, which may escape altogether, whilst the other maladies almost invariably commence with swelling of the tonsils.

Attention should always be paid to the mode of onset, but in many instances it must be admitted that it is almost impossible to recognise the complaint in the incipient stages. At a later period, the coalescence of the spots to form superficial ulcerations has very little in common with diphtheria. Syphilis is perhaps the only other disease which might produce a condition resembling the tubercular disease under consideration. The mucous patches of secondary or inherited syphilis would be distinguished by the absence of any surrounding inflammation or papules, by their co-existence on the mucous membrane of the cheeks and gums and on the tongue, and by their very slight tendency to undergo ulceration. Gummatous infiltration or ulceration would present no difficulty in the

earlier stages; in the later ones, the rapid melting down and the deep ulceration should suffice to characterise it. The general symptoms should also be taken into consideration, and it would then be hardly possible that a mistake should be made.

Believing as we do that the condition is only a part of general tuberculosis, the prognosis is absolutely unfavourable. There is little to be said as to treatment. The general treatment would of course be tonic and supporting, but the dysphagia renders it very difficult to do much. Mackenzie recommends insufflations of morphia to relieve the pain, and we are inclined to think that cocaine spray might be used cautiously with a view to rendering possible the thorough application of iodoform, a trial of which is certainly indicated by its success in tubercular ulceration in other parts of the body. Gougenheim has reported a case where chronic tubercular ulcerationof the fauces in a patient who presented no other sign of tuberculosis was thus cured.

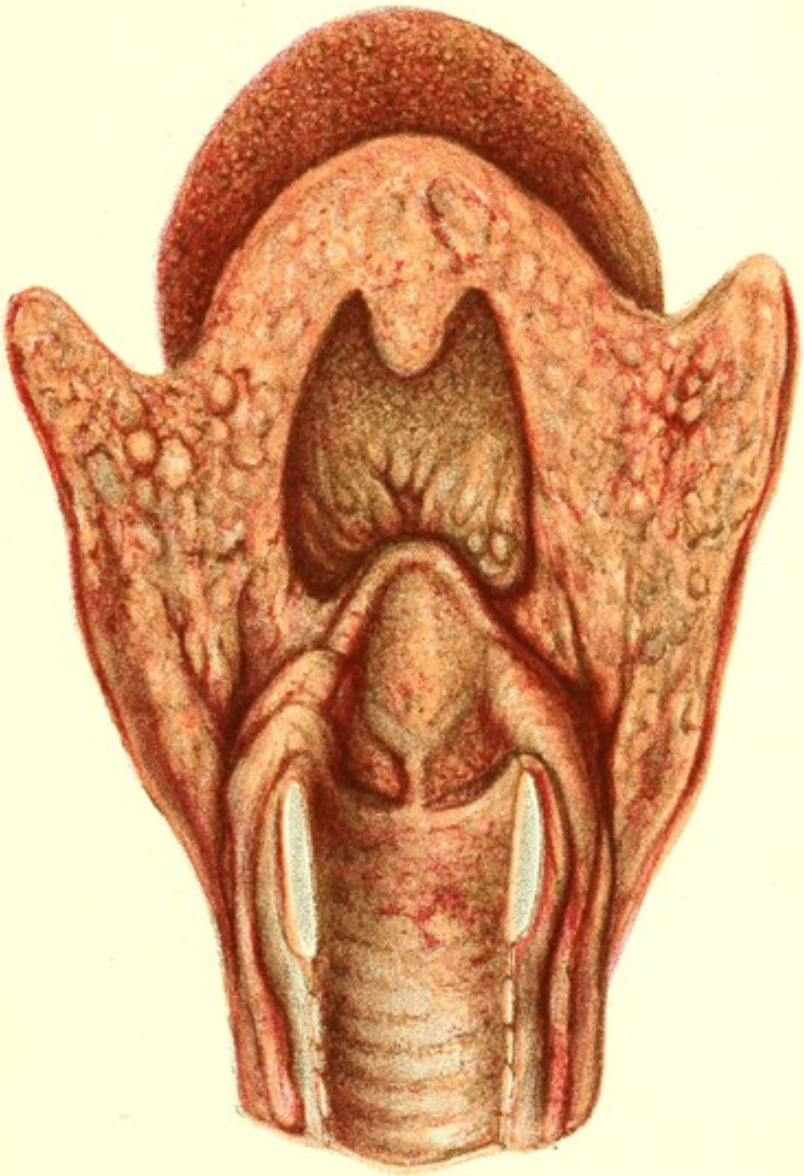
We think our cases entirely bear out Dr. Gee's conclusions: "First, that there is a form of angina which must be called tuberculous, and which forms a part of general tuberculosis; and next, that there is a form of tuberculosis which may be called anginal, inasmuch as during life the chief symptom relates to the fauces."

(For report of the discussion on this paper, see 'Proceedings of the Royal Medical and Chirurgical Society,' New Series, vol. ii, p. 150.)

DESCRIPTION OF PLATE III.

Acute Tubercular Ulceration of the Fauces.

(From a drawing made by Mr. E. Burgess.)



ON THE

ACTIVE PRINCIPLE OF CASTOR OIL.

BY

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(COMMUNICATED BY DR. LAUDER BRUNTON, F.R.S.)

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IT has long been known that the seeds of castor oil are exceedingly active, but the active principle has never been isolated. The object of the following paper is to try and throw some light upon the connection between the activity of the seeds and that of the oil. Previous observers having either obtained no definite result or contradictory results, the further investigation of the subject was suggested to me by Professor Schmiedeberg, of Strasburg. The last investigation previous to mine was that of Bubnow, who found that by extracting the seeds with acid, filtering the solution, and then precipitating with an alkali, a precipitate was obtained which was active when dried. This young investigator having been unfortunately removed by death, without publishing his method, I had to recommence afresh. After removing the oil from

the powdered and shelled seeds by alcohol and ether, a process which required eight days, I extracted with acid; but Bubnow having left no details I was obliged to make a number of experiments in order to ascertain the proper strength of acid. These experiments showed that hydrochloric acid containing 1 per cent. of absolute acid yielded a precipitate with carbonate of soda which was inactive, and so was that obtained with any stronger acid. An acid containing 4 per 1000 of HCl was found serviceable, as it yielded an active precipitate, although it did not appear to remove the whole of the active principles from the seeds, inasmuch as the residue still remained active after being treated with acid for twelve hours. When the acid was weaker than 1 per 1000 no precipitate was obtained. When the precipitate was treated with sulphate of copper and caustic soda it gave the reaction characteristic of albumen very strongly, so that it evidently contained an albuminous substance. I next tried to make a watery extract of the powdered seeds, but as it could not be got clear by filtration I obtained from Italy some of the seed cake, that is, seed from which the oil had been expressed. This seed cake and the active seeds I had experimented upon were obtained from the same source. Numerous observations have already shown that the symptoms of poisoning by castor-oil seeds are vomiting, purging, and collapse, occasionally with a subsequent febrile condition, and sometimes albuminuria. In some experiments I found that if 5 grammes of the shelled seeds in the natural state were given to a rabbit, nothing was to be noticed within the first few hours; for example, if given at 4 p.m. nothing would be observed that evening, but next morning the animal would suffer from violent diarrhœa and would die any time after 7 p.m. on the second day. The only abnormal post-mortem appearances were in the intestine, with the exception that as a rule the stomach was slightly inflamed at the pylorus. Close to the pylorus the intestine was red with scattered extravasations, the glands were swollen, and though not much

reddened towards the mucous surface they were reddened towards their subserous aspect. At the end of the small intestine there was usually a dark purple patch, and this often occurred also at the entrance of the bile-duct into the pylorus. The whole of the small intestine was full of a greenish-yellow fluid with greenish flakes floating in it; the mesenteric glands were swollen and dark red. The two thirds of the cæcum nearest the small intestine were reddened. All the other organs appeared to be normal with the exception of a few small scattered extravasations in the lung.

The effects of the powdered seed deprived of oil, and also of the cake, were the same as those just described; but it was noticed that sometimes subserous extravasations were present upon the peritoneal surface of the intestine and in the mesentery, and these were also seen after the administration of Bubnow's extract. A quarter of a gramme of this extract injected subcutaneously had a very remarkable action, for the next day the animal would have slight diarrhœa and die on the second day. On post-mortem examination the only differences observed between the effect of the extract when administered subcutaneously and by the mouth were that after subcutaneous administration the purple extravasated patches at the beginning and end of the intestines were absent, while subserous and mesenteric extravasations were invariably present. There was no inflammation at the site of injection. Sometimes the lower part of the large intestine contained stiff gelatinous masses, and the lymphatics of the neck, axilla, &c., were congested. Bubnow's extract appears from the facts already mentioned to be a substance which is precipitated along with albumen either as a compound with it or mechanically by it. I therefore thought that it might be possible to get an active solution from the cake by extracting it with water, and this idea proved to be correct. I bruised the oil cake with about twice its weight of water, pressed out the liquid, and after allowing it to stand for twenty-four hours so as to clear, I filtered

it. I thus obtained a clear yellow, transparent, semi-syrupy solution of extreme activity. On adding four times its bulk of alcohol to this, and letting it stand, a precipitate was formed which was not increased by the further addition of alcohol. This precipitate when dried proved to be extremely active. I invariably obtained the reaction of a glucoside whenever the substance was active. All attempts to obtain an active principle without albumen failed, and an albuminous body was always present in the solution when it was physiologically active. The same was the case when attempts were made to purify Bubnow's extract. Amongst the means used for this purpose were precipitation with acetate of lead and ammonia, fractional precipitation with alcohol and dialysis. After this active substance had been extracted from the castor-oil seeds the residue was no longer active. This active substance keeps well when dried, but if the first precipitate thrown down by alcohol from the aqueous extract of the seeds be again treated with water, it does not redissolve completely as one would expect, but only partially. The filtrate is exceedingly active and the residue is also active, but its activity appears to depend upon the difficulty of washing out the active principle completely. But if the process of precipitation by alcohol and re-solution be continued we at last get a small quantity of an insoluble residue, which is inactive; a soluble residue, which is also inactive, would be obtained as well. In other words, the mere action of alcohol is sufficient to render the active principle inert. Mere contact with chloroform for a few days has the same effect. Many attempts to obtain the substance pure by the use of various chemical reagents ended in yielding an inert substance.

Considering that a glucoside reaction was always present, and that the active substance always became inert under chemical treatment, it is possible that some light may be thrown upon the nature of this active principle by the following fact. If the slightly acid aqueous solution of castor-oil cake be precipitated by adding

chloride of calcium and afterwards soda, a precipitate of hydrate of lime falls, and along with this there is also precipitated a compound of lime with a physiologically inert glucoside. By continued washing with water, rendered alkaline by soda, the albumen present can be almost entirely removed, leaving the hydrate of lime and the compound of calcium with the glucoside behind. By now adding oxalic acid the glucoside can be set free, and as it is insoluble in alcohol and ether it can be precipitated by the addition of two parts of alcohol or of ether to one of the solution. The glucoside then falls as a flaky substance, very like the active substance containing albumen obtained from the seeds. This fact, which was discovered by Professor Schmiedeberg, would seem to indicate that we are here dealing with an anhydride of a glucosidic acid, which becomes inert whenever it is altered from its anhydrous condition, and becomes either hydrated or combined with a base. In this character it appears to resemble other bodies of a similar nature, such as euphorbin and other substances of the acrid group, or cathartic acid, which was lately shown by Stockman to be an insoluble, non-nitrogenous glucoside of this kind. Although the glucoside has been prepared, I have not further examined its composition.

Referring to the crude but active substance, I may state that, when injected hypodermically, it is excessively active, causing the usual congestion of the intestine with small extravasations over all the serous membranes. Thus after ·0059 gramme injected hypodermically there were extravasations into the wall of the intestine on its subserous aspect, into the mesentery, into the pericardium (visceral), slightly into the lungs, into the fold between the cerebrum and cerebellum, and into the dura mater of the spinal cord. Large doses of the substance or of the seeds taken by the mouth cause similar effects.

The active body obtained by Bubnow is the same as that which I have obtained, and he supposes it to be the active principle of castor oil.

But there are certain remarkable differences between castor oil and this active body. The reputation of castor oil as an aperient is a well-justified one, but that of the seeds is so far questionable that it seems by no means a true aperient, and acts as such only by producing an effect upon the vessels, which in cases of poisoning is not confined to those of the intestine. The points of difference are as follows :

1st. It was found by Bubnow that boiling the seeds for a short time was enough to render them inert. This I found to be invariably the case ; even exposure to heat below boiling point, *e. g.* mere boiling in absolute alcohol (98 per cent.), was enough to destroy the activity. But castor oil may be boiled hours, till, indeed, it evidently is decomposing without losing its activity.

2nd. This substance is soluble in water, not in alcohol ; but castor oil never yields any precipitate, no matter how much alcohol or ether be added to it. Hence this substance cannot be present in the oil. But what is still more striking is the fact that if a rabbit be poisoned by castor oil in such a dose as to cause death, diarrhœa invariably occurs, but death never occurs before the second day, and the intestine never shows anything beyond a muco-purulent appearance, and not a trace of congestion or inflammation is to be seen anywhere. In other words, the action of the oil seems to consist only in a stimulation of the intestinal secretion, death being due to exhaustion from the great drain. But with the seeds death may occur in about twelve hours, without positive diarrhœa, and the intestine always shows excessive vascular engorgement, even when the dose of the seeds given is so small that the animal does not die. I performed this experiment several times, and always with the same result. Last summer I found thirty grammes of oil sufficient to kill small rabbits in two days ; this winter, for large rabbits, sixty grammes were necessary, but the result was the same, and in both cases there was no inflammation. Moreover, it is impossible to get water to take up any

active principles from castor oil, when shaken up with it for a long time. Again, I never found the oil prepared by me, nor that left by Bubnow, stronger than the ordinary commercial oil; and I can only say that it seems as if the statement made by Parola and repeated in various books (*e. g.* Flückiger and Hanbury), to the effect that oil extracted from the seeds by alcohol keeps better, does not cause nausea or vomiting, and is four times as strong as ordinary oil, is based upon an error of observation. It does not seem that they ever filtered their oil (extracted by alcohol) through paper, but this I always did. In fact on one occasion where I had filtered only through cloth as they did, the solution showed only a very slight cloudiness indeed, but was much more energetic than usual. It was not until I refiltered through paper that I found that it was this mere trace of impurity which made the difference in activity. It is evident that if further proof be required it is only necessary to take the oil cake, and as it has lost its milder oil by pressure, to extract it by alcohol. One thus gets almost 20 per cent. of the weight of cake of oil of a rather mawkish almondy smell, but it is not one whit more active than the usual oil.

Ricinoleic acid I found, like others, to be perfectly inert in a fifteen-gramme dose, which would be a powerful dose were it like castor oil in activity. Olive oil soaked on the castor-seed powder did not take up any active principle. Some of the substances which give rise to the characteristic disagreeable flavour of castor oil can be got during saponification of it, but they are inactive. The taste of ricinoleic acid is quite different, though also nauseous. As many experimenters have tried to get the active principle of castor oil by saponification, and it was evident that it disappeared *en route*, I tried all the different bodies left in the solutions, but found none active. It is thus evident that it is destroyed by saponification, and I therefore tried, but in vain, to see if it were possible to get a substance differing in activity and in solubility in diluted alcohol. Thus absolute alcohol mixes in any quantity

with castor oil; if one add 25 parts of water to, say, 200 alcohol and 100 of oil, the whole becomes cloudy, but on heating to boiling point it clears up, again becoming cloudy on cooling, and an alcoholic layer rising to the surface. No matter how I varied the proportion of water I never found any difference in the activity of the body dissolved in this alcoholic layer. Now, is it possible that after all there is a *tertium quid* besides the ricinoleic acid and seeds, or is it the glycerine which causes the aperient effect, the castor oil simply decomposing with sufficient readiness to allow the glycerine time to act? It is perfectly possible, I fancy, but I did not unfortunately think of this till it was too late for me to carry out the experiment, but I hope to do so soon. If it be so it is evident that there is no need of seeking for a resin, as one will never find it. Certainly the appearance of the bowel is such as one would expect from glycerine. There is one thing against this theory, viz. the fact that castor oil injected into the bowel is said to be aperient. This statement is, however, not a very easy one to prove, seeing that the physical nature of the castor oil would cause aperiency. Of course ricinoleate of magnesia would cause aperiency due only to the magnesia, an experiment of no value as regards this question. Seventeen grammes of ricinoleic acid given to a rabbit caused but a very slight aperient effect, corresponding to about eight grammes of the oil itself.

Before concluding this I may state that it seemed to me that the castor-oil cake, although a deadly poison, might be made to subserve beneficent purposes. Thus in Italy there remains about one fifth part of the oil in the seeds, in India, where the mode of extraction is cruder, there remains more. It appears to me that if greater heat were used, that is, 212° or even much less, the oil could be better extracted, and as the oil is not always for internal use, this would in general do no harm to the oil, while much of the above waste would be avoided. The heat would render the oil cake harmless. Now, as the cake has rather a pleasant smell and taste, and is full of

albumen, we have before us what might become an article of diet.[1] As an example of the similar transformation by heat of a poisonous substance into an article of diet we may cite cassava. The only difficulty that I see here is the husks of the seeds ; these would be very indigestible and nauseous, but I fancy could be easily removed before the expression of the oil. As regards the possible importance of this body I may mention that there are some 700,000 gallons of castor oil exported from India, which would mean some 3000 tons of castor-oil cake, and as this is highly albuminous, and by the mere cooking becomes harmless, it simply implies that there are so many tons of highly nutritious food thrown away. The value of this in India is inestimable, because it would be much more valuable than so much rice, and would help to eke out the rice. The importance of a nitrogenous food, where religious superstitions forbid the use of animal food, is self-evident. I suggest this as worthy of consideration by those who could investigate the matter further. In times of famine what a blessing it would be, and, so far as I can see, it would keep exceedingly well in a very dry climate.

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[1] I may state that for comparison, and with the hope of obtaining the substance free from albumen, I examined some leaves fresh from Italy, but obtained no active substance; there seemed to be a substance like the altered glycoside present. The so-called ricinin of Tuson, &c., is probably not originally present in the seeds, but is a product of decomposition. The statement made by Power in the 'American Journal of Pharmacy,' xxxvi (1854), p. 207, that the seeds have a protein substance and a body like amygdalin may be true; but though I had not any description of the experimental grounds for this, I can only say that if we make a watery extract after an alcoholic one, and add the products to each other, the result is nothing physiologically.

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A CASE

OF

MULTIPLE EMBOLISM OF THE ARTERIES OF THE EXTREMITIES

FOLLOWED BY THE

FORMATION OF ANEURISMS.

WITH REMARKS ON THE RELATIONS OF EMBOLISM TO ANEURISM.

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Alice D—, æt. 20, was admitted into St. Bartholomew's Hospital on Nov. 2nd, 1885, with a pulsating tumour in the bend of her right elbow, and another in the right popliteal space.

The patient is a domestic servant, and she states that four weeks ago, whilst at her work, she felt an aching and stiffness in the right knee, which she attributed to rheumatism. A week afterwards she complained in a similar manner of her right elbow. She was then seen by a medical man, who ordered her to apply some iodine to the elbow. On November 2nd she attended the sur-

gical casualty department of St. Bartholomew's Hospital and was immediately admitted.

On inquiry into her history, she stated that she has had a winter cough for several years, and has lately suffered from shortness of breath on the slightest exertion. There is no history of syphilis, rheumatism, or chorea. Her family history is good.

She is poorly nourished, has a troublesome cough, and expectorates some viscid frothy mucus. The cardiac area is increased, and there is a double aortic murmur, with a doubtful presystolic murmur at the apex. Resonance over the lungs is good, but there is much rhonchus and sibilus everywhere. Abdomen natural. Situated immediately above the bend of the right elbow is a swelling of the size of a Tangerine orange. The skin over it is rather œdematous and discoloured as if from bruising. The superficial veins are slightly fuller than natural. The swelling is tender to the touch, easily compressible, so that it can be much reduced in size by steady pressure. If after this partial emptying of the tumour pressure be made upon the brachial artery above, it remains collapsed, but fills up quickly again when the pressure is released. It pulsates synchronously with the heart, and the pulsation is distensile in all directions. A distinct thrill can be felt and a loud bruit heard over the swelling. Passing over the aneurism for nearly two thirds of its length can be felt the pulsation of a large vessel, which is deemed to be the brachial artery. This gradually lessens and becomes lost towards the distal end of the aneurism. The pulse at the right wrist can scarcely be recognised. No venous distension or œdema below the swelling. The circumference of the arm over the broadest part of the aneurism is nine and a half inches. The finger-ends of both hands are remarkably clubbed.

In the right popliteal space there is a swelling of the size of a large orange, presenting the same characters as the one in the arm. It is most prominent on the outer side of the ham and behind the head of the fibula. It

extends also to the inner side of the calf to about four inches below the knee-joint. No pulse can be felt in the tibials of the right side or in the femoral artery of the left side, although it can be easily felt in the left tibial vessels. A small patch of extravasated blood is noticed in front of the right outer ankle. Circumference of the limb over the most prominent part of the aneurism is $13\frac{5}{8}$ inches. There is no sign of any similar tumours elsewhere.

The patient was put upon a liberal non-stimulating diet, and confined to her bed.

On the first night after her admission into the hospital her temperature rose to 103·4°, but it fell next morning to normal.

Three days afterwards she was seen in consultation, when Mr. Langton stated that he believed the aneurisms to be embolic in their origin, and the result of secondary changes taking place in the arterial walls from the presence of the emboli. He proposed gentle compression of the main vessels above the aneurisms, but not upon the sacs themselves, as they gave the impression of being thin walled, and probably not consisting of the coats of the vessels. Moreover, long delay was not advised, unless manifest improvement resulted, before proceeding to ligature the arteries. In this opinion, both of the nature and the treatment of the aneurisms, he was substantially supported by his colleagues.

November 6th.—Pressure was applied to the brachial and the femoral arteries by pads and bandages, the latter being gently continued over the aneurisms. The following day it was observed that the pad failed to control the pulsation of the popliteal swelling, so a tourniquet was applied very gently. Towards the evening the patient complained of much burning pain in the foot.

9th.—Has a good deal of pain in the arm, but slept well after a morphia draught. Measurement of the popliteal aneurism a quarter of an inch smaller, of the elbow tumour a quarter of an inch larger.

12th.—Since the application of pressure, the elbow tumour has diminished to its size on admission, and is manifestly firmer. Altogether the patient is much relieved.

14th.—No pulsation can be detected in the aneurism at the bend of the elbow. On examining the front of the right knee pulsation is detected in the arteries, which are much enlarged, and with the stethoscope a bruit can be heard. There is also slight pulsation in both tibial arteries of the same side. Circumference of the popliteal tumour is $11\frac{5}{8}$ inches, and of the elbow, $9\frac{1}{2}$ inches.

17th.—The elbow tumour yesterday suddenly enlarged to $10\frac{1}{2}$ inches, and this morning measures $11\frac{5}{8}$ inches.

19th.—The patient had last night a sudden remission of pain in the elbow, and to-day there is a brownish discolouration of the skin over the tumour, due probably to blood leaking through the walls of the aneurism. Ligature of the brachial artery was determined upon, and was performed a short distance above the aneurism, by tying the vessel with carbolised catgut ligatures in two places and dividing the artery between them. All pulsation ceased, and the edges of the wound were brought together by carbolised catgut sutures, the operation being performed antiseptically throughout.

21st.—The patient has been much more comfortable generally and locally since the operation ; sleeping and eating well. The arm was dressed, all the sutures were removed as the wound had united throughout its entire length. The measurement of the popliteal aneurism remains as before.

27th.—The popliteal tumour having increased during the night with the same discoloration over it as was noticed in the elbow, the superficial femoral artery was tied at the apex of Scarpa's triangle in the same manner as the brachial a few days before.

December 1st.—She is able for the first time since admission to bend her right elbow. To-day the crural wound was dressed and had united except at the upper

part. No pulsation can be detected in either of the aneurisms although they both remain of the same size.

7th.—The wound over the ligatured femoral artery has re-opened, and was dressed with a stimulating lotion, as the granulations were pale and flabby. Circumference of the popliteal aneurism 10¼ inches, of elbow, 9½ inches.

11th.—Last night about seven o'clock the patient became suddenly faint and pulseless. Half an ounce of brandy was administered, and on her rallying it was observed that her mouth was drawn to the right side. When seen early in the morning she was quite conscious, but complained of great pain in the back of her head. She had lost all power of the left half of her body, although she could raise her right leg. She had no reflex action of her left leg. There was conjugate deviation to the right side; mouth much drawn to the right, and on protruding her tongue it inclined to the left side. She perspired profusely over her forehead, more upon the left than upon the right side. No pulsation could be detected in the right common femoral artery. Both aneurisms were smaller and softer.

12th.—Passes urine involuntarily. Several gangrenous spots were noticed to-day for the first time on the dorsum of the right foot and on the leg, and over the patella. A few bullæ have appeared on the plantar surface of the three inner toes of the left foot.

18th.—The whole of the foot and the greater part of the leg on the right side are involved in the gangrenous process, and numerous bullæ have formed in different parts of the limb. At times the patient is delirious. She sank and died on December 28th.

Post-mortem examination.—The body was much emaciated. *Thorax, lungs,* and *pleura* normal. *Pericardium* normal.

Heart: Weight 15½ ounces; left side much hypertrophied. Muscular substance natural. Tricuspid and pulmonary valves natural. Mitral valve natural. The aortic valves were hidden in a mass of recent clot, which

adhered loosely to numerous vegetations. On washing off this clot it was found that one of the cusps was almost completely ulcerated away. The two other cusps were covered by numerous vegetations, some of which had undergone calcareous degeneration, and formed large, pendulous growths, loosely attached to the valves by slender pedicles, and swaying to and fro when subjected to a gentle stream of water. The aortic valves themselves were softened and lacerable. They had evidently been the seat of recent inflammation.

Immediately beyond the valves the *aorta* was dilated into three pouches or aneurisms corresponding to the sinuses of Valsalva. The largest of these was behind the left posterior segment, and bulged so as to come into close proximity to the left auricular appendage. It was as large as half a walnut and of about the same shape. In none of the pouches was there any clot, ulceration of the endothelium, or evidence of atheroma. Extending up the aorta from these three aneurisms were some granulations, some old, others apparently of recent date. They were very thick near the valves, but rapidly thinned further up the vessel, the highest granulation being placed about one inch and a half from the aortic orifice. At this level there was a small ulcer the size of a split pea, which had completely penetrated the inner and middle coats of the aorta, and had encroached upon the outer coat. Beyond this point the aorta in its whole extent, as well as all its main branches to the neck, arms, and trunk, was perfectly healthy.

Abdomen : Liver much enlarged considering the emaciated state of the patient. It weighed 82 oz. and was fatty and congested.

Spleen enlarged ; weight 18 oz ; surface puckered and scarred from old infarction. No recent infarcts.

Kidneys : Surface puckered from old infarcts.

Intestines natural.

Uterus, ovaries, and *vagina* natural.

The brain : Upper surface natural. The right middle

cerebral artery was occluded at its first bifurcation by a small calcareous embolus, which completely blocked the vessel at this spot. A little farther on another branch of the same vessel was filled with a firm, but not calcareous embolus. At both of these places the vessel was much dilated. A couple of inches beyond the last embolus, another plug had been lodged in a small branch, whilst a fourth embolus was found in a branch of the right anterior cerebral artery.

The right lenticular nucleus was in a pulpy condition, and the softening had extended to the white matter in its immediate neighbourhood, and to the convolutions of the island of Reil.

Right upper extremity.—In front of the elbow was an oval swelling extending equally above and below the level of the joint. From this the skin was reflected, and all the tissues around the aneurism, together with the brachial, ulnar and radial arteries were removed. On laying open the brachial artery it was found to be in a perfectly natural condition as far as a point about two inches above the elbow-joint. Here it was quite occluded and matted to the surrounding tissues. It was at this spot that the ligatures had been applied. From here to its termination the vessel was filled with blood-clot partly decolorised and adherent to the endothelium. Just above the bifurcation the brachial artery on its anterior surface was perforated by a small hole, not much larger than a pin's head, blocked by a firm, dark, and closely adherent clot. On the removal of the latter, the whole was found to open directly into the swelling in front of the elbow already mentioned.

This swelling was composed of a mass of blood-clot encapsuled merely by the soft tissues of the part condensed by pressure. The clot was firm, but its outer layers only showed a slight trace of lamination. In no part was it limited by an expansion of the artery, or of its sheath.

Immediately below the aperture in the brachial artery, the ulnar and radial vessels were found to contain the

remains of an embolus, consisting of calcareous material of precisely the same nature as that met with on the vegetations attached to the aortic valves, and hidden to a great extent by blood-clot. The greater part of this calcareous matter was found in the ulnar artery, and here it formed a very distinct and hard lump. The wall of the radial, however, had been completely ulcerated through, and small portions of gritty material here protruded through its walls into the tissues outside the vessel. Below the seat of embolism both the radial and ulnar arteries were very small, and filled with clot as far as the middle third of the forearm.

Vessels of the Right Lower Extremity.

The right common iliac artery was occluded immediately above its bifurcation by a large embolus, composed chiefly of calcareous matter similar to that found on the aortic valves. Around this was some recent blood-clot. The common iliac above the seat of embolism was perfectly healthy, but where the embolus was lodged the artery was thinned, and distinctly bulged to the outer side. On partly separating the embolus from the vessel, the endothelial lining of the latter was found to be irregular and pitted, as though from recent inflammation or ulceration.

The internal iliac artery was patent, and its walls natural.

The external iliac artery was shrunken, and filled with dark firm clot.

The common femoral was in a similar condition.

The superficial femoral was filled with clot in its upper two inches, and below this, for about a similar distance, the artery was represented by a fibrous cord. It was at this place that the ligatures had been applied, but no traces of them could be found. The vessel was filled with clot in almost the whole remainder of its course, but the popliteal artery was patent.

Immediately below the bifurcation of the latter vessel, the posterior tibial artery was shrunken and filled with clot, and, at a distance of about half an inch from its origin, was perforated by a small aperture, similar to that already described in the brachial, and leading into a large swelling in the calf. This swelling contained several ounces of dark and rather soft clot, confined merely by the surrounding tissues, and nowhere very definitely circumscribed. The posterior tibial artery was in no way dilated; on the contrary, below the spot where it communicated with the collection of clot it was not possible to trace the vessel, and no remains of an embolus were to be found.

Vessels of the Left Lower Extremity.

The common iliac was healthy.

The internal iliac was also healthy, but, relatively to the other vessels, was large.

The external iliac in its upper two inches was universally dilated, so as to form an aneurism of globular shape the size of a pigeon's egg. The sac of this aneurism appeared to be composed of all the coats of the vessel except at its most anterior part. Here the sac had partly given way, and the contents were limited by the superjacent sheath and fascia. The inner surface of the sac was quite smooth, and there was no appearance of atheroma. The sac contained dark soft blood-clot. There was no laminated fibrin.

Below the aneurism the external iliac and the upper part of the common femoral arteries were so contracted that it was difficult to find them. They were not as large as a normal radial, their calibre was practically obliterated, and they contained a slender cord of decolourised fibrin, in which were embedded granules of calcareous material similar to that found in the right common iliac and brachial vessels.

The lower part of the common femoral, and the superficial and deep femoral vessels, were all patent and healthy.

A microscopical examination of the diseased vessels revealed the following conditions.

Several portions of the right common iliac were examined, including the outer and thinnest part of the vessel where it was most distinctly bulged. The whole arterial wall in contact with the embolus was the seat of an inflammatory process, and, in its whole thickness, was infiltrated with cells. This cell-infiltration was most evident in the inner coat, and in almost every section examined the latter was found to be distinctly ulcerated,



FIG. 1.—Section of common iliac at the seat of embolism. *a*. Middle and outer coats infiltrated with cells. *b*. Portions of calcareous embolus. *c*. Clot with fibrin mesh-work.

its endothelium being destroyed and the subendothelial tissue and fenestrated membrane crowded with cells. Where the arterial wall was thinnest this ulceration had entirely penetrated the intima and involved the middle coat. The muscular fibres of the latter had been exten-

sively destroyed, and the sections consisted rather of the products of inflammation than of the normal structures.

Sections were made of one of the plugged branches of the middle cerebral, the most distal one being selected.

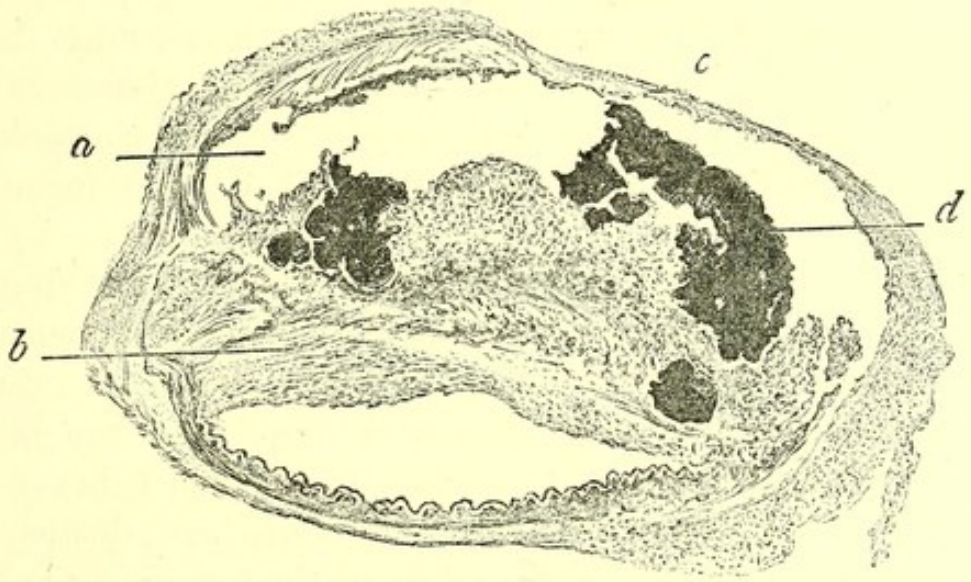


FIG. 2.—Section of plugged anterior cerebral artery, showing destruction of arterial wall in the neighbourhood of the embolus. *a*. Lumen of vessel. *b*. Clot. *c*. Arterial wall partly destroyed. *d*. Embolus (calcareous).

The embolus was in parts calcareous, but was surrounded by decolourised clot. The vessel wall, in about three fourths of its circumference, was only slightly infiltrated with cells, and the endothelium was intact. At one part, however, the coats were considerably destroyed, the inner coat being entirely removed, and the muscular coat partially ulcerated. At this point the arterial wall was extensively infiltrated with cells.

Sections of the wall of the aorta taken from one of the aneurismal pouches showed a general disorganisation of the ordinarily dense structure of the vessel, and an infiltration with inflammatory products. The endothelium also was partially destroyed.

Sections of the sac of the aneurism on the left external iliac presented precisely similar appearances.

Remarks.—In studying this case the state of the heart and aorta is the first consideration.

It is evident that, in spite of the absence of any marked symptoms of septic infection, "ulcerative endocarditis" was in progress immediately before death. It is further evident that the aortic valves had been long previously diseased, for the calcareous masses, at least, must have been of some years' standing, and it must therefore be concluded that the recent destructive inflammation attacked, as it so frequently does, valves which had been formerly damaged.

It seems to us probable that the emboli were due to ulceration and destruction of the valvular attachments of the vegetations, which, together with the calcareous masses adherent to them, were thus cast loose into the blood stream, and, looking at the large calcareous concretions yet left on the two aortic valves which are least destroyed, and the very extensive destruction of the remaining valve, it is likely that the large calcareous mass in the right common iliac was derived from the latter valve.

The condition of the aorta already described is evidently due to an extension to it of the inflammatory process which attacked the valves; and it is to the softening of the walls caused by inflammation that we attribute the aneurismal pouches we have mentioned. Aneurisms such as these, in connection with aortic valvulitis, have previously been described, and a very good specimen is in the museum of St. Bartholomew's Hospital.[1] The ulcer on the aorta, which had nearly penetrated the vessel, we attribute also to the same cause. It is evident that the process by which the valves themselves were destroyed might, by extension to the aorta, cause the formation of an ulcer in its walls. It is worthy of remark that this ulcer is beyond the reach of direct mechanical irritation by the diseased valves, or by the calcareous masses attached to them.

There can be no reasonable doubt that the calcareous embolus in the right common iliac artery was derived

[1] No. 1430 A.

from the diseased aortic valves, and it is of much interest and importance to note accurately the changes in the artery where it had lodged; for a microscopical examination shows that it had been the seat of extensive inflammation, which had not only resulted in softening of its walls, but, in parts, had almost completely perforated them by a process of ulceration. In consequence of these changes, the artery at the time of death was actually in process of aneurismal dilatation.

The occlusion of the left external iliac and common femoral vessels we consider to be of long standing, and for these reasons: that they are extremely small and shrivelled; that their contents are not recent clot but old, adherent, decolourised fibrin with calcareous matter; and that whilst the patient was under observation the circulation in the left lower extremity was generally good and the pulse in the tibials strong. Whether the occlusion of these vessels was due to the lodgment of an embolus in past years it is impossible to say for certain; nevertheless, considering the condition of the heart, the presence of calcareous matter in the occluded artery, and the embolism of other vessels, we consider such an explanation to be probably correct.

The aneurism on the left external iliac we consider to be of more recent origin than the obliteration of the vessels beyond it, for its sac was thin; the tissues over it were but little matted or adherent; there was no laminated fibrin in it, and it contained merely soft recent clot. It also may have been caused by the lodgment of an embolus. If so, the latter must have been subsequently disintegrated.

The aperture in the right brachial artery appears to have been certainly due to the lodgment of an embolus. It was situated immediately above the bifurcation. The vessel in the rest of its course was perfectly healthy, and even immediately around the aperture there was no disease, whilst in the vessels below, and in the tissue immediately around the hole, we found the remains of a calcareous embolus which could scarcely have come from any

other situation than the valves of the heart. This calcareous material was certainly not derived from the wall of the vessel itself, for, apart from the fact that calcification of arteries is never met with at the early age of nineteen, the vessels in question, as we have already said, were healthy. Finally, to the best of our knowledge, idiopathic ulceration of an artery at a single spot has never been seen except, as we shall presently show, in vessels which have probably been plugged. We therefore conclude that the artery wall was ulcerated at the place where it was plugged, and that the blood, escaping into the surrounding tissues, at first in small quantities, formed a so-called false aneurism, without there being any true dilatation of the vessel wall itself.

A considerable number of cases are now on record in which aneurisms occurring in young subjects have been associated with aortic valvular disease, and this coincidence, together with the situation of such aneurisms at the bifurcation of arteries, has suggested the probability that the disease of the vessels has been the result of their occlusion by an embolism.

In the great majority of cases no embolism, however, has been actually discovered at the seat of aneurism, although vessels in other parts of the body have been plugged. As far as we are aware, Ponfick is the only writer on this subject who has found an embolus in the diseased vessel itself, and there are yet many surgeons who doubt the direct relationship between embolism and aneurism.

The following is a list of arteries in which aneurisms have been found on post-mortem examination and have been attributed to embolism:

Axillary.—Guy's Hospital Museum, No. 1502[20].

Brachial.—Goodhart, Pathological Soc. Trans., vol. xxviii, 107.

Ulnar.—Ogle, Brit. and Foreign Med.-Chir. Rev., vol. xxvi, p. 500. Guy's Hospital Museum, No. 1503[30].

External iliac.—Moore, Path. Soc. Trans., vol. xxxiv,

p. 71. St. Bartholomew's Hospital Museum, Heart, No. 1299[A], Artery, No. 1460[A].

Common femoral.—Parker, Med.-Chir. Soc. Trans., vol. lxvii, p. 57. Museum, Royal College of Surgeons, Heart, No. 3017[B], Artery, No. 3241[A]. Bryant, Path. Soc. Trans., vol. xxvii, p. 98, and specimen in Guy's Hospital not yet added to the Museum. Guy's Hospital Museum, No. 1519[21].

Popliteal.—Bryant, Path. Soc. Trans., vol. xxviii, p. 98, and Guy's Hospital Museum, No. 1519[39]. Tufnell, Dublin Quart. Jour., 1853, p. 371.

Posterior tibial.—St. George's Hospital Museum, No. 129.

Superior mesenteric.—Wilks, Path. Soc. Trans., vol. xi, p. 44, and Guy's Hospital Museum, No. 1504[45]. Guy's Hospital Museum, No. 1504[46]. Ogle, Path. Soc. Trans., vol. viii, p. 168.

Splenic.—Guy's Hospital Museum, No. 1504[47].

Internal carotid.—Holmes, Path. Soc. Trans., vol. xii, p. 61.

Cerebral arteries.—Church, St. Bartholomew's Hospital Reports, vol. vi, p. 107. Goodhart, Path. Soc. Trans., vol. xxviii, p. 107. Several specimens in Guy's Hospital Museum. Ponfick, Virchow's Archiv, 1873, vol. lviii, p. 528.

The various authors who have written on this subject are not agreed as to the exact manner in which the aneurism is produced. Some maintain that the artery above the seat of embolism becomes dilated, others believe that the vessel yields at the seat of plugging itself. In all cases, however, except that recorded by Ponfick, the process of formation of the aneurism has not been demonstrated.

We believe that in the case recorded by us the mode of formation of the aneurisms is clearly demonstrated. An examination of the specimen both by the naked eye and by the microscope reveals that where the embolus has lodged the artery wall has been inflamed. The inflam-

mation has progressed to softening and ulceration of the vessel, and in those arteries which had been most recently plugged, as evidenced both by the clinical symptoms and by the post-mortem examination, the ulceration was in progress at the time of death.

In the arteries of the arm and leg, on the other hand, the ulceration had caused complete perforation of the vessel, and, the blood escaping into the surrounding tissues, a sac had been formed by their condensation and by the clotting of the extravasated blood. In the left external iliac artery, however, the wall had merely been softened and had become the seat of an aneurismal dilatation. In the brain one artery at least had ulcerated at the seat of the embolism, and its condition was such as to render it probable that, had the patient survived, complete penetration would have followed.

In the right common iliac the arterial wall was both ulcerated and dilated.

We believe that in the case we have described it is proved that embolism may cause aneurism, and that the disease and subsequent dilatation of the vessel occur at the seat of the lodgment of the embolus and not above it.

The next point for consideration is the reason why, in the vast majority of cases, embolism should not cause aneurism, or rather, why it should only exceptionally do so?

It may be argued that the causation of aneurism is purely fortuitous, and that in any given number of cases of embolism a certain percentage will result in the development of aneurism, just as in the case of ligature of vessels in their continuity, aneurism has resulted in a few instances.

We believe this reasoning to be defective, and first we would point out that although aneurism has occasionally occurred after ligature of an artery in its continuity or in a stump, there is no evidence to show that the aneurism has been produced by the mechanical dilatation of a healthy vessel above the seat of the constriction. It is probable that the aneurisms that have followed ligature,

have resulted either from a softening of the artery walls by suppuration, as in the cases recorded by Mr. Symonds in the Clinical Society's 'Transactions,' vol. xix, p. 140, 1886, and in the Pathological Society's 'Transactions,' vol. xxxv, 1884, p. 146; or from a failure to obliterate the vessel at the point of ligature, as in the case recorded by Mr. Thomas Smith, in which the ligature of the superficial femoral with catgut for the cure of a popliteal aneurism failed in its object, and also produced an aneurism at the site of the operation.

We would therefore urge that not only is there no proof that an artery ever becomes mechanically dilated behind an embolus, but we deny that such an event as mechanical dilatation has ever been known to follow the obstruction offered to the blood-stream by a ligature.

Again, it will be readily conceded that, considering the frequency of embolism, the percentage of cases which result in aneurism is infinitesimal, and it is difficult to suppose that in a single individual two arteries should be completely ulcerated through and several others in process of destruction at the time of death without the presence of some specific cause.

It appears obvious to us that the theories of dilatation occurring above the seat of embolism, of the distension of the vessel by the current of blood through an artery which is narrowed and not obliterated, and others which have been propounded, are not sufficient to meet the case.

Two other explanations remain: the one originally suggested, we believe, by Dr. Goodhart, is that aneurism only occurs when the embolus is the "product of a fungating endocarditis in which the vegetations are large, and often, though not always, accompanied by ulceration;"[1] the other is that of Ponfick, who attributes the formation of aneurism to the calcareous nature of the embolus, and argues that by its mechanical irritation it produces an ulceration of the vessel at the seat of plugging.[2]

[1] 'Path. Soc. Trans.,' vol. xxviii, p. 107.

[2] 'Virchow's Archiv,' vol. lviii, p. 528.

Taking the last of these theories first, we think that the explanation applies to Ponfick's cases alone, and not to any of the others to which we have alluded in the appended table, for in none of them was any calcareous embolus found, nor, with one exception, were the vegetations on the valves noted to be calcified.

On the other hand, we find that of twenty-five recorded cases in which the condition of the heart was mentioned, there was ulcerative endocarditis in progress at the time of death in ten, and recent endocarditis with production of warty vegetations, such as are described by Dr. Goodhart, in twelve others. From the descriptions given, it is probable that in several of the latter there was ulceration of the valves, but this is not definitely stated. In the three remaining cases there was certainly some recent endocarditis, but whether warty or ulcerating is not clear from the descriptions.

Further, it is noteworthy that although Ponfick attributes the aneurisms in his cases to the calcareous nature of the emboli, yet in all of them he describes the heart as being the seat of "recurrent warty endocarditis," and in two of his six cases he describes ulceration as being present.

In our own case, although the emboli were calcareous, we do not attribute the formation of the aneurisms to this fact, but rather to the presence of a distinct "ulcerative endocarditis," and a corresponding septic condition of the emboli.

In support of this view we submit the following arguments:

1st. The aneurisms of the sinuses of Valsalva were not due to ulceration of the arterial walls, but to their softening by inflammation. This could not result from the mechanical irritation of a calcareous embolus, and consequently must be considered to be due to an extension to the aorta of the diseased condition of the valves. This proves that the infection of the vessel may result in softening and dilatation without pre-existing ulceration.

2nd. The ulcer in the aorta just above the valves was independent of either mechanical irritation or the lodgment of an embolus. It was evidently the result of an extension to the artery wall of the ulceration which was in progress in the valves themselves, thus showing that in an artery ulceration may result from infection by contact with, or in proximity to, valves affected with ulcerative endocarditis.

3rd. It is well known that in pyæmia and allied conditions emboli are capable of conveying to distant parts septic particles which infect the tissues in which they lodge, and set up in them changes of a precisely similar nature to those which are in progress in the part from which they themselves have been derived. Considering that direct extension to the aorta of the valvular inflammation is capable of causing both softening and dilatation as well as ulceration, we submit that it is eminently probable that the ulceration of the brachial and posterior tibial vessels, the ulceration and dilatation of the right common iliac and cerebral arteries, and the dilatation of the left external iliac, were all alike due to an inflammatory process set up in them by infective emboli, some of which were found *in situ* after death.

4th. The fact that in a very great proportion of all published cases the form of heart disease, found after death, was warty or fungating, and in 40 per cent. combined with ulceration and destruction of the valves, affords the strongest argument in favour of the theory that not only in our case but in all others of aneurism due to embolism, the peculiar form of heart disease present is the real explanation of the arterial lesion.

5th. Of fourteen specimens examined by the writers, exclusive of that which is the subject of this paper, the diseased artery has seemed to be distinctly ulcerated and not the seat of aneurism in three instances, viz.:

Ulnar artery.—Guy's Hospital Museum, No. 1503[30].

Axillary artery.—Guy's Hospital Museum, No. 1502[20].

Common femoral artery.—Guy's Hospital Museum, not numbered.

In the following cases an examination rendered it probable that the artery was ulcerated rather than dilated. This could not, however, be distinctly proved.

Common femoral artery.—Guy's Hospital Museum, No. 1519[21].

Popliteal artery.—Guy's Hospital Museum, No. 1519[39].

Superior mesenteric artery.—Guy's Hospital Museum, No. 1504[45]. St. George's Hospital Museum, No. 115.

Posterior tibial.—St. George's Hospital Museum, No. 129.

The ulceration in the case recorded by us, and in the other cases to which we allude, certainly cannot be the result of a mere mechanical distension, and, in our opinion, affords strong evidence in favour of a specific action of the embolus itself.

In conclusion, we beg to submit the following propositions :

1st. Embolism may undoubtedly cause the formation of an aneurism.

2nd. The aneurism is developed at the seat of embolism and not above it.

3rd. The aneurism may be due to an inflammatory softening and consequent dilatation of the artery, or to its ulceration, and the subsequent formation of a sac by the surrounding soft tissues.

4th. Aneurism only occurs when the emboli are derived from a heart, the valves of which are affected by a special form of disease, resulting in the production of fungous growths, and often of ulceration and destruction of the valves themselves.

(For report of the discussion on this paper, see 'Proceedings of the Royal Medical and Chirurgical Society,' New Series, vol. ii, p. 154.)

DESCRIPTION OF PLATES IV, V, AND VI.

(A Case of Multiple Embolism of the Arteries of the Extremities, followed by the formation of Aneurisms. By JOHN LANGTON, F.R.C.S., and ANTHONY A. BOWLBY, F.R.C.S.

PLATE IV.

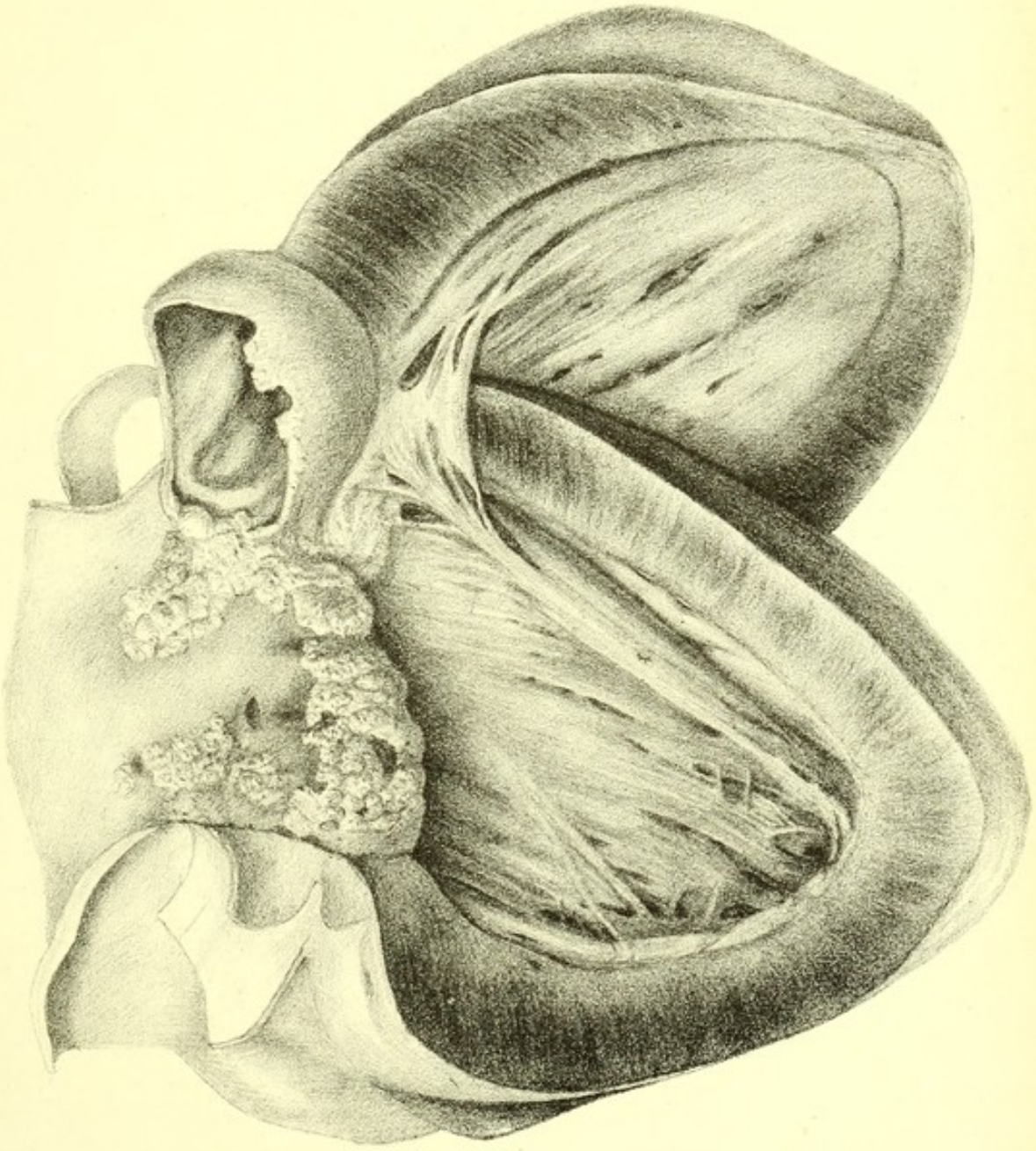
The left side of the heart, showing warty growths on the aortic valves with ulceration of one of the cusps, and the formation of an aneurismal pouch behind the left posterior segment; the aorta is in part covered by granulations, and about an inch above its origin presents a small ulcer.

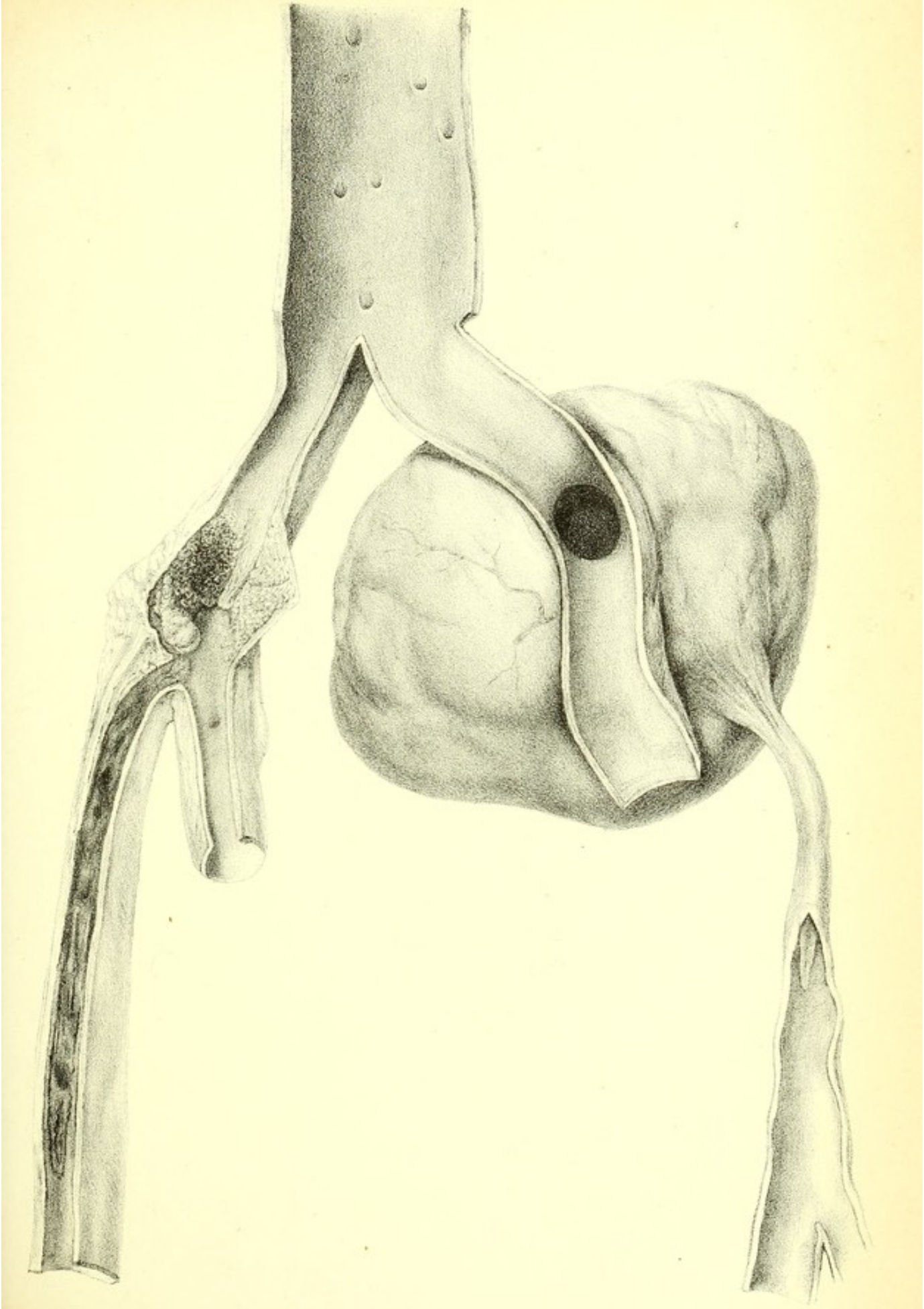
PLATE V.

The aorta, with the iliac and femoral vessels. The right common iliac contains a calcareous embolus just above its bifurcation, and in this situation the vessel is a little bulged and its wall ulcerated. The left external iliac is the seat of an aneurism, and the left common femoral is shrunken and filled with an old adherent clot.

PLATE VI.

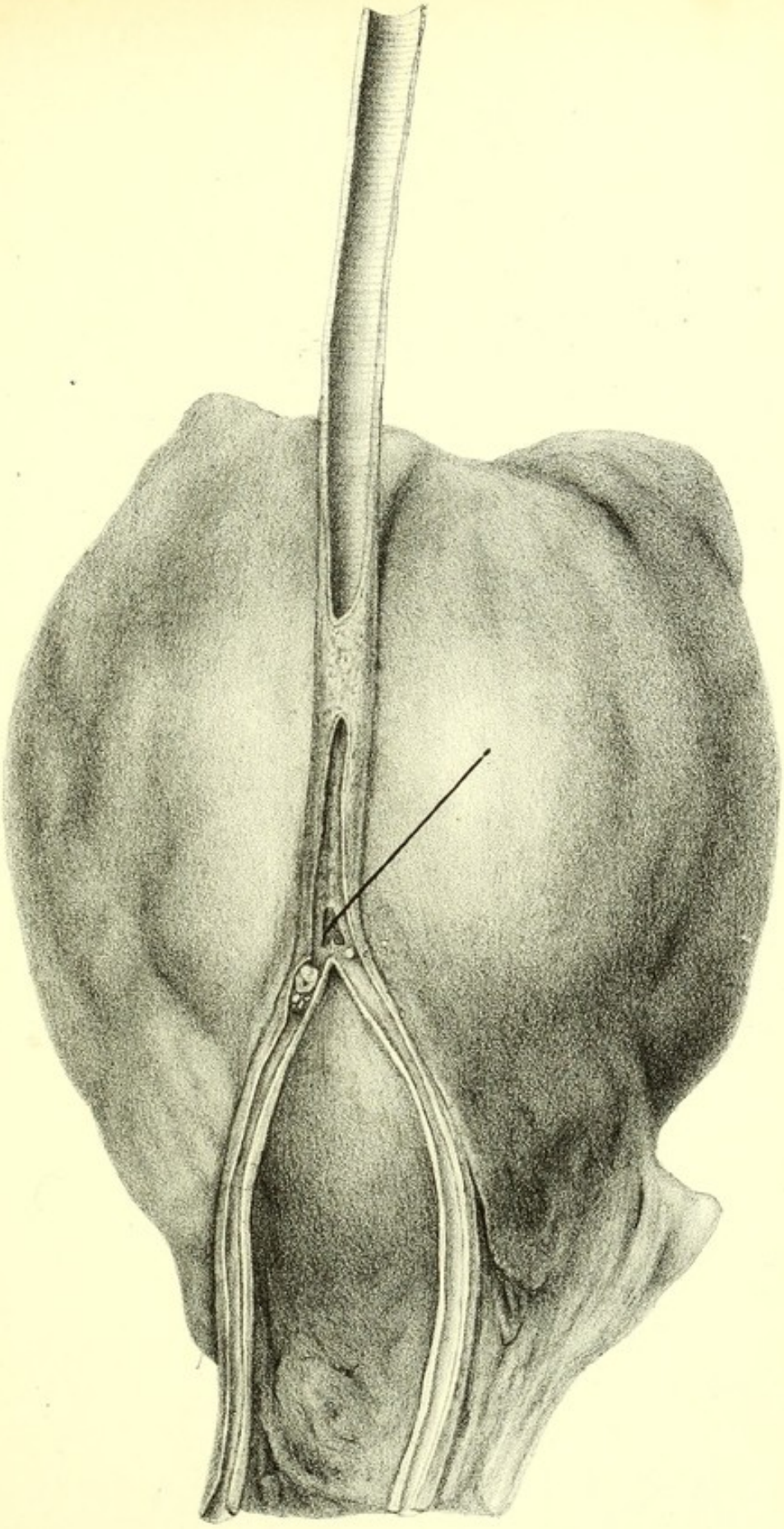
The right brachial and ulnar vessels. A bristle has been passed through an ulcerated aperture in the brachial, just above its bifurcation, into the sac of a diffused aneurism. The radial and ulnar arteries contain portions of a calcareous embolus. The brachial artery has been tied a little above the place where it is ulcerated.





T. Godart del.

Mintern Bros. lith.



Godart del.

Mintern Bros. lith.

ON THE

EFFECT UPON THE FEMORAL ARTERY OF ITS LIGATURE

FOR THE

CURE OF POPLITEAL ANEURISM.

BY

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Received June 8th—Read December 14th, 1886.

When the femoral artery is tied at the apex of Scarpa's triangle for the cure of popliteal aneurism, what effect is produced on the vessel between the point of ligature and the sac? In what state is the artery found at a subsequent period? Is it pervious or impervious? With the view of obtaining a satisfactory answer to this question I have examined all the preparations of popliteal aneurism in which the artery had been tied that I could find in the museums of the London hospitals, and the result is set forth in the following table.[1]

[1] I am greatly indebted to Mr. D'Arcy Power for the important help he has given me in examining and describing these cases, and I am glad to have this opportunity of acknowledging it.

Table of Cases in the Museums of the London Hospitals in which the Femoral Artery has been tied in Scarpa's triangle for the cure of Popliteal Aneurism.

Hospital.	Duration of life after operation.	State of artery between ligature and sac.	State of aneurism.
St. Bartholomew's	6¾ years	Pervious from ligature to anastomotica magna; from anastomotica to sac plugged by dark-coloured, imperfectly laminated clot	A firm, dense mass of fibrous tissue.
,,	6 years	Pervious from just below ligature to aneurism	A solid mass of fibrous tissue.
,,	11 years	Obliterated for two inches below ligature; then pervious, but much contracted	The aneurism is cured.
,,	18 months	Pervious, but lined with a thin clot of blood of variable consistence, as if in process of absorption	Filled with firm clot.
,,	A long while	Obliterated at seat of ligature; only two inches of artery shown below; this is pervious	Not shown.
St. George's	2 months	Only one and a half inches of artery left above the sac; this is quite pervious	The sac is full of laminated clot.
,,	No history	The artery is quite pervious throughout from the point of ligature to the sac	The sac contains a small amount of discoloured clot.
,,	4 years	Artery between point of ligature and sac pervious and injected	The sac is now quite empty; the aneurism was not cured; pulsation reappeared.
,,	7 days	Artery pervious throughout	The sac is full of dark-coloured laminated clot.
Guy's	12 days	Pervious	Not shown.
,,	11 days	Upper half filled with firm clot, tapering below; lower half contracted, but pervious	Lined with laminated clot.
St. Thomas's	Some days only	Upper portion only of the artery is preserved, which is pervious, except where a very small clot lies below ligature	Not shown.
,,	Some days only	Pervious; the ligature was placed above the profunda femoris	Partially filled with laminated clot.

Hospital.	Duration of life after operation.	State of artery between ligature and sac.	State of aneurism.
University College	6 days	The femoral artery bifurcated half an inch below profunda, and the divisions reunited seven inches lower down; a clot extended from the ligature on one of the divisions to the point of their reunion	Completely filled with clot.
„	Some days	Only a portion of the artery in connection with the sac is preserved; this is pervious	Almost empty; only a thin layer of laminated clot.
„	3 months	Obliterated at seat of ligature; pervious below	A solid mass of dense fibrous tissue.
„	43 days	Pervious, but very much contracted	Filled with firm coagulum.
King's College	18 days	Occupied by clot for three inches from sac; the only portion preserved	It contains some fibrous coagulum.
London	A few days only	Pervious, except a plug for one and three quarter inches below ligature	Not shown.
„	7 years	Occluded for two and a half inches below ligature; then patent to origin of anastomotica magna; below this again occluded	A mass of fibrous tissue.
St. Mary's	2½ years	Pervious; injected throughout	Aneurism controlled for some time; but recurred after many months.
„	Some days	Artery patent throughout; even pervious through aneurism	Sac filled with stratified clot.
„	2 or 3 years	Solid for an inch below ligature; the rest of it pervious throughout	Aneurism much shrunken. Cured.
„	Several years	Clot for an inch below ligature; artery then pervious for three inches; then clot again for three inches	Aneurism shrunken. Cured.
„	Many years	Pervious throughout	Cured. Shrunken.
Middlesex	12 years, then amputation	Pervious	A mass of dense fibrous tissue.

These cases may be thus arranged:

Total number of cases 26
Complete specimens . . 17
Incomplete specimens . . 9

In the complete specimens:		
Artery pervious throughout . . .		13
Artery partially pervious . . .		4
Number of cures		15
In these artery pervious .	11	
In these artery partially closed	4	
Number of failures		2
In these artery pervious . .	2	
In these artery partially closed	0	

Now, I think that most surgeons will hardly be prepared for these results. That in many cases after ligature of the femoral artery in Scarpa's triangle the trunk of the vessel below remains to a greater or less extent pervious is a fact well known to all of us; but I think, also, most of us would have said that in many cases, perhaps in the majority, the canal becomes obliterated. But this table of twenty-six cases furnishes no single instance in which after the operation the artery was rendered impervious throughout. Of seventeen specimens which are perfect, in four instances it is to a greater or less extent pervious, and in thirteen instances it is patent throughout. Very probably, in almost all cases, it is diminished in size, but this point cannot be determined clearly in the preserved specimens, and the extent to which it remains patent is no doubt greatly influenced by the size, number, and arrangement of its branches. The specimens, however, hardly afford demonstration of this, but it is clear that while in some cases branches arise from the patent portions, in others they arise from a part that has become impervious.[1]

[1] The obliteration of the trunk of an artery below a ligature has been, perhaps, generally regarded as a very gradual process. For instance, Sir Astley Cooper wrote—

"When a ligature has been applied it is some time before the artery below becomes obliterated. Mr. Forster, surgeon of Guy's Hospital, has a drawing in his possession of a case of popliteal aneurism for which the femoral artery

But the fact is abundantly shown that obliteration of the artery is by no means necessary to the cure of the aneurism. Here in fifteen cases the aneurism is cured, and yet in all the artery is to a large extent pervious; in eleven it is pervious throughout. It is clearly enough, as there is ample evidence to prove, that the pressure of the blood stream on the sac be reduced. This, indeed, may be said to be the principle of the operation which Hunter performed as distinguished from all previous ones; that branches may be allowed to intervene between the site of the ligature and the sac, and so blood may still find its way freely though circuitously from above downward to the main trunk and thence to the aneurism, and yet nevertheless by thus breaking the force of the main stream a cure will follow. Not invariably, indeed, on this account, for occasionally, as is well known, although the artery as usual becomes obliterated at the seat of ligature, the supply remains too free and the pressure too strong on the aneurismal sac and so it continues to yield. In such instances the ligature of the main trunk does not from various causes—among the chief of which must be the freedom of the collateral current and the character and condition of the aneurism—suffice to restrain the circulation to a sufficient degree, and these instances throw a strong light on the principle of cure in the treatment by compression above and the frequent failure of this method.

The following case is of interest from this point of view,

was tied in August; the man died in January following, and when the limb was injected, the femoral artery, below where the ligature had been applied, was found to have received a part of the injection by communicating vessels.

"It is for this reason that the pulsation in some aneurisms where there are free communicating vessels, will remain for a considerable time after the operation. However, in twelve or fourteen months, when the femoral artery has been tied, it becomes obliterated, above the ligature to the arteria profunda, and below it, to the origin of the anterior tibial artery; and sometimes the remnant of the vessel is not only converted into a cord, but is ossified" ('Medico-Chirurgical Transactions,' vol. ii, 1811, p. 253).

But this table clearly shows that many years may elapse, 6, 7, 11, 12, and yet the artery may be either in part or throughout pervious.

but it is instructive chiefly in showing that while a ligature upon the artery far away from the aneurism may fail, a ligature lower down, nearer to the sac, may succeed.

J. J—, æt. 37, a hawker, was admitted into St. Bartholomew's Hospital on the 18th of January, 1883, with a popliteal aneurism in the left ham in the form of a well-defined globular tumour about two and a half inches in diameter. It was much reduced when the femoral was controlled and there was a distinct murmur over the sac. He had been conscious of something wrong behind the left knee for three months. There was no history of syphilis or evidence of intemperance or of disease in the arterial system elsewhere.

On February 13th the femoral artery was tied at the apex of Scarpa's triangle with a silk ligature. All pulsation in the sac at once ceased. The wound healed by the first intention throughout except where the ligature passed out. This, which was rather stout, did not separate till the thirty-first day. On the next day very slight pulsation could be detected on the inner side of the sac, but not elsewhere. The tumour had gradually diminished in size and become more solid. On the 24th of March he was discharged.

In February, 1884, he was re-admitted with distinct pulsation in the tumour, which was then quite as large as before the operation. The pulsation was greatly reduced, but not entirely controlled by pressure on the femoral above the scar of the operation. Since his discharge he had been, for many hours of each day, on his feet following his business as a hawker. On one day in the previous November, after a walk of twenty miles, the tumour increased in size, and became painful.

By rest and the continual application of an elastic bandage the pulsation almost subsided, and he left the hospital in the end of May.

In February, 1885, he was again admitted with the aneurism in much the same state as when he came to us the year before. The tumour felt solid, but with distinct

pulsation, most plainly felt on the inner side. This was almost completely stopped by pressure on the femoral above. The man was kept at rest for some months with the limb bandaged and firm pressure over the tumour. On three occasions Esmarch's bandage was applied so as to control the circulation in the limb, for from half an hour to an hour. But no perceptible effect was thereby produced on the aneurism.

On August 6th I cut down on the popliteal artery between the opening in the adductor magnus and the tumour. The artery which was raised by the tumour and comparatively superficial, came readily into view. It appeared healthy, hardly diminished in size, and pulsating with almost normal force. It was tied about two inches above the point where it joined the surface of the sac, by a catgut ligature in two places, and divided between them. No other artery could be seen in relation to the sac. All pulsation in the tumour immediately ceased and never returned. The wound healed throughout by the first intention. The tumour rapidly subsided, and when the man left the hospital a month afterwards, it was greatly reduced. He has not been since seen.

Other cases similar to this can be produced, in which, while ligature of the artery at the distance of Scarpa's triangle failed to effect a cure by not sufficiently restraining the current through the sac, the aneurism was forthwith controlled and cured when the artery was subsequently tied much closer to it. And, in view of the patency of the artery below, after ligature in Scarpa's triangle, I would suggest that when this operation fails to cure a popliteal aneurism, in the absence of any special objection, the artery should be tied lower down; if practicable, in the upper part of the popliteal space, so as to be below the anastomotica magna. This may make all the difference, as in the following case:

A man, æt. 48, was admitted into St. Bartholomew's Hospital under the care of Mr. Smith in October, 1881, with a popliteal aneurism. Fifteen months previously he

noticed a swelling in the right ham for which, seven months later, the femoral artery was tied in Scarpa's triangle. This was followed for three months by a reduction of the tumour, but afterwards it steadily increased.

On admission the aneurism was as large as a common orange with considerable impulse. The femoral was occluded in the apex of Scarpa's triangle, but the popliteal pulsated strongly. Pressure on the common femoral stopped all pulsation in the limb. Bandaging and flexion having failed, the superficial femoral was exposed in Hunter's canal, and as it was found to be pervious though small, it was tied in two places and divided between the ligatures. This did not stop the pulsation in, or control the aneurism. In May, 1882, after another trial of pressure and flexion without avail, the popliteal artery was tied in two places and divided between the ligatures, an inch above the aneurism. Then all pulsation at once ceased and the sac rapidly diminished.

I would go even farther than this, and suggest that in some, perhaps in many, cases of popliteal aneurism it would be better to tie the popliteal artery in the first instance rather than the femoral. The one single objection of any weight to this operation is in the risk that the artery so near the aneurism may prove to be diseased. But is not the chance of this somewhat exaggerated? At least in many cases of popliteal aneurism it shows no signs of it, and the frequent occurrence of aneurism in the popliteal artery is perhaps more largely due to the position of the artery and the strain to which it is occasionally subjected than to any material deterioration of its structure. Moreover, let it be remarked that we are already accustomed, without much fear of the consequences in this respect, to place ligatures on other arteries for the cure of aneurism elsewhere at a very short distance from the sac, as in ligature of the subclavian for axillary aneurism and of the carotid in the neck below for aneurism above.

The increased difficulty of ligature of the popliteal over

the femoral need hardly be taken into account. And for the rest, with regard to the two operations, the advantage appears to me to be with ligature of the popliteal, mainly on the score of the greater certainty of its success in the cure of the aneurism. In view then of the number of cases known to us in which ligature of the femoral artery has failed I would ask whether we may not reconsider the question of ligature of the popliteal artery for the cure of popliteal aneurism ?

(For report of the discussion on this paper, see 'Proceedings of the Royal Medical and Chirurgical Society,' New Series, vol. ii, p. 162.)

RICHTER'S HERNIA

OR

PARTIAL ENTEROCELE.

BY

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Received July 12th—Read December 14th, 1886.

THE variety of strangulated hernia with which the present paper deals has attracted but little attention in this country. In the majority of English text-books it receives no notice, and in those treatises in which it is mentioned it is usually disposed of in a few lines. The hernia is rare, but not so rare as to be merely a curiosity. The only two specimens of it that exist in the museum of the College of Surgeons were recently presented by myself, and yet, on the other hand, I have collected over fifty recorded instances of this peculiar lesion.

The main features of the hernia are these. A part only of the circumference of the bowel is engaged and strangulated in the hernial orifice; the involved segment may rapidly pass into gangrene, and yet the lumen of the gut remain free; the portion snared projects from the surface of the intestine as a small, rounded, bud-like diverticulum.

The nomenclature of this hernia is involved in no little confusion. English writers either describe it without special name or speak of it as Littre's hernia. This latter term is employed by most German and French surgeons, but is applied to a variety of rupture quite distinct from that now under notice.

In 1700, Littre[1] (who at that time signed his name De Litre) published an account of "a new form of hernia," and detailed three cases. Two of these were examples of a Meckel's diverticulum finding its way into a rupture. The third case was of too vague a character to be of any scientific value.

Littre believed the diverticulum to have been developed in the hernial sac, as the result of constriction and pressure.

The following were the diagnostic points of this "new form of hernia:"—abdominal pain with prostration, absence of constipation, of meteorism, and of hiccough, vomiting that was of slight severity and never feculent.

The precise nature of the diverticle was not demonstrated until the early part of the present century, when its anatomical character was established by Joannes Meckel.[2] Before Meckel's time, however, it had been discovered by several observers in hernial sacs. Thus Méry,[3] in 1701 reports a case of strangulation of the diverticulum in a hernia; other examples of this form of rupture were provided by Wrisberg,[4] Gunz,[5] Ruysch,[6] Morgagni[7] and others.[8] Some of these writers offered no explanation of these occurrences, others, like Méry, adopted the theory propounded by Littre. Since Meckel's time,

[1] 'Mémoires de l'Académie Royale des Sciences,' 1700, p. 300.

[2] 'Handbuch der menschlichen Anatomie,' Halle, 1815-20.

[3] Mém. de l'Acad. Royale des Sciences,' 1701, p. 272.

[4] Loder's 'Journal für die Chirurgie,' Bd. 1, p. 182, Jena, 1797.

[5] 'De Herniis,' Lips., 1744, p. 6.

[6] 'Thesaurus Anat.,' Amsterdam, 1707.

[7] 'Adversaria Anatomica,' 1741.

[8] See Cazin, 'Sur les diverticules de l'intestin,' Paris, 1862, p. 91, and Reicke's work.

a large number of examples of hernia of the diverticulum, both strangulated and not strangulated, have been placed on record. The symptoms that arise when the process alone is strangulated, conform very generally to those described by Littre, and also bear resemblance to those that attend strangulation of a partial enterocele.

It is to the rupture that contains a Meckel's process that the term Littre's hernia should alone be applied.

French authors do not employ the name of Littre in an adjectival sense in connection with any kind of rupture, but use the term diverticular hernia (hernie diverticulaire) for this especial variety. German writers strictly limit the title Littre's hernia—a term very generally adopted—to the rupture that contains a Meckel's diverticulum, employing also as a synonym the expression diverticular hernia (Divertikelbrüche, Darmanhangsbrüche).

The history of the partial enterocele is obscure, and that the two varieties of hernia have been confused together, is a matter for no surprise. It is usually said that Fabricius Hildanus[1] was the first to recognise a partial enterocele. A reference to the works of this writer does not fully confirm this. In 1598 Hildanus attended a lady of sixty-three, who had a hernia of the groin of seventeen years standing. It became strangulated, and was apparently reduced *en masse*. Gangrene occurred, and an artificial anus followed, which closed spontaneously in two months. Hildanus never saw the bowel, and only hazards a conjecture that it was engaged in the ring without forming a fold or loop. This conjecture would seem to have been founded upon the circumstance that the fæcal fistula healed spontaneously.

In 1714 Littre[2] published a case of strangulation of a part of the colon, the lumen of the tube being still patent. Le Dran[3] in 1731, describes what is apparently a genuine

[1] Cent. 1, 'Obs. Chir.,' 55, 1606.

[2] 'Mémoires de l'Acad. Royale,' 1714, p. 200.

[3] 'Observations de Chirurgie,' T. ii, p. 37, Paris, 1731.

example of this form of hernia. George Arnaud[1] in 1749, makes brief mention of it in his work on hernia.

In 1757 a remarkable paper was published by Louis[2] on gangrene after hernia. In it he describes the partial enterocele. "The gut," he writes, "is not always engaged in the ring by a portion large enough to form a loop. Often it is only nipped, and that over a surface of varying extent." He states that such strangulated herniæ are attended by milder symptoms, and that they quite commonly lead to gangrene. The illustrative cases, however, quoted by Louis do not all bear examination. He appears to have considered that if spontaneous cure followed the establishment of a fæcal fistula in these cases that the hernia was of the kind just mentioned. Examples of the partial enterocele were also described by Morgagni[3] and de Haen[4] about 1761.

It remained, however, for Augustus Gottlob Richter[5] to give, so far as I know, the first really scientific description of this particular hernia. His treatise on hernia was first issued in 1778. In this work he devoted a separate chapter to what he terms "small ruptures," and therein gives a full, faithful, and elaborate description of the hernia in which a part only of the circumference of the bowel-wall is strangulated. His account of the clinical aspect of the condition is excellent, he recognises Louis' work, and also appreciates the true nature of the hernia that was first described by Littre.

Of the surgeons who have noticed this hernia since Richter's time little need be said. A list of the principal writers will be found in the bibliography at the end of this paper.

One word now on the subject of nomenclature. The French surgeons give to this variety of strangulated

[1] 'Traité des Hernies,' Paris, 1749.

[2] 'Mém. de l'Acad. Royale de Chirurgie,' 1757, T. viii, p. 26.

[3] 'De Sedibus et Causis Morborum,' Venet., 1761, Epist. 34, Art. 18.

[4] 'Ratio Medendi,' Lugd. Bat., 1761—75.

[5] 'Abhandlung von den Brüchen,' 1778, 1785.

hernia the titles "Pincement herniaire de l'intestin," "Pincement latéral," "Hernie partialle." The German writers employ the terms "Darmwandbrüche," "Lateralbrüche," "Partialbrüche." In English there is need of a more convenient title. The term "partial enterocele" is not quite clear, and exempts the colon from participation in this variety. The title "small hernia," employed by Cooper and others, is not sufficiently expressive, and the title "incomplete hernia," as used by Gross and some other surgeons, is confusing, since in connection with inguinal ruptures it is usually applied to cases where the gut has not left the inguinal canal.

I would venture to suggest the title "Richter's hernia," partly because a definitive title is not forthcoming, partly to make still more clear the lines that should separate this hernia from that known as Littre's, and partly because with Richter must rest the main credit of establishing the individuality of this lesion.

That the herniæ of Littre and of Richter should have been confused with one another can be no matter of wonder. Such confusion is to be especially found in the reports of English surgeons. By some the term Littre's hernia includes only the partial enterocele, by others it embraces both the partial enterocele and the diverticular hernia, while by a third class of writers all expressive titles are avoided.

Before describing the clinical aspects of Richter's hernia I might give a brief account of four cases that have been under my care at the London Hospital.

Case 1. A woman, æt. 62, admitted July 24th, 1883. She presented a very small femoral hernia of the right side that had appeared suddenly during exertion three days previously. She was at once seized with abdominal pain, lost her appetite, and became much prostrated. On the second day—but not before—vomiting set in and persisted. On the day of admission the abdomen was a little distended, there was much colicky pain, and frequent vomiting. As the patient would consent to no operation

an ice-bag was applied. On the fifth day of the strangulation, the patient's symptoms being in no way relieved, she consented to herniotomy, which was at once performed. A portion of ileum was held in the femoral ring; the piece so strangulated did not represent more than one third of the circumference of the gut, and projected from the surface of the intestine like a bud. It was gangrenous. Two inches of the bowel were excised, the canal restored by two lines of suture, and the intestine reduced into the abdomen. The patient was much exhausted at the time of the operation, and died two days after its completion. The autopsy showed that the suture line in the gut was well healed, and there was no peritonitis. Throughout the progress of the case there was absolute constipation. The vomiting never became feculent. The specimen is in the museum of the College of Surgeons.

Case 2. A woman, æt. 48, admitted in August, 1884, with a strangulated hernia in the right femoral region. This rupture had existed for twelve years, had been always reducible, and had never been supported by a truss. The symptoms began with abdominal pain, vomiting did not set in until twelve hours had elapsed. The bowels were well open on the first day. Herniotomy was performed upon the third day of the strangulation. A well-marked Richter's hernia of the ileum was discovered. The bowel was not gangrenous, and was reduced in the usual way. The vomiting continued. The bowels were well opened on the fifth day after the operation. The patient died, apparently of a low form of peritonitis, six days after the herniotomy. No autopsy was permitted. In this case constipation was absolute after the first day. The vomiting was not urgent and never feculent.

Case 3. A man, æt. 60, admitted March 13th, 1885, with a strangulated right femoral hernia. The rupture had been noticed for eighteen years, but formed a tumour no larger than the top of an adult's thumb. The patient was very thin. No truss had been worn, and the rupture had never given trouble. Three days before admission—after

an attack of coughing—the hernia became painful. Violent taxis was at once employed, and as the swelling was not diminished it was poulticed. The bowels were well opened on the evening of the first day. On the second day vomiting set in. It became frequent, but was never feculent. The abdominal pain increased, and the patient suffered from distressing hiccough. Herniotomy was performed on the fourth day of the strangulation. A Richter's hernia was found. The lumen of the ileum was not entirely occluded. The sac contained omentum. As the bowel appeared in fair condition it was reduced. For some time the patient did well. On the fifth day after the operation he left his bed—against orders—and walked about the ward in the absence of the nurse. The next day (the sixth) he died suddenly. The autopsy revealed a small perforation in the lately strangulated segment through which fæcal extravasation had taken place into the peritoneal cavity. In this instance constipation was absolute after the first day. The vomiting was never feculent. The specimen is in the museum of the College of Surgeons.

Case 4. A man, æt. 44, admitted August 6th, 1885, with strangulation of a right oblique inguinal hernia. The rupture was of two years standing, was about the size of a walnut, and always reducible. A truss had been worn irregularly. On August 3rd, after exertion, the hernia became painful and irreducible, colic appeared, and in two hours slight vomiting. In three hours the bowels acted. A copious motion was also passed on the next day, but after that the bowels remained confined. On this same day (August 4th) the hernia was reduced after violent taxis lasting thirty minutes. It proved to be a reduction *en masse*. The patient was sick seven times on this day, and three times on August 5th.

On admission the man's condition was good, the tongue clean, the abdomen not distended. The colic and vomiting had persisted. The inguinal canal was at once cut down upon. A reduction *en masse* was discovered. The

sac contained a little omentum, and a portion of small intestine that formed a Richter's hernia. The piece of bowel picked up was about the size of a cherry. It was of a deep purple colour. The lumen of the intestine was free near the mesentery. The strangulating agent was the neck of the sac. The sac was removed and ligatured, and the canal closed in the usual way. The patient made a rapid recovery.

The account that I will now give of *the morbid anatomy and clinical aspects* of Richter's hernia is founded upon the examination of fifty-three recorded cases, among which the four cases just described are included. The number of reputed examples of Richter's hernia that surgical literature presents, greatly exceeds that just enumerated. I have met with some eighty cases in all. About thirty of these, however, are either so imperfectly reported as to be valueless, or do not afford substantial evidence as to their identity.

An analysis of these cases is furnished at the end of the paper. Richter's hernia is more common in women than in men, and is limited to adults, the average age being fifty-three. It is met with more frequently in the femoral than in the inguinal region, and upon the right side than upon the left. It occurs in old herniæ in preference to those of recent formation, and is more often associated with ruptures that have been reducible than with those that have become irreducible. The tumour that existed was in all the recorded cases very small, varying in size from a nut to a hen's egg. In nearly 50 per cent. of the cases the rupture was neither recognised nor suspected during life. In all these instances death followed. In no less than seven of the cases of femoral hernia (the total number of which was thirty-eight) an enlarged gland was found immediately in front of the femoral opening. This is a feature that is deserving of notice. In some of these cases the gland hid the hernia entirely, while in others it led to the diagnosis of acute adenitis as the cause of some of the local symptoms.

In each instance the gland would appear to have been solitary, and of recent appearance. It is well known that certain lymphatic vessels having left the femoral glands make their way through the crural canal to reach the intra-abdominal glands. If a hernia accurately engages the femoral ring it is conceivable that these efferent vessels would be compressed. Such impediment in the lymph circulation may affect the more superficial glands, they may be engorged with lymph and become enlarged either on that account alone or by reason of some adenitis induced by the lymph stasis. The presence of a solitary enlarged gland immediately in front of the crural canal may at least excite suspicion that the lymph-vessels in that canal are exposed to compression.

The segment of bowel engaged is nearly always the ileum, and especially the lower portion of it. In only three cases is the jejunum said to have been involved, and in only one case the colon. A part of the circumference of the bowel only is strangulated. In the case of femoral hernia the constricting agent would appear to be the crural ring, and in the case of inguinal ruptures, most usually the neck of the sac. The amount of bowel that is strangled varies. In some cases as much as four fifths is involved, in others three fourths, two thirds, or one half. Probably in the majority of cases less than one half of the circumference of the tube is held. In many instances the portion strangulated represented only one sixth. Gross states that he has seen several such cases. The strangulated portion is invariably of circular outline, and forms a prominent projection from the surface of the unimplicated bowel. The little projection, which tends to become slightly pedunculated, may be no larger than the tip of the little finger, or may reach the size of "a pea-nut without its shell," or attain the dimensions of a cherry. Excellent drawings of the condition are given by Gross[1] and Dent.[2]

[1] 'System of Surgery,' 1872, vol ii, p. 613.

[2] 'Trans. Clinical Society,' vol. xv, 1882, p. 16.

In no less than twelve fatal cases that were examined after death it is expressly stated that the lumen of the gut was in part clear. In some cases nearly two thirds of the lumen was patent, while in other examples no more than a director could be passed by the strangulation. Scarpa's experiments, as detailed in his work on hernia, showed that when two thirds of the circumference of the ileum was artificially strangulated, the passage of water along the tube was more or less completely arrested by reason of the bending of the bowel, but that when only one third was involved fluid passed easily.

In every example in which the description is complete it is shown that the particular part of the bowel-wall involved is that most remote from the attachment of the mesentery. In no instance does the mesentery ever fall within the grip of the constricting ring.

The mode of formation of Richter's hernia is a matter open to dispute. Many surgeons believe, and the belief is of ancient foundation,[1] that a piece of small intestine becomes adherent over the hernial ring, and that partly from intra-abdominal pressure and partly from pressure of the intestinal contents, the segment of the bowel wall that covers the ring is gradually forced through it. In this way a lateral protrusion is formed, and if at a future date the little diverticulum becomes strangulated, the phenomena of this particular rupture are produced.

Schmidt[2] fully accepts this theory. He goes further and doubts the existence of a free Richter's hernia, *i. e.* of one where no adhesion has taken place between the sac and the intestine.

While there is good reason for assuming that the theory that underlies Schmidt's belief is, in part at least, well founded, there is no evidence to show that it can apply to all cases. I can find but few instances in which it was quite clear that the gut was adherent to the sac. In the

[1] Riecke, 'Ueber Darmanhangsbrüche,' Berlin, 1841.

[2] "Die Unterleibsbrüche," Pitha und Billroth's 'Handbuch,' Band 3, Abth. 2, Stuttgart, 1878.

majority of cases the two were free, and it is remarkable that in instances where herniotomy was performed there was no difficulty in liberating and reducing the bowel. If no other evidence existed than that afforded by my own specimens at the College of Surgeons, they would be alone sufficient to refute Schmidt's assertion. They would serve to disprove also the grounds upon which this writer's scepticism is founded. He believes with Roser[1] that the reported cases of Richter's hernia as an acute condition are valueless, that the reporters have been deceived by appearances, and that the bladder-like projection on the bowel is produced really by the whole loop being firmly pinched in at its base while it has become distended at its summit. Roser gives some ingenious diagrams to illustrate his views. On the other hand, that a portion of small intestine may become adherent over the hernial orifice, and that the bowel wall so placed may become protruded through that opening must be allowed. It may also be conceded that a diverticulum may form when a portion of the bowel is adherent to the wall of a very small hernial sac. Albert[2] describes this condition as the chronic form of the partial enterocele. He states that the pouch that is formed in the bowel interferes with peristaltic movements, and that in time some stenosis is produced with all the symptoms of chronic obstruction of the small intestine. Quite apart, however, from the formation of any diverticulum, it is known that adhesion of the lesser bowel to any unyielding parts may produce such changes as will in time lead to the symptoms of chronic stenosis.[3]

Observations that apply to the small intestine cannot be made in equal degree when the colon is concerned. Many cases have been noted where a part of the circumference of the colon has been found to be engaged in a hernial sac. This has been especially observed in connec-

[1] 'Handbuch der anatomischen Chirurgie,' 5 Aufl., p. 327; and 'Centralblatt f. Chirur.,' No. 24, 1886.

[2] 'Lehrbuch der Chirurgie,' Vienna, 1885, vol. iii, p. 316.

[3] Treves, 'Intestinal Obstruction,' London, 1884.

tion with small umbilical herniæ. An instance of strangulation of a part of the cæcum is reported by Lorenz.[1] So far as the partial enterocele is concerned, the strangulation would appear to be sudden and to ensnare a piece of bowel that had been in no way prepared for the accident. In many of the cases there is reason to believe that the actual formation of the hernia and the strangulation of its contents were the simultaneous effects of the same accident. In five cases of Richter's hernia (two inguinal and three femoral) omentum was found in the sac. In each instance the hernia had existed for two or more years.

The symptoms of this rupture as gathered from an examination of fifty-three cases are as follows: In about one third of the examples the symptoms differed in no way from those that attend typical cases of strangulated hernia. There is the same prostration, the same condition of pulse and tongue, together with absolute constipation and persistent vomiting. It is interesting to note that in several of these instances the lumen of the bowel was found to be free, to a varying extent, at the time of death. With regard to the remaining cases, it may be said that the symptoms generally were much less severe and pronounced than are those usually attending strangulation. In about one tenth of these a motion was passed on the first or first and second day of the strangulation. In the other cases the bowels continued to act from time to time. Sometimes a motion was passed without aperients every day. For example, in a case of eight days' duration, which ended in gangrene, the bowels remained perfectly regular. In other instances the bowels responded to aperients whenever given during the progress of the case. Thus in a patient who presented evidences of strangulation for ten days, an aperient was followed by three loose motions on the ninth day. The gut was gangrenous, and four fifths of its circumference were involved. In the remaining examples, a stool was passed without artificial aid on the third, fourth, fifth, or sixth day of the strangulation. As

[1] Quoted by Albert, loc. cit.

regards prognosis, no difference is to be noticed between the cases attended by constipation and those in which the bowels were regular. In three cases there was persistent diarrhœa throughout the whole progress of the trouble. This appears to have been due to enteritis. All the patients died. In one case where death occurred on the ninth day, no motions were passed, but a purge was followed by a discharge of bloody mucus.

With regard to vomiting, it is upon the whole less frequent and less severe than in the usual form of strangulation. It may not appear until the second day, and may become less urgent as time progresses. In several instances the patient did not vomit more than three times a day throughout the progress of the affection. There is no connection between the condition of the bowels and this symptom. In some cases where the bowels were opened regularly, the vomiting was marked and persistent, while in others of like nature the vomiting was slight and tended to become less. The vomiting is rarely feculent, the fifty-three cases affording only six examples. The earliest appearance of this symptom was on the fourth day of strangulation, and the latest appearance on the eighth day. In three of the cases there was absolute constipation, in two motions were freely passed on the second day, and in the remaining instance no motion, but bloody mucus was passed, and that after a purge. One of these cases serves to illustrate the uncertain progress of the vomiting. The patient was a man aged 65, the hernia femoral and of ten years' standing. The symptoms began with pain, prostration, and colic. Vomiting commenced on the second day, and on that day the bowels were relieved. On the third and fourth days there was no vomiting at all. On the fifth day there was copious bilious vomiting, and on the sixth day the ejected matters became feculent.

Hiccough is quite uncommon, and I can find only five cases in which it occurred. In one of these it was one of the earliest symptoms, and persisted until the patient's

death on the sixth day. In the remaining cases it appeared late.

Meteorism and distension of the abdomen are very rarely met with. In those cases in which the bowels were freely opened this symptom was entirely absent. In other instances it was influenced by the same conditions that influence tympanitic distension in the simple forms of strangulated hernia.

The tumour in these cases being very small is difficult to examine, but when large enough for its physical conditions to be made out it presents no other than the features of an ordinary strangulated rupture.

In some cases a truss was worn, in others not. In some, the rupture was reducible in others irreducible. One striking feature in Richter's hernia is the great difficulty experienced in effecting reduction by taxis. This end was only accomplished in two cases. In each instance the hernia was femoral, and death rapidly followed from acute peritonitis. In the majority of the recorded examples taxis had been tried. In four cases there had been reduction *en masse*. Of these three were femoral and one inguinal.

Another striking point in this variety of rupture is the frequency with which the gut becomes gangrenous. This may be explained by the fact that the constricting force comes direct upon the gut. The mesentery that may afford some protection is not involved. In many instances also the hernia escapes notice. Gangrene is met with in over 50 per cent. of all cases, and occurs more frequently in connection with femoral than with inguinal ruptures. It has been found as early as the third day of strangulation. On the other hand, in cases where strangulation symptoms have persisted for seven, eight, and even nine days the gut has been found still viable. Both these extremes are afforded by femoral herniæ.

The average *duration of strangulation* before death or relief by operation is in femoral hernia five and a half days, in inguinal seven days, and in ventral four days.

The *mortality* in Richter's hernia is, as may be supposed, very high, being represented by 62·2 per cent. It is relatively higher in femoral than in inguinal ruptures. The mortality also after herniotomy in these cases reaches a very high figure. Details upon these points are furnished in an appendix to this paper (see pp. 164, 165).

This high death-rate depends partly upon the frequency with which gangrene occurs, partly upon the fact that the character of the symptoms often encourages delay or leads to an incorrect diagnosis, and partly upon the difficulty of effecting reduction by taxis.

In the matter of *treatment* there is nothing very special to be said. It is evident that these herniæ demand early attention. I would urge that no attempt should be made to reduce the protrusion by taxis, but that herniotomy should be performed as soon as the condition is recognised. As an operative procedure herniotomy is at the present day peculiarly simple, whereas taxis involves unusual risk to the gut, and unusual risk of reduction *en masse*. The sac should, in every instance, be freely opened, and steps taken to secure the protruded bowel before the stricture is divided. There is, so far as I know, no special point in connection with the treatment of the gut when gangrenous. One cannot, however, fail to be struck with the frequency with which spontaneous cure has followed in cases which have been practically left to themselves.

APPENDIX.

Total number of cases 53

	Males.		*Females.*			
"Femoral" .	4	...	14	=	18	
R. femoral . .	5	...	10	=	15	= 38 Femoral.
L. femoral . .	3	...	2	=	5	
"Inguinal" .	8	...	3	=	11	
R. inguinal .	1	...	0	=	1	= 13 Inguinal.
L. inguinal .	1	...	0	=	1	
Ventral . .	0	...	2	=	2	= 2 Ventral.
	22		31		53	

Age.

Males . .	Average 54	...	Youngest 17	...	Oldest 80
Females .	„ 52	...	„ 30	...	„ 80

Duration of Hernia.

Recent . .	Femoral . . .	11	
	Inguinal . . .	3	= 16
	Ventral . . .	2	
Duration not stated—	Femoral . . .	13	= 16
	Inguinal . . .	3	
Old . . .	Femoral . . .	14	
3 months to 18 years.			
Average—9 years.			
	Inguinal . . .	7	= 21
2 years to 13 years.			
Average—5 years.			
			53

Presence of Gangrene.

	Femoral.		*Inguinal.*		*Ventral.*
Gangrene of bowel . .	21	...	6	...	1
No gangrene present .	16	...	5	...	1
Condition not stated .	1	...	2	...	0
	38		13		2=53

Average Duration of Strangulation at time of Operation or Death.

Femoral = 5½ days. Extremes 2 days and 10 days.
Inguinal = 7 „ „ 4 „ 11 „
Ventral = 4 „

Mortality.

	Femoral.		*Inguinal.*		*Ventral.*		
Deaths . . .	29	...	3	...	1	=	33
Recoveries . .	9	...	9	...	1	=	19
Not known . .	0	...	1	...	0	=	1
	38		13		2	=	53

Herniotomy (34).

	Femoral.		*Inguinal.*		*Ventral.*
Deaths	18	...	1	...	0
Cures	6	...	4	...	0
Recovery with artificial anus .	3	...	1	...	1

No operation (16).

Deaths	9	...	2	...	1
Recovery with artificial anus .	0	...	4	...	0

Enterotomy or Colotomy.

Deaths	2	...	0	...	0
	38		12		2

One unknown.

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(For report of the discussion on this paper, see 'Proceedings of the Royal Medical and Chirurgical Society,' New Series, vol. ii, p. 167.)

CLINICAL OBSERVATIONS

ON

"INDURATION" IN THE PRIMARY LESION OF SYPHILIS IN WOMEN.

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THE description of the primary lesion of syphilis in works on that disease has, no doubt, been based mainly on its characters and course, as met with in the male sex. Under ordinary circumstances it comparatively rarely comes under notice in women; still more rarely is it seen in them in its earliest stages, and it does not present the same facilities for observation throughout its course.

If syphilis present any peculiarities in women distinctive from its effects on the other sex, they may fairly be specially looked for in those lesions developed during the active period of the disease, upon those organs peculiar to the sex, and specially implicated by the disease at that period; that is to say in the primary lesions situated on

the genital organs, and in those consecutive lesions in the same situation, to which women seem remarkably prone; rather than in the general manifestations of the fully-developed disease and its later effects, as occurring after ordinary contagion, in which it seems to be identical in the sexes.

The establishment of the Government Lock Hospitals for Women under the Contagious Diseases Acts, in operation for about eighteen years, but now recently abolished, furnished excellent opportunities for observing fully those manifestions of syphilis in women alluded to; not only because of the large number of cases, but also because the patients, being compulsorily detained in hospital as long as symptoms lasted, were continuously under observation throughout the course of the disease. Primary sores thus came under notice from the time of their commencement, or very soon after, and remained so till their disappearance. In a considerable number of instances the primary sores made their appearance whilst the patients were under detention in hospital for some other form of venereal disease; ran their whole course, and were followed by typical courses of general syphilis, thus affording thoroughly authenticated cases, complete throughout, in a manner possible only under the system of the Contagious Diseases Acts.

The cases upon which these clinical remarks are based were those admitted into the Royal Albert Hospital, Devonport, during a period of six and a half years, and were about 3300 in number. Every case was kept, and all patients thoroughly examined three times a week as a matter of routine.

Space will not allow dealing here with more than a very limited part of the subject of the primary lesion of syphilis in women, and it is intended to consider that character of primary syphilitic sores which has always been looked upon as of special diagnostic significance, viz. "Induration;" to inquire into the peculiarities of its characters in women, its development, the question of its

absence from true syphilitic primary sores, how far it is an essential character to such sores, and its value as a diagnostic sign.

In dealing with cases met with in the class of women whence these are derived there is difficulty in establishing the authenticity of cases, in being sure that the sore observed is really the primary sore, and the general symptoms the first outbreak of such, and not a recrudescence of symptoms in already syphilised patients; hence, out of the large number of admissions only a relatively small number of primary sores are forthcoming which may be relied on as well authenticated, many probably authentic but open to doubt being omitted. The authenticity of cases here given is based on, first, the known antecedents of the patients, a large proportion having come into hospital several times for gonorrhœa before contracting syphilis, a common event with young girls commencing a life of prostitution, and their total freedom from all signs of syphilis during those admissions. Next, the occurrence of a sore followed by development of typical general symptoms, the latter marking the true nature of the sore, the diagnosis not resting on the characters of the sore. Lastly, the subsequent course of the case being marked by repeated admissions into hospital for recurrences of syphilitic symptoms mainly of the nature of mucous patches and tubercles to which women, and especially this class of women, are so liable, contrasting thus with the known freedom of the patients from syphilis before the occurrence of the sore.

Seventy-one cases have been selected. Of these no less than twenty-six developed the primary lesion whilst under detention in hospital at periods varying from four to forty-one days after admission, affording further proof of their true nature by having passed through incubation periods, and all being followed by typical outbreaks of general symptoms. These seventy-one cases have been tabulated, the characters and courses of the sores being briefly given. The table is appended (Table A).

The characters of induration, as found in the primary sore in women, may now be considered as exemplified in the cases included in this table, and its peculiarities in the sex described. In the male sex induration is described as being always or nearly always present, and in a well-marked form, circumscribed and confined to the base of the sore, sharply defined in its boundary, frequently assuming a remarkably characteristic form and peculiar density and hardness, like a ring of cartilage set in the surrounding soft tissues.

From a consideration of the cases whence these notes are derived it is found that there are many interesting peculiarities of the induration of the primary sores in women which contrast with these points in men. First, as to the degree of the development of the induration or its quantity. It is stated as a general fact that in women it is less marked and less abundant than in men. These observations substantiate the truth of this in a large proportion of cases, but tend to show that it is not the minimum amount only that occurs in women, but that, whilst in a large proportion the degree is slight, in a good many cases a maximum amount of induration is reached; in fact, there is the greatest latitude in this respect, the induration varying from large tumour-like masses or wide-spread patches to cases where it is with the greatest difficulty tangible or visible. Between these extremes every degree is met with. As to the proportion of the cases in which induration is well- or ill-marked it will be found on reference to the table (A) that, roughly estimated, in one third of the cases it was much developed, in another one third it was moderately so, and in the remainder it was so slight as to be appreciable with difficulty or not at all.

As illustrations of the large development of the induration may be cited Case 111, where the sore formed a hemispherical swelling as large as half a Tangerine orange, set upon the outer surface of the labium; and Case 136, in which there were two primary sores, one of which

became raised into a tubercle-like prominent sore the size of a walnut shell, while in contrast may be noticed Case 75, where the sores were merely three slightly raised pale spots on the mucous membrane, and Case 80, where the mucous membrane was merely a little thickened and slightly altered in colour. Certainly to be noted as a peculiarity is the large proportion of cases (here about one third) where the degree of induration is so slight that it can hardly be appreciated; an important point connected with its diagnostic value.

Next as to position and the circumscription of the induration; whilst in men it is described as being distinctly circumscribed and confined to the base of the sore itself, and clearly marked off from the surrounding tissues, this is very far from being the case always in women. As to its diffusion the thickening is very commonly widely spread far beyond the base of the sore; thus about a dozen cases will be found in the table, where the induration spread widely through the tissues, and it is not at all uncommon, as in several of these, to see the sore confined to a portion, say one extremity, of a labium, whilst the whole labium is infiltrated, thickened, and resistant. It is remarkable in connection with this matter that in Case 143, where the primary sore consisted of a circular reddened moist surface on the mucous membrane, neither raised nor palpably thickened, the induration which developed in connection with it did not form in its base, which was never thickened, but spread away from it to the adjacent part of the labium (it being in the posterior commissure), which became thickened and infiltrated.

In extent, too, the induration not unfrequently spreads in a flattened form widely through the circumjacent skin, rendering it hard, thickened, and also discoloured, as in one case (109) where it extended up from the labium into the groin, where there were indurated glands, and in another (28), where a large patch of skin on the buttock became superficially thickened in immediate con-

nection with the sore. In depth and thickness the induration also varies greatly, in some cases forming a tumour-like mass, as in cases already cited, in others forming a thin superficial layer, which has been likened by writers to parchment or even paper, only to be recognised by pinching edgewise between the finger and thumb. Indeed, it may be said that induration shades off in some cases to the vanishing point.

If an attempt be made at comparison between the degree of development of the primary lesion in connection with the amount of its induration and size on the one hand, and the severity of the fully developed disease on the other, it is found that there is apparently no dependence of the latter upon the former; slightly marked sores and those of large size being followed respectively by both severe and mild cases of general syphilis. It is stated on the authority of Bassereau that when the primary lesion shows a tendency towards destructive changes the general disease will be severe. Another point refers to the particular form of the induration, as to whether the typical Hunterian chancre, characterised by its cartilage-like density and its clearly-defined and ring-like outline, is met with in women. It seems evident that in them it is at least very uncommon. Fournier mentions this fact, and it is well sustained by an examination of the cases in the table, and by the general impression which one gets from an acquaintance with the characters of their primary sores. As before stated, a very large proportion, perhaps half, have not the thickening strictly confined to the base of the sore.

The impression is also given that the induration in women is very seldom abruptly bounded off from the surrounding tissues, indeed it is often evidently a progressive infiltration; neither does the hardness ever exceed that given by a firm fibrous tumour in the most developed cases, firm, resistant, and resilient. Very far from anything like this, with their slight or moderately developed induration, are the majority of the sores; the

term "*thickening*," diminishing as before said in some cases to zero, conveys more correctly the true sensation conveyed by them to the touch. The thickening when well developed has a solid resistant feel not quite resembling other growths or swellings in the skin or mucous membrane, and seems to occupy a position between solid inflammatory infiltration and neoplastic tumour, which corresponds also with its pathological position.

The amount and degree of induration has been thought to be dependent in some way on its situation. These cases do not bear out any connection of this kind. Fournier states that induration is least marked in those sores which occur just external to the vaginal orifice, with which statement these observations agree thus far, that whilst sores as little indurated may occur anywhere else, those occupying this position are especially apt to be but slightly indurated. Beyond this there seems to be no connection between position and the degree of induration.

The mode and time of the development of induration may next be considered. For this purpose a group of twenty-seven cases, whose date of onset, having occurred whilst the patients were under observation in hospital, is accurately known within a day, may be referred to. They are appended in Table B.

These sores, arising under the circumstances they did, at once possessed additional interest and were carefully watched for the development of induration as a confirming sign of their true nature. Of twenty-three of these cases included in Table C, in only five was there any sign of anything approaching to thickening at the onset; and it will be noticed how slightly marked it was in these cases, by reference to Table B. Of the remainder, the time at which thickening became appreciable is given in Table C, in eighteen cases, in days intervening from the outbreak of the sore; it will be seen the time varied from seven days to thirty-four days. In one case (Case 6) no thickening appeared in the sore, but seven days after it had healed thickening occurred in its site.

In the other four cases no thickening at all occurred throughout. In four the sores were rather elevated than palpably thickened to the touch. Indeed, in eight cases out of the twenty-three it will be noticed how slightly, if at all, this character was present.

It is evident then that though thickening may be present in the sore from its commencement, yet judging from this small number of cases it is not so in the great majority (here about four to one). Next it appears evident that there is no fixed or even usual time in the course of the sore at which thickening may be expected to appear. Table C shows the very varying times in the age of the sore at which it occurred, up to forty days. If it is fair from so small a number of cases to take an average time of appearance, the nineteen cases would give fifteen days, or about the end of the second week, as an average time. Where the length of interval seems to be so inconstant, however, there is little use in taking the average except for the sake of comparison with the statements of other authors.

Taking the average duration of the primary sores included in the table, which is found to be forty-five days, the sores would then have run just one third of their course, on the average, before thickening appeared.

In fifty-seven other cases where the patients were admitted, bearing already the primary sores, twenty-five showed induration on admission. These cases would as a rule have existed less than a fortnight, that being the interval of the periodic examinations. In eleven of the remainder, the interval after admission before induration or thickening appeared is noted, and varies from three to thirty-two days, showing again how inconstant the time of its appearance is.

I am aware that the general belief is that induration occurs early in the course of the sore; and that it is thought to appear by the end of the first week or certainly before the end of the second. I have not, however, met with any details or statistics attempting to prove this,

except a table quoted from Sigmund as follows: 71 cases on ninth day; 84 cases on tenth day; 76 cases on fourteenth day; 15 cases on seventeenth day; 12 cases on nineteenth day; 3 cases on twenty-first day.

This gives the middle and end of the second week as the usual time for the appearance of induration. Considering, however, how little to be relied on are the statements of patients with regard to the time of appearance of the sore, too much weight must not be attached to it.

Some of the older writers, influenced no doubt by the theories they had conceived, were dogmatic in their teaching with regard to the time of the appearance of induration. Thus Ricord is stated to have proved induration in every sore by the fifth day; and elsewhere he is quoted by Bumpstead as stating that he had never seen a sore become indurated after the third week. With this, however, the latter says he cannot agree.

In considering the discrepancy between what is generally taught concerning the usual time of the occurrence of induration, and the large proportion of cases here given in which it was delayed till later in the course of the sore, it is to be remembered that these have to do with women only, in whom it has been shown that in a large proportion of cases induration is ill marked and slight throughout the duration of the sore; possibly, therefore, it is slower and weaker in its development. The cases on which opinion has generally been formed have probably been mostly or wholly in men, and dependent entirely on the statements of patients. The cases here given were not dependent on the statements of patients; and the dates of origin must be correct within a day.

The nature of the syphilitic cell-infiltration of the primary lesion, which is the basis of the clinical character of induration or thickening, is of a chronic or at most of a subacute variety; yet exceptions are occasionally met with in which the action is of a rapid, acute, and inflammatory kind, even when due to the uncomplicated effect of the syphilitic poison. This is well illustrated in Case

145, where the patient had been in hospital three weeks when the sore appeared, having recovered from the attack of gonorrhœa for which she was admitted. Here the sore was a slight and quiet one for seven days, when suddenly it assumed an angry surface, and infiltration with redness quickly spread throughout both labia. This gradually subsided into chronic and lasting thickening.

With such exceptions of sudden development it would appear that thickening is seldom if ever highly developed in the early course of the sore. We have seen, indeed, that it is in the large majority of cases quite absent then, and that when present it is only slight and soft. It may not develop beyond this grade, but continue in the same degree throughout. It is more common, however, for it to become more marked with the progress of the sore, and in a considerable number of cases to become much more developed in the latter half of the course of the sore and up to the time of the commencement of its healing or even at the time of its healing. It is during this period especially that the highest degree is reached and the sores most of all assume the characters of typical "induration."

This marked accession of induration will be found noted in a good many, seventeen at least, of the cases in table A (Nos. 7, 53, 137, 6, 45, 77, 20, 139, 144, 149, 61, 71, 28, 96, 54, 38, 21).

Induration usually retains or increases its degree, at least to the time of the commencement of healing; it may subside *pari passu* with the healing, but frequently when considerable its disappearance is slow and gradual, and may take many weeks to accomplish.

The clinical characters of the induration must depend upon, and be in accordance with, the pathological changes which are its cause. At first a slight infiltration of young cells would cause no hardness, which can only result from the sclerosing process, which is only gradually accomplished in an abundant mass of exudation cells. The pathological process being as a rule gradual and slowly developmental, the progress of the clinical characters

would also be so, increasing up to the point when involution commences, which usually corresponds with the commencement of healing.

It has been shown that induration is absent in the early period of the primary sore much more frequently than it is present; that its appearance may be delayed for very variable and considerable periods even till quite late in the course of the sore, and lastly, that in a considerable proportion of cases it is so slightly developed that it can be detected only with the greatest difficulty.

It remains to prove that the gradations of amount of induration or thickening which have been shown to exist in women, pass down to cases in which it is impossible to appreciate any thickening exceeding the resistance afforded by the surrounding normal tissues.

The question is so constantly being asked, "Can a non-indurated chancre be followed by syphilis," and answered in a general way on one side or the other, without an appeal to facts, or else left unanswered, that there is much indecision in most people's minds as to what is the truth. When the doctrine was formulated, and no doubt correctly in the main, that there were two distinct classes of sores, only one of which was followed by general syphilis, thickening and induration were held to be characteristic marks of that class, and were never met with in the other. The supporters of the doctrine taught that induration was never present in the latter class, and this has never been called in question. But they also taught that induration is *always* present in the former and is an unfailing sign. Such hard-and-fast rules are seldom so fully borne out by facts in pathological and clinical investigations as not to be liable to exceptions; further experience has more recently tended, whilst confirming the general truth of the rule, to allow more latitude for exceptions. It seems certain that in women, where induration is in the majority of cases a less developed sign than in men, many exceptions are to be met with.

Fournier, who has written especially with regard to

women, and is a firm supporter of the old doctrine, states in his 'Leçons cliniques sur la Syphilis' that induration is so constant in syphilitic infecting chancres that its absence is an actual curiosity; later on, however, he admits that even to an experienced observer its presence may sometimes be doubtful. He then adduces 100 cases of such sores, and admits that in thirteen he was unable to detect any induration; eight of these he explains away as being in such a position, viz. at the vaginal orifice, as not to be able to be taken between the finger and thumb; otherwise he assumes induration would have been detected. There still remain five cases in the hundred which even to his experience presented no induration. So large a proportion as 5 per cent. hardly justifies us in considering the absence of induration as a curiosity for its rareness; rather such an admission from one seeking to uphold the universality of induration is significant.

Although many observers have stated in a general way that they have seen cases where syphilis has followed non-indurated sores, such statements convey merely a vague impression, and it is only by carefully recorded facts that any fixed conclusion can be arrived at.

The writer of these notes commenced clinical observations on the subject with the belief that marked and characteristic induration was the special distinguishing mark of the infecting chancre; experience, however, soon proved that this was far from being the case.

It is to be kept in mind that the question here raised is not whether that class of sores known as the soft suppurating chancre or chancroid is ever capable of infecting the system, an important question requiring separate investigation; but whether syphilitic infecting chancres ever lack the special character, induration. In other words whether "induration" is an essential character of the infecting chancre.

Amongst these recorded cases there are nine which go to substantiate the fact that syphilitic infecting sores may be totally wanting in the character of induration or

tangible thickening, giving a greater sense of density and resistance above that of the neighbouring unaffected parts. Of the nine, five are chosen for being fully reported, as sufficiently accurate in detail and authenticity to bear investigation; and some remarks are appended to them.

The persistence of the characters of syphilitic chancres and their slowness to change will account for the few notes made as to their condition; though examined thrice weekly, notes would only be called for when any changes were observed in them.

In conclusion, I think we are justified, on the evidence I have submitted, in believing that induration, palpably evident, cannot be regarded as a character essential to the primary lesion of syphilis; the infective poison may exist in a sore without evidencing its presence by that character. Now that the morbid anatomy and pathology are better known, it can be understood how histological changes essential to the development of the disease may take effect at the seat of inoculation without necessarily producing such gross changes as to cause appreciable alteration in the form and substance of the tissues affected.

Further it is evident that induration, at any rate in the case of women, does not possess that paramount importance which has been attached to it by the school already alluded to, who sought to elevate it into a sign peculiar and essential to the very nature of the sore. It cannot now be regarded as a character of vital importance, but must be assigned its proper place as an important, indeed the most important, clinical sign for diagnostic purposes which the primary sore presents. Nor is it by any means the only character for diagnosis which the primary sore possesses. There are others, into which it is not the purpose of this paper to inquire. Nor is induration peculiar to the primary sore only, for abundant examples are forthcoming from the cases on which these notes are founded of consecutive or secondary sores in syphilised individuals, presenting induration exactly similar to that of primary

sores. Nor, again, is induration always or indeed often present throughout the whole course of a sore; sometimes it is wanting throughout. So ill-marked is it in many cases as to lose much of its value as a diagnostic sign. Even when present, it does not of itself absolutely distinguish the primary character of the sore; and absent, it does not conclusively prove the absence of the syphilitic poison.

The cases are here appended.

Table A.—*Table of* 71 *Primary Sores.*

75. Three spots on mucous membrane, slightly raised, looking merely a little paler than the rest of the mucous membrane.
80. Mucous membrane of prepuce and one labium minus thickened and slightly altered in colour.
24. A patch of the mucous membrane in posterior commissure looking reddened; a little raised and thickened.
50. A portion of the mucous membrane on inner surface of labium, thickened but even with general surface; it became hard a week later.
145. In posterior commissure a patch of mucous membrane, looking merely a little swollen, raised, and reddened, soft; followed by inflammatory infiltration in labia; sore took on a pultaceous surface and congested look.
97. A round patch of mucous membrane in posterior commissure, reddened, thickened, with smooth abraded surface.
7. On outside of labium a sore, with smooth, glazed-looking, scarcely secreting surface, a little thickened round its edge; becoming later very large on a wide and thick base.
53. Sore on labium, even with general surface, with almost dry, scarcely secreting surface on a widely thickened base; its surface became very large, and its base very thick.
66. Sore on labium, a superficial abrasion on a raised and thickened base.
72. Sore consisted of a superficial abrasion with smooth, red surface, on thickened prepuce; also a small, round abrasion on inside of labium, red, without any elevation or thickening.
108. Sore on labium minus, round, with nearly dry surface on a slightly raised, flattened base; whole labium swollen and infiltrated.
141. Sore on front of labium, circular, slightly raised, with smooth, pinkish, moist surface, partially covered with adherent scab; whole labium infiltrated.
48. Sore deeply seated in hollow between urinary papilla and labium

minus; mucous membrane there being thickened and nodulated, and presenting a fissured sore.

18. Sore on labium minus, surface slightly raised, covered with greyish pultaceous material, subsequently becoming dryer, and scarcely secreting. Was never indurated.

104. Sore within posterior commissure, small, with yellow surface; a little thickening about its margin.

109. Sore on outside of labium, large and raised, with pultaceous material on surface; soon changed into dry, bright red patch with silvery scales; infiltration spread very widely.

127. Sore just outside orifice of vagina, a patch of mucous membrane covered with pultaceous material, becoming slightly raised, but never thickened; also several roundish sores with slightly raised surfaces on labia.

105. On red and swollen prepuce an oval excoriation, with adherent pultaceous material and thickening; later it became dry, red, and raised a little.

137. On outside of labium a papular elevation, changing to an elevated and slightly thickened circular, smooth, flat surface, moist and glazed looking; became large on a wide and thickened base; whole labium became enlarged and of a congested hue.

138. Two little thickened infiltrations, surrounding hair-follicles; also a superficial excoriation on prepuce, which was swollen and thickened.

111. Sore on outer surface of labium; a large hemispherical tumour, size of half a Tangerine orange, projecting, firm, and elastic; its rounded surface smooth, of a yellowish-red colour, scarcely secreting; labium otherwise not swollen nor thickened.

136. On labium, perinæum, and near anus, three sores in form of raised tubercles, one becoming the size of a walnut shell, with smooth, moist, greyish-pink surfaces.

142. Sores on front of labium, six or seven in number, superficial circular abrasions, level with general surface; leaving reddish discoloured spots a little raised, but not thickened.

112. Within interlabial folds and posterior commissure, three or four sores, superficial, with yellow surfaces, edges looking a little swollen and raised, but never thickened.

90. On outside of labium a small circular spot with moist abraded surface, never becoming thickened.

146. Around vaginal entrance several superficial sores, with reddish or yellowish surfaces, not raised, nor thickened; becoming later a little raised, but continuing quite soft.

6. On front of labium three small, superficial, circular abrasions, not altering their characters throughout; but seven days after healing their sites became thickened.

45. On fold of nates two round, quite superficial sores, which spread

into one, presenting a slightly raised surface; when healing it became elevated and thickened.

77. On labia and fold of nates several small round excoriations, with dry, glazed-looking surfaces; late in their course they became elevated and thickened.

140. On front of labium a roundish, superficial abrasion; later it formed a raised, flattened, circular tubercle, with slightly moist surface.

143. In posterior commissure a round patch of mucous membrane, with red, rather moist surface, neither raised nor thickened; later infiltration in adjacent right labium; none in sore.

165. On labium minus a small, round, reddened spot, becoming slightly raised and thickened; infiltration spread through the labium minus.

20. About vulva several rounded sores, with yellow surfaces, adherent pultaceous material; surfaces became non-secreting, one became raised on a thickened base.

68. In extralabial furrow a large superficial excoriation, covered with greyish, pultaceous material; becoming a little raised, then again flatter and drier.

107. On labium minus two or three superficial excoriations, with adherent pultaceous material, spreading into one large, circular thickened sore; labium swollen and thickened.

22. Just external to orifice of vagina two sores, with adherent greyish pultaceous material, becoming later a little raised, with non-secreting surfaces; later still thickened.

126. On vaginal wall three superficial sores, with yellow surfaces spreading into one with dryish surface; it became raised and thickened on one side of its margin.

139. On urinary papilla a patch of the mucous membrane, neither raised nor thickened, but covered with grey pultaceous material; becoming afterwards raised and thickened, and later still a hard raised patch.

144. In posterior commissure a superficial sore, with greyish-yellow adherent pultaceous material; a little later thickened; left hard thickening on healing.

148. On outer surfaces of labia four circular sores, level with general surface; surfaces smooth, with a little pultaceous material; thickening soon occurred, and spread through labia.

149. On labium minus a small circular abrasion, level with general surface, with greyish surface; spread and eroded somewhat; later became thickened.

113. On cervix uteri two circular sores, with yellow surfaces; one formed a large raised tubercular elevation, leaving a thickened whitish patch on healing.

114. At entrance to vagina an irregular sore, with yellow surface, spreading and eroding irregularly; never raised nor thickened.

89. In posterior commissure a small excavated sore, which later assumed an even, granular surface, but was never raised nor thickened.

61. On labium minus three ulcerated sores; they soon became superficial, but spread; thickening appeared very gradually, becoming in time well marked.

70. On inside of labium two ragged, eroded sores, with yellowish-grey surfaces, no thickening; they filled up, and later on induration developed in them and throughout labium.

99. Just outside entrance to vagina several little irregular, deeply eroded, yellow-surfaced sores, which later spread into one, with red, granular surface, and margin slightly raised, perhaps slightly thickened.

100. At entrance to vagina a rather eroded sore, with yellow surface which gradually increased; later the whole adjacent labium was infiltrated; no separate induration in sore itself.

147. On inner surfaces of labia two rather deeply eroded sores, with yellow surfaces; changing after to slightly raised, red places, smooth, moist, and a little thickened.

28. On fold of nates four small sores, a little ulcerated, with a little red, thickened infiltration around, forming later large sores, level with general surface, with greyish pultaceous surfaces; induration later, with great thickening and redness around.

26. On inside of labium a large ulcerated sore, with unhealthy greyish surface; whole labium infiltrated and thickened throughout.

44. At orifice of duct of Bartolini a large sore, with grey, sloughy-looking surface; thickening in subjacent tissues.

85. At entrance to vagina a small sore, which spread by ulceration, a little thickening about it.

96. On outside of labium a round sore, superficially ulcerated; much inflammatory infiltration in adjacent part of labium; later it became rather prominent, with glazed surface; later still much larger, with much widespread thickening.

3. In extralabial furrow a small sore discharging pus, with redness around; it became larger, with almost dry surface, glazed looking; without any thickening throughout.

46. On inside of labium a superficial sore discharging pus, changing into a reddish patch, looking as if merely denuded of epithelium; it never became thickened.

41. On inside of labium, which was swollen and red, several superficial excoriations; later on some superficial induration in labium, which continued swollen.

58. About vulva several large superficial sores discharging pus; two of them soon became indurated, and left well-marked induration on healing.

65. On the labia, and opposed, are two sores with suppurating surfaces,

not thickened; later they became thickened and increased in size, their surfaces covered with sloughy pultaceous material.

103. On folds of nates three or four vesicles on reddened patches of skin, leaving little excoriations, which became papular and red; later, one formed a raised patch with shining red surface.

43. On folds of nates three vesicles, leaving little excoriations, which later became raised patches of skin with dry surfaces, forming tubercles, which subsided into thickened, brown, scaly marks.

95. In extralabial furrow a large sore, with granular surface, discharging pus, a good deal of thickening in its large base; later, congested, sloughy looking; later still irregularly nodulated.

54. Labium minus with sub-inflammatory infiltration, which, breaking down, formed a large ragged sore; later, extensive induration spread around it.

55. Swelling and redness of left labia; on the inner aspect a large irregular surface excoriated and discharging pus; much thickening and enlargement continued after the healing.

56. Redness and swelling of labia extending up to groins, with extensive excoriations on former, which slowly healed and left discoloured patches.

86. Labium red and swollen, with a large excoriation covered with grey pultaceous material, which left thickening when healed.

34. On folds of nates two sores looking like simple excoriations of the skin, so continuing; but later, on healing, leaving reddish slightly scaly patches; no thickening throughout.

49. On folds of nates two large superficial excoriations; later, forming large reddened patches of the skin, neither perceptibly raised nor thickened.

38. On perinæum a small and superficial sore, without any particular characters; increasing, its surface later became thickened and nodulated; later, raised into a tubercle.

115. On outside of labium a superficial excoriation, soon changing into a raised patch, with nearly dry surface, superficially thickened.

21. On folds of nates two simple-looking excoriations, which, later, became raised and tubercular in form.

TABLE B.—*Table of 27 Primary Sores, showing characters at earliest period.*

This table includes those tabulated in Table C. Those here marked with a * are not included in Table C.

Case.	No. of sores.	Position.	Characters.
24	1	Posterior commissure	A red patch on mucous membrane within commissure slightly raised and thickened.
41	Several	Labium	Several small, superficial excoriations on mucous membrane of labium, which was infiltrated throughout; infiltration sub-inflammatory in its nature.
75	3	Labium	Slightly raised spots on the mucous membrane, looking altered and whiter than the rest.
97	1	Posterior commissure	Circular patch of the mucous membrane raised and thickened, with smooth, red surface. (Patient not seen for past 7 days owing to menstruation.)
145	1	Posterior commissure	Patch of the mucous membrane looking swollen, red, and a little raised, soft, not thickened.
6	1	Labium	Small circular erosion of epithelium on mucous membrane, no thickening or elevation.
45	2	Buttock	Round superficial excoriations of the epidermis on fold of nates; no thickening or elevation.
77	Several	Mucous membrane about vulva and folds of nates	Small round excoriations of epithelium and epidermis, without thickening or elevation.
165	1	Labium minus	A little round spot on mucous membrane which merely looks a little redder than the rest.
142	Several	Labium	Circular superficial abrasions of epidermis on front of labium, neither thickened nor elevated.
*20	1	Cervix uteri	A greyish-surfaced patch, circular, on mucous membrane of cervix, even with surface.
90	1	Labium minus	Round patch of mucous membrane, with moist surface abraded of epithelium, not raised nor thickened.
143	1	Posterior commissure	Round patch of the mucous membrane, presenting smooth surface, red, scarcely moist, level with surface, not thickened nor elevated.

Case.	No. of sores.	Position.	Characters.
23	1	Labium	Superficially ulcerated, not thickened; like a simple chancre; on mucous membrane.
89	1	Posterior commissure	A small excavated sore, not thickened.
113	1	Cervix uteri	A small sore with yellow surface, round, with surrounding inflammation on mucous membrane of cervix.
21	4	Folds of nates, labium minus	Superficial excoriations on nates; no specific characters; considered accidental; superficial circular excoriations on labium minus a few days later, no thickening.
38	1	Perinæum	Small and superficial sore place, without any particular characters.
43	3	Folds of nates	Little excoriations of epidermis on folds of nates, no thickening.
49	2	Folds of nates	Large, widespread excoriations of the epidermis, quite superficial, not thickened, dryish.
140	1	Labium	A superficial abrasion of epidermis on front of labium, without elevation or thickening.
146	Several	Labium	Excoriated surfaces on mucous membrane around vaginal entrance, without any particular characters, no thickening.
*62	1	Perinæum	A sore on perinæum, entirely without specific appearance, not suppurating, nor thickened.
*59	2	Labium	Small superficial sores, without any thickening.
*144	1	Posterior commissure	A superficial sore, with greyish-yellow surface, not thickened.
28	2	Folds of nates	Small ulcerated sores, with dryish-looking surfaces, a little thickening and redness around.
103	3	Folds of nates	Three little vesicles on an inflamed patch of skin, leaving little erosions (turning later to papules).

TABLE C.—*Showing the age of 23 Primary Sores in days when thickening first became apparent.*

These cases are included in Table B.

Case.	No. of days.	
24 ...	From beginning.	
41 ...	From beginning.	
75 ...	From beginning.	... Elevated; scarcely thickened.
97 ...	From beginning.	
145 ...	From beginning.	... Raised and soft; not thickened.
167 ...	7	
146 ...	7	... Elevated only; never thickened.
113 ...	11	
143 ...	14	... Thickening in adjacent tissue; none in sore itself.
21 ...	16	
43 ...	17 (about)	
45 ...	18	
23 ...	19	
103 ...	19	
28 ...	22	
38 ...	26	
77 ...	28	
140 ...	34	... Elevated only; never thickened.
6 ...	40	... Sore had healed six days; thickening in site.
142 ...	None throughout.	
90 ...	None throughout.	
89 ...	None throughout.	
49 ...	None throughout.	

Five Cases of Infecting Primary Sores in which there was no Induration.

CASE 6.—E. Y—, æt. 19. Admittted for the first time.

April 9th, 1879.—Gonorrhœa only; no other sign of disease, local or general.

May 5th.—There is a small circular superficial abrasion on right labium minus.

8th.—There are two fresh sore places; all are circular superficial sores without any induration; no enlarged inguinal glands.

June 7th.—(There has been no note of any change in the sores.) Sores

have now healed over, no induration at all in them; no enlarged inguinal glands. During the past week there have been aching pains about hips.

14th.—The right labium minus, on which the sores were situated, has now become thickened and swollen.

17th.—There are enlarged and indurated glands in right groin. Labium minus still thickened. A syphilitic macular exanthem has appeared on limbs.

July 1st.—The thickening has disappeared from labium.

September 15th.—Left recovered.

Within the next three months fresh exanthem appeared, and mucous patches on the folds of buttocks. During the next four years she was admitted many times, always or nearly always with recurrence of syphilitic symptoms.

The primary sore appeared in this case after she had been in hospital and removed from source of infection twenty-six days, which time the incubation period must have exceeded. There was no induration in the sores, nor in the inguinal glands, until seven days after the former had healed over, when thickening appeared at their site, and three days later the glands were found affected, at which time also the early exanthem appeared. The case continued to show repeated outbreaks of syphilitic signs during a long subsequent period.

In this case the antecedents were not known. The incubation stage was typical. The appearances of the sores were characteristic with the exception of the absence of induration. The case is peculiar in the fact of induration appearing where the sores were situated seven days after they had healed. The inguinal glands, which maintained their freedom till induration had appeared, shortly after became typically affected. The development of the constitutional symptoms with pains in the limbs, and the appearance of a syphilitic macular exanthem, was typical. The further course of the disease observed for four years was also regular.

Case 49.—A. O—, æt. 19. Fourth admission. Under previous observation in hospital during the under-mentioned periods:—1st admission, December 24th, 1879, to January 8th, 1880; 2nd admission, March 10th, 1880, to March 18th, 1880; 3rd admission, April 21st, 1880, to April 27th. All these admissions were for slight gonorrhœa only; there were no other signs of disease, local or general.

June 14th, 1880 (fourth admission).—Gonorrhœal discharge with redness of internal parts. No other signs at all of disease except a distinct gland in each groin.

July 22nd.—There are two patches of the skin of the folds of nates, one on each side, looking as if superficially excoriated, of rather large size.

27th.—The patches, which were excoriated, have now dry surfaces, look red, but are neither raised nor thickened.

August 9th.—For some days past she has complained of scalp pains. She has now some brownish, slightly-raised spots of syphilitic eruption generally scattered about the body.

14th.—Excoriations have disappeared and also the eruption. She did not again come under observation.

In this case the patient was under observation several times in hospital during the seven months preceding the appearance of the sores, and was under periodical examination outside during that time. She did not exhibit any sign of syphilis. The incubation period was well marked, and must have exceeded thirty-eight days, as that was the period she remained in hospital before the sores developed. The characters of the sores were typical, with the exception of the absence of induration and thickening. There being two enlarged inguinal glands, probably from irritation of a non-specific kind on admission, preventing the time of their affection by the syphilitic poison being fixed. The outbreak of early symptoms was typical. The subsequent course of the disease did not come under observation.

Case 3.—A. T—, æt. 18. Fifth admission. She was under observation in hospital during the under-mentioned periods:—1st admission, January 6th to 16th, 1880, for gonorrhœa only; 2nd admission, February 23rd to March 16th, 1880, for gonorrhœa only; 3rd admission, April 14th to May 1st, 1880, for gonorrhœa and a pus-discharging sore; 4th admission, May 28th to June 8th, 1880, for gonorrhœa. During these admissions she presented no signs of syphilis.

June 18th, 1880 (fifth admission).—On account of menses not examined until 24th. There was then a small sore, with suppurating surface and redness around it, in the left labio-crural hollow.

July 1st.—The sore has spread and has now become larger, with a smooth, glazed-looking surface, scarcely secreting at all, now considered syphilitic. There is no induration about it, but there is a distinct gland in corresponding groin.

20th.—Sore has nearly healed over; gland has become large.

31st.—Sore is healed; there has been no thickening throughout.

August 20th.—The site of the sore has broken out afresh; the enlarged gland remains; a copious syphilitic exanthem has appeared over the back.

31st.—Mucous patches have appeared on tonsils.

September 11th.—Signs of disease have all subsided except that the maculæ have not quite disappeared.

Recurrence of macular eruption, patches on tonsils, and papular infiltration on labium followed two months after, and during the next two years there were repeated outbreaks of syphilitic symptoms.

In this case the antecedents had been observed for five months during which she was several times in hospital, free from all signs of syphilis, and under regular medical examination during the intervals. The sore at first presented signs not usually characteristic of syphilis, but later on assumed typical appearances, lacking only induration, which never occurred—the former a very frequent occurrence. There was typical enlargement and induration of an inguinal gland. The outbreak of constitutional symptoms was typical. The subsequent course of the disease was regular.

Case 142.—B. K—, æt. 18. Admitted for the first time.

Sept. 5th, 1884.—There is purulent discharge from the vagina and Bartolini's duct; no other signs of disease, local or general.

20th.—A very small roundish superficial excoriation on one labium without any thickening.

27th.—There are now six or seven little circular excoriations on the labia, chiefly on the skin surface, level with the general surface; not ulcerated; no thickening in them, no enlarged inguinal glands.

Oct. 2nd.—All the sores are larger; one has a centre where the epidermis is sound, surrounded by a zone of excoriation.

11th.—Sores have all healed over; the places remain a little reddened; they look slightly higher than the general surface, but do not feel thicker to the touch than neighbouring parts; no enlarged glands.

21st.—Copious syphilitic exanthem has appeared.

Nov. 21st.—All signs of syphilis have now subsided.

In this case the incubation period lasted more than fifteen days, the time she remained in hospital before the primary lesions appeared. The primary lesions presented typical characters with the exception of the absence of induration; they were situated on the front surfaces of the labia, where they presented every facility for examination; attention was fully aroused as to their true nature, and the sores were constantly examined with respect to the presence or absence of induration. There was an absence of induration of the inguinal glands, a point which will be referred to later on, and the occasional but apparently rare occurrence of which will be illustrated from these notes. The outbreak of general symptoms occurred in a typical manner.

Case 34.—M. H—, æt. 17. Admitted for the first time.

April 12th, 1880.—Gonorrhœal discharge. There are no enlarged inguinal glands nor signs of general syphilis; there are two patches of excoriation, one on either fold of the nates.

17th.—Sore places look to be drying up and healing.

22nd.—The places look nearly dry, yet have not quite healed over; they have scarcely any secretion; there is no thickening in them or about them.

May 6th.—The excoriations have now healed over; there is no induration about them, nor are there any indurated inguinal glands.

13th.—The excoriations have left discoloured reddened places on the skin, which is there a little scaly; there has been no induration throughout either on the places or in the inguinal glands.

She now left the hospital and continued under medical examination outside; she was re-admitted.

July 6th (second admission).—There were now enlarged and indurated inguinal glands, some macular syphilitic eruption on the back, and on the vulva some tubercular and papular infiltrations and dry, scaly infiltrated patches of the skin.

August 7th.—All signs have disappeared.

Recurrence of secondary syphilitic symptoms took place eight months later.

The primary sores presented typical characters, in their persistence without any marked change, in the nature of their surfaces, in their course, and in the condition of the skin they left after healing; they were favorably placed for examination. The glands again in this case presented no enlargement or induration until after the sores had healed; secondary symptoms followed in due course, being fully developed seven weeks after the healing of the primary sores.

(For report of the discussion on this paper, see 'Proceedings of the Royal Medical and Chirurgical Society,' New Series, vol. ii, p. 193.)

ON A CASE

IN WHICH THE

URINARY BLADDER WAS TWICE RUPTURED;

THE FIRST TIME BY VIOLENCE; THE SECOND TIME (SEVEN YEARS AFTER THE FIRST) BY THE GIVING WAY OF THE ADHESION WHEREBY THE RENT HAD BEEN CLOSED.

BY

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Received November 9th, 1886—Read February 22nd, 1887.

The following report will, I hope, be deemed worthy of the attention of the Fellows of the Royal Medical and Chirurgical Society for two reasons; first, because the complete history of the two accidents which befell this man's bladder is fully known, and recorded; and secondly, because it is, I believe, the first time that a description has been published of the post-mortem appearances of the bladder some years after recovery from an intraperitoneal rupture of its coats.

The view very generally expressed respecting rupture of the urinary bladder is an illustration of the not uncommon process of "begging the question." It is an argument in a vicious circle, and amounts pretty much to

this : " Intra-peritoneal rupture is necessarily fatal, because the escape of urine into the peritoneal cavity always excites fatal peritonitis ; if, therefore, a patient who has suffered an injury which is supposed to have caused an intra-peritoneal rupture of the bladder recovers, the diagnosis must have been erroneous."

This case will, I think, be a satisfactory refutation of such an argument, for we have evidence that the peritonitis which followed the original injury was recovered from ; and there can be no reasonable doubt entertained from the appearances after death, that an intraperitoneal rupture was caused by the injury.

Whether the peritonitis after the original injury was due to extravasation of urine, or to the violence of the injury, I will not here discuss. I will merely remark, in reference to this question, that there are good reasons for the opinion that healthy urine may occupy the peritoneal cavity without exciting peritonitis.

As the case presented itself on the first occasion it was published in the ' Medical Times and Gazette ' of 1879 (vol. ii, p. 603, *et seq.*) as one of ruptured bladder ; but in order to give here a concise and connected account of the whole case I will quote a portion of that report.

"On Friday, June 6th, 1879, William H—, æt. 39, a spare man of middle height, was admitted into Brodrip Ward of the Middlesex Hospital under my charge. He stated that he was an upholsterer, and that on Wednesday, June 4th, he was drinking in a public house, when some difference arose between him and another man, which they thought they ought to settle by wrestling. The patient was thrown with much force, and whilst lying with his back on the ground his opponent knelt with both knees upon the lower part of his abdomen. William H— seems to have lost consciousness for a little time, but on 'coming to' he with much difficulty walked home and went to bed, taking over an hour to do the distance, which is about one mile. He tried several times to pass water, and could not ; but within an hour or two after getting into bed he voided a small quantity of blood. He was visited by a doctor twice during the night and the next day, who ordered hot fomentations to the abdomen, and effervescing draughts to check sickness, and recommended his removal to the hospital. The patient stated that prior to the injury

he had not passed water for 'an hour or two,' and that he had been drinking up to the moment of wrestling. He also said that during the thirty-six hours between the accident and his admission into the hospital he had 'certainly not passed more than three quarters of a pint of blood and urine together;' that he had made several efforts before he voided anything at all; that which first passed being like pure blood, but that which was passed at subsequent attempts became less and less blood-charged, and more and more like natural urine. He began to suffer intense pain in the hypogastrium immediately after his belly had been kneaded, but as the hours wore on the pain, though it never left the lower part of the abdomen, 'got higher and higher up his stomach;' and he added that he had vomited frequently, bringing up a quantity of greenish fluid, and had also had a good deal of hiccup.

"*On admission* his face looked pale, sunken, and anxious; his skin was covered with cold perspiration; his abdomen was tympanitic, distended, and extremely tender, the slightest pressure causing him to wince and cry out; the most acute tenderness seemed to be at a small umbilical hernia and just over the symphysis pubis. Nothing abnormal could be felt *per anum*. He had micturated voluntarily, but with great pain and difficulty. A No. 7 silver catheter was introduced with ease into the bladder, and between three and four ounces of clear, normal-looking urine were withdrawn. After the urine ceased to flow a very slight pressure made with the finger-tips upon the hypogastric wall caused a spasm, whereby four or five small soft black bodies, the size of melon-seeds, were expelled through the catheter; these proved to be blood-clots. The point of the catheter was then moved over the inner surface of the contracted bladder in search of a rent in its walls, but no indication of such was detected. The patient was frequently vomiting a bright green, bilious fluid. Pulse 120, soft and compressible, and fairly full. Respirations shallow, but of normal frequency. Tongue moist. Ordered hot fomentations to the abdomen; half a grain of powdered opium in a pill every six hours if awake; to take no fluid and as little solid food as possible, and to quench his thirst by sucking a small quantity of ice. A gum-elastic catheter, with india-rubber tubing attached, was fixed in the bladder, so that the urine might flow off into a vessel beside the bed.

"June 6.—*Vespere:* Temperature 100°, pulse 108. Patient has ceased vomiting since 6 p.m. Has hiccup badly. Abdomen less tender. Has eaten some bread and butter, and not vomited after it. Took one slice at 3.30 p.m. and another at 11 p.m.

"7th.—Temperature 101·6°, pulse 120. Was sick once in the night, the vomit being bilious. Two pints and a half of clear yellow urine have flowed off through the catheter. Abdomen less tender, but pressure over hypogastric region produced vomiting. Had another pill at 3 a.m.

Vespere: Temperature 101·8°, pulse 116. Vomited after the bread and butter to-day.

"8th.—Temperature 99°, pulse 90. Tongue moist and clean. Abdomen distended and tympanitic, but less tender. No sickness to-day after food. Catheter still in; one pint and a half of urine has been passed. Ordered four ounces of brandy daily.

"9th.—Temperature 101°, pulse 98. Complains of a good deal of pain in hypogastrium. Catheter removed, washed, and re-introduced. The point was covered with what appeared to be pus. Bowels opened twice during the day; motions soft and clay-coloured. No more sickness. *Vespere:* Temperature 100·4°, pulse 100. No further change.

"10th.—Still much, sometimes very acute, pain in hypogastric region; to-day, moreover, there is considerable thickening and hardness above the symphysis pubis as high as half-way to the umbilicus, but extending farther to the right than to the left of the median line. The abdomen is somewhat less distended, and, except in the hypogastrium, less tender. Bowels open once this morning, the stools being less loose than yesterday. Catheter to be removed daily and washed out.

"11th.—Temperature 100°, pulse 104. Complains of griping pains in hypogastrium and in the course of the ascending and descending colon, which last a minute or two and then pass off. Abdomen more swollen and tense.

"13th.—Temperature 99·6°, pulse 72. Complains of more pain to-day in hypogastrium; the tenderness in this region is more considerable than it has been during the last few days, but the induration is somewhat less. There is a free thick white discharge by the side of the catheter, and no urine is flowing through the catheter. On removing the catheter it was found plugged with muco-pus. No. 10 catheter was introduced, and about three ounces of clear urine drawn off. Ordered to continue the pills and hot fomentations. The bladder is to be washed out with warm water containing a few drops of a mixture of eight grains of thymol in two ounces of glycerine.

"14th.—The patient complained of so much increase of pain in the hypogastrium after the injection that it is not to be repeated; it was only used once. He passed this morning a stool consisting only of opaque, gelatinous matter, like the white of egg, which, on boiling with liquor potassæ, became quite clear. Urine contains pus; specific gravity 1015.

"16th.—Bowels open slightly this morning, the action being attended with much pain. Examined *per rectum:* no induration or thickening about the bowel, which is full of soft, yellow, solid fæces. Up to yesterday the urine had been pretty clear, but on re-introducing the catheter this morning a quantity of thick, dirty-looking pus first passed, and then the urine flowed pretty clear. On removing the catheter the end which

had been retained in the bladder was seen to be darkly discoloured. The tenderness and induration in the hypogastrium are less pronounced. Feels very low and hungry. Ordered a simple enema, and a mixture containing chloric ether, spirits of ammonia, and tincture of calumba; still continuing the pills and the hot fomentations. Is to have chicken for dinner to-morrow. The catheter is no longer to be fixed in the bladder, but to be introduced two or three times daily if patient cannot micturate without it. *Vespere:* Temperature 101·6°, pulse 96. Bowels acted freely after the enema. Stools normal. Passed urine voluntarily after a little straining. The urine was thick and opaque, being largely charged with pus, and became very ammoniacal on standing.

"From this time the notes show an almost uninterrupted progress towards recovery. The catheter was not again required. The amount of pus in the urine, which for several days was large, gradually diminished. The induration behind the hypogastric wall gradually cleared away, and the pain, distension, and tenderness of the abdomen ceased.

"On June 26th the urine was acid, and contained neither blood, pus, nor albumen, though there was a slight deposit of mucus. On June 30th he was allowed to get up, and the next day to go into the hospital garden. On July 2nd there was a slight reappearance of pus, but by July 9th the urine was acid, sp. gr. 1015, free of pus and albumen, but contained a slight excess of phosphates.

"Quinine mixture was substituted for the alkaline and hyoscyamus mixture. On July 10th the patient left the hospital quite well, and was not again heard of until his return on August 4th, 1886."

The conclusions then arrived at were that there had been a wound high up in the urinary bladder so that the organ had preserved some of its power of retaining and expelling urine, and that the wound had been closed by inflammatory adhesion.

When readmitted on August 4th, 1886, the man made the remark that he was suffering from "the same trouble as that for which he was treated by Mr. Morris seven years before," and when seen in the out-patient department his account of his present illness, and his symptoms, excited the suspicion of ruptured bladder. The following report was made by Mr. Nash, one of the physicians' resident assistants.

Present attack.—On Monday, August 2nd, patient was at a "beanfeast;" but states that he only drank three or

four glasses of wine and three or four of beer during the day. He passed water three or four times, and returned home quite sober.

3rd.—He got up as usual, feeling quite well. His bowels were open, and he passed urine, the latter act being accompanied by slight pain. He next got pain in his abdomen during the morning, which increased as the day advanced. In the evening he was unable to pass water, and was suffering great pain. His wife applied hot fomentations to his abdomen to relieve pain, but without effect.

4th.—On Wednesday afternoon he came to the hospital complaining that he had passed neither urine, fæces, nor flatus, since the previous morning.

The house surgeon introduced a No. 3 catheter (there was no stricture of the urethra) and withdrew about three ounces of turbid urine, which was highly albuminous, and contained a trace of blood.

He has vomited several times since yesterday (Tuesday) morning and suffered a good deal from hiccup. The vomit in the surgery consisted of the contents of the stomach. He was admitted into a medical ward as a case of suppression of urine.

State on admission.—A spare man, with anxious drawn face, complaining of intense pain in the abdomen, and inability to pass urine. Temp. 99°, pulse 124, small, fairly regular, somewhat wiry in character. Tongue coated with yellowish-white, moist fur.

Abdomen : Walls very tense. Extreme tenderness all over the front, flanks, and back. Seat of greatest pain just above symphysis, of greatest tenderness at umbilicus. At the latter spot there is a small hernia, the opening of which just admitted the tip of the index finger.

4th.—Resonant on light percussion all over.

Thorax : Movements slight, percussion and breath sounds normal.

Heart : Apex beat not localised, sounds normal.

Treatment.—Four dry cups applied to loins followed by hot fomentations to loins, and over the front of the

abdomen. All pain and tenderness in the back then disappeared.

10 p.m.—Dr. Pringle and Mr. Sutton, being in hospital, saw the case and recommended opium gr. j every four hours, and the abdomen to be painted with glycerine and belladonna, and covered with hot fomentations. During the evening patient vomited a large quantity of dark green bilious matter.

5th.—This morning he is much better, but has passed neither urine, fæces, nor flatus since admission.

November 3rd, 5th and 7th.—Catheter passed, and one pint of thick, turbid urine drawn off.

Urine: Acid, 1024, contained a large quantity of pus, mucus, albumen, and a small quantity of blood, and indican in excess. Under the microscope no casts were seen, only pus and a few blood-corpuscles.

12 noon.—Dr. Fowler having seen the patient, ordered the treatment to be continued, and an enema, Ol. ricini, to be given at once.

6 p.m.—Half of the enema was returned, and with it a quantity of hard, scybalous masses came away. Patient now seems very comfortable. Has very little pain in abdomen, which is still tender. Has not vomited all day. Bladder not distended.

6th.—Slept well during the early part of the night. About 7 a.m. passed a little flatus and a large quantity of urine, and suddenly became very sick. At 8 a.m. suddenly became collapsed, surface of body covered with cold sweat, pulse not felt at wrist. Quick, calm, and rational; says he does not feel much pain, but feels very weak and exhausted. Abdomen very distended, tympanitic, liver dulness obliterated.

He never rallied, and died at 11.5 a.m.

A *post-mortem* examination was made by Dr. Fowler on August 6th. His report states: The abdomen was distended and tympanitic, and on opening the peritoneal cavity there was evidence of recent acute peritonitis. The contiguous coils of intestine were united by recently

effused lymph, the peritoneum was everywhere injected, and in the pelvis, where the inflammatory changes were most advanced, there were fifteen ounces of purulent fluid, and a quantity of lymph. The stomach and small intestines were irregularly distended. The rectum and sigmoid flexure contained a quantity of semi-solid fæcal matter.

A band one inch long united the fundus of the bladder to the anterior wall of the rectum. The vesical end of the band was hollowed out, and through a rupture in its walls, close to the bladder, a string of mucus was projecting. Slight pressure caused puriform-looking urine to ooze from the bladder, showing that there was at the rupture a direct communication between the bladder and the peritoneal cavity. The kidneys were congested, and the pelvis of each contained some purulent urine, and its lining membrane was inflamed.

The other organs were normal.

The rectum, bladder and penis, were removed *en masse*, and submitted to the examination by a Committee, which met at the Royal College of Surgeons, consisting of Mr. Savory, Mr. Bryant, Dr. Goodhart, and Dr. Fowler.

The following is the report of the Committee :

Report on the case of Rupture of the Bladder.

August 13th, 1886.

The undersigned met to-day at the Royal College of Surgeons, at the request of Mr. Henry Morris, to examine a specimen removed from a man who was under the care of Mr. Morris in June 1879, with what was then diagnosed as a ruptured bladder, and who left the hospital recovered.

The patient was readmitted into the Middlesex Hospital, under the care of Dr. J. K. Fowler, on August 4th, 1886, suffering from acute peritonitis, caused, as was eventually shown, by the giving way of the bladder at the site of the old lesion, and he died on August 6th.

The specimen consisted of the penis and bladder laid

open along the anterior aspect, and the rectum with the surrounding structures. The urethra and prostate were of normal size, and free from disease; the bladder also was of normal size, and with the exception of the condition herein described, quite healthy.

The peritoneum covering the back and fundus of the bladder, and that lining the pelvis, was considerably thickened, apparently the result of old inflammation. The bladder wall, for a wide area around the seat of injury, was also thickened from a similar cause. The peritoneum was coated with a layer of quite recently formed lymph, and an opening into the bladder was visible at the base of a band of adhesion between the bladder and rectum.

About an inch below the fundus of the bladder, and on its posterior aspect, slightly to the right of the mesial line, there was, as seen from within the viscus, a rounded opening of a quarter of an inch diameter. This opening led to a funnel-shaped recess, which was bounded at its wider or vesical end by the thickened and plicated vesical walls, and tapered off within a hollow cord-like adhesion into a mere blind sinus just large enough to admit a probe, which could be passed to within one eighth of an inch of its posterior extremity. This tubular band of adhesion was one and a quarter inches in length, and extended between the bladder and the front wall of the rectum. It was in a part of its length divided into two unequal strands, which were united at their extremities, like some of the musculi pectinati of the ventricles of the heart.

The wall of this funnel-shaped recess, beyond the level of the outer surface of the bladder, was membranous, not being thicker than a sheet of stout note paper.

This adhesion had been partially torn through at its vesical attachments during life, thus permitting a free escape of urine into the peritoneal cavity.

The lining of this recess was smooth, and in direct continuity with the mucous coat of the bladder, and was marked by finely injected parallel vessels extending its whole length.

The opening into it from the bladder was finely plicated and had somewhat the appearance of cicatricial tissue, but no microscopical examination was made to determine whether it was cicatricial tissue or attenuated mucous membrane, as to have done so would have injured the specimen.

There were no peritoneal adhesions in the pelvis, other than the one above described, but some old pigmented patches were noticed in the deepest part of the recto-vesical pouch.

The explanation of the foregoing changes, taken with the clinical history of the case, seems to us as follows :

1. This pouch marks the site of an injury to the bladder and surrounding structures received by the patient in June, 1879.

2. That injury was in all probability a rupture of the bladder attended with extravasation of urine into the peritoneal cavity, and into the cellular tissue and structures of the bladder wall.

3. At the site of the injury, the bladder and rectum became united by inflammatory lymph.

4. The contractions of the bladder and bowel, hollowed out this adhesion by their traction upon it; thus leading to the formation of the infundibuliform recess above described, and thereby predisposing to the fatal rupture.

5. The perfectly normal appearance of the rest of the bladder, and the characters of the recess itself, negative the supposition that this was an ordinary congenital diverticulum or acquired sacculus.

6. The specimen, in our opinion, exhibits all the features which might be expected at a remote period after a rupture of the bladder.

(Signed) THOMAS BRYANT.
W. S. SAVORY.
J. KINGSTON FOWLER, M.D.
JAMES F. GOODHART, M.D.

Note.—The Chairman of the evening, Dr. John W. Ogle, having written to ask me the question, I desire to state that there seem to me to be two possible causes of the giving way of the funnel-like cicatrix: one is that, owing to its extreme thinness, it yielded under distension of the bladder; the other, that softening and ulceration occurred from the retention and decomposition of urine in the funnel-like recess, just as a sacculus occasionally yields from the same cause. The account of the fatal illness points to the latter cause as the more probable.

(For report of the discussion on this paper, see 'Proceedings of the Royal Medical and Chirurgical Society,' New Series, vol. ii, p. 203.)

ON SOME

DANGERS CONNECTED WITH THE USE OF THE ASPIRATOR

AS A MEANS OF

RELIEVING THE DISTENDED URINARY BLADDER,

WITH SUGGESTIONS FOR THE TREATMENT OF CERTAIN CASES OF RETENTION OF URINE.

BY

WILLIAM H. BENNETT, F.R.C.S.,

ASSISTANT SURGEON TO ST. GEORGE'S HOSPITAL, AND LECTURER ON ANATOMY IN THE MEDICAL SCHOOL.

Received December 14th, 1886—Read February 22nd, 1887.

The operation of aspirating the bladder, by means of a fine needle introduced above the pubes, for the immediate temporary relief of retention of urine, has become a common proceeding on account of its simplicity and supposed harmlessness.

It is recommended by the majority of the accredited authorities in the text-books of the day, and is variously spoken of as being "safe," "perfectly safe," "entailing no danger," "simple and admirable," &c. In one authority only can I find any caution as to possible danger from urinary extravasation, and that applies to the

proceeding when repeated, being associated with a reference to the fatal case recorded by Dr. Mackie Williams in the 'British Medical Journal' for February 21st, 1880.

Personally I have long thought, and indeed have been in the habit of teaching, that the operation, trivial as it may appear in itself to be, is open to serious objections, and may be connected with grave risk, for it has always seemed to me that the production of a distinct breach of surface in the walls of a distended bladder, by the introduction of a needle, however fine, the organ being one of great elasticity and muscular power, would directly tend to a giving way of the coats at the point of puncture, *unless an immediate and decided relief of tension could be effected by a free and rapid flow of urine from the needle*, and that even if no actual rupture were to occur there may be a liability under similar circumstances to an oozing of urine through the wound by the side of the instrument.

This liability would be increased by the spasmodic contraction which the act of puncture would probably excite in the bladder walls.

In aspiration by means of a fine needle, this immediate relief of tension does not, in my opinion, appear to be sufficiently decided to obviate the risks alluded to, since the flow of urine is of necessity slight, seeing that the canal of the needle is of so small a size.

Herein there lies, I believe, a strong objection to the operation in many cases.

With reference to the question of vesical tension, I may perhaps say that I have been for some time watching with considerable care the immediate effect upon the contraction of the bladder coats, produced by the introduction of instruments *per urethram* as well as through the walls of the viscus, and, so far as I am able to judge, the following conclusion, based partly on the sensations of intelligent patients, and partly upon careful observation of the force of the expelled stream of urine, may be taken as fairly accurate.

The introduction of a catheter or canula into a bladder

distended to any extent short of that which would cause a temporary paralysis is followed immediately by a jet of urine, through the instrument, the force of which is determined by the degree of elastic tension in the organ. After an interval, momentary in itself, the stream becomes much stronger from violent spasmodic contraction of the muscular elements of the vesical walls, aided by the action of the abdominal muscles, which, under these circumstances, are, as a rule, beyond the control of the patient.

It follows, therefore, I assume, that the period of greatest tension is not at the moment of the entry of the instrument, but shortly afterwards; *unless the bladder is enabled to empty itself very rapidly*. It is at this moment that the liability to rupture or oozing is the greatest, and then must be the time at which a free flow is most called for, such a flow as would be provided by the use of the ordinary trocar and canula usually hitherto employed for tapping the bladder through the rectum or above the pubes as the case may be.

I may at once state that I believe the aspirator may be used without risk of rupture or exudation for cases in which the bladder coats are *healthy*, as they may of course be in a case of recent acute retention, but in cases of old-standing obstruction, whether from stricture or prostatic enlargement, where the bladder walls are hypertrophied, varying in thickness, and often rotten or easily lacerable, the proceeding may be productive of serious harm, and is altogether inferior to the older method of tapping with the large trocar and canula.

An objection of a minor kind to the use of the aspirator seems to me to present itself in those cases in which the bladder contains putrid urine, for it must surely be a proceeding of doubtful advisability to withdraw, through the perivesical cellular tissue, a needle which has been directly before steeped in a decomposing and virulent fluid.

Whatever value may be attached to the foregoing remarks, the following case will sufficiently demonstrate the serious complications which may follow the simple

proceeding of aspiration of the bladder under certain circumstances.

J. P—, æt. 38, a shopkeeper, a steady and intelligent man, was admitted into St. George's Hospital under my care on September 19th, 1886, with the following history.

Sixteen years previously, whilst he was the subject of a urethral discharge, he had been thrown violently across a horse's withers whilst riding. Immediately afterwards some blood came from the urethra, and the stream of urine commenced to diminish gradually in size. In three months he could urinate only with great difficulty, and therefore went to St. Bartholomew's Hospital as an in-patient, where a catheter was passed and tied in; ultimately he was made an out-patient, after having been instructed to pass a catheter for himself.

In a few weeks he neglected to use the instrument, and again the stream became very small, and for years before his present illness he had been unable to micturate without much straining and frequent pain.

Three days before he came under my treatment complete retention supervened. Twelve hours later the urine began to dribble away, and had continued to do so up to the time of his admission into the hospital.

He had applied at another hospital on the previous day, where catheterisation had been attempted without success, and had been followed by copious bleeding.

On admission.—The patient was in great pain and distress. Urine was dribbling from the urethra, the bladder was much distended.

The house surgeon, having failed to pass a catheter, introduced an aspirator needle into the bladder above the pubes, but drew off only a small quantity of foul urine which contained no blood.

Immediately after the introduction of the needle the patient was seized with acute pain, and commenced to vomit. I was accordingly sent for at once, and on my arrival found the man vomiting and rather collapsed; there was dulness above the pubes, some rigidity about

the abdominal parietes in the hypogastric region, and tenderness on slight pressure.

Without very much difficulty I passed a No. 4 catheter (silver) through a hard stricture into the bladder, and drew off a small quantity of urine, which contained a considerable amount of blood. The dulness and rigidity above the pubes were very little, if at all altered, by my proceeding, but the patient was so well satisfied with the fact that a catheter was in the bladder that he declined to have anything further done. The instrument was therefore loosely tied in.

On the following morning, September 20th, he thought himself rather better; the pulse was very quick, the tongue rather dry, the temperature normal, there was occasional vomiting. Only a little bloody urine had passed through the catheter.

The dulness above the pubes was rather more extensive, but the rigidity seemed slightly less marked.

The catheter was withdrawn, and a No. 6 passed without difficulty, but the patient begged that it might not be tied in, as he ascribed the vomiting to the presence of the instrument in the bladder. It was therefore ordered to be passed at frequent intervals.

By the 21st only a few ounces of urine had been drawn off by the catheter; the patient was worse in all respects. The dulness had much increased, there was great tenderness over the hypogastrium, but he still declined further interference. On the 22nd he was obviously sinking, and I at length obtained permission to deal with the case as I thought fit. I therefore cut down in the middle line immediately above the pubes, sixty-four hours after my first seeing the patient. Upon getting through the parietes a large quantity of dark-coloured, intensely ammoniacal urine rushed out through the incision. On introducing the finger, a large cavity could be felt with a softish mass at the bottom, which was presumed to be the bladder; the walls of the space appeared to consist of a quantity of sloughing cellular tissue. The cavity was

thoroughly washed out, and a large drainage tube inserted.

So much relief followed the operation that the patient immediately rallied, and by the 25th was in a fairly satisfactory condition.

The vomiting ceased, the rigidity about the abdomen gradually subsided, the pulse improved, and nourishment was taken well, but in spite of the persistent use of the catheter, all the urine passed through the abdominal wound, which was full of sloughing tissue.

On September 30th, therefore, I performed a median cystotomy, introduced a large tube through the wound, and tied it in.

By October 4th great improvement had taken place, very little urine was coming from the hypogastric wound, at the bottom of which there still remained a large flabby slough.

On October 8th this slough came away, and was followed by a considerable flow of urine. This fact, combined with the appearance of the slough, seemed to indicate pretty certainly that a portion of the bladder itself had become gangrenous. After this date, although there was a continuous full flow of water from the peritoneal tube, a considerable stream constantly came from the hypogastric wound. That which came through the perinæum was clear, contained a little blood at times only, and showed but a small quantity of albumen.

Upon the evening of October 13th some swelling of the right leg was noticed, and upon examination the iliofemoral vein was found to be blocked. There was very little pain, but the pulse was quick, and the temperature, which had up to this date varied from 98° to 100°, now rose to 102°.

The thrombus soon commenced to clear up, and the swelling of the limb gradually subsided. The general condition of the patient suffered seriously from this complication, and great weakness followed, which was much increased by the extreme disinclination of the patient to take food of any kind.

By October 20th all the slough seemed to have come away, the urine was still passing freely by both wounds, and the strength was being regained as more nourishment was taken.

No great change occurred, although there was some improvement in the general health and a steady decrease in the quantity of urine which came from the supra-pubic wound, until November 4th, when a fluctuating swelling was observed in the left groin. On the 6th there was a distinct change for the worse, the abdominal wound was very foul, and a considerable quantity of fæcal matter was seen to come from it. From this date the case became hopeless, the fæcal discharge increased in quantity, and all nourishment was refused. He gradually sank and died on the 9th.

Post-mortem examination.—The bladder, contracted to the size of a small hen's egg, lay, denuded of peritoneum, in a cavity below and behind the pubes into which the hypogastric wound directly led. On the anterior aspect of the viscus was a circular orifice rather larger than a shilling, the result of the rupture and sloughing referred to in the notes of the case. The opening from the neck of the bladder into the urethra was patulous and gaping from having lodged the perinæal tube for so long a time.

The cavity in which the bladder lay was bounded above and behind by coils of large and small intestine matted together. In a knuckle of the latter, which projected somewhat into the space, were two openings, with sloughy margins, from which fæcal matter freely oozed.

There was no evidence of peritonitis, old or recent, excepting in the immediate neighbourhood of the walls of the cavity, nor was there any obvious obstruction caused by the matting together of the intestinal coils.

On dividing the ureters a little pus exuded from that on the left side. The kidneys were in a condition typical of chronic suppuration in an early stage.

The occurrence of this case recalled to my mind one of a less serious kind, having some bearing on the question

of the use of the aspirator, which came under my notice in 1875, when I was house surgeon to St. George's Hospital, and of which I have some brief notes.

A man, æt. 65, applied for treatment on account of retention of urine due to enlarged prostate. Having failed to introduce a catheter, and as he declined to become an in-patient, I aspirated the bladder above the pubes and drew off a large quantity of foul urine.

The patient complained of a good deal of pain at the time of the withdrawal of the fluid, and it struck me that there was rigidity about the seat of the puncture directly afterwards.

He came again on the following day, having succeeded in the meantime in introducing his catheter into the bladder, as he had been for some time accustomed to do. There was then distinct tenderness and rigidity above the pubes, but not much pain. Three days later he again presented himself, having suffered a good deal of pain since his last visit. He had also noticed on the previous day some pain and tenderness about the back of his perinæum, where I found on one side, rather in front of the ischial tuberosity, a boggy, ill-defined, fluctuating swelling. Upon incising this, some very foul, dark, and thin discharge came away ; at the same time I withdrew from the cavity some long string-like sloughs.

The parts above the pubes were still tender and hard, and pressure over that situation caused some discharge from the perinæum. He again refused to remain in the hospital, went away, and I did not see him again.

In two other cases which have happened under my observation aspiration of the bladder has been followed by pain, supra-pubic rigidity, and nausea, in one case lasting five hours, in the other nine, after which the symptoms subsided without further complications. In both instances the urine evacuated was very foul.

The first of these cases presents an undoubted instance of the extra-peritoneal rupture of a diseased bladder, the direct result of its puncture by an aspirator needle.

So far as its causation is concerned, I believe it is the first case of the kind recorded. It is interesting as showing the remarkable relief afforded by clearing out the urine which has escaped from the bladder into the pelvic cellular tissue, even in the case of a patient who was apparently moribund.

Considering the great tenacity for life exhibited by this man, I have very little doubt that, could I have operated a few hours after the rupture occurred, the case would have probably terminated in recovery.

Case 2, I believe, was an instance of the escape of a few drops of decomposing urine into the cellular tissue around the bladder wound, resulting in inflammation and sloughing, fortunately somewhat limited in extent.

In the other cases mentioned, it is not unreasonable, I think, to suppose that the temporary pain and rigidity may have been accounted for by the passage of the needle over the cellular tissue about the puncture, immediately after having been steeped in foul urine.

A careful consideration of the cases described in this paper, and the importance, in my opinion, of the points mentioned in connection with the objections to producing a wound in the diseased bladder walls, without effecting an immediate and decided relief of tension, as well as the obvious risk which may occur in the withdrawal of a foul needle through healthy perivesical cellular tissue, lead me to submit the following conclusions:

1. Aspiration of the bladder for the relief of retention of urine is a more serious proceeding than is usually supposed, and can only be resorted to with safety in recent and acute cases in which the bladder walls are presumably healthy.

2. The operation is absolutely inadmissible in cases of long-standing stricture with chronic difficulty in micturition ending in complete retention, the bladder walls being in such cases hypertrophied, rotten, or otherwise diseased, the appropriate treatment for these cases, if the stricture be impassable, being drainage of the bladder through a

perinæal incision, the stricture being divided or not at the time of operation as required by the conditions of the case or in accordance with the custom of the operator.

3. The aspirator can be used only with some risk in cases of retention where there is reason to believe that the urine is foul, unless the means are at hand by which an antiseptic solution may be introduced into the bladder before the withdrawal of the needle. Even under these circumstances it is only applicable to cases in which the retention is due to prostatic enlargement where there has been no long-continued previous suffering from cystitis, irritable bladder, or vesical atony; such cases being more appropriate for treatment by prostatotomy and perinæal drainage.

4. In cases of retention, from any cause whatever, where it is probable that the bladder walls are diseased, or the urine foul, if the surgeon decide to tap the bladder instead of performing the major operation, the paracentesis should be done with a *large trocar and canula*, and not with a fine aspirator needle, for reasons already stated.

5. In the event of an attempt at aspiration being followed by pain and rigidity, with or without vomiting or nausea, lasting for twelve or fifteen hours, the good of the patient would be best consulted by free incision above the pubes, and drainage of the bladder through the perinæum at the same time, if the urethral or prostatic obstruction be impassable by catheter.

(For report of the discussion on this paper, see 'Proceedings of the Royal Medical and Chirurgical Society,' New Series, vol. ii, p. 203.)

SOME NEW FACTS

CONCERNING

GOUTY PAROTITIS AND GOUTY ORCHITIS.

BY

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OF CONTREXÉVILLE (VOSGES).

(Communicated by Sir ALFRED GARROD, M.D., F.R.S.)

Received July 12th, 1886—Read March 8th, 1887.

On May 12th, 1885, I read a paper before the Academy of Medicine of Paris on gouty affections of the glandular system, bearing more particularly on gout in the parotid gland and in the testicle. The paper was intended to fill a lapsus existing even in the most recent treatises on gout, in which scarcely any mention is to be found of gout as affecting glands, especially the parotid gland. I am now anxious to bring this subject before the notice of the Royal Medical and Chirurgical Society, with certain additional facts which have come to my knowledge, in the hope that the great experience of English medical men in this country, in the matter of gout, may throw a new light on the subject.

In the first place, *unmistakable attacks of gout are met*

with, having their seat in the glands and alternating with articular manifestations. In May, 1881, I had for the first time an opportunity of observing an attack of gout localised in the parotid gland. I had searched in vain in the various works on the subject for some allusion to this peculiar form, when in August, 1882, Dr. Garrod informed me that he too had had a similar case under his care. In the following year Dr. Rotureau, of Paris, noted another case of gouty parotitis. Lastly, in July, 1885, I myself had a fourth case of this kind under my care at Contrexéville.

The first case which came under my observation was that of M. D—, æt. 68, a patient of Professor Teissier, of Lyons, and a decidedly gouty subject, who had presented, in a more or less marked degree, almost all the symptoms of the uric acid diathesis; attacks of unmistakable articular gout, nephritic colic, eczema, dyspepsia, and even some amount of glycosuria. Notwithstanding his disinclination to take care of himself, the nephritic colics had not recurred since 1869, and the attacks of gout had become much less frequent, when in May, 1881, I was called upon to visit M. D—, who at that time resided in Paris. My patient, who had suffered severely from the financial crisis of January, 1881, told me that, having called on his dentist the day before for the purpose of an operation on one of his teeth, he awoke in the night with a very painful swelling at the angle of the jaw on the left side, on a level with the parotid region. I thought that the dental operation had given rise to an abscess, and had recourse to the usual remedies; but at the end of forty-eight hours there was not the slightest alteration in the appearance of the tumour, and I began to think I was mistaken in my diagnosis. I requested Dr. Bucquoy, of the Hôtel Dieu, to give us the benefit of his opinion, and this gentleman thought that the swelling could only be attributed to the dental traumatism, and that surgical interference would probably soon prove his opinion to be correct. On returning the next day, however, we found

that the swelling had almost completely disappeared, with relief of the local pain, its disappearance coinciding with an attack of gout in the right knee. I did not see this patient again until the 3rd of July following, when he came to Contrexéville. He told me that the other parotid gland and the left knee had been successively invaded by the gout. At the time of his arrival, except a slight induration of both parotids, no trace remained of his attack beyond a saline taste in the mouth at the beginning of each meal. After a careful examination of the glands I convinced myself that this saline taste was due to the presence of urates in the saliva secreted by the parotids, this after all being one of the epiphenomena of the gouty crisis. Dr. Garrod's case was very similar, and I hope to see it published in detail.

The case observed in Paris by Dr. Rotureau is still more instructive, because it shows the attacks of gout following the same course five times in succession in the same patient at intervals of from one to two years. In this patient the attacks invariably began in one of the parotids, rapidly invading the knee on the opposite side, and then successively the other parotid and the other knee.

Judging from these three examples one might be inclined to suppose that the glandular and articular manifestations followed a certain regularly alternating course; but that this is not always so, the following case will show.

Mr. L. E—, æt. 54, of robust constitution and sanguine temperament, had suffered from gout and gravel for upwards of twenty years. He has had nephritic colic and attacks of gout, which until 1885 had been clearly articular. He visited Contrexéville in 1885, when he was attended by Dr. Boichox. Two days after his arrival he suffered from an attack of gout in the right wrist followed by a very painful swelling of the parotid region *on the same side*. After applying the usual external remedies without benefit, Dr. Boichox requested me to see his

patient. Under the impression that I had to deal with a new case of parotid gout I did not hesitate, in order to relieve the excruciating pain, to prescribe fifty drops thrice daily of a tincture of the fresh flowers of colchicum together with sulphate of quinine. The patient was promptly relieved by this treatment, and was enabled to return to Paris, and to business forty eight hours afterwards. In accordance with the axiom *naturam morborum curationes ostendunt,* we must admit that this was a genuine case of parotid gout.

I will deal more briefly with the subject of gouty orchitis, the existence of which has often been pointed out by English authorities, by Hunter and Sir James Paget among others. In January, 1885, several cases were described by Dr. Guyot and Dr. Millard at a meeting of the Medical Society of the Hospitals of Paris, and gave rise to a discussion. Lastly, in February, 1886, a thesis on the manifestations of gout in the genital organs was read by Dr. Legalchez before the Faculty of Paris. For my own part I have only met with two cases of gout in the testicles.

The first case, that of a man æt. 62, has already been reported on to the Academy of Medicine of Paris in May last; the second, under my own care in March, 1885, has not yet been published. Both are cases of orchitis not accompanied by any urethral discharge, occurring in confirmed gouty patients, and followed by well-marked attacks of articular gout. Although the absence of any urethral discharge was carefully ascertained, it has excited doubt in the minds of certain medical men who have not had to do with cases of genuine gouty orchitis. I can only refer them to the observations published by Dr. Latil of Aix[1]. The patient suffered from orchitis ending in an attack of gout in the big toe, twenty-six days after a fracture which obliged him to keep his bed with a surgical apparatus on, thus excluding any suspicion of urethral contagion.

My own patient was a man æt. 35, whose father and

[1] 'Bull. et Méms. de la Soc. Méd. des Hôp. des Paris,' 1885, p. 36.

mother both suffered from gout. When twenty-eight years of age he underwent an operation for incurved toe-nail, and the toe was rendered insensible to pain by the application of the ether spray. Shortly afterwards his first attack of gout declared itself in this toe, recurring two years later and subsequently affecting both feet and both elbows. In March, 1885, the patient suddenly became aware of a swelling of the left testicle accompanied by acute pain, without any history of a traumatic or urethral origin. This swelling involved the testicle as well as the epididymis. The testicle was three times its natural size and the swelling was general; there was no effusion into the tunica vaginalis ; it was moderately tender on pressure, but was the seat of intense shooting pain. Poultices, belladonna fomentations and position, failed to effect any improvement in the patient's condition until the swelling suddenly disappeared on the supervention of an attack of gout in the big toe. I am sorry now that I did not at once have recourse to colchicum as I did in the case of my patients suffering from gouty parotitis.

In the various observations published by medical authors we remark in most cases the alternations which I have already pointed out with reference to parotid gout—the left testicle and the right foot—for in all the cases hitherto published the left testicle has been the sufferer. This rule, however, is not absolute since in the case of my other patient, the right testicle and the right foot were affected. I may add that so far I have never met with gouty manifestations in the testicle and in the parotid in the same patient.

(For report of the discussion on this paper, see 'Proceedings of the Royal Medical and Chirurgical Society,' New Series, vol. ii, p. 218.)

A CASE OF EMPYEMA,

WITH

PULMONARY GANGRENE FOLLOWING ENTERIC FEVER, TREATED BY PERFLATION.

BY

WILLIAM EWART, M.D.Cantab., F.R.C.P.,
AND
R. FITZROY BENHAM, M.R.C.S.

WITH REMARKS ON THE METHOD OF PERFLATION, AND ITS VALUE IN THE TREATMENT OF EMPYEMA.

By WILLIAM EWART, M.D.

Received August 23rd, 1886—Read March 8th, 1887.

[The early notes of the case are by Mr. Benham, who attended the patient throughout his illness.]

"On April 24th, 1886, I first saw Charles C—, æt. $10\frac{3}{4}$, who was suffering with rigors which were followed by fever; and I was informed that prior to my visit he had been feeling unwell for about two or three weeks. The family history was healthy, and there had been no previous illness except scarlet fever in early childhood and chorea minor a few months prior to the present illness, for which I attended the patient. I soon discovered, by the special features of the temperature chart, of the evacuations, and of the cutaneous spots, that

this was a case of enteric fever. Astringents were prescribed to check the diarrhœa, and the pyrexia was partly controlled by antipyrin, quinine and acid having been tried in vain. The violent delirium, which lasted continuously for sixteen days, also abated soon after antipyrin was administered. Even during the delirium localised tenderness could be detected over the right iliac fossa. By the end of the fourth week the patient had so far improved as to be able to play with his toys. A few days later, however, an attack of general peritonitis set in which was marked by the usual symptoms. The urine deposited lithates, but was otherwise normal. The diet at that time was still restricted to fluids.

"Five days after the onset of the peritonitis, which had meanwhile greatly diminished in severity, pain was felt on deep breath, and on cough at the upper part of the left parasternal region. The pain gradually increased, and on the third day some dulness on percussion at the left base was recognised, together with exaggerated breathing at the seat of pain. The patient rapidly lost ground, and after about eight days from the onset of the chest-trouble he was suffering from severe exhaustion and dyspnœa on the slightest attempt at movement. He lay on his left side breathing rapidly (at about 40 per minute), emaciated, suffused, with parched red tongue, and mouth extensively studded with aphthæ. The cough was feeble and hacking, and he had a hectic temperature. The left chest, which I found the previous day to measure one inch more than the right, was absolutely dull from the second rib downwards, the dulness extending to the right for one and a half inches beyond the manubrium sterni. No heart-beat could be detected on the left side, but the cardiac impulse was plainly visible in the right chest, and was felt and heard with greatest intensity at a spot one and a quarter inches to the right of the right nipple in the fifth intercostal space. The left chest was motionless and bulged in its upper part. Loud blowing breathing and coarse râles were audible above the left

second rib, and less abundantly over the upper part of the right lung anteriorly. Loud blowing breathing was also heard to the right of the manubrium. The voice-sounds were not anywhere tubular in character. Feeling that paracentesis should be performed without delay I consulted Dr. Ewart on June 11th."

The physical signs described in Mr. Benham's report having been confirmed, and the patient's pulse being of fair strength, we determined to aspirate the empyema immediately. General anæsthesia was induced and the needle was inserted into the fifth intercostal space, close to the anterior axillary line. After escape of three quarters of a pint only of a shreddy pus, not fœtid, but possessing a tenacious, stale "animal" odour, the flow ceased, and could not be re-established, although the cannula was carefully probed and found patent. The heart receded only a little way towards the left, but the dyspnœa was considerably lessened. We determined to open the chest next day, and on June 12th two free openings were made (under the carbolized spray from a Benham spray-producer), one at the site of the previous aspiration, the other dorsally in the ninth interspace, in vertical line with the angle of the left scapula. The pus resembled closely that obtained by aspiration; its amount was estimated at about three pints. A probe introduced at the anterior orifice could be moved freely to a depth of four or five inches, and could be pushed upwards and inwards as far as a spot corresponding to the level of the second right costo-sternal junction. Avoidance of all fluid injections having been agreed upon as a principle of treatment, a continuous drainage-tube was introduced and the wounds were dressed with a thin bag of carbolized gauze loosely filled with wood-wool impregnated with corrosive sublimate, and covered with the usual mackintosh. This formed not only a most efficient absorbent pad but also completely sealed the wounds. Meanwhile the patient was much relieved. His breathing, which had been very rapid, became quiet and he was able to lie on his right side. Owing to the free

oozing of discharge it was necessary to renew the dressings the same evening.

On the 13th breathing was audible over the upper part of the lung, especially at the back; the discharge was moderate in amount. On the 14th the discharge was rather foul but not abundant; two short tubes with flanges were substituted for the single long tube. On the 15th (being the fourth day from the date of the operation), perflation of the chest with carbolized air was performed with the help of a Richardson's bellows, and of a wash-bottle containing 10 per cent. solution of carbolic acid in water. Care was taken that the air, delivered into the chest through the *anterior* wound by an india-rubber tube, should not escape *anteriorly*, but issue through the *posterior* orifice.

A few seconds after the beginning of perflation the posterior opening, which in the recumbent posture of the patient was the most dependent part of the chest, became blocked, and its lips bulged outwards, causing slight pain. In another moment, under the influence of rising pressure, a solid substance was forced through the aperture; this was followed by a large mass of shreddy false membrane and by upwards of one ounce of fœtid pus. The pain immediately subsided, as well as the slight cough which had been set up at first.[1] Ventilation was kept up for fifteen minutes, but nothing further was discharged. The carbolized spray had played on the wounds throughout and the same kind of dressing was reapplied. (A few days later, finding that the skin had become irritable and scattered with red spots, Mr. Benham dusted the surfaces with a powder consisting of oxide of zinc and boracic acid.) The temperature, which for the last three days had returned to normal degrees, did not rise, and the patient spent a comfortable night. The solid lump was found on inspection to be a wedge of whitish-grey, solidified and necrosed pulmonary substance, which measured 2 centimetres by 1·4 and by 0·9, in its longest diameters. On June 16th was

[1] Cough was not observed at subsequent dressings in this case.

painlessly expelled, by the same method, a fresh supply of false membrane, with a little pus ; and again on the 17th a small shreddy mass, weighing about four drachms, together with two fluid drachms of pus. An examination of the chest, made on the 15th, had revealed the presence of loud bronchial breathing opposite the left first and second cartilages ; this was replaced, on the 17th, by vesicular breathing. The vesicular murmur was audible, before the dressings were detached from the wounds, as low as the fourth rib anteriorly, and as the seventh rib behind. After their removal the breathing became less vesicular and mixed with amphoric notes. At the base, front and back, the breathing was purely amphoric. The heart had not returned to its normal apex-site, although it closely approached that spot. A probe inserted anteriorly still reached with facility the region covered by the second left costo-sternal junction, and could be sent far and free towards the posterior opening ; but in the axillary direction the mobile surface of the lung could be felt at the level of the third rib. Owing to the narrowness and obliquity of the intercostal spaces the posterior part of the pleura could not be explored satisfactorily.

On the 18th, 19th, and 20th the dressings showed only stains of pus ; and hardly any discharge was expelled by perflation. On the 22nd (being the tenth day from the date of operation), about one ounce of slightly turbid, blood-stained serum escaped ; and about six drachms of the same fluid, but of clearer quality, were discharged on the 24th. On this date auscultation yielded, before any air was admitted, vesicular sounds posteriorly as far as the base of the lung and anteriorly close up to the wound, but not in the interval between the two wounds. Air having been let in, the vesicular breath-sound was transformed into a harsh blowing, which in the scapular region was accompanied with a faint amphoric sound ; whilst true amphoric breathing extended over an area one and a half inches in diameter between the wounds. No abnormality of the voice-sounds was found except a little way above the

posterior wound, where they possessed a bleating character. When the probe was introduced it came into contact with the lung within a distance of three inches in the upward direction.

It was inferred from the results of this day's examination that the lung was almost fully expanded during the periods of air-tight closure between successive dressings, leaving only a small cavity at the base of the pleura, and that it was partly adherent to the chest wall in the posterior mid-axillary region.

On the 28th the fluid exudation was about the same in amount; the probe could still be sent into a large free space. On July 4th, being anxious to close the anterior wound, and finding that the posterior opening was becoming uncomfortably small, we agreed that the latter should be enlarged, and this was done under ether. When the finger was introduced through the enlarged wound it impinged upon the tough surface of the lung, and below this it was possible to pass a probe in the direction of the anterior opening. On this occasion, in order to avoid the escape into the chest of any of the blood from the incision (which bled rather freely) the wound was kept exposed for a considerable time, and for upwards of three quarters of an hour, from first to last, perflation was applied under spray. On July 5th not more than one drachm of serum was secreted; on the 6th, 7th and 9th, only a few drops. The anterior tube was removed on the 10th, and a smaller tube was inserted behind. On the 12th, exactly one month from the date of thoracentesis, the posterior tube was found lying out of the wound; the next day only a pin-hole opening remained, and on July 15th the wound was healed. Breathing was now everywhere audible, and was of good quality except at the axillary base. The left shoulder was drooped from nervousness, but there was hardly any permanent deformity. The right chest measured $12\frac{5}{8}$ inches at the level of the nipple; the left $12\frac{3}{8}$ inches.

Remarks on the case.—Without being pathologically remarkable, the case is of some interest, inasmuch as it

presents an unbroken chain of pathological events. That the bowel was extensively ulcerated may be safely assumed from the severity of the pyrexia, and of the typhoid symptoms. The enteric lesions were probably responsible directly for the peritonitis, and indirectly, by way of embolism, for the pulmonary gangrene; and the latter was followed by suppuration within the pleura. In favour of the view that the gangrene was the outcome of thrombosis or embolism strong support is derived from the appearances of the specimen expelled from the pleura. Its pulmonary nature is readily identified by the smooth glistening surface covering two of its sides and by the faint pigmentary tracings of the lobules, whilst its shape shows it to belong to the pulmonary fringe. The line of separation is indicated on one of the smooth surfaces by a rim of yellow inspissated pus closely parallel to the ulcerated edge. On one of the two surfaces roughened by ulceration is seen a small cylindrical pit resembling the bed of an absent vessel. This, it is assumed, was the site of the original pulmonary lesion. (Attention may be drawn incidentally to the fact that the specimen was evacuated through an external opening of apparently smaller diameter than would allow of its easy passage.) The shreddy false membranes were yellowish-grey, and consisted of fibrin mixed with pus; only one of the smaller pieces possessed a red tinge such as would identify it with the blood-clot. The presence within the pleura of material such as we have described would in itself lend a feature of gravity to an empyema irrespective of any constitutional exhaustion preceding, as in this case, the chest affection. That in a case thus complicated suppuration should have ceased as early as the tenth day, and made place for a serous flow, is a result perhaps not exceptional, but worthy of record.

This result is, in our opinion, to be ascribed to the successful evacuation of the cavity within the first week; and the early expansion of the compressed lung was in great measure due to the care bestowed on the dressings with a

view to air-tight closure of the wounded side. It must be confessed that the final operation for enlarging the posterior opening was undertaken unnecessarily under the impression that the expansion of the lung and the obliteration of the cavity were not keeping pace with the healing of the external wounds. The auscultatory evidence of the extensive re-establishment of breathing during the intervals of closure of the wounds should have been more implicitly trusted. Had this evidence been acted upon the empyema might perhaps have been permanently closed within fourteen days after the paracentesis. With an actual duration of four weeks the case may nevertheless be looked upon as successful in point of time.

Remarks on the Method of Perflation and on its value in the Treatment of Empyema.

(Before proceeding with these remarks it is my pleasant duty to express my indebtedness to Mr. Benham, not only for having agreed to the application of a novel method, but for his valuable assistance in the endeavour to render it a success, and for practical suggestions in the manipulation of the apparatus. To his able surgical co-operation is due in a large measure the successful result obtained in the case.)

Hippocrates, although free from the indiscriminating dread of air which arose after his time (his only fear was lest the lung should become parched, a danger which he sought to obviate by injecting oil and wine), does not appear to have utilized air surgically in the treatment of empyema. Modern English works contain no mention of the injection of air as a means of treatment in this affection. But in a pamphlet by Audouard ('De l'Empyème,' Paris, 1828), the following suggestive remark appears as a mere parenthesis: "Et pourquoi ne se servirait on pas, de l'air comme moyen médicamenteux?"

This suggestion remained unnoticed, and the prevailing prejudice against air held continued sway. In 1841, in an inaugeral thesis on empyema, which was supposed to reflect recent knowledge and practice, Sédillot laid down the following proposition (p. 173) : "Une indication complexe, très importante pour la cure, est celle de laisser toujours dans la poitrine assez de pus pour éviter l'introduction de l'air et trop peu pour écarter les parois thoraciques." The deep-rooted objection to the escape of air into the chest had its origin in the observation that pus in contact with air underwent putrefaction. Cases, however, were not wanting in which putrescence occurred even after its exclusion, and Sédillot was driven to use the lame argument that air did harm by its bulk, mechanically preventing the expansion of the lung.[1]

Within recent times the lingering distrust of air suggested to Bowditch the aspiration method, a valuable addition to surgical resources ; and the success of this method may have served for a time to emphasize an erroneous belief. But since the beginning of the Listerian era we have learnt to regard air with less diffidence, and the treatment of empyema may now be expressed in a formula of surprising simplicity : "a timely and sufficient incision to be kept aseptic and open." In this form it was applied by Dr. Markham Skerritt, in 1876,[2] without the complication of any fluid injection.

Air was capable, however, of something better than harmless neutrality. It was pressed into the service by Roser and subsequently by Quincke. To Roser[3] I must resign the claim of priority for a method which, up to three days before writing these lines I had held to be not only original but novel. Roser's method differs from my own less in its principle than in some of its details. The air injected is

[1] The discussion on thoracentesis before the Académie de Médecine in 1872 is of historical interest in connection with the alleged dangers from the admission of air into the pleura.

[2] 'Brit. Med. Journal,' July 22nd, 1876.

[3] 'Archiv der Heilkunde,' v, p. 84, 1864.

unwashed, an ordinary air-syringe is used, and a second chest-tube is provided for the outflow. Roser's views and his practice entirely agree with my own with regard to the mechanical uses of the injected air, but from his short description it does not appear to what extent he employed perflation systematically as a healing agent. Roser's results, as far as reported, were highly satisfactory, even in the presence of such complications as pulmonary or bronchial fistula. It is surprising that in spite of this success the method should have failed to become generally known, or fairly tested.

H. Quincke[1] suggested as an improvement an ingenious apparatus, whereby air and fluid were alternately injected. The leading pretext for this innovation was the alleged danger arising from shifting the patient's position during the first days after incision. Quincke's method, which can be applied strictly *in situ*, is excellent theoretically, but its results do not sufficiently commend it.

Perflation, as I have practised it, consists in systematic "through-ventilation" of the suppurating cavity. I have selected the term perflation as the shortest and most explicit word conveying that the chest is not inflated, nor merely injected with air, but "blown through." The method has been described in the 'Lancet' (July 31st, 1886, p. 226), and need only be briefly sketched here. The following apparatus is necessary: (1) a Richardson's bellows; (2) india-rubber tubing to conduct the air from the bellows into the bottle and from the bottle into the chest; lastly, (3) a tall wash-bottle containing a short column of strongly carbolised water ($\frac{1}{10}$), to act as a purifier and as a valve for the air to be forced through it. This bottle, in winter, should stand in warm water.

In cases where a single opening has been made into the chest, if the tube be introduced to a sufficient depth, air conveyed by the tube from the bellows will pervade the whole space before it escapes at the orifice by the side of the tube; and if any obstruction should exist near the

[1] 'Berliner klin. Wochenschrift,' 1872, p. 65.

orifice the air would be delivered into the cavity beyond. Where two openings have been made, one of them will admit the tube and will require to be closed air-tightly during perflation, the other remaining patent. In either case obstruction may arise from some internal plug as soon as the patient has been so placed that the wound of exit is absolutely dependent. If air be now injected, its tendency will be to drive out the plug.

The following mechanical principles are at the root of the method : (*a*) air diffuses into every recess of a given space, whereas fluids gravitate ; (*b*) a current of air penetrating with a certain force into an air-containing cavity will distribute some of this force in every direction ; (*c*) owing to the specific lightness of air, fluids or solids will not be suspended by it, but will inevitably fall in it to the most dependent spot.

It is freely admitted that fluid injections can also be made to enter into universal contact with a cavity, and that they also are capable of transmitting even pressures. With these objects in view, various authorities[1] have utilized the principle of the syphon. But with all fluid injections it remains a matter of chance whether the solid to be expelled will gravitate through the fluid medium to the exact spot desired. Moreover, several objections must be raised against the method of filling, emptying, and refilling the chest by means of a syphon. Two, three, or more pints of fluid make up a weight which can hardly be tolerated by the pleura with *comfort* nor, if hydraulic pressure be superadded, with *safety*. The lung is thereby submitted to sustained compression of a very inelastic kind ; and further, in virtue of its great buoyancy it is not unlikely to be floated up, out of position, thus straining its vessels.

But the chief practical defect common to the syphon method, and to all methods involving fluid injections, is the difficulty of absolutely emptying the pleura by their

[1] F. Cresswell Hewett, 'Brit. Med. Journal,' March 11, 1876; Kashimura of Tokio, 'Berliner klinische Wochenschrift,' No. 3, 1880.

means, and the uncertainty in which the operator is left on this score. Solid material may remain behind as well as fluid. To speak of fluids only, injections of any kind, if allowed to stagnate in the pleura, cannot fail to set up or perpetuate irritation.

From the patient's point of view, perflation is mechanically preferable to fluid injections. It is simple, painless, free from shudders and drenchings, and performed with a minimum of disturbance, and, as far as my experience goes, with practical freedom from danger.

Judged by its results in the case narrated above, perflation seems to possess the following advantages as a method of treatment for empyema :

(1) It facilitates the early removal from the chest of putrid residues or of the recent products of inflammation.

(2) In the same measure it tends to shorten the period of suppuration ; and

(3) To lessen the danger of permanent shrinking of the lung ; and, therefore, also

(4) To lessen the necessity for resection of ribs.

(5) It leaves the chest relatively dry, and practically free from any foreign material, since air is rapidly absorbed by the pleura.

This paper would not be complete without some allusion to the question of the surgical value of local ventilation. Some authorities appear to have seen good results from this form of treatment. Bouisson[1] submitted superficial wounds to the direct draught of a bellows for frequent periods of fifteen minutes. A scab usually formed, under which reparative processes advanced more rapidly than under moist dressings or ointments. The same method appears to have been successfully tried by Sir James Simpson. Dr. Ogilvie Wills[2] saw wounds treated in this manner unite without one drop of pus. Dr. Krönlein[3] devotes an important work to the study of the treatment

[1] 'Brit. and Foreign Medico-Chirurg. Review,' i, 1859, p. 546.

[2] 'Lancet,' i, 1873, p. 907.

[3] 'Die offne Wundbehandlung,' Zürich, 1872.

of wounds without any applications impeding local ventilation, and appends to his book a useful bibliography.

Roser (loc. cit.) observed in cases treated by his method a very favorable influence on the abscess walls. In his own words, "their shrinking proceeded so rapidly that one might assume that the stimulus of air had a special action in that direction." My own case gives countenance to the same assumption.

It may be questioned whether in single empyemas, or in the healing stage of empyemas originally complicated with gangrene or false membrane, the pleural surface presents any true granulations. More probably granulations are to be found only in cases of inveterate suppurations of the pleura with fistulous opening. If the injection of simple or of medicated air should prove to possess a tendency to change the latter condition into the former, to convert a pus-secreting into a serum-secreting surface, or if in the absence of any inherent virtue attaching to air the general conditions of the treatment could be shown to favour such a result, then the therapeutical value of the method which I advocate would be established.

Perflation, in this communication, has been considered exclusively in connection with empyema. Its applicability to other pulmonary conditions and to other regions of the body will suggest itself without need of any remarks from me. The mode and duration of its application might be varied to any extent, as well as the antiseptics used in its performance. These are matters for experimental surgery. For my part, even should the usefulness of perflation fall short of larger expectations, I shall feel content if in many cases of empyema it may, in the future, be found as beneficial as in the cases we have related.

Dr. Delépine's Report on the small mass removed from the pleural cavity: The microscopical examination shows that the specimen is a small mass of lung tissue. The pleura and subjacent alveoli, with some carbon pigmentation, can be easily recognised, and the alveoli are distended

with granular débris, probably the remains (decolourised) of some extravasated blood.

(For report of the discussion on this paper, see 'Proceedings of the Royal Medical and Chirurgical Society,' New Series, vol. ii, p. 221.)

ON A CASE

OF

OBSTRUCTION OF ONE URETER BY A CALCULUS,

ACCOMPANIED BY COMPLETE SUPPRESSION OF URINE.

BY

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It is hoped that this isolated case will be deemed worthy of consideration by the Fellows of the Society on account of the occurrence of certain unusual symptoms which have an important bearing on the diagnosis of this often obscure disease; but, if this excuse were insufficient, it might be added that the patient was an indefatigable member of our own profession and most anxious that his own sufferings should, if possible, be utilized for the general good.

The only serious illness from which he had suffered occurred during his student days in 1872, when he was about nineteen years of age. He had then a poisoned wound which resulted in symptoms of general septicæmia and was accompanied by left-sided pleurisy and no doubt pericarditis. He was much reduced by this but gradually recov-

ered, and from that time until June, 1884, he enjoyed very good health interrupted only by occasional attacks of colic, thought by him to be intestinal, and passed over as matters of small consequence. The first of these occurred about 1881; it only lasted for two hours and was relieved by the administration of chloroform. A second, a year later, lasted four hours and yielded to the hypodermic injection of morphia. They both consisted of severe abdominal pain, not localized, and accompanied by much sweating, and on the second occasion by vomiting, but there was nothing in either to suggest the ureter as the seat of the disorder, nor in the appearance of the urine to prompt the patient to examine it. At times, after this second attack, he experienced sharp shooting pains in the right testicle, which he thoughtlessly attributed to a large left-sided varicocele from which he suffered, but otherwise he considered himself quite well.

The illness for which he asked my advice began therefore without any other warning recognised as such on June 4th, 1884, with increased frequency in micturition and a pain in the region of the bladder if he attempted to retain his water. Before this time, and indeed afterwards, he was in the habit of emptying his bladder at rather remarkably long intervals. The trouble in passing water lasted for a week, when on June 11th there was a good deal of blood in the urine, and under the microscope oxalate of lime and perhaps some uric acid crystals were discovered ; but next day the urine was normal in the morning though blood again appeared in the evening. On the following day, June 13th, abdominal pain like that from which he had suffered in the two previously described attacks, came on and was accompanied by sickness ; it was so severe that he kept his bed on this and the two following days, but on the morning of the 16th it had disappeared and he was getting about again. In the evening of this day, however, it returned with great severity, "doubling him up," and becoming localized in the region of the right kidney. His temperature was now 102° F.

I saw him on the evening of the 17th. He was then in great pain of a spasmodic character. There was considerable tenderness over the right kidney, but no tumour was discovered on careful palpation. There were anorexia and nausea, and a temperature of 102°. The urine was clear, but contained a few pus-cells and rather more than a trace of albumen ; no casts were seen.

After a few days the pain, which had been somewhat relieved by hot fomentations and morphia, disappeared, and at the same time the temperature, which had remained high, fell to normal and there was a considerable increase of pus in the urine.

As it was assumed that some obstruction to the ureter had been overcome he was allowed to get up and to go out, and it was intended that he should go into the country in order to recruit, but after he had been walking rather more than usual and, it should be added, after the bladder had been sounded without the discovery of a calculus, the pain returned again and the temperature assumed a hectic character, varying from 99° in the morning to 102° at night, whilst the urine became less turbid. The pain increased very much and the tenderness reappeared, the former shooting down into the groin, the latter being most marked in the back between the ribs and the right iliac crest. The urine was now noticed to contain flocculent masses, which under the microscope were seen to consist chiefly of long felted chains of short bacteria. It was acid and free from putrefactive smell. It is most probable that it also contained granular and hyaline casts, as they were present in large quantities a few days later, but they were not carefully looked for at this time. The most peculiar feature about the urine—and it is one to which I would direct special attention—was the enormous increase in the amount of albumen without any increase in the quantity of pus, so that by July 12th it had reached the proportion of one half, at which period the granular and hyaline casts were exceedingly abundant.

It seemed almost certain that there was some deeply-

seated matter in the neighbourhood of the kidney, and therefore on July 13th, after consultation with Dr. Broadbent and Dr. Poore, I made a free incision in the lumbar region, intending to expose the kidney. A deep abscess was found, containing about an ounce of thick pus, situated rather in front of and below the kidney. The tissues were quite confused from old inflammatory changes, and I did not succeed in making out accurately the locality of the kidney itself, though several punctures were made with a needle into what was supposed to be the organ, with the object of discovering the stone which it was thought was present. Looked at in the light thrown upon the case by the post-mortem examination, it is probable that the kidney could not be felt because it was even then a mere soft bag of pus embedded in dense inflammatory tissue, while the stone could not have been struck because it was already half way down the ureter. Had I used a perforated, instead of a solid needle, it is probable that I should have tapped the kidney and by opening it at this time have discovered the whereabouts of the stone. But this was not done, so the search was relinquished, and the anterior part of the wound was sewn up after inserting two large drainage-tubes to the bottom of it.

The amount of albumen after the operation was not constant. On the following morning it was only $\frac{1}{12}$, but it afterwards rose to $\frac{1}{4}$ and $\frac{1}{3}$. It gradually, however, sank to a much smaller quantity, while that of the pus remained the same; and though there was an occasional and unaccountable increase, it may be said that after three days it did not amount to more than the pus would account for.

The casts, which were in enormous abundance on the 14th, had quite disappeared by the 17th.

There was no hæmaturia after the operation, and no urine ever came through the wound. The amount of urine passed in the twenty-four hours now varied from 70 to 110 ounces, the sp. gr. being usually about 1012 to 1014.

This part of the case may now be summarised by saying that all the symptoms were at once relieved; the nausea disappeared and the appetite returned; the local pain went away, but some remained for a short time in the groin, preventing the patient from extending his hip, the temperature sank at once to normal, and only once or twice rose above 99° F. after this, these slight rises being, curiously enough, sometimes associated with a very temporary increase in the amount of albumen in the urine; this, however, gradually fell to about $\frac{1}{20}$, and the pus steadily diminished in amount, but the bacteria were always present, notwithstanding the administration of quinine, benzoic acid, and other drugs supposed to be bactericidal.

He left his bed about August 10th and went into the country about August 16th, at this time complaining only of an occasional slight pain at the tip of the penis after micturition, but principally when the urine happened to be particularly thick.

From this time forward the patient much improved and became as well as he had ever been in recent years, and in fact his weight was more than it had been for a long time. But though between 18th August, 1884, and December, 1885, he was able to return to his practice, and kept in fair general health, he was always thin and pale, and had much of the aspect of a patient suffering from tubercular disease, though repeated examination failed to discover any evidence of the presence of this condition. He had occasional attacks of colic, always situated on the right side, accompanied generally with some rise of temperature and always with sickness and a temporary clearance of the urine, followed by a discharge of pus when the symptoms abated. These attacks were often preceded by an inability to hold his water, or rather by a frequent and irresistible though painless necessity to micturate, lasting for some days before the onset of acute pain. But for long periods he was quite free from any renal symptoms, except that the urine, though scanty, always contained pus and masses of the minute organisms before referred to,

and a small amount of albumen, perhaps no more than would be accounted for by the pus.

The recurrence of these attacks, while the general health did not suffer, determined me, at his request, to undertake a further operation with the object of removing the stone which I presumed was present in the kidney ; but, after his mind was made up, there followed a period of such excellent health that the investigation was put off again and again, and on Thursday, December 10th, 1885, after some of the usual preliminary troubles, the last and fatal attack set in. I have no authentic record of the earlier symptoms, but it seems to have been quite similar to the previous ones and continued until Monday the 14th. During this time he administered to himself hypodermic injections of morphia pretty freely, taking (I think he said) two and a half grains subcutaneously. On the 14th the colic passed away and on the 15th complete suppression of urine set in.

I saw him on the 17th, when he seemed unaware of the serious import of this new symptom. He had then all the appearance of a patient under the influence of opium. His pupils were small and he was very drowsy, dropping off to sleep whenever he was left undisturbed. His skin was dry and the temperature subnormal ; the pulse hard and incompressible, but the heart-sounds indicating an incomplete systole, a slight systolic mitral murmur which had always been present from the first being audible. The respirations were very slow, the expiration especially being prolonged. Nothing could be felt of either kidney as he lay upon his back, nor was there any local pain or tenderness.

Dr. Broadbent and Mr. Beck saw him with me in the evening, and the question of attempting any further operation was carefully discussed and, as the event showed, unfortunately discarded. We concluded that in all probability the right had for a long time been his only working kidney (the left, it was assumed, having been rendered useless either as the result of calculous or strumous disease), and

that the present complete suppression resulted from the fact that now the right ureter was at last altogether plugged. We discarded as unlikely the hypothesis of the simultaneous plugging of the two ureters with symptoms referred only to the right side, and also the possibility of the suppression being merely functional and sympathetic with the acute trouble on the right side; but I have no doubt that we should have paid more attention to the fact that during the previous attack a fair quantity of moderately healthy urine had been passed whilst there was every reason to suppose that the right ureter was completely or almost completely plugged. It was therefore decided that if anything were to be done it should consist in cutting down on the right kidney, but that the risk of administering chloroform in what appeared to be an advanced state of uræmia (with convulsive symptoms probably imminent) was too great to allow of an operation which, as we thought, could at best only prolong life for a short time. At this time I confess that I came over to the diagnosis of strumous kidney, and was thus quite willing to fall in with the general conclusion. We contented ourselves, therefore, with giving hot-air baths, which produced a copious perspiration, with opening the bowels, which were remarkably sluggish, as indeed were all the functions of the body, and with administering copious warm water enemata in the hope of stimulating the action of the kidneys in case the suppression should after all be functional. For nearly a week the suppression was complete, viz. from the Tuesday till the following Monday morning. During this time the uræmic symptoms became more and more pronounced and we were expecting momentarily the final convulsion. But on Saturday the 19th, as he lay on his right side, I was able distinctly to feel the left kidney, which seemed to be slightly tender. I therefore punctured it with an aspirator needle through a small incision in the loin, and, as the autopsy showed, did actually pierce the lowest calyx of the pelvis, either on this occasion or on the following morning, when the process was repeated, but no water was drawn

off. In the early morning of Monday the 21st he passed 14 oz. of urine, thick and turbid with much tenacious pus, overloaded with albumen (four fifths), but with a very small amount of urea (0·8 per cent.). This, no doubt, came from the right kidney as will be afterwards seen. In the evening of this day he passed 6½ oz. more, showing a smaller amount of albumen (one fourth) and a larger proportion of urea (1 per cent.); a few casts were seen. The uræmic symptoms naturally diminished, but the general condition became worse. Hiccough set in, and the vomiting, which had been very troublesome for some days, preventing all attempts at feeding by the mouth, now became incessant. He lived till the 26th (five days), passing each day about 30 oz. of urine, which became nearly clear, containing but little pus and a few casts. The percentage of urea steadily increased, reaching 1·85 per cent. on the 25th, and the albumen remained small in amount (usually about one tenth) but he did not rally at all and died exhausted by the constant hiccough and vomiting.

At the post-mortem we found the right kidney dilated and nodular, of a large size and quite soft, so that it was easy to understand how it happened that it was impossible to feel it during life, and to detect it when the perinephritic abscess was opened, for even at the post-mortem examination it felt like a piece of intestine. Some kidney substance remained, and the pelvis, which was converted into a large branched cavity contained a large quantity of pus, and the mucous membrane was thickened from old inflammation. The ureter was much distended throughout and half way down it was obstructed by a stone 1·25 inches long and ·5 inch in diameter, which had evidently occupied this position for a long time; but it is remarkable that the distension, though not so great, is very palpable below the obstruction. This is not now very apparent owing to the effect of the spirit upon the preparation.

The left kidney was large and congested; but to the naked eye it showed but little sign of disease, and the

pelvis was quite free from inflammation, showing clearly that there had been no obstruction on this side. The left ureter was normal and patent. The only abnormalities visible to the naked eye consisted in a few small cysts exposed by removing the capsule, and in the fact that the cortical part was perhaps proportionally rather small and the whole kidney substance somewhat opaque.

The bladder appeared to be quite healthy. The urethra was not examined, but as the urine up to the last passed without difficulty, it may be assumed that there was no obstruction in it.

There was no sign of tubercle in any organ examined.

It was interesting to note that the adhesions, resulting from the left pleurisy which occurred thirteen years previously, were quite soft and spongy, and could be easily broken down; and also that the pericardium was throughout adherent by adhesions of similar density. No suspicion of this condition was raised during life, though the heart was frequently examined. There was, indeed, as has been mentioned, a slight mitral murmur, though the mitral valve seemed to be perfectly healthy and competent. The heart was not hypertrophied, the left ventricle was perhaps a little thicker than normal, but the right was certainly thinner. This condition no doubt originated at the same time as the pleurisy, namely, thirteen years before.

Under the microscope the left kidney shows signs of chronic interstitial inflammation, in increase of the connective tissue between the tubules and around the Malpighian capsules; but there is little if any evidence of acute interstitial change. The epithelium, on the other hand, is swollen and cloudy, in some places quite plugging the tubules, in others being partly shed; these changes are no doubt to some extent chronic but principally acute. The kidney must therefore be considered to be in the state which we recognise as one that is particularly apt after operations about the bladder and urethra to develop into a surgical (suppurating) kidney; but it is, I think, clear that it was a good working kidney which might have continued func-

tional for a very considerable time. The stone consisted of uric acid coated by a thin layer of phosphates.

I scarcely venture to refer to the dilatation of the right ureter because its appearance has been so much changed by the spirit in which it has been preserved. Nothing will, I think, be found in the specimen to account for this dilatation below the point of obstruction, and yet this was, at the time of making the post-mortem, very marked. It cannot be supposed that the presence of the stone had set this up, for this is opposed to the experience of other cases in which it is found small below the seat of obstruction. I would suggest that it depended upon previous obstruction at the entrance of the ureter into the bladder, either by inflammatory thickening of the mucous membrane, or by the presence of plugs of pus or mucus; in fact that the dilatation occurred before the stone began its descent. It will, I think, be allowed that dilatation of the ureter may occur without obvious mechanical causes. Such instances have been met with post mortem, and as possible examples of the same condition observed during life I might mention two cases of pyonephrosis with dilated ureters, on which I have operated by incising the kidney, in which no cause of obstruction is known. In one, a large instrument could be passed for a long distance down an enormously dilated ureter and no distension can be found on vaginal examination. The discharge from the wound in this case has, after three years, ceased, and it is clear that all the secretion from this kidney now finds its exit by the natural channel, or else that the diseased kidney has ceased to secrete urine at all, which is highly improbable.

But the point of all others which makes this case noteworthy is the remarkable sympathy exhibited on two occasions between the two kidneys; for it is clear that both in the first and second attacks the sound kidney suffered severely. In the first, the obstruction to the right ureter had clearly been overcome, at least in part, and the remaining lesion consisted practically only in a collection

of pus around the damaged organ ; but the concomitant symptom, and apparently the result, was a secretion of highly albuminous urine containing very large numbers of hyaline and granular casts. As soon, however, as the pus was evacuated, the albumen began to diminish coincidently with the fall of temperature, so that in a very few days it was no more than the pus would account for, and the casts had entirely disappeared.

On the second occasion there were two factors at work, one the obstruction of the right ureter, and the other the presence of a certain amount of morphia in the system, which, when the left kidney struck work, could only be eliminated by the perspiration and the breath. The result was absolute suppression for nearly a week, not accompanied by the symptoms of uræmic intoxication which are met with in acute Bright's disease, but by those which we are accustomed to associate with some mechanical obstruction to both ureters, *i. e.*, to borrow the term used by Sir William Roberts, there were symptoms of obstructive rather than of non-obstructive suppression. We thought we were justified in assuming that such a cause existed, and it is of the greatest importance to know that this was not the case ; for it is at least probable that if the distension of the right kidney had been relieved by an early incision, the function of the left would have been restored. It was the assumption that the obstruction was mechanical which led us to believe that the damaged kidney was the only one which was functional, and that prevented me from undertaking what would certainly have been a dangerous, and probably, as was then thought, a useless operation. How far the morphia was an active cause of the suppression cannot be accurately estimated, but it is noticeable that, two days after the suppression set in, the patient had remarkably the appearance of a man suffering from opium poisoning, and that the drowsiness and contraction of the pupils certainly appeared earlier than is usual in suppression due to a mechanical cause.

It might, of course, be maintained that, both on the first and the second occasion, acute inflammatory processes may have been at work in the left kidney, and have thus influenced the secretion. In answer to which it can only be stated that on neither occasion was the urine smoky, and that after death the only appearance of acute change was shown in the cloudy swelling of the epithelium, which, after such an illness, one would naturally expect to find.

It was suggested that the plan so ably urged and practised by Mr. Thornton should be adopted in this case, viz. that the kidneys should be explored through an abdominal incision. Had this been done, the *left* kidney would certainly have been felt to be enlarged, but I do not think it would have been possible to make out the *right* one because it was as soft as a piece of intestine. If the stone had been detected it might possibly have been pushed up into the kidney and removed through the loin with the best results, but if, as is not improbable, it had not been discovered, we should have been left more perplexed than before.

Lastly, it may be asked why, when the secretion became re-established, the patient did not recover? This is a question not easily answered. The percentage of urea was never more than 1·85, and the quantity of urine in the twenty-four hours never more than 32 oz. It is thus clear that the kidney was not doing its proper amount of work, presumably not enough to put the patient in a condition to overcome the tendency to vomiting and hiccough which finally exhausted him.

To recapitulate, then, the points which seem to make this isolated case worthy of record, it appears probable from a consideration of the symptoms observed:

1st. That the presence of an abscess round a damaged kidney may cause intense albuminuria, and the occurrence of a large number of casts in the secretion of the opposite, comparatively healthy organ, which symptoms rapidly disappear on the evacuation of the pus.

2nd. That irritation in such a diseased kidney (aided, perhaps, by the presence of some morphia in the system) may cause absolute suppression of urine lasting for a week, similar to that caused by mechanical obstruction, and not resembling that which occurs in the course of Bright's disease.

And it is interesting to note:

3rd. That in this instance the whole of the ureter was dilated, although the stone was lodged half-way down it.

Appendix.

The distinction between *non-obstructive* and *obstructive* suppression of urine was first clearly pointed out by Sir William Roberts,[1] and is lucidly explained by the late Dr. Hilton Fagge.[2] Non-obstructive suppression, which occurs in acute Bright's disease, the later stages of chronic Bright's disease, in certain acute fevers, in the algide stage of cholera, and after instrumentation of the urethra, when the kidneys are diseased and perhaps in other conditions, is usually fatal if unrelieved in two or three days, and though it is impossible to give a clear account of the symptoms caused by it which shall fit all cases, it may be said to be characterised by headache, vomiting, the rapid onset of unconsciousness, and what are known as uræmic symptoms, while any urine that may be passed is either bloody or highly albuminous, and there is often more or less anasarca.

In obstructive suppression which follows the complete blocking of both ureters,[3] "the patient, instead of dying within a day or two, goes on for seven or eight days without any other grave symptoms, so that both he himself and his relations find it easy to imagine that there

[1] 'Roberts on the Urine,' 3rd edition, pp. 24 and 26.

[2] Fagge's 'Principles and Practice of Medicine,' 1886, vol. ii, p. 387.

[3] Dr. Fagge, loc. cit.

cannot be anything serious the matter with him. He is calm and free from distress, with an unclouded intellect, and with natural pulse, respiration, and temperature. He may be able to take food fairly well, the tongue being clean, and there being neither nausea nor vomiting. The muscular strength, however, may be observed to fail, and there is often marked sleeplessness at night. There is no desire to micturate, and sometimes no urine at all is voided. Generally, however, at very irregular intervals, the bladder discharges a few ounces, or even a pint or more of urine. This is always pale and watery, and of very low specific gravity, and unless blood be mixed with it, is usually quite free from albumen. At the end of about a week symptoms appear which are commonly followed by a fatal termination within two or three days at the latest. The most distinctive of these are muscular twitchings, which Dr. Roberts says are never wanting. Contraction of the pupils also constantly occurs. The muscular weakness now rapidly increases, and, as a result of its involving the respiratory muscles, the breathing is slow, panting, and laborious. The appetite is entirely lost, and the tongue and the palate become dry. There is increasing drowsiness, with short fitful snatches of sleep and a little rambling delirium. Convulsions and coma rarely set in, the intellect being commonly preserved to the last, so that the patient has in more than one instance spoken sensibly the instant before his death. Diarrhœa is of quite exceptional occurrence, and so is severe vomiting. The skin is moist, and often sweats profusely. There is never any ammoniacal or urinous odour from the surface of the skin or with the breath, nor does the body give off such odours after death. In one instance slight anasarca appeared when the suppression first took place, but it passed off entirely on the third day. The duration of life is stated by Dr. Roberts to be as a rule from nine to eleven days, and he remarks that the passing of a few ounces or even of two or three pints of a dilute urine does not seem to prolong it by more

than a few hours. He knows of only three instances in which the patient survived beyond the eleventh day."

I have thought it best to insert this quotation because we thus obtain a *résumé* of the opinion of two most careful observers, Sir William Roberts and the late Dr. Hilton Fagge. If I ventured to differ from this description in any way it would be by suggesting that death may take place as the result of a single convulsive attack, and also that the profuse perspiration described is not a constant symptom. In the body of the paper want of space has prevented me from describing very fully the patient's appearance, but almost every word of the above quotation would apply to it most accurately, especially that which describes the symptoms of the later stages of obstructive suppression.

(For report of the discussion on this paper, see 'Proceedings of the Royal Medical and Chirurgical Society,' New Series, vol. ii, p. 229.)

ON A CASE

OF

SUPPRESSION OF URINE,

FOLLOWING INJURY TO A SACCULATED KIDNEY CONTAINING CALCULI, THE OTHER KIDNEY BEING ENTIRELY DISORGANISED.

BY

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Samuel C—, æt. 13, first came under my observation at the East London Hospital for Children, in February, 1886, on account of hæmaturia, which followed immediately on a fall over some steps. We were led to suppose that his health had always been good before the accident. The boy was well-nourished and strong and met with his accident while playing at school.

On January 21st he fell over some steps, striking his right side in the region of the kidney; he lay in the school-yard for some time in great pain, and had difficulty in getting home; he was then put to bed. Two days later "he passed blood instead of urine," as his mother expressed it. For a week he vomited every day, and passed bloody

urine, and suffered more or less pain, after which he gradually regained his previous health.

On February 2nd, *i. e.* about fourteen days later, the pain and bloody urine returned, and he was then brought to the hospital. The day after his admission it was noted that he had complained of pain in his side but had not vomited. Careful palpation, which did not appear to cause pain, failed to detect any local abnormality. The urine was pale in colour, sp. gr. 1005, faintly acid, free from deposit and from albumen. He remained in the hospital about three weeks, but beyond the slight pain and some little hæmaturia for a day or two, there were no symptoms to point to injury or disease of the kidney, and he was accordingly discharged. His temperature was raised for the first few days to about 100° F., but subsequently went down, and continued normal. Nothing more was heard of him until the end of April, when he was brought back and readmitted.

It was then stated that he had been very variable since his discharge, sometimes being apparently well, at others complaining of severe pain in the side, and vomiting; for the last five days there had been pain in both sides, and he had vomited and had had shivering fits occasionally. On further questioning, the mother stated that he had had symptoms of this kind for twelve months previous to his accident but that they had got worse since. His urine had not contained any blood previous to the accident.

On readmission (April 27th) he was found to have a prominent swelling, occupying the space between the costal arch and the crest of the ilium, reaching forward as far as the mid-line of the body, and backwards, nearly to the spine; it was smooth on the surface, tense, with obscure fluctuation, dull on percussion and sharply defined at the front margin. The boy complained of pain and some tenderness over the whole swelling, back and front. The first urine passed was dark in colour from the presence of blood. The temperature was normal. He was kept in bed; the urine continued to contain blood until April

30th, when it was found to be considerably less dark in colour.

After consultation with my colleague, Dr. Eustace Smith, the swelling in the loin being more tense and painful, I decided on tapping it, and I drew off fifty-five ounces of clear amber-coloured urine; this was found to contain a considerable amount of albumen; under the microscope plenty of blood-corpuscles were found, but no casts, and no crystals of any kind. The temperature rose slightly after this operation (101° F.) but fell again the next day.

May 3rd.—The urine has averaged forty-five and a half ounces per diem since his admission, but the quantity has gradually become smaller. On April 28th fifty-six ounces were passed; on May 2nd only thirty-four ounces; it has been lighter in colour (that is, freer from blood) since the tapping. During the last twenty-four hours only five ounces have been passed; this was pale in colour, alkaline in reaction, and contained albumen; there were no casts, but an abundance of blood-corpuscles, and a few crystals of phosphates. He is free from pain; the cyst is again gradually filling.

4th.—Ten ounces of urine passed during last twenty-four hours.

5th.—Eighteen ounces of urine passed.

6th.—Urine, twenty ounces.

7th.—Urine, one ounce in the twenty-four hours. The swelling was now as large as, or larger than, at first; its anterior margin reached well beyond the mid-line of the abdomen; its size and tension caused considerable distress. A fine capillary cannula was introduced, and fluid, to the amount of seventy-four ounces, slowly drained away.

8th.—The tumour had again filled up, and was again tapped. Towards night the boy became very restless, and complained of great pain in the abdomen. Hot fomentations were applied and afforded some relief.

9th.—I found the boy very ill; he had been very restless, and at times delirious, through the night, and was

now evidently suffering from uræmia. A vapour bath was ordered, which caused the patient to sweat very profusely; while this was going on, he became convulsed on the left side. Not more than two ounces of urine had been voided during the past thirty-six hours.

The convulsive attacks continuing, and having obtained the consent of the parents, I proceeded to cut down upon the kidney. Chloroform was carefully administered by Dr. Richards, one of the resident officers; as anæsthesia came on the convulsive movements gradually ceased, and the breathing became regular. Mr. Battams, resident medical officer, assisted me at the operation. An incision three inches long, slightly oblique from above downwards and forwards, was made just below the twelfth rib down to the perirenal fat; this was caught up with two pairs of dissecting forceps and separated sufficiently to admit a finger, with the further aid of which a sufficient area of the kidney, or rather of the peri-nephritic sac, was exposed. A small cannula was introduced and a quantity of the fluid let out. After the tension of the sac had been relieved an opening was made with a scalpel sufficiently large to admit three fingers. Between eighty and ninety ounces of fluid were evacuated; it was of a dark port wine colour with a faint urinous smell; it contained several blood-clots (small) which from their dark colour appeared to be recent.

The interior surface of the sac-wall was smooth and velvety, and there existed in different places a few firm adhesions between it and other parts of the cyst cavity. This sac extended far beyond the reach of the finger, downwards and forwards towards the pelvis, and in front beyond the umbilicus. The exact nature of the sac was not made out at the time; it appeared to be connected with the external border and adjoining parts of the anterior and posterior surfaces of the kidney. At its attachment to the kidney the sac was small and narrow, but towards its apex it broadened out and was very extensive, not unlike an immense pear. The narrowness of the attached part of the sac, and the strength and thickness of the walls,

quite prevented exploration, by the finger, of the anterior and posterior surfaces of the kidney and of its pelvis and ureter. The kidney itself was of a livid colour and studded here and there with small cysts. As there appeared to be some fluctuation, and with a view to examine for calculus, I passed in a long needle in several places without, however, striking a calculus. As urine dribbled out of these punctures I subsequently introduced a fine cannula and let out about six ounces of blood-stained urine. After the sac had been evacuated its edges were sewn to the margin of the wound; a suitable drainage-tube was put in, it being deemed inexpedient to proceed further at the time.

My colleague, Dr. Dawson Williams, was kind enough to examine the fluid for me. He reported a sp. gr. 1012 with neutral reaction, and that 100 cc. yielded 1·95 grammes of albumen, and that the filtrate gave on two estimations ·7 and ·6 per cent. of urea, an average of ·65 per cent. The proportion of blood was probably 6 or 7 per cent., assuming all the albumen to have been due to blood.

May 10th.—The boy passed a quiet night, sleeping for several hours. There was no return of the convulsions. On seeing him in the early afternoon I was disappointed to find the dressings quite dry and the sac quite empty. He had voided no urine; a catheter was passed into the bladder and about ℥ss of blood-stained urine was drawn off. Under the circumstances I considered further operative interference contra-indicated. After consultation with Dr. Crocker a wet pack was ordered for an hour, small doses of elaterium were to be given internally and a poultice of digitalis leaves was applied over the region of the kidneys.

May 13th.—The boy died. Between this and the preceding date not more than three ounces of urine were passed. No fluid escaped from the opening in the loin.

Autopsy.—Only a partial autopsy was permitted. The two kidneys, ureters, and bladder were removed together. Connected with the external border of the right kidney was a sac which reached downwards to the brim of the

pelvis, and forwards almost to the mid-line of the abdomen. Between this sac, near its pedicle and the abdominal wall there were some recent adhesions. The sac was completely empty; its interior was shreddy, and dark in colour from blood staining. No direct communication between it and the pelvis of the kidney could be found. It is thus not easy to explain how the urine found its way into the sac. The wall of the sac appeared to be quite independent of the normal capsule, for this latter, though somewhat thickened, peeled off with ease; the sac wall was about a quarter of an inch in thickness, fibroid in appearance, well organised, and therefore of some standing.

The kidney was about twice its normal size, pale and mottled in appearance, with some small cysts on its surface. When cut into it was found to be undergoing cystic degeneration; the calyces were much dilated and thickened; in one of these a calculus, as large as a broad bean, was found. The pelvis of this kidney was also dilated, and the ureter as low down as the brim of the pelvis was thickened and dilated so as to readily admit the little finger. At the level of the brim of the pelvis a small calculus was found in it; beyond this point the ureter was of normal size.

The left kidney was degenerated into mere sacculations, several of which contained small calculi. One of these sacculations contained a calculus similar to, but rather smaller than, that found in the right kidney. The ureter was slightly larger than normal. The bladder was healthy.

As far as could be seen, the other viscera appeared quite normal, but we were not allowed to remove them.

Remarks.—(1) *Diagnosis.*—When the boy first came under observation, a diagnosis was made of rather severe bruising of the right kidney, with consequent hæmaturia. When he came under observation for the second time, with the additional history that was elicited of his condition previous to the accident, and with more or less continuous symptoms of localised pain, vomiting, and intermittent hæmaturia since his discharge from the hospital, a diagnosis

was less easy. Moreover, his symptoms had been greatly aggravated for five days previous to readmission, and besides this, a soft fluctuating swelling had developed in the region of the injury. On the whole, the diagnosis became injury to the kidney with subsequent development of hydronephrosis. But the comparatively late onset (upwards of three months after the accident) of the adventitious sac pointed, I thought, to some further condition, and this was suspected to be a calculus, which had been displaced at the time of the accident, and was now interfering with the outflow of urine from the pelvis of the kidney, possibly in consequence of inflammation set up around its new site. This view was thought to be strengthened by the continued presence of blood in the urine.

(2) *Symptoms.*—An examination of the amounts of urine passed in periods of twenty-four hours is interesting:

In the first period of 24 hours, 56 oz., blood stained.
In the second ,, ,, 56 oz., ,, ,,
In the third ,, ,, 40 oz., less ,, ,,
In the fourth ,, ,, 43 oz., again clearer.
In the fifth ,, ,, 34 oz., ,, ,,

An average of $45\frac{4}{5}$ oz. per diem.

During the next two periods the amount fell to five ounces and ten ounces respectively, while in the next two periods the amount rose to eighteen ounces and twenty ounces respectively, an average of only thirteen and a quarter ounces per diem. In the next period of twenty-four hours, only one ounce of urine was passed, but it was not until two days later that I recognised that suppression of urine had come on. The nephrotic sac had refilled each time after tapping so rapidly that I concluded all urine from the one kidney passed into it, and that the small quantity voided *per urethram represented the product of the other kidney*, and I further thought it not impossible that owing to pressure on its ureter or pelvis, the outflow was interfered with, for at this time there was nothing to lead me to suspect disease of the left kidney. On May 9th, however, I found unmistakable symptoms of uræmia,—somno-

lence, offensive breath, and convulsions, and that not more than two ounces of urine had been passed in two days.

(3) *Treatment.*—Under the circumstances, which was the best treatment to pursue? The diagnosis was far from clear. Had I to deal with a case of complete suppression of urine, brought about by the pressure of a hydronephrosis on the renal nervous ganglia? Had I a case of solitary kidney, and that diseased? or had I to do with double calculous disease with symptoms chiefly confined to one side? To discuss these questions with all the details of the case complete, and with the notes of the post-mortem examination before us, would serve no useful purpose. A more interesting issue appears to me to be whether, even with full knowledge of all the details, I could have dealt successfully with the case.

This opens up the question whether the small calculus impacted in the ureter could possibly have been removed through an opening in the loin, and whether, therefore, an intra-abdominal operation would not have been better. The latter would have permitted me to discover the disorganised condition of the left kidney; also the calculus in the right ureter just at the brim of the pelvis, and possibly to push this calculus back through the enlarged ureter into the pelvis of the kidney. I might also have dealt more successfully with the right kidney, after incising it, had I opened the abdomen, and had eyes as well as fingers to assist me. Finally, it would have demonstrated the absolute infeasibility of removing the right kidney, it being practically a solitary one, a proceeding I might otherwise have contemplated, and which, I think, would have been surgically indicated, in a case where there was an obstruction, more or less complete, in its ureter, which could not be removed.

(For report of the discussion on this paper, see 'Proceedings of the Royal Medical and Chirurgical Society,' New Series, vol. ii, p. 232.)

A CASE

OF

ANEURISM OF THE ABDOMINAL AORTA

TREATED BY

LAPAROTOMY AND THE INTRODUCTION OF STEEL WIRE INTO THE SAC.

BY

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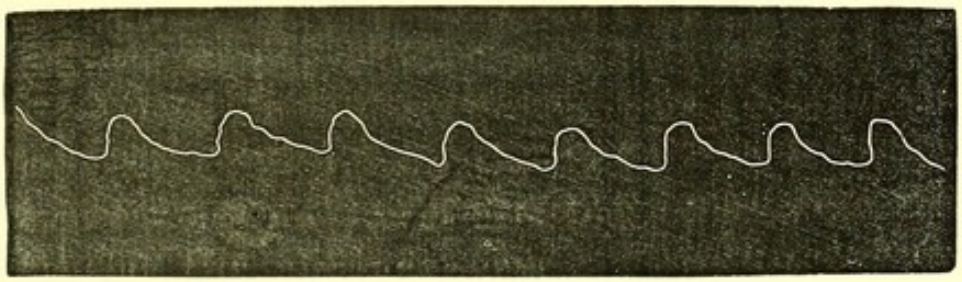
J. K—, æt. 46, a German, by occupation a tailor, was admitted to the Middlesex Hospital under the care of Dr. Finlay on September 9th, 1886, and subsequently came under my care in Dr. Finlay's absence during the autumn vacation.

The patient stated that he had contracted syphilis in Geneva in 1864, and that about the year 1880 he had several "spots" or "sores" scattered over his body, some being as large as a sixpenny-piece. He had been twice married. His first wife died in her first confinement; his second wife had given birth to five healthy children, and had had no miscarriages. He had never

suffered from gout or rheumatism, and denied habitual alcoholic excesses. In 1884 he first complained of pain in his left leg and thigh without ascertainable cause, which he was told was "sciatica;" a year later the pain had become most severe in the left hip and loin, and for about four months previous to admission he had experienced a throbbing sensation below the margin of the ribs on the left side, accompanied by intense, constant, fixed pain, referred to the back and diminished by bending the body forwards.

State on admission.—An emaciated foreigner with demonstrative manner, expressing himself in an almost incomprehensible "patois;" temperature normal, respiration tranquil, radial pulse beating 88 to the minute, feeble and rather incompressible. The cardiac dulness was obliterated by hyper-resonant lung note; the apex-beat was localisable only by the stethoscope at the fifth rib within the mammary line; the sounds were feeble and free from bruit, but the second sound was comparatively accentuated over the aortic base. The breathing was everywhere weak with prolonged expiration; there were no adventitious pulmonary sounds. Micturition was not abnormally frequent; the urine was of good specific gravity (1020—1025), free from deposit, and contained neither albumen, sugar nor casts. There was no evidence of skin, lymphatic, or throat syphilis. The abdominal walls were thin, lax, and sunken; some prominence and visible systolic pulsation in the epigastrium, especially to the left of the middle line, were noted; on palpation the pulsation was recognised as distinctly expansile in character, and its limits could be accurately determined as extending vertically from one inch below the xiphoid cartilage to the umbilicus, and laterally, from the middle line to three and a half inches to the left; percussion over the area thus demarcated was dull. The tumour appeared to be fixed posteriorly and its pulsation could not be felt by the hand on the abdomen when the patient was placed in the knee-elbow position. A loud, blowing, systolic

bruit was heard over the area of pulsation without exerting pressure with the stethoscope. There was also a tender region in the back to the left of the three upper lumbar vertebræ, over which the heart-sounds were plainly audible. Pulsation in the femoral arteries was forcible, equal, and synchronous on the two sides, and tracings, subsequently taken, presented characteristically aneurismal oblique line of ascent and loss of summit wave.



The *diagnosis* of aneurism arising from the abdominal aorta, probably sacculated with narrow aperture and eroding the vertebræ, was made; absolute rest was enjoined; the diet was regulated on Tufnell's principles, applied as rigorously as the fractiousness of the patient would permit; iodide and bromide of potassium were administered four times daily, the former in gradually augmented doses of fifteen to sixty grains, and hypodermic injections of morphia were given at night or according as pain or restlessness indicated.

Progress.—On October 1st pulsation was plainly felt and systolic murmur heard in the left ilio-lumbar region. On October 4th the advisability of surgical treatment was discussed in consultation with Mr. Morris, who considered that proximal compression of the aorta was feasible only if a well-made cone-shaped pad were fitted to the abdominal tourniquet, and as the pulsation extended so low it was very doubtful if distal compression would do more than control one of the primary branches immediately beyond the bifurcation of the aorta.

Laparotomy and the introduction of steel wire was, on the whole, thought to be the operation best suited to the

case and it was decided to adopt this treatment. The patient, after due explanation, gave his consent.

On October 5th the pulsation had extended in an upward direction almost as high as the xiphoid cartilage, and laterally, two inches to the right of the middle line; a loud, musical, diastolic bruit was superadded to the systolic over the point of maximum intensity of the pulsation, and there was a trace of albumen in the urine. The pain in the back was daily becoming more and more excruciating, necessitating the employment of morphia injections every two or three hours day and night, and the patient's strength was obviously diminishing. As it was considered that there was evidence of rapid increase of the aneurism —now absolutely precluding the applicability of proximal compression—and imminent risk of its rupture, recourse was had to the procedure about to be described.

Operation.—The patient having been fed for two days exclusively by the rectum, and the bowels having been cleared out with a dose of castor-oil, an incision was made on Friday, October 8th, at 9.30 a.m., by Mr. Morris from the xiphoid cartilage to the umbilicus along the middle line. The abdomen having been opened, the surface of the aneurism was first exposed by teasing an opening in the great omentum between the transverse colon and the stomach. This was found not to give sufficient room for manipulation, and some difficulty was experienced in keeping back the viscera—considerably distended by flatus—with flat sponges and the fingers of an assistant, to sufficiently expose the sac for the introduction of the cannula. The sac was therefore sought, and easily exposed, above the upper edge of the stomach, and by dragging that viscus downwards and the left lobe of the liver and edge of the thorax upwards an ample view of the sac was acquired and maintained. The trocar and cannula were introduced and the cannula held between the forefinger and thumb of the left hand in such a direction as to pay in the wire downwards and to the left, *i.e.* in the course of the blood current. A sharp spurt of blood followed the

withdrawal of the trocar but ceased as soon as a few inches of the wire were introduced. Now was found the extreme difficulty of the operation owing to the coiling of the wire and the depth of the cannula. The wire, which was of fine steel, similar to that used in Dr. Cayley's case[1] reported last year and similarly purified, was coiled upon a roller which, being too large to enter the abdominal wound, was necessarily held at some distance from the cannula; thus, several inches of wire had to be unwound before it could reach the cannula, and there was, of course, a strong tendency for it to coil into the same circles as upon the roller. It was both a tedious and difficult task to straighten the wire as it was being paid through the cannula. Another difficulty was caused by the depth at which the cannula had to be held by the left hand of the operator and the tendency to lift the cannula which the force of the pulsation exerted, so that it was not always easy to be sure how much pressure was required to adjust the cannula between the outward force of the pulsation and the inward force of pressing the wire into the sac. The result was that the cannula slipped out of the sac, and after about eight inches of wire had been paid through, it was found that the greater part of this wire had passed simply into the abdominal cavity. The wire was therefore severed close to the sac, and the cannula was again introduced at a point near to the first puncture and more wire was paid into the aneurism. At length, after—as it was believed—about a foot of wire had been introduced, an insurmountable kink stopped its further progress. The cannula was withdrawn, and the wire was cut off close to the surface of the sac. By this time the aneurism was felt to be very appreciably harder than at the beginning of the operation. There was no hæmorrhage throughout excepting the free flow from the aneurism each time the cannula was introduced, but this ceased as soon as a short length of wire had been fairly introduced into the sac. The withdrawal of the cannula was not followed by hæmorrhage.

[1] 'Med.-Chir. Transactions,' vol. lxix, p. 267.

The abdominal wound was closed in the usual manner, and covered with a dressing of boracic lint, cotton wool and a roller.

The patient rallied well after the operation and for two days his progress continued satisfactory; the circulation in his lower extremities was unimpeded; he stated decidedly that the pain in his back had disappeared; the wound caused him little or no discomfort, and he retained nutrient suppositories and peptonised enemata containing brandy, under which his pulse improved, as well as small quantities of beef-tea and milk given by the mouth. It was, however, necessary to continue the free employment of morphia in order to control his restlessness. On October 11th he refused food, returned his enemata, developed hallucinations from which he had suffered previous to the operation, became almost uncontrollable in his attempts to get out of bed, and finally began to sink gradually. He died of asthenia on October 13th, five days after the operation, during which time his temperature had never exceeded 100·6° F.

The *autopsy* was made thirty-four hours after death; post-mortem rigidity had passed off, and there were evidences of commencing putrefaction in the abdomen, which was moderately distended. The edges of a linear wound extending from the xiphoid cartilage to the umbilicus were maintained in accurate apposition by five deep sutures and were healthy in appearance. The costal cartilages were ossified; on the anterior aspect of the right ventricle was a "milk-patch" the size of half-a-crown; both sides of the heart contained a large amount of mixed clot, a considerable proportion of which was pale, firm, and adherent to the endocardium, especially of the valve-cusps. From the right side a fibrinous clot extended up the pulmonary artery as far as its bifurcation. All the valves, as well as the myocardium, were normal, the heart weighing 10¾ oz. The ascending and transverse aorta and the arteries arising from them were healthy in appearance, but considerable atheromatous change was present in the

descending thoracic aorta, increasing in amount and intensity from above downwards. There was marked emphysema of the anterior portions of the upper lobe of both lungs and of the anterior edge of both lower lobes with congestion, œdema, and subpleural ecchymoses in their posterior portion.

On opening the abdomen there was found to be strictly localised, recent, adhesive peritonitis connecting the parietal peritoneum corresponding to the operative wound with the left lobe of the liver, the small omentum, the anterior surface of the stomach and transverse colon, and the great omentum, which latter was very short. The stomach was somewhat dragged downwards by these adhesions, and a mass, considered to be an aneurismal sac containing clot, could be felt above its lesser curvature, but the seats of puncture could not be distinguished. The sac was more easily reached by turning up the transverse colon with its mesentery and was found to lie chiefly to the left of the middle line, extending as far as, and displacing the left kidney outwards, on the inner aspect of the upper portion of which it made a deep indentation. The pancreas lay across, and was arched forwards by, the aneurism, but neither it nor any other of the viscera were firmly adherent to the sac. The left renal artery, however, coursed for nearly an inch at its origin in the thickness of the retro-peritoneal tissue reinforcing the sac and the corresponding vein, which ran transversely across the sac near its centre, seemed slightly compressed from behind forwards. The absence of firm adhesion between the sac and the viscera was considered as evidence in favour of its rapid extension.

The stomach was dilated and contained about an imperial pint of thick fluid; it and the intestines, which were empty, showed no sign of disease. The liver, spleen, and kidneys were healthy. These and the hollow viscera having been removed, the more intimate relations of the aneurism were investigated. The fibres of the left crus of the diaphragm above, and of the psoas muscle on

the left side and below, overlay, and were incorporated with, the sac. Seen from the front, the aneurism was trilobate in form, the central portion being the most prominent, and the aorta lying between it and the right lobe. Its diameter, both vertically and transversely, measured four inches. At its most prominent part, immediately to the left of the origin of the superior mesenteric artery, there was localised thickening and induration of the sac, which probably corresponded to the seat of the punctures; and extending vertically downwards from this to the lower boundary of the sac, some dark clot infiltrated the matted tissues reinforcing the sac, due presumably to oozing from the punctures during the operation. The aorta having been opened along its anterior aspect the opening of the aneurism was perceived to be situated entirely on its posterior wall, directed slightly towards the left side and behind the origin of the cœliac axis and superior mesenteric artery; it was oval in form, measuring one inch vertically by five eighths of an inch transversely, and was occluded by dark clot.

The aneurism was then removed along with the last dorsal and three upper lumbar vertebræ, to which it was firmly adherent, and the mass immersed in alcohol for three weeks. A vertical, antero-posterior section having then been made from the centre of the vertebral canal to the most prominent portion of the sac, the progress of extension of the aneurism from its point of origin could be observed (*a*) backwards, to erode deeply the last dorsal and two upper lumbar vertebræ whilst sparing, to a comparative degree only, the intervertebral discs; (*b*) to the right of the aorta for three quarters of an inch, displacing the inferior vena cava; (*c*) upwards for an inch to the upper border of the last dorsal vertebra; (*d*) downwards for nearly three inches, to within half an inch of the level of the aortic bifurcation; and (*e*) to the left for a distance of two inches and three quarters. The result of the pressure around the aorta was a marked diminu-

tion in calibre of that vessel for a distance of two inches, best seen at the level of the renal arteries, where the bulging forwards of the posterior wall was very notable.

The section described divided the aneurism into two unequal parts, the left being the greater; both parts were filled with clot of which more than one third, lining the interior of the sac throughout the section, was considered to be of ante-mortem formation (see Woodcut, p. 275). It may probably be fairly assumed that in the part on the left side of the section the proportion of ante-mortem clot was even greater. In order, however, not to destroy the specimen the clot was not removed in searching for the wire; but its presence was easily detected by the introduction of two steel needles connected with the poles of a galvanic battery —of which only two cells were in action—and with a very delicate galvanometer which at once indicated when a complete circuit was established by contact with the wire, by a marked deflection of the needle. The wire had obviously fallen from its point of introduction and lay embedded in the middle of the left portion of the aneurism at a distance of about an inch from the centre of the section described. It was, unfortunately, impossible by this method to verify the amount of wire introduced.

Remarks.—I have been emboldened to bring this case before the Royal Medical and Chirurgical Society by the fact that many of the most important contributions to our knowledge of the treatment of abnominal aneurism are contained in the Transactions of the Society, and because I am convinced that the Fellows whose criticisms will, I trust, follow will discuss the principles of the treatment adopted in a philosophical spirit rather than decry the operation on the mere ground of its want of success in the present instance.

From a medical point of view the case presented no features of novelty and but few of special interest, the confirmation post mortem of almost all the surmises of the physician only demonstrating the facility of diagnosis of certain conditions by physical examination. The presence

of a loud, musical diastolic, in addition to the more usual systolic bruit, was considered as evidence of narrowness of the aperture of the sac, and as it developed along with other signs indicative of its rapid extension, as confirmatory of these. It is universally admitted that a diastolic bruit over an aneurism is of exceptional occurrence, but this case, as well as one narrated by Dr. Wickham Legg[1] and one by Mr. Bryant,[2] invalidates the absolute truth of the dictum of Quincke[3] and differs from the experience of Dr. Walshe[4] and Dr. George Balfour[5] that such a diastolic bruit is never present.[6] Nor can the presence of a to-and-fro bruit afford occasion for wonderment, if free to-and-fro circulation take place between a large artery and a large aneurismal sac containing little or no coagulum, separated one from the other by a narrow and hard-edged aperture.

The main importance of the case, however, lies in the treatment resorted to. I admit that I am somewhat sceptical as to the value of drugs in such cases, and am inclined to consider the benefit attributed to them in many cases published as successful, as due mainly to the absolute rest and dietary restrictions with which they have been invariably combined. Da Costa's[7] six consecutive cases treated by iodide of potassium in full doses are certainly, however, more convincing than the solitary triumphs ascribed to acetate of lead,[8] sulphate of iron,[9] aconite,[10] or ergotine[11] (employed subcutaneously by Wolff in a case of

[1] 'St. Barthol. Hosp. Rep.,' vol. xvi, p. 258.

[2] 'Med.-Chir. Trans.,' vol. lv, p, 225.

[3] 'Ziemssen's Encyclopædia,' vol. vi, p. 419.

[4] 'A Practical Treatise on Diseases of the Heart, &c.,' London, 1862, 3rd edition, p. 494.

[5] 'Clinical Lectures on Diseases of the Heart and Aorta,' London, 1876, p. 351.

[6] Dr. Balfour, however, cites an instance of diastolic murmur in abdominal aneurism in his second edition, 1882.

[7] 'Boston Medical and Surgical Journal,' vol. xcix, p. 813.

[8] Daly, in 'London Hosp. Rep.,' vol. iii, p. 179.

[9] Bone, in 'Lancet,' 1864, vol. ii, p. 207.

[10] Grimshaw, in 'Med. Press and Circ.,' 1876, vol. i, p. 406.

[11] 'Berliner klinische Wochenschrift,' 1873, p. 314.

more than dubious diagnosis), whilst the utility of atropia and digitalis as recommended by Mr. Barwell,[1] receives no support from recorded experience. Moreover, the undoubted value of the iodide of potassium in some thoracic aneurisms renders it probable that abdominal aneurisms may to some degree be amenable to its influence, however exerted, and it will probably not be contested that surgical interference ought to be invoked in the treatment of all internal aneurisms only when that drug along with absolute rest and restricted diet have proved futile after prolonged trial.

Such, unfortunately, was the case in this instance; indeed I cannot but believe that had surgical measures been employed earlier, before the patient's strength was at such a low ebb, his life might have been materially prolonged, and its burden of pain lightened.

Even at the time of the first consultation with my surgical colleague, Mr. Morris, I was dubious as to the applicability of proximal pressure, not sanguine as to its results, and fearful of the risks of direct pressure on the sac, whilst a subsequent study of the literature of the subject has convinced me of the dangers of any compression applied to the aorta above the level of the duodenum. Below that level pressure upon the intestine or other viscera may be avoided by careful application of the compressor. The fact that five successful cases of proximal pressure have been recorded by Murray,[2] Greenhow,[3] Moxon and Durham,[4] Heath,[5] and Philipson,[6] shows that such treatment *may* be successful despite the occurrence of hæmatemesis, albuminuria, jaundice and threatened gangrene of the lower extremities during, or as the direct results of, the operation; whilst in one case, included in five

[1] 'Ashhurst's Encyclopædia of Surgery,' vol. iii, p. 476, *et seq.*

[2] 'Med.-Chir. Trans.,' vol. xlvii, p. 187.

[3] Ibid, vol. lvi, p. 385.

[4] Ibid., vol. lv, p. 213.

[5] 'British Medical Journal,' 1867, vol. ii, p. 287.

[6] Ibid., 1877, vol. ii, p. 247.

unsuccessful cases collected by Mr. Barwell, gangrene resulted, the external iliac was tied and amputation performed; the remaining four cases died from peritonitis and bruising of the abdominal contents. One cannot but agree with the author when he writes,[1] "Doubtless more (cases) have failed than are made available by publication, but we may conclude with almost complete certainty that all successful instances are known." I am inclined, on the whole, to limit the applicability of proximal pressure to those aneurisms in the abdomen which arise below the aortic bifurcation.

It is, however, to be borne in mind that the great majority of abdominal aneurisms arise from the aorta or its branches about the level of the cœliac axis, that is, at a point above which compression of the aorta is impracticable. Dr. Sibson's[2] experience that 133 out of 177 cases occupy this position remains unchallenged; thus the principles of treatmeut of the present case are those of a great preponderance of abdominal aneurisms.

What alternatives, then, to the operation performed presented themselves, and with what prospect of success comparable to that obtained by similar treatment by Loreta, of Bologna,[3] in his memorable and in many respects similar case? The records of distal pressure employed in five cases are far from encouraging. Mr. Bryant's case[4] died eleven hours after the second application of the tourniquet from peritonitis and obstruction of the bowel; Mr. Lunn and Dr. Benham's case[5] died eleven days after the operation from gangrene of the intestine and blocking of the superior mesenteric artery, the aneurismal sac being nearly filled with clot; Dr. Markham Skerritt's[6] case died seven weeks after the operation from diffusion of the

[1] Loc. cit.

[2] 'Medical Anatomy,' p. 57.

[3] 'Memoire dell' Academia delle Science dell' Istituto di Bologna,' vol. vi, 1885, p. 215.

[4] 'Med.-Chir. Trans.,' vol. lv, p. 225, *et seq.*

[5] Ibid., vol. lxviii, p. 191, *et seq.*

[6] 'British Medical Journal,' 1878, vol. i, p. 405.

aneurism ; in a case under Sir James Paget in St. Bartholomew's Hospital in 1865, the facts of which have been kindly communicated to me by Mr. Astley Bloxam, then house surgeon, the results of the compression comprised peritonitis, gangrene of the gut and the reduction of the pancreas to a pulp ; whilst in an unrecorded case under the care of Mr. Barwell, to whose courtesy I am indebted for permission to cite it, distal compression was applied continuously for six days without either temporary or permanent benefit. Not only does the treatment by distal pressure appear to me to be fraught with danger from pressure upon the abdominal contents involving, it may be, their obstruction, laceration, rupture, inflammation or gangrene, but also to be theoretically faulty, as the branches arising from the upper portion of the abdominal aorta are too numerous and too large to afford any reasonable prospect of sufficient arrest of the circulation above the point of compression to determine coagulation.

Another danger, common to all measures of pressure, whether proximal or distal, less obvious than the above, but probably none the less real, lies in the increased tension in the aorta and its branches above the seat of pressure and the consequent risk of rupture of diseased vessels. This increase of tension, transitory though it may be, has been graphically represented in Dr. Mahomed's tracings from the radial pulse in the paper by Dr. Moxon and Mr. Durham.[1] Without specially investigating the point, I have incidentally come across a narrative of three instances, and have myself seen one instance of coexistent thoracic aneurism, whilst in four others a second, unsuspected abdominal aneurism was found to exist on post-mortem examination.

The results of galvano-puncture in the treatment of internal aneurism, in this country at all events, have been admittedly disappointing. As far as I have been able to ascertain, it has been employed in only two cases similar to the present. The more successful of these is reported

[1] Loc. cit.

by Keyes.[1] The patient, a woman, æt. 42, died about a month after galvano-puncture had been performed for the third time, probably of exhaustion from an unsuspected thoracic aneurism. The author honestly states his doubts whether the action of the current had anything to do with the presence of the stratified clot found in the sac, as it was not firmer about the seats of puncture than elsewhere, and did not differ from the clot of other aneurisms in the same person. The less successful galvano-puncture was performed by Felice dell' Acqua[2] upon an Italian nobleman, but "the patient quickly died."

The treatment of aneurism by the introduction of foreign bodies into the sac has been only recently discussed before the Society, and its principle has received general acceptance and approbation.[3] Mr. Bryant's opinion "that it seems likely to be of use in certain aneurisms in which none of our ordinary methods are applicable, and in which under many circumstances we have only to stand by and wait for the end" represented, I think, the feeling of the Society at the discussion and stimulated me to hope for some measure of success in a case presenting so many points of resemblance to Loreta's, although the age and unsatisfactory *morale* of the patient materially reduced the chances.

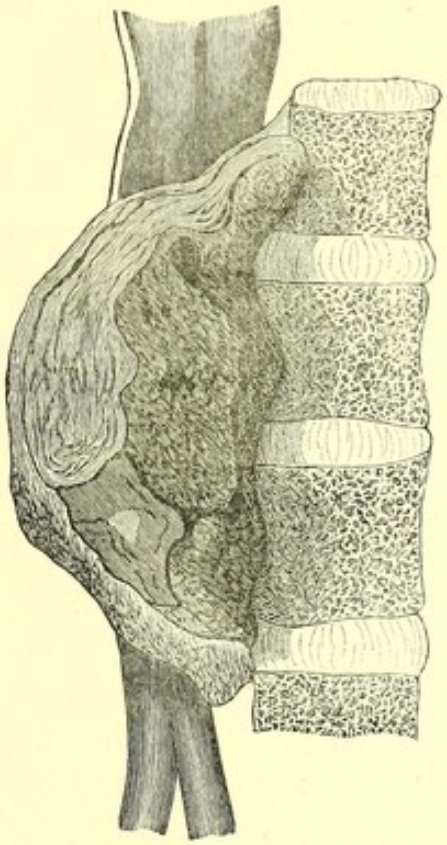
The operation presented some unforeseen difficulties, upon which Mr. Morris will remark, but these were, on the whole, successfully encountered, and the patient's death was certainly not directly due to the operation, as in the majority of recorded cases of the most feasible alternative, *i.e.* distal compression. The amount of clotting produced by even so short a length of wire as it was found possible to introduce, the insignificant hæmorrhage from, and complete occlusion of the punctures, the absence of peritonitis or other abdominal complications, despite the unruly conduct of the patient, all to my mind justify the procedure

[1] 'New York Medical Journal,' vol. xiv, p. 569, *et seq.*

[2] 'Gazetta Medica Italiana Lombardia,' No. 28, 1870, p. 217.

[3] 'Proceedings Roy. Med. and Chirurgical Soc.,' New Series, vol. ii, No. 2.

adopted and render the epithet "inaccessible," hitherto frequently employed with regard to aneurisms thus situated for the future an unjustifiable one.



Remarks by Mr. Henry Morris.—There was no difficulty in exposing to view the aneurism sufficiently for the purpose of introducing the cannula. Whether in another case this would be accomplished better by drawing down the stomach or by drawing up the transverse colon and omentum will depend upon the precise situation of the aneurism and the nature of the adhesions round about it.

The great difficulty attending the operation was the management of the wire. We had thought it desirable to have the wire coiled upon a long and very slender rod so that there should be a strong disposition for it to regain its twist and so occupy but a small space within the aneurism. This difficulty could be greatly diminished or altogether removed by having the wire wound upon a reel which could be held in the right hand of the operator within the abdominal cavity, and thus the wire could be paid off the reel directly into the mouth of the cannula.

Another way of obviating it would be to substitute fishing-gut or horsehair for the wire, but the advantage which the wire has over either of these materials is twofold: it coils up within the aneurismal sac, and is therefore less likely to find its way through the opening between the aneurism and the artery, and it can be introduced in one continuous piece instead of presenting a number of ends to be pushed onwards into the sac.

There is no doubt that but for the kinking of the wire more of it would have been inserted in this case; but it had never been my intention to introduce more than two feet because I have long held the opinion that for the perfect cure of aneurism the contraction of the sac must follow the coagulation of blood within it. It seems to me therefore that one of the points in connection with this mode of treatment as to which it is important that we should obtain information is "what is the smallest quantity of the foreign body which need be introduced to excite the requisite formation of clot?" The smaller the quantity the greater the probability, *cæteris paribus*, of a cure, because the contraction of the walls of the aneurism is more likely to be safely and perfectly accomplished when one foot instead of many feet of wire forms part of the contents of the solidified tumour.

(For report of the discussion on this paper, see 'Proceedings of the Royal Medical and Chirurgical Society,' New Series, vol. ii, p. 241.)

A CASE

OF

SACCULATED AORTIC ANEURISM

TREATED BY THE

INTRODUCTION INTO THE SAC OF THIRTY-TWO FEET OF STEEL WIRE.

BY

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Received March 8th—Read April 12th, 1887.

A CASE of aortic aneurism treated by Moore's operation was recently under our care at the Royal Hospital for Diseases of the Chest, and as this mode of treatment is attracting some amount of attention at this time, and has not been followed by the success of which it appears to be capable, it seems desirable to place the case on record.

The patient was a tall, well-developed, powerful-looking man, a smith by trade, forty-eight years of age, who gave the following history.

History.—His parents both died at the age of seventy; there was no hereditary disease in his family. He had always been a smith, and had been in the habit of drink-

ing freely while at his work, but not to excess. At fourteen years of age he had typhoid fever, and he once had a slight attack of gonorrhœa; he had not had syphilis. Five years ago he had pleurisy with effusion on the right side, for which he was tapped. He recovered in two months. Five months ago he noticed a swelling of the front of his right chest which slowly increased. Two months ago a severe cough came on, attended with dark, thick expectoration, and at this time he had pain in the swelling. The cough lasted three weeks, and since then he has noticed that he has been short of breath on exertion, and that the swelling has been larger. He has not had any pain either in the chest or down the arm. He has not suffered from palpitation. He has recently been losing flesh, in spite of his appetite remaining good.

State on admission to hospital (November 20th, 1886).—The man was stout, florid, with congested facial capillaries, iron-grey hair, prominent suffused eyes, free from pain, and said he felt "quite well."

On the front of the right chest was a smooth round swelling, in shape and position like a mammary gland, extending vertically from the second to the fifth rib, and laterally from the right edge of the sternum to the axilla. This swelling was the seat of a forcible expansile and heaving pulsation synchronous with the heart's action. Over it was heard a harsh high-pitched systolic murmur. The second cardiac sound was very loud and accentuated.

The heart's impulse was heaving and very forcible, one inch outside the left nipple line in the sixth interspace; the cardiac dulness reached up to the third cartilage. There was a loud systolic murmur heard at the apex, and conducted into the axilla, and above and outside the apex the second sound, previously clear, was replaced by a coarse diastolic bruit, audible over a very limited area only. The pulse at the left wrist was of good force and volume. On the right side it was smaller in volume, but this was thought to be explained by the presence of a large branch

coming off from the radial artery above the wrist, and coursing round the lower end of the radius to the back of the thumb.

Over the uppermost third of the abdomen, and along the right side of the chest below the swelling, were numerous dilated veins, the blood in which would flow in either direction. The pupils were equal and active. There was no dysphagia.

Above the swelling anteriorly, and down to the angle of the scapula behind, the resonance of the right chest was much impaired, and the respiratory murmur was distant and weak. No râles; vocal vibration diminished. The base of the lung was resonant and breath-sound normal. Nothing abnormal was detected in the left lung or in the state of the abdominal viscera. The urine was acid and free from albumen.

Progress.—The man was kept in bed on a restricted diet, and was ordered iodide of potassium in gr. xv doses three times a day. He was of a very excitable temperament, and it was impossible to get him to keep quiet in bed, but in spite of this it was noted on December 3rd that there was "rather less force in the pulsation of the tumour." Over the posterior apex of the right lung there was loud bronchial breathing; no râle.

Matters went on in this way without material change until the second week in January, when the patient was a good deal excited by the death of two patients in the same part of the ward as himself.

On January 11th the following note was made:—"For the last few days the swelling has been increasing rapidly in size, and more noticeably so up to yesterday morning. The lower part of the tumour has become much more prominent, not extending into the axilla, but hanging down like a slightly pendulous mamma. Pulsation is very marked and appears more superficial. He has had severe throbbing pain about the nipple, and shooting pain in the shoulder, which is now less. The cardiac signs are unchanged in character, but the intensity of the diastolic

bruit in the left axilla is increased. He is paler, has no cough or difficulty in swallowing; no change in the general physical signs."

In consequence of this change Dr. White asked Mr. Gould to see the man with him with a view to operative interference for his relief. It was evident that the aneurism was undergoing rapid enlargement and that, if left alone, death from external or subcutaneous rupture of the sac was imminent. After explaining the position to the patient it was decided to introduce some fine steel wire into the sac, and this was done on January 11th at five o'clock in the afternoon.

Operation.—The part was rendered insensitive by a subcutaneous injection of five minims of a 20 per cent. solution of hydrochlorate of cocaine, and then a fine Southey's trocar was thrust into the aneurism about the level of the third intercostal space, immediately above the right nipple and at a spot where the pulsation seemed to be most superficial.

Bright blood spurted from the cannula, through which fine steel wire was slowly passed. When about nine or ten feet of the wire had been thus passed in, it broke and it was found impossible to push the broken end through the cannula. Accordingly, a second puncture was made immediately below the first, and twenty-two feet of the same wire was passed into the aneurism; as there was now distinct and increasing resistance to the passage of the wire the cannula was slowly withdrawn and the wire cut close to the skin, and then the same was done to the first cannula. A pad of boracic lint was strapped over the punctures. It was estimated that from ten to twelve ounces of blood escaped through the cannula while the wire was being introduced, but the hæmorrhage was at once controlled by the pad. The operation lasted one hour and a quarter. The patient was nervous and excited throughout, and at times became faint, and for this an ounce and a half of brandy was given. He did not complain of any pain. Immediately after the opera-

tion, when very faint and with feeble cardiac impulse, there was hardly any pulsation to be detected in the mamma-like projection of the aneurism. Two hours later he had recovered his ususl appearance and was warm and comfortable save for some shooting, throbbing pains in the back of the shoulder. There was very slight oozing of blood from the punctures. Pulse 96, respirations 32. He was ordered gr. x of chloral and gr. xv of bromide of potassium at once and to be repeated as required. He failed to get sleep in the night, and was very uncomfortable owing to his constrained position in bed and to numbness in the shoulder and arm. The little pad of lint had been stained through by blood but was quite dry. The tumour was much harder than before, and inelastic; the pulsation was more of a heaving character than before. The radial pulse was of fair force and volume. He was rather restless through the day, and in the evening was sick once, being troubled with flatulence.

January 13th.—After the injection of a quarter of a grain of morphia, patient had a quiet night; his pulse was somewhat bounding, 96; respirations 24. There was now a bruise of bluish-yellow colour, extending over the upper part of the swelling. The skin here was glazed but not red. The cardiac phenomena were unchanged, but the second sound was feeble over the aneurism, and the systolic bruit was less marked. The bowels were confined, and a dose of compound senna draught was given, which had the desired effect. The pad of lint was removed in the afternoon because it was soaked through, and there was noticed a considerable and continuous oozing of slightly blood-stained serum from the second puncture. There was slight tenderness over the punctures, the swelling was firm and the pulsation felt in it seemed to be deep. The discharge of blood-stained serum increased and became very troublesome.

14th.—Pulse less full and less bounding in character; patient quiet, free from pain in the chest. Discharge of

thin blood-stained serum through the second puncture was much more profuse. He was troubled with diarrhœa and tenesmus which abated towards evening, and after a bromide and chloral draught he slept well.

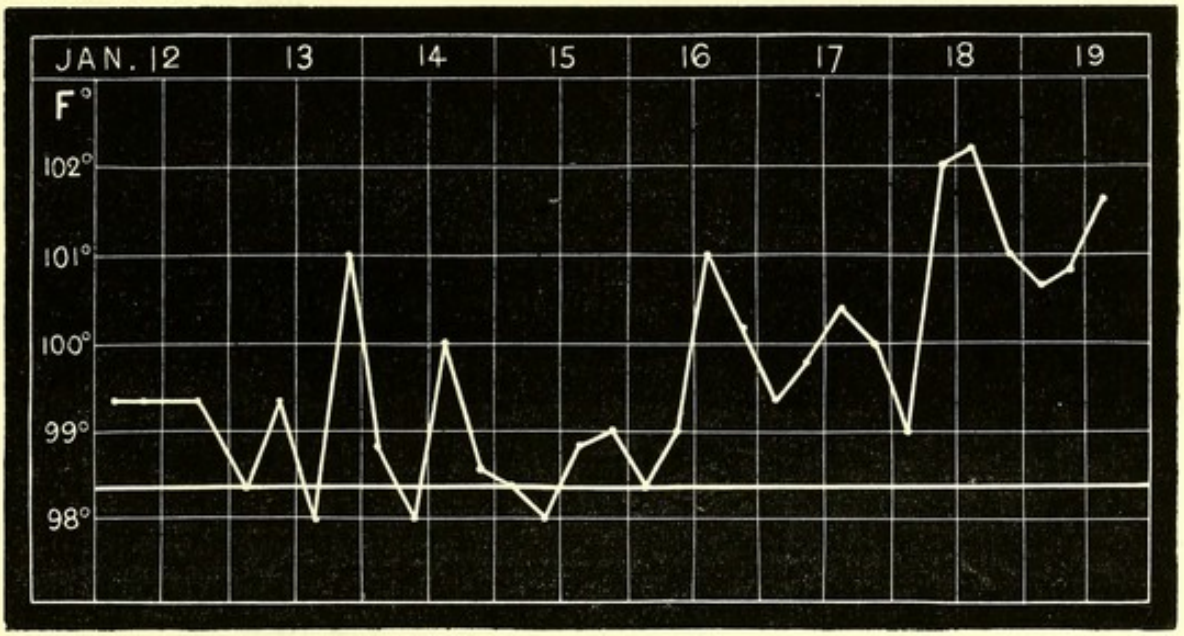
15th.—Was quieter this morning; no pain; no cough; pulse of fair force, soft and regular. The serous oozing still very profuse.

17th.—The discharge continued, and was estimated at ℥v to ℥vj in the day; it was unchanged in character, being thin, blood stained, and not coagulating spontaneously. The ecchymosis was fading, and there was no redness, tenderness, or other sign of inflammation over the aneurism. There was no change in the heart-sounds, but the pulse was smaller and softer. The bowels had been freely opened with Friedrichshall water. Finding that pressure over the puncture very easily arrested the leakage Mr. Gould applied a pad of boracic lint with four "tails" of strapping. This was in the afternoon; in the evening the patient became very restless and complained of the pressure of the pad, but he slept after the injection of a quarter of a grain of morphia.

Next morning (18th) he was in pain; the pulse was small and feeble. The draining of serum had completely stopped. There was no change in the limits, appearance, or pulsation of the tumour. During the day the pain in the right chest increased, and he complained of a sense of tightness in the axilla. It was found that a general swelling of the subcutaneous tissue over the aneurism had taken place, and extended into the axilla, and upwards to an inch above the clavicle. The skin had a slight purplish tinge, and two bullæ filled with dark fluid developed over the aneurism.

Next morning (19th) the extremities were cold, the pulse could scarcely be perceived at the wrist, and the patient was evidently moribund. There was very extensive œdema of the subcutaneous tissue of the chest, involving also the shoulder and arm; the skin over it was mottled in different shades of purple and becoming gangrenous. At 1 p.m.

he died. The temperature throughout the last ten days of life, is shown on the accompanying chart.



Post-mortem examination (twenty-six hours after death) by Dr. White.—Rigor mortis marked; much hypostatic congestion of the dependent parts. The considerable swelling of the right side of the chest and neck and right arm wore a purplish aspect, and large bullæ containing blood-stained serum were present around the right nipple. Arborescent lines of brown discolouration were scattered over the chest and extremities. The tumour of the chest felt hard. The skin, subcutaneous fat, and the pectoral muscle were reflected from the aneurism, and in doing this much sanguineous fluid escaped. The tumour protruded through the chest wall, between the second and third rib cartilages close to the sternum, and further out, between the third and fourth ribs. Over its outer part the muscle was blended in the sac wall. Two small portions of the sac, each about the size of a threepenny piece, and close to the punctures, were obviously sloughy, and during the dissection one of these gave way, and some of the wire protruded from the aperture, and a considerable quantity of dark fluid blood ran out.

The heart, aorta, sternum, and tumour were then removed in one piece and set aside for further examination.

The heart was displaced downwards and outwards, and was covered with a thick layer of subpericardial fat. The left ventricle was considerably dilated and its walls moderately thickened. The aortic orifice was enlarged; the three cusps of the valve were healthy. Immediately above its origin the ascending aorta was enlarged into an irregularly globular dilatation, the size of a large orange, and the interior of this part was very irregular from atheroma. The transverse part of the arch, as far as the origin of the left subclavian artery, was slightly dilated; beyond that point the thoracic aorta was of its normal calibre. Springing from the upper part of the tubular aneurism above mentioned, on its anterior and outer aspect, was an ovoid sacculated aneurism of about the size of a cocoa-nut. Rather more than half of the sac was contained within the chest, and was firmly adherent to the sternum, right ribs, and rib cartilages and the right lung. It projected through the chest wall at the level of the third right rib, the cartilage and a part of the rib having been destroyed in the process. The outer portion of the aneurism formed a tumour adherent to the second and fourth ribs.

The intra-thoracic portion of the aneurism had a thin but well-marked sac. The extra-thoracic portion was destitute of any definable sac, and the tissues limiting the cavity were composed of soft, shreddy, muscular tissue abundantly infiltrated with blood and serum.

The aneurism contained a confused mass of steel wire, in the meshes of, and partly surrounding which, was a considerable quantity of firm, red, fibrinous clot. One loop of the wire projected just through the mouth of the aneurism into the dilated aorta. The entire mass of wire and clot formed an extremely firm mass; it was nowhere adherent to the sac.

The mouth of the sacculated portion of the aneurism was very large, easily admitting four fingers.

Permission to examine the other organs could not be obtained.

Remarks.—In spite of the large size of the aneurism, of its wide mouth, and of its rapid growth, there was one circumstance that rendered it favorable for treatment, and that was the large sacculated portion well marked off from the tubular portion. Although the first part of the arch of the aorta was dilated, yet the external tumour was a distinct sacculated pouch springing from it. Tufnell's treatment was never fully carried out. The man himself when first he came under treatment could not be made to realise the very serious nature of his ailment, and would not submit to a very rigorous limitation of his diet, and the most explicit and often-repeated directions failed to make him observe the perfect rest that forms so essential a part of this treatment. He was of so excitable a disposition that not only would he turn about in bed, but do so quickly. It was no matter of surprise therefore that when he became considerably upset by the death of two near neighbours in the ward the aneurism rapidly increased in size. It was evident then that unless something was done which would speedily excite coagulation in the sac, death would quickly ensue, and it was equally evident that our choice was limited to one or other of the means of directly exciting coagulation within the sac. Electrolysis has not met with the success which was at one time anticipated for it, and the urgency of the case seemed to particularly contra-indicate it. It is true that Moore's treatment of aneurism may still be spoken of as in its experimental stage, but the partial success obtained by Dr. Cayley and Mr. Hulke in a case not unlike our own encouraged us to hope that it might prove of benefit to our patient, and at any rate prevent the threatened rupture of the external portion of the aneurism. The result achieved showed that while our expectation was not groundless we had not made sufficient allowance for the danger always attendant upon the sudden coagulation of a large aneurism lying in a bed of loose connective tissue,

nor for the special mechanical disadvantage of having a large mass of steel wire within a thin sac. The particular points in the case to which we would especially direct attention are as follows:

The fatal termination of the case was due to the sloughing of the tissues and the aneurism, and this, we fear, was largely due to the treatment pursued. At the time of operating the external portion of the aneurism was enlarging rapidly and the sac had no doubt given way. The operation itself led to an extravasation of blood beneath the skin, and a free discharge of bloody serum occurred from one of the punctures. But it was only when a compress was applied with sufficient firmness to arrest this oozing that really serious symptoms arose; and it would seem that the moderate pressure of the pad of lint over the firm unyielding mass of wire in the aneurism sufficed to produce gangrene of the intervening tissues. It is well known that the sudden coagulation of the blood in a large aneurism is liable to cause sloughing of the sac and inflammation of the superjacent tissues, and this has been especially noticed in the case of axillary aneurisms where the tumours are surrounded by loose connective tissue. The reason assigned for this occurrence is the altered nature of the pressure exerted upon these tissues when the fluid within the sac becomes solid. Still more is this the case when the contents of the aneurism are not a mass of fibrin and blood-clot only, but a tangled coil of steel wire.

Looking back upon our case, therefore, we think that the partial rupture of the sac which had no doubt taken place at the time of the operation rendered it an exceedingly unfavorable case for any treatment, perhaps especially so for the one we pursued. Further, the length of wire introduced was too great, and the unyielding mass it formed in the aneurism was distinctly a source of danger. Had we stopped when the wire broke, and been content with some ten feet or so coiled up in the cavity, it may be that we should have excited coagulation without

exposing our patient to the danger which proved fatal to him. We were under the impression that the aneurism was even larger than it proved, and, guided by previous operators, we continued to pass in the wire until distinct resistance was met with, and did not regard, as we should have done, the serious risk this entailed.

The hæmorrhage during the operation was annoying to the operator but probably beneficial to the patient.

The following is a list of all the cases which we have been able to find in which Moore's treatment has been carried out.

1. Moore. 1864. Aortic aneurism; iron wire, 26 yards; death. ('Med.-Chir. Trans.,' xlvii.)
2. Levis. 1873. Subclavian; horsehair; death.
3. Bryant. 1873. Popliteal; horsehair, 20 feet; "almost complete consolidation;" death in five days from ulcerative endocarditis.
4. Rubio. 1874. Innominate; iron wire, 2—3 mètres; death.
5. Paul. 1874. Innominate; horsehair, 15 feet; death. ('Med.-Chir. Proc.,' New Ser., 12.)
6. Levis. 1876. Innominate and subclavian; horsehair, several yards; death.
7. Levis. External iliac; horsehair, several yards; inflammation, suppuration of sac; death from hæmorrhage.
8. Baccelli. 1884. Aortic; watch spring; death.
9. Baccelli. 1884. Aortic; watch spring; death.
10. Van der Meulen. 1879. Brachial; catgut; recovery.
11. Loreta. 1885. Abdominal. Silvered copper wire; recovery.
12. Ransohoff. 1886. Aortic; silver wire; death.
13. Maury. Subclavian; horsehair; death.
14. Cayley and Hulke. Aortic; steel wire; improvement; death.
15. Pringle and Morris. Aortic; steel wire; death.
16. White and Gould. Aortic; steel wire; death.

Among these sixteen cases there are but two of complete recovery: those of Loreta and Van der Meulen; but in two more, those of Bryant and Hulke, the result was at any rate a partial success. Ashhurst mentions that Domville, Montenoveri, and Stimson have also had unsuccessful cases, but we have not been able to find details of these.

Conclusions.

1. Moore's treatment is worthy of further careful trial in properly chosen cases.

2. The operation should be performed before there is reason to suspect rupture of the sac.

3. Only a small quantity of wire should be introduced at any one time.

4. No firm pressure should be made over the aneurism afterwards.

(For report of the discussion on this paper, see 'Proceedings of the Royal Medical and Chirurgical Society,' New Series, vol. ii, p. 241).

A CASE

OF

ANEURISM AT THE ROOT OF THE NECK

AND IN THE UPPER PART OF THE CHEST IN A VERY ADVANCED STAGE

TREATED BY THE

INTRODUCTION OF STEEL WIRE.

BY

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SURGEON TO THE MIDDLESEX HOSPITAL.

Received March 23rd—Read April 12th, 1887.

The number of cases of aneurism treated by the introduction of foreign substances into their cavity with the intention of inducing coagulation of the blood they contain in a mechanical way is yet so small that this plan of treatment must be considered as still upon trial. No final consensus of opinion can be expected respecting its remedial value until much larger experience of it has been gained. At present widely different estimates are held of the benefits that may reasonably be expected from it, of the risks to which it exposes the patient, of the relative value of the different materials which have been or may be used, and some technical points in the procedure are doubtless susceptible of improvement.

As a contribution to the statistics of this method I offer to the Royal Medical and Chirurgical Society the following report of a case so treated recently under the care of Dr. Cayley and myself in the Middlesex Hospital. The case presents other features of much interest involving questions of diagnosis and of distal ligature, but I intentionally exclude the discussion of these on the present occasion, my concern now being solely with the plan under consideration. I will only remark that at the stage of the aneurism in which the patient entered the Middlesex Hospital the supposition of its being innominate and aortic was a reasonable inference from the dulness of the right chest apex; next, that distal ligature was proposed, but this, and indeed every surgical measure, was firmly and persistently declined by the patient until he re-entered the hospital last February, shortly before his death.

On Aug. 19th, 1886, E. B—, æt. 31, seaman, a sunburnt, dark-haired, muscular man, was admitted into the Middlesex Hospital under the care of Dr. Cayley. He had in the root of the neck immediately above the right collar-bone a pulsating expansile swelling which displaced the trachea and larynx to the distance of one inch beyond the middle line. Percussion on the clavicle and over the first intercostal space disclosed dulness of the right chest apex continuous with the swelling apparent in the neck. The expansion of the chest was good; the respiratory and the cardiac sounds were normal. The pulse was 60; that at the left was thought weaker than that at the right wrist and slightly retarded. Axillary temperature 97° F. The left pupil was slightly smaller than the right, and this not from synechiæ. His voice, he said, was weaker than formerly; for this no cause, unless a slight congestion of the laryngeal mucous membrane, was apparent.

Previous history.—Thirteen years ago he had had palpitation of heart, for which he was treated in the Naval Hospital at Haslar and quite recovered. Two years later he had cholera in Russia. He had once had gonorrhœa but never syphilis. These, he said, were his only illnesses

until the present, otherwise his health had always been good; he had always been strong and well able to do the heavy work incidental to a seaman's life.

History of present illness.—On October 13th, 1885, he stumbled, and in falling struck his neck upon the edge of the tail-board of a cart, receiving at the same time a cut through the lip. For this he went to St. Mary's Hospital, and he continued, he said, to attend there until about six weeks before he entered the Middlesex Hospital in August of the following year. He first noticed the swelling in the neck about three weeks after the date of the fall. It had gradually increased, and latterly the enlargement was attended with much pain. The pain spread from the swelling up the right side of the neck to the shoulder, the armpit, and the arm. He had had occasionally slight difficulty in swallowing, and also slight embarrassment of breathing, but not to the degree of causing actual distress.

The diagnosis formed by Dr. Cayley, in which Dr. Powell, who also examined the patient, concurred, was aneurism of the innominate artery and aorta.

The patient remained in a medical ward until September 27th, when he was transferred to my care for the greater convenience of subjecting him to the plan of treatment known as Tuffnell's. The treatment hitherto had mainly consisted in confinement to bed, the topical application of ext. belladonnæ, and the internal exhibition of potass. iod.

Under this, with transitory semblances of slight improvement, the aneurism had continued to enlarge. Its upper border now reached the level of the middle of the thyroid cartilage, and its outward extension was not less evident. The right common carotid artery was found to be displaced forwards and to the left of its normal course by the advancing aneurismal swelling, and the pulsation in this vessel was distinctly weaker than that in the left common carotid.

On October 15th Tufnell's plan of treatment was begun.

This is too well known to need description; it is enough to say that the details were very strictly carried out. On November 13th, having latterly become restless and sleepless at night, and his resolve to submit to the restraint imposed beginning to fail, his midday meal was increased by ℥j of solid and ℥j of fluid; and on December 2nd a further increase of ℥ss of solid and ℥ss of fluid was sanctioned; his diet at the latter date then consisted of buttered bread ℥ij with milk ℥ij for breakfast, the same solids with the same quantity of milk or wine in the evening, and of ℥iv of meat, boiled fish and bread, with ℥iv of milk for dinner. This continued to be his diet allowance until January 23rd of this year, when at his urgent wish he was allowed to return to his home in a suburb of London.

A daily report of his progress during the above time would be very tedious to read, and it is not necessary. Within a few days the pulsation of the aneurism was found to have become less forcible, some decrease in the size of the swelling was observed, and some firmness suggestive of the formation of clot was noticed; the pain also had abated so much that he spoke of it as being quite bearable, and occasionally absent for a short while. His nights, on the whole, were better than for several previous weeks, although he was sometimes roused by the collection of mucus in his throat, which he could not easily expel.

The patient was intelligent; he fully realised his condition, and the marked relief of pain and the other signs of improvement made him for a time very docile; but the improvement did not continue, and with slight fluctuations in its rate the swelling enlarged, and pain increased. With this he lost heart, he became intolerant of restraint, and requested his discharge.

At that date (January 23rd) the right common carotid artery, which before could be easily felt and even seen pulsating as it ascended upon the inner and anterior border of the swelling, was traceable only as a band in which none or an extremely feeble pulsation was discover-

able. The pulse in the right facial and temporal arteries was also very weak.

He was taken home recumbent in an ambulance, but the journey tried him, and for some days afterwards he felt less well. About ten days before his readmission into the hospital a rapid enlargement of the aneurism began, and with this the pain in the neck and shoulder became so severe that on the February 24th he re-entered, anxious then to submit to any measure that might be proposed.

The aneurismal swelling then measured 8 inches across horizontally, $6\frac{1}{2}$ inches in its vertical diameter, and $16\frac{1}{2}$ inches round its base. Its inner border reached 2 inches to the left of the middle line; its lower extended to the level of the second rib; its outer was one inch distant from the acromio-clavicular joint, and its upper border was in the level of that of the thyroid cartilage. The whole swelling pulsated forcibly, and at its outer part, where the skin seemed very thin, a peculiar wavy motion was discernible. The outer end of the collar-bone, displaced downwards and forwards, could be traced upon the lower and outer part of the aneurism for a distance of about $1\frac{1}{2}$ inches where the continuity of the bone was interrupted; its sternal end could not be made out. The right radial pulse was synchronous with and slightly fuller than the left.

At a consultation with Drs. Cayley, Powell, Fowler, and Biss, the proposal to pass wire into the cavity of the aneurism was concurred in. This was carried out the same afternoon; between 10 and 11 yards of pianoforte wire being introduced through a small trocar. At the moment of withdrawing the piercer, blood jetted from the cannula, and subsequently during the passage of the wire it continued to issue freely. This loss seemed to lessen the great tension of the aneurism, and then a small fragment of the collar-bone could be felt and indeed be seen to move up and down under the thin structures forming the anterior wall of the cavity. At the end of

about a quarter of an hour the swelling was thought to have become firmer, and as there appeared to be some resistance to the entrance of the wire this was severed and the cannula withdrawn. The puncture was secured with a very fine pin and twisted suture. The patient, who during this procedure had been under the influence of ether, had become by this time very cyanosed. One hour afterwards the blueness of his surface had disappeared; his wrist-pulse was good; and he complained only of some pain in the right arm. Next day the aneurism was thought to be distinctly firmer. The patient said that he "felt well." The suture was removed and the little puncture, after being dusted with iodoform, was covered with a piece of boric lint secured and covered with collodion. On March 1st, in the forenoon, just above the site of the puncture, bloody serum was found to ooze from a slight crack in the cuticle. Around this spot the swelling felt softer and it pulsated more strongly than elsewhere. This oozing continued through the day. In the evening, the left wrist-pulse being full and bounding, 120, and the aneurism heaving strongly, and the pain in it and in the arm having become very severe and the patient being in great distress, my house surgeon, Mr. W. K. Sibley, took ʒivss of blood from the left arm with marked relief. This was followed by sleep lasting several hours, and on the following morning (March 2nd) he said that he had scarcely any pain. The aneurism was thought harder, and it pulsated less forcibly. He expressed himself as feeling better and more comfortable. His principal source of distress was the collection of mucus in the throat.

On March 3rd the circumference of the base of the swelling was 21 inches, the horizontal diameter 8½ inches, and the vertical diameter 7½ inches. On March 5th—three days later—the circumference had increased to 26 inches. On the 6th he passed a very restless night, his breathing being occasionally very embarrassed. The sputa were slightly bloody. The superficial veins of head and neck, particularly those of the right side, were turgid. On March

8th some bloody serum oozed from a purplish spot at the outer side of the swelling, quite remote from the puncture. The sputa continued slightly bloody. The difficulty of breathing increased; it seemed referable to pressure on the trachea. Death occurred on March 9th at 11 a.m., nearly thirteen days after the date of the introduction of the wire, apparently from exhaustion. His temperature ranged during the three first three days between 98° and 99° F.; on the third day it touched 99·9° F., its maximum, and during the last three days it was subnormal.

For the following account of the post-mortem examination I am indebted to Mr. Leopold Hudson, the pathologist to the hospital.

"On dissection the aneurism was found to spring from the first part of the right subclavian artery. It formed a large, roughly spherical swelling which measured 6½ inches in the vertical, and 7 inches in the horizontal direction. It projected into the neck 4½ inches above the clavicle, measuring from the middle of that bone, where its eroded end could be felt beneath the skin. From its highest point it passed obliquely downwards, traversing the median line one inch above the episternal notch, displacing but not compressing the trachea, and gaining a firmer attachment to the inner half inch of the left clavicle; from this point it passed downward just internal to the left first chondro-sternal joint, then crossed the sternum obliquely to the right second costal cartilage, and continuing in this line became attached to the right third rib 2½ inches from the sternum. It followed this rib round the thorax for 4 inches, then turned up obliquely across the second and first ribs behind, and became attached to the right side of the bodies of the fifth, sixth, and seventh cervical vertebræ, the cancellous tissue of these being eroded and laid bare, the corresponding intervertebral discs being also attacked, but destroyed to a much less extent than the bone. Looked at from behind, its attachment to the sternum followed a line from the left clavicle

to the third rib, exactly similar to that on the front of the bone. The concavity of the pleural dome was replaced by a bulging, and the wall of the sac was here so thin that it appeared in places to be formed merely by the serous membrane. There were no pleural adhesions, the right lung being depressed and compressed, but on section healthy. It extended but slightly into the apex of the axilla, and the acromial end of the clavicle projected for two inches out of its upper and outer part."

"On laying open the sac from behind the coils of steel wire appeared to traverse almost equally all parts of it except the apex. On the wire, especially at the outer, lower, and posterior parts, there had been deposited a considerable layer of white fibrin, and where these covered coils abutted on the sac wall, the fibrinous material had extended over the interior of that structure. In the upper part of the sac, however, clotting had not yet taken place, only very soft black coagulation being found here. Digital examination of the interior revealed a soft shreddy wall everywhere except in the immediate neighbourhood of the large vessels, where endothelium still remained. The clavicle to the sternal side of its middle had lost more than an inch of its substance by erosion, and the bone, so far as it could be felt in the sac, was soft, spongy, and bare of periosteum. The first rib about its middle was also eaten through, and it, with the second and upper border of the third ribs, and right half of the manubrium sterni, was bare and rough as far as it lay in the sac."

"The aortic arch was slightly dilated, and showed in the intima raised opaque patches of atheroma. The innominate artery, an inch and a quarter in length, contained no clot, and admitted with difficulty a forefinger; its walls were healthy. The common carotid was much smaller than the vessel of the opposite side, was somewhat flattened from pressure, but was pervious. That part of its lower outer wall which abutted on the aneurism had been by stretching drawn upwards, so as to make its lower orifice three-quarters of an inch above the opening of the inno-

minate. The third part of the subclavian artery ran in a channel on the upper posterior wall of the sac; it was small and flattened from before backwards, but like the carotid contained no clot. The sac above approached the skin so nearly that the intervening matted tissues could only with great difficulty be recognised. The scalenus anticus was reduced to an attenuated fibrous cord, which blended with the sac wall, three inches external to the orifice of the innominate, and immediately beneath it the subclavian opened into the sac. The thyroid axis was represented by a minute branch filled with firm clot; its transverse cervical and suprascapular branches could not be found. The vertebral, internal mammary, and superior intercostal were each filled with firm clot. The right innominate vein was plugged immediately distal to the point of entry of the inferior thyroid branch, and the clot extended up into the internal jugular. The subclavian vein, filled with firm grey coagulum, passed directly into the wall of the sac, and could not be isolated from it. The recurrent laryngeal nerve sprang from the posterior surface of the wall of the sac; it was much flattened; the main trunk of the vagus in a similar way passed down and became lost in the upper and inner part of the sac. The left common carotid and subclavian were pervious and of normal size; the thoracic portion of each was displaced to the left. The left innominate vein was slightly narrowed from pressure. The belly of the right sterno-mastoid, where it crossed the sac, was inseparably blended with it; at its attachment to the manubrium the tendon had been quite hollowed out, and its anterior fibres formed the boundary wall of the sac at this point. The only visceral changes found were signs of subacute inflammation of the brachial tubes and incipient fatty infiltration of the liver. By wish of the friends the head was not examined."

The post-mortem examination disclosed what had been previously quite unsuspected—that the aneurism arose from the first part of the subclavian and not from the innominate artery and aorta. It showed that the wire

was nearly evenly distributed through the cavity and that the formation of firm clot was less than might have been expected from the distinctly greater firmness of the aneurism noticed towards the close of the operation (and during the first few days afterwards). It is possible that this illusion was produced by the support afforded by the wire to the wall of the cavity and by the contraction consequent on the lessening of tension by the loss of blood through the cannula. Whilst I may not claim that the introduction of the wire attained the measure of success which my earlier case had led me to hope, I think that I may at least assert that it proved innocuous, and that its failure here should not weigh against its further trial in suitable circumstances.

Remarks.—Scope of wire, method, &c.: From my present standpoint I would restrict the application of that which may be termed the *induction of clotting by mechanical entanglement*—for shortness I shall speak of it as the wire-method—to those classes of cases in which either through the situation of an aneurism, or in consesequence of its very advanced stage, or upon both these grounds, pressure and ligation in their several modifications are inapplicable, and where the plan of treatment known as Tufnell's has failed or cannot be practised.

For such cases the wire-method will, I suggest, favorably compare with the injection of chemical coagulants into the sac, with the injection of constringents (*e. g.* ergotine) into the tissues outside the sac; and with galvano-puncture. It is not my intention to reopen a discussion of the relative merits of these latter methods. To the wire-method it is objected that it exposes the patient to very great danger from embolism and from septic infection, and also from endarteritis set up by friction of the wire against the inner surface of the sac. To these objections it may be replied that in the truly desperate circumstances incidental to the advanced stage of a large aneurism some danger may well be faced. The probability of the occurrence of embolism will be largely dependent on the size and direct-

ness of the opening of communication between the sac and the large blood-channel on which it is seated. I am not aware that these matters can be ascertained even approximately by any diagnostic signs yet known.

There must always, then, in every case be an inherent chance of embolism which the surgeon cannot control, but his share of the risk can be diminished by the employment of a material which he can ensure will remain in the neighbourhood of his puncture, not stray indeterminately and possibly pass into the blood-channel. On this account, and also because its thorough asepticity can be more certainly ensured, I prefer to employ spirally coiled hard iron wire, which again coils up over the cavity on escaping from the tube. Horsehair, fishing-gut, and catgut cannot be depended on to do this; they are more difficult to manipulate, and from the character of their surface they are more likely to carry infective matters.

In neither of the two cases reported by Dr. Cayley and myself, in which I have used such wire, have embolism or septic infection or endarteritis occurred. This number of instances is, of course, much too small for a basis from which to generalise, but it encourages the hope that these disasters will not often occur. As regards the method of procedure the patient was anæsthetized, less because it was apprehended that the operation would be very painful, than because it ensured his thorough quietude and freedom from nervous agitation with accompanying vascular excitement. A small exploring trocar was used. Its cannula proved a trifle too wide. The calibre should be such as will just allow the easy passage of the wire. If the latter fits the tube too loosely there may occur inconvenient leakage; if too tightly there is risk of the wire being jammed, an accident which may immediately be followed by the wire kinking in front of the cannula. I have found it easy to pass the wire through the tube by seizing it with two pairs of dissecting forceps each used alternately, "hand over hand." For ensuring its asepticity the wire, as was done for the first case reported, was boiled in a

solution of potassic hydrate to remove grease, washed in distilled water, immersed in a solution of carbolic acid, and as it was being drawn off the cylinder on which it was coiled it was passed through a sponge wet with the same solution. With the idea of lessening as much as possible hand contact with the wire, and of facilitating its passage through the tube into the sac, Messrs. Maw and Thompson have constructed the little apparatus exhibited. Its principal parts are a small cistern in which the wire coiled on a revolving cylinder is immersed in an antiseptic solution. Connected with this is a tubular needle for use as a trocar, and intermediately placed are two small rollers which grip the wire between them, and by turning the milled head of the upper roller the wire is drawn off the cylinder and passed through the needle-tube.[1]

[1] In carrying out these ideas much ingenuity has been shown by Mr. Jones, the surgical mechanician to the hospital, a workman in the employ of the above firm.

(For report of the discussion on this paper, see 'Proceedings of the Royal Medical and Chirurgical Society,' New Series, vol. ii, p. 241.)

AN ANALYSIS OF NINETY-THREE CASES

OF

WRITERS' CRAMP AND IMPAIRED WRITING POWER;

MAKING, WITH SEVENTY-FIVE CASES PREVIOUSLY REPORTED, A TOTAL OF ONE HUNDRED AND SIXTY-EIGHT CASES.

BY

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On February 12th, 1878, I had the honour of communicating to this Society a paper containing an analysis of 75 cases of "Writers' Cramp and Impaired Writing Power."[1] I wish now to supplement that paper by communicating some of the details of 93 additional cases, making a total of 168.

For the purpose of reference, and to avoid confusion, the cases which form the basis of the present paper are numbered from 76 to 168.

My first series of cases was arranged in 6 groups, viz.:

1. Paralytic (6 cases).
2. Spasmodic (5 cases).
3. Degenerative (9 cases).

[1] 'Medico-Chirurgical Transactions,' vol. lxi.

4. Neuritic or neuralgic (20 cases).
5. Writers' cramp proper (31 cases).
6. Anomalous (4 cases).

With the present 93 cases certain changes in this classification are necessary.

Class 1 as then constituted is omitted, because with the exception of 2 cases of paresis from pressure on nerves the present series contains no case of definite nerve paralysis.

In Class 2 (spasmodic) 4 out of the 5 cases were consequent on cerebral lesion. These 4 will therefore be placed in the new paralytic (cerebral) group. Cases 16 and 17 clearly were cerebral, and so also was probably Case 75 (congenital left-handedness).

Nos. 73 and 74 (locomotor ataxy and general paralysis) will be placed in the degenerative group, and I shall omit No. 72.

Further, for the purposes of comparison I shall include groups 4 and 5 in the same class, which I shall speak of as the neuro-muscular groups.

Thus, by the omission of Nos. 1, 2, 3, 4, 5, 6, 11, and 72, there remain 67 of my previous series available for comparison with the present 93, and these 67 may be thus arranged:

1. Paralytic (cerebral), 7 cases.
2. Degenerative, 9 cases.
3. Neuro-muscular, 51 cases.

Adopting the classification for my present 93 cases I have:

1. Paralytic, 13 cases (10 cerebral).
2. Degenerative, 14 cases.
3. Neuro-muscular, 66 cases.

Paralytic group.—20 cases, of which 13 belong to the new series. Of these 13 cases No. 76 was possibly (?) due to lead-poisoning, and Nos. 77 and 86 were cases of paresis due to pressure on nerves; 8 (78, 79, 80, 81, 82, 83, 84, 85) were cases of paresis due probably to long antecedent cerebral paralysis. None of them were coarsely

hemiplegic, and in all the evidence of previous cerebral paralysis had to be carefully searched for. An attack of hemiplegia, even though it be slight and occur no matter how early in life, always, I believe, leaves its mark. Patients are often said to "recover" from attacks of right hemiplegia and are able to follow their occupations without apparent difficulty. It is, however, in the performance of a delicate muscular act such as writing, which requires a maximum amount of steadiness and firmness, that the previous cerebral damage asserts itself. The writing is shaky or slowly executed, or executed with a certain sense of difficulty.

In elderly people who come complaining of writing with difficulty the evidence of slight hemiplegia must always be looked for. Persons also who state that "they have always had a difficulty in writing" must be examined carefully for evidence of hemiplegia, a trouble which is by no means uncommon in early life, but which, I believe, is often not suspected, because the movements of an infant are too little specialised to enable defects to be noticed.

When I speak of "hemiplegia," a term which it is convenient to use, it must be understood that I include under it such a condition as brachial monoplegia from cortical lesion.

Among the cerebral cases are included 3 of congenital left-handedness (1 in the previous paper and 2 in this), because I believe this defect to be due to lesion or anomaly of the left cerebral hemisphere. Of these cases (75, 87, 88) only one wrote with his left hand, which he did fluently, and gave a normal slope to his letters. He was left-handed for fine work (writing, playing flute, and violin) and right-handed for coarse work (hammer, knife, cricket). The left hand and foot were bigger than the right, and the left boot was the first to wear out. Both this patient and No. 87 had injured the left arm (dislocation of shoulder and fracture of arm) in early life, but this had not cured the left-handedness. In 87 the right clavicle was shorter than the left, a defect which was noted by

me in another case of left-handedness not included in this paper, and in whom there was excess of knee-jerk on the right side. In No. 83, who had presumably suffered from infantile hemiplegia, there was observed a faulty development of the right clavicle. Nos. 87 and 88 were left-handed in everything except writing, and it is, perhaps, to be regretted that they were not taught to write well with the left hand instead of badly with the right.

In 16 out of the 17 cerebral cases there was a general clumsiness in delicate acts other than writing. There was altered irritability of one or other of the muscles of the right hand in 6 cases.

Nerve tenderness was observed in 5 cases, and tremor in 5 cases.

Degenerative group.—23 cases, of which 9 (12, 13, 14, 15, 18, 19, 20, 73, 74) belong to the old series and 19 (89 to 102) to the new.

Of these 2 (12, 93) were apparently slight cases of *disseminated sclerosis,* 2 (89, 90) of *paralysis agitans,* 3 (73, 92, 102) of *posterior sclerosis,* 2 (94, 97) of *lateral sclerosis,* 5 (13, 15, 18, 19, 20) of *muscular atrophy and fibrillary tremor,* 1 (74) was a case of *general paralysis,* 1 (14) had marked tremor of right hand and foot, 1 (91) showed signs of emotional weakness, was colour blind, but had no change of the discs; 3 (96, 98, 101) had rheumatic thickenings of finger-joints or muscles; 1 (95) was gouty, and 2 (99, 100) were alcoholic.

Of these 23 cases 6 had diminution in the faradic irritability of certain muscles, 2 only had nerve tenderness, and in 14 tremor was a marked symptom. In 11 it was noted that there was difficulty in other acts than writing.

This and the preceding group embrace 23 cases. Thus rather more than a fourth of the total 168 cases have been referred by me to lesions of the nerve-centres.

Neuro-muscular group.—117 cases (51 in the old series, and 66 in the new).

This group is formed partly *per viam exclusionis, i. e.* by the exclusion of cases marked by symptoms which are

generally acknowledged to point to lesions of the nerve centres.

The most important objective symptoms observed in this group were (in addition to writing difficulty)—

(*a*) Tenderness of one or other of the nerves of the arm.

(*b*) Changed faradic irritability of muscles.

(*c*) Tremor.

The presence of nerve-tenderness is frequently recorded in my previous series, but it was not so systematically looked for as it has been in the cases of the present series.

My observations on cases of writing difficulty, extend over a period of sixteen years, and it must be remembered that in the earlier years I was not alive to the importance and frequency of nerve-tenderness as a symptom. I believe, however, that in all the cases of the present series nerve-tenderness has been searched for.

The discovery of nerve-tenderness requires care and method, and exact comparison of the relative tenderness of the corresponding nerves of either arm. The best method of procedure is, I believe, as follows:

The patient stands facing the observer with the arms bare; he is then requested to *rest* his hands with the palms upwards on the shoulders of the observer; the arms must *really rest* on the shoulders, and be without muscular effort of any kind. To test the tenderness of the median nerves, the observer must cross his hands, and, grasping the patient's elbows, exert pressure with the pulp of either thumb just to the inside of the biceps tendon. The pressure on either side is made simultaneously, and if one median nerve be more tender than the other, this becomes clearly manifest, indeed, in well-marked cases the patient will shriek out or writhe under a pressure on the one side, which causes him no discomfort on the other. I think there is no difficulty in arriving at a just conclusion as to the relative tenderness of the median nerves.

To test the musculo-spiral nerves the observer's hands need not be crossed. He must grasp the lower ends of

the arms, and exert pressure with the thumbs above the outer condyle of the humerus, close to the fold of the supinator longus. The ulnar nerves are best reached just above the olecranon.

As to the pathological significance of nerve-tenderness, I shall have something to say hereafter.

Out of the 66 cases (of the present series) which fall into the neuro-muscular group, there were 13 in which nerve tenderness existed apart from change of muscular irritability, 21 in which change of muscular irritability existed without nerve-tenderness, 32 in which these symptoms concurred.

The neuro-muscular group is thus divisible into three sub-groups.

SUB-GROUP A.—*Nerve-tenderness without change in muscular irritability* (13 cases).

In 12 the right median nerve was tender.

In 1 both medians were tender, in 1 all the nerve trunks were tender, in 1 the musculo-spiral and median were tender, and in 1 the nerve-tenderness was very doubtful.

Seven cases suffered from gout, rheumatism, dyspepsia, or alcoholism.

Four cases had tremor.

Three are noted as being crippled in acts other than writing.

The previous duration of the trouble in this group was relatively not long. In the 9 cases in which this fact is recorded these periods were, eighteen months, thirteen months, twelve months, nine months, six months, "two or three months," "some weeks," "a few weeks," one month.

SUB-GROUP B.—*Change of muscular irritability without nerve-tenderness* (21 cases).

By change in muscular irritability is meant merely a change in the irritability to faradism, tested by a comparison of the effects of equal currents applied to corresponding muscles in either hand or arm.

The hands are placed symmetrically and at absolute rest (usually hanging over the back of a chair). One rheophore is then fixed to the nape of the neck, while the other (a rheophore of small surface) is applied delicately to the muscles which it is wished to test. Having determined the least strength of current which will cause the contraction of a muscle (and I usually begin with the first dorsal interosseous) on the left (and presumably healthy) side, the same current is then applied to the corresponding muscle on the right side, and any difference in the effect is noted. When it is stated that the muscular irritability is depressed or heightened it means that the muscle responds to the current less readily or more readily than the same muscle on the left side.

To detect the differences requires some care but not more care than is required to detect the alterations of tone observed in the eye-muscles in severe cases of diplopia.

Each of these 21 cases had muscles manifesting a decrease of irritability. In 3 there were muscles manifesting an increase of irritability alongside of others which showed a deficiency.

The first right dorsal interosseous (1st R. D. I.) was affected thirteen times.

The abductor and opponens pollicis eight times.

The second right dorsal interosseous six times.

The fourth right dorsal interosseous and flexor longus pollicis were affected twice, the third dorsal and fourth palmar interosseous each once, and the extensors of the thumb and small muscles of the little finger each once.

In 8 cases there was trouble (pain, enlargement, creaking) in one or other of the joints, and in a ninth there was a family history of rheumatic gout.

In 11 cases there was tremor.

Four are noted as being crippled in acts other than writing.

In 18 cases the previous duration was noted, and it will be observed that it is longer than in the previous group in which no change in muscular irritability was detected. Thus we have recorded periods of fifteen years, thirteen years, "many years," "some years," "years," six years, six years, three years, three years, fourteen months, "some months," one year, "a few months," four months, two months, two months, one month.

Sub-Group C.—*Nerve-tenderness and change of muscular irritability combined* (32 cases).

Nerve-tenderness occurred in the median only in 28 cases, the ulnar only in 2 cases, and in the median and musculo-spiral combined in 2 cases.

As regards muscular irritability, decrease only occurred in 20 cases, increase only in 8, and a combination of the two conditions in 4.

As regards the muscles affected we have:

The first right dorsal interosseous, affected nineteen times.

Other interossei seven times.

Abductor and opponens pollicis seven times.

Extensors of thumb four times.

Flexor brevis pollicis three times.

Flexor longus pollicis twice.

All the muscles of the hand three times, and supinator longus, supinator brevis, serratus magnus, and pectoralis major each once.

In 2 cases there was a history of sprain of the wrist

(153, 162), and one (140) had had his right hand smashed in a mangle twelve years previously.

Creaking of the right shoulder occurred in 3 cases, 9 are noted as being dyspeptic, gouty, or alcoholic.

Tremor was noted in 18 cases.

In 11 cases there was disability in acts other than writing.

The previous duration was for the most part considerable, in only 3 cases was it less than a year. In the whole 66 cases of the neuro-muscular group the previous duration was less than a year in 11, or 16·6 per cent.

In sub-group A the previous duration was less than a year in 6, or 46 per cent.

In sub-group B the previous duration was less than a year in 5, or nearly 24 per cent., and

In sub-group C the previous duration was under a year in 3, or 9·4 per cent.

It is dangerous to form conclusions on averages of small numbers, but these figures point in the direction that some time is, as a rule, necessary for the production of change in muscular irritability.

In the whole series of cases in both papers we find that of the muscles whose irritability was found altered:

The interossei were affected seventy-nine times.

Abductor and opponens pollicis twenty-eight times.

Extensors of thumb twenty-three times.

Flexors of thumb thirty-two times.

Muscles of little finger nine.

And other muscles only rarely and exceptionally.

In the present series of 93 cases nerve-tenderness (N. T.) was noted fifty times, and in the subjoined table its distribution in the groups and its association or otherwise with altered muscular irritability (A. M. I.) is shown.

	N. T. only.	A. M. I only.	N. T. + A. M. I.	Neither N. T. nor A. M. I.
Paralytic (13 cases) . . .	4	4	1	4
Degenerative (14 cases) . .	0	3	0	11
Neuro-muscular (66 cases) .	13	21	32	0
Total cases (93) . . .	17	28	33	15

Of the 50 cases in which nerve-tenderness was noted it is to be observed that in all but 8 it occurred in the *median nerve*. I wish to call particular attention to the fact that the *muscles* in which change of irritability has been most frequently noted (the interossei) are supplied by the *ulnar nerve*.

What is the pathological significance of altered muscular irritability?

We have seen it occurring in every group, cerebral, degenerative, and neuro-muscular, so that it has no absolute value for localising the writing trouble in the brain, cord, nerve, or muscle.

It is probable due to some change in the muscle or intra-muscular nerves, and it seems probable also that such change may be primary or secondary to a lesion in brain, cord, or nerve.

This paper is strongly confirmatory of the fact pointed out in the previous paper that depressed muscular irritability in cases of "writers' cramp" is observed more often in the muscles of *pen-prehension* than in those of *pen-movement*, *i. e.* in muscles which are subjected to very prolonged contraction and therefore very liable to get over-fatigued.

It is possible that fatigue in a muscle is produced with undue readiness when the voluntary motor stimulus is impeded at any point. The man with cerebral paresis is very conscious of muscular effort when he tries to write, and the same is true of patients in whom the conducting power of the nerves is interrupted by inflammation, congestion, or pressure.

Case 77 is very instructive on this point. It was that of a boy, aged twelve, in whom a congenital fracture of the clavicle had caused pressure on the brachial plexus. The case was brought before the Clinical Society by Mr. Barker. The boy had peculiar trouble in writing, so that the case had been spoken of as one of writers' cramp. In this case there was very slight but quite obvious wasting (or non-development) of the pectoralis major, and

the muscles of the right arm and hand. The electric irritability was, however, normal in all the muscles except the first right dorsal interosseous, in which the irritability was depressed. The patient was a lad of unusual intelligence, and had worked hard at his writing lessons, which caused him fatigue and pain. It would seem as though the pressure on the plexus had necessitated increased effort, and had predisposed to the fatigue of the muscles of penprehension and the lessening of the electric irritability in at least one of them, and that probably the most important. The pressure on the nerves of the brachial plexus was relieved by operation, and some weeks later I saw the boy writing without difficulty, and I found on examination that the depressed irritability of the first right dorsal interosseous no longer existed. It must be remembered that not only had the pressure been removed but the boy had had a prolonged period of rest.

Occasionally the irritability of one or more muscles has been found exalted. The reason of this is doubtful. Occasionally I have found a muscle *supplied by a tender nerve* manifesting exalted irritability, but this is by no means invariable. I feel sure that the practice of massage increases the irritability of muscles.

It must be remembered that change in the irritability is judged by no fixed standard but only by comparison with the opposite limb, so that we are always liable to the fallacy that a relative change may not really mean an absolute change from the physiological normal. For example, when we find the right first dorsal interosseous more irritable than the left, this fact may really be due to an abnormal depression in the irritability of the left first dorsal interosseous.

It must be remembered also that a muscle which manifests excess of irritability is not necessarily more efficient than the corresponding muscle of the opposite limb. Some authors have spoken of a condition of "irritable weakness," a condition which is probably a fact. Not only have the muscles shown great differences in their

readiness to respond to equal electric currents, but their mode of contraction has often been obviously different to the eye ; the muscle, which is presumably healthy, contracting forcibly and moving its point of insertion vigorously, while the muscle which shows deficiency of irritability contracts in a hesitating, jerky, and unsatisfactory manner.

The feeling of contraction (under electric stimulation) is often very different on the two sides, at least this is what patients say ; but the fact has not been systematically recorded because of the great difficulty of making a satisfactory record of such a fact.

For the same reason the record of cutaneous sensibility has not been systematically made although, to judge from expressions used by patients, change has frequently been present.

What is the pathological significance of nerve-tenderness ? It is recorded fifty times in the present series of 93 cases, viz. forty-five times in the neuro-muscular group, five times in the paralytic group, and not at all in the degenerative group. The conditions under which nerve-tenderness occurs are very varied.

It is oftenest and best observed in neuralgia of the fifth nerve. Whether the neuralgia be definitely due to central change or peripheral irritation or constitutional cause we often find every accessible branch of the nerve more or less tender on pressure, and the tenderness, marked during a paroxysm, is absent or scarcely noticeable in the intervals between the attacks.

In paralysis of the facial from exposure to cold, we usually find the trunk of this purely motor nerve more or less tender at its point of emergence from the stylo-mastoid foramen.

In "sciatica" every accessible branch of the nerve is often tender, and even when the sciatica is due to intra-pelvic irritation we find tenderness of the ultimate branches, notwithstanding that the source of trouble may be nearly a yard distant from some of the tender twigs.

I have noticed great nerve-tenderness in cases of lead palsy and in cases of muscular atrophy, but without any other evidence of neuritis.

In true neuritis of a mixed nerve we find not only nerve-tenderness, but changes in sensibility, neuralgia, causalgia, trophic change, muscle wasting, and often distinct evidence of swelling of the nerve involved.

In chronic rheumatic conditions and in gout the nerve-trunks are often tender, and this without any good evidence that they are irritated by thickenings or bony out-growths.

Nerve-tenderness may certainly be produced by excessive muscular exertion. I have seen such a condition in laundresses, who state that they have strained the arm from wringing clothes, and also in men who have made excessive muscular effort, and notably in one gentleman whose musculo-spiral nerve had become excessively tender from prolonged use of a heavy fishing-rod.

In some of my cases the nerve-tenderness was associated with dyspepsia and alcoholism.

Mr. Barker's case (No. 77) may be again alluded to. In this patient all the nerve-trunks of the arm were very tender before the removal of the pressure on the brachial plexus, but after the successful operation this tenderness quickly disappeared.

From this résumé it appears, therefore, that nerve-tenderness may, in regard to its cause, be

1. Functional (neuralgia).
2. Central.
3. Peripheral.
4. Due to cold.
5. Due to rheumatism.
6. Due to inflammation (neuritis).
7. Due to muscular exertion.
8. Toxic (lead, alcohol, gout).

In my case of impaired writing power the median nerve has most often been found to be tender. Muscular failure is most often evident in the muscles of pen-prehension and

notably in the interossei. When these small muscles of pen-prehension fail, bigger muscles (such as the superficial and deep flexor of the fingers and the long flexor of the thumb) take their place, and I believe that the tenderness of the median nerve is dependent upon the over-use of these (median) muscles.

In the early days of these cases it is possible that one or more of the deep branches of the ulnar nerve may have been tender in association with over-use of ulnar muscles, but this, of course, is only a surmise.

No thickening of any nerve-trunk was detected in these cases.

Trophic lesions were not common. In No. 77 (Mr. Barker's case) the nails of the affected arm grew slowly. No. 78 was troubled by coldness and blueness of the hand. No. 102 had a congenital brown mark on the inner side of the right arm. No. 128 had pitting of the right finger-nails. No. 130 had a crop of moles on the right arm and a lichenous rash on the right forearm. No. 144 had brittleness and furrowing of the right thumb-nail, and No. 149 had throbbing of the thumb after writing.

"Gout," "rheumatism," and creaking and thickening of joints were tolerably common.

Tremor was a very common symptom and is recorded in 87 out of the total of 168 cases. It is a symptom of varied significance and appears to be liable to occur with defects at any part of the motor path.

In some few instances the tremor was of very limited extent. No. 121 had tremor of the thumb only, accompanied by slight wasting of the abductor and opponens pollicis. No. 122 had tremor of the middle finger only. No. 150 had tremor of the far-phalanx of the thumb only, and in this case there was deficient irritability of the extensors of the thumb as well as of the abductor and opponens. No. 160 had tremor of the thumb from a similar cause.

Tremor of limited area may thus point to the fact that

the muscles moving the joints about which the tremor occurs are especially affected.

Obvious spasm was rare. No. 82 had slight athetotic movements, but, as a rule, it was hardly possible to say, from merely watching the act of writing, why the writing failed.

There were some exceptions to this rule, however, and these were instructive.

No 76 suffered from extreme flexion of the far-phalanx of the thumb, associated with paralysis of the extensor of that phalanx.

No. 117 suffered from a collapsing of the phalangeal joint of the thumb and rotation towards the index, associated with weakness of the extensor primi internodii and abductor and opponens.

No. 127 (like 76) suffered from flexion of the far-phalanx of the thumb associated with weakness of the extensor. Nos. 140 and 144 were very similar cases.

In Nos. 129, 131, 139, and 140 there was inability to keep the forefinger on the pen, associated with weakness of the first right dorsal interosseous.

No. 156 did not use his thumb while writing, and in this case there was weakness of the flexor brevis pollicis and tremor of the thumb.

In No. 163 the phalangeal joint of the thumb collapsed, and it was noticed that faradisation of the flexor longus pollicis caused tremor of the thumb.

When writing is made impossible by some decided and recurring spasmodic action, we may expect to find either *deficient* irritability in the muscles which normally antagonise, or *excess* of irritability in the muscles which normally produce such movement.

The examination of the ninety-three cases comprised in the present paper tends to confirm the statements put forward in the previous paper.

The most important change has been the suppression of what in the previous paper was called the group of "true writers' cramp" and its amalgamation with the neuritic

group to form the new neuro-muscular group with its three subdivisions.

This is a distinct gain as constituting a step towards a *pathological* as opposed to a *symptomatic* nomenclature. Impaired writing power or "writers' cramp" is but a symptom, and our object must be to refer every case of impaired writing power to its proximate cause.

More than a quarter of my total cases of impaired writing power have been due apparently to recognised morbid states of the brain and spinal cord, and it must be borne in mind that the act of writing, an act which requires at once so much delicacy and firmness, would certainly afford evidence of slight changes in the nerve-centres, of changes so slight as not to be likely to materially interfere with the performance of coarser and less sustained muscular exercises.

Every case of so-called "writers' cramp" requires a very careful and prolonged examination, for my experience tends to show that hardly any two cases resemble each other in all particulars. By methodical examination we must first exclude all recognised morbid states of the nerve-centres or nerves, joints, ligaments, and muscles. The results of our examination having been to this end negative, we find nothing objectively wrong except altered muscular irritability, nerve-tenderness, or tremor; but it must be observed that I have never failed to find one or more of these conditions present. These conditions must indicate some pathological change, although we may be in great doubt as to the precise nature of this change.

In my previous paper I spoke of the condition as "chronic fatigue," and I see no reason why this name should not be maintained.

Dr. Augustus Waller, in his very able investigations into the process of fatigue, reported in the 'British Medical Journal' for July 17th, 1886, states that a fatigued muscle is (*a*) less excitable, (*b*) shortens less, and (*c*) can do less work. In my clinical investigation I have shown that in cases of impaired writing power certain muscles have been

found manifesting these three conditions, and that the affected muscles are precisely those upon which the prolonged strain of the act has mainly fallen. Dr. Waller has shown that in fatigue induced by over-stimulation of a nerve, at death, and after nerve section, it is at the motor end-plate (the junction between nerve and muscle) that conduction is first interrupted. Dr. Reid, working with Dr. Waller, has shown that when stimulation takes place from the brain, failure of conduction occurs first in the cord where the ganglionic cell forms a junction between the fibre of the pyramidal tract and the fibre of the anterior nerve-root.

Dr. Waller has also found that muscle, the nerves of which have degenerated, is more rapidly exhausted than normal muscle, a statement which finds some clinical confirmation at p. 310 of this paper, where I stated that "it is possible that fatigue in a muscle is produced with undue readiness when the voluntary motor stimulus is impeded at any point." This is because, as is well known, when a block occurs in the motor path, degeneration of the nerve takes place in the parts beyond the obstruction.

Finally, a few words may be added as to the treatment of conditions of impaired writing power.

Clearly to talk of "a cure for writers' cramp" is about as rational as to talk of a cure for lameness. The first step towards treatment is diagnosis.

It seems needless to insist on the necessity of rest, or to enter at all fully into methods of treatment discussed in my previous paper.

It is necessary to state that the great majority of the patients whose cases are recorded in the present paper were seen by me only once or twice, so that where no record is made of the result of treatment it must be inferred that the result is not known.

The results, where known, were as follows:

No. 76 (possibly due to lead) is reported as "improved somewhat after a ten weeks' use of the galvanic current."

No. 77 was completely cured by Mr. Barker, who removed the pressure on the brachial plexus.

Not much improvement was to be expected in Cases 78—97, in which the trouble was presumably central.

No. 98, a rheumatic clerk, æt. 39, with marked tenderness over the cervico-dorsal vertebræ, completely recovered after blistering the tender vertebræ and the administration of saline purgatives with iodide of potassium.

No. 99, who had been told that he was suffering from "writers' cramp," "a disease of the spinal cord," was treated for alcoholic dyspepsia and completely recovered.

No. 101, who had rheumatic finger-joints, was relieved by blistering certain tender points in the hand, and cured, at least for a time, by massage.

No. 107 completely recovered after blistering the tender nerves, undergoing massage, and taking a tonic.

No. 109 completely recovered after giving up alcohol and taking a holiday.

No. 110 completely recovered after a course of saline purgatives and a holiday.

No. 113 was completely cured by wearing a piece of capsicum plaster over his tender median nerve.

No. 114 was completely cured by saline purgatives.

No. 115 (with no objective symptoms) was completely cured by massage.

No. 119, whom I saw in October last, and for whom massage was prescribed, is, I hear, no better. He has wasting of the right dorsal interosseous muscle.

No. 120 (a dyspeptic and rheumatic clerk) was improved by wearing a capsicum plaster over the shoulder, massage, arseniate of iron, and a dinner pill.

Nos. 121, 122, and 123 derived no benefit from massage. (The previous duration of these three cases was six years, thirteen years, and "years.")

No. 126 was much improved by massage.

No. 143 was cured by blistering the tender nerve and massage.

No. 146 had derived no benefit from massage (his

nerves were exquisitely tender when I saw him, and he was gouty).

No. 149 found massage of no use.

No. 158 (a rheumatic secretary) was much improved by a course of hot baths, blistering the tender median nerve, and taking Donovan's solution.

No. 159 was much improved by blistering the median nerve and massage, which caused marked increase in the size of the arm.

No. 162 was much improved by blistering the median nerve and regulating the bowels.

Nos. 163 and 167 were much improved by similar measures.

No. 168 was much improved by blistering the median nerve and using the galvanic current.

As shown in the above record, the methods of treatment which met with a fair measure of success were directed :

1. To the improvement of the general health by diet, regulation of the bowels and the administration of tonics and alteratives (especially arsenic).

2. To applying counter-irritants over painful nerve-trunks wherever they were to be found.

3. To promoting the circulation and nutrition in the affected limb by means of massage, hot baths, and the electric current.

It is probable that the mode of action of massage is similar to that of the galvanic current.

It will be remembered that I have previously recorded a few cases in which the persistent use of galvanism was productive of great benefit. I have now to record several others in which the employment of massage proved very serviceable. At the same time it must be borne in mind that the paper contains the record of six cases in which massage was of no use.

I think I may state conclusively that massage is not useful so long as the nerve-trunks are tender. Indeed, in one or two cases I have seen patients with markedly

tender nerves made worse by massage. The tenderness of the nerves must first be subdued by blistering before massage is commenced.

With regard to massage, I have come to the conclusion that a great mystery is made of it without cause. Doubtless it is an art which requires strength, practice, patience, and common sense. I have employed three or four masseurs and with good results.

No mechanical contrivances for throwing the strain of writing on to a new set of muscles is in any sense curative, although this may prove of service in enabling a certain small amount of writing to be done.

In the employment of type writers only such are to be recommended as are worked by a key-board. Those which involve the permanent grasping of a handle are often very fatiguing.

For details of cases see Appendix.

(For report of the discussion on this paper, see 'Proceedings of the Royal Medical and Chirurgical Society,' New Series, vol. ii. p. 250.)

APPENDIX.

Tabulated details of the 93 cases specially dealt with in the preceding paper.

GROUP 1.–

No.	Name, age, sex, occupation.	Date.	Previous duration.	Loss of function other than writing.	Muscles.					Tremor.	P
					Which affected.	Paralysis.	Spasm	Wasting.	Electric irritability.		
76	H. W., 47, M., Manager	4/3/80	4 or 5 years	Buttoning, carrying a bag	R. & L. ext. sec. internod. poll.	...	...	...	No response	Slight, of both hands	
					R. & L. ext. ossis metacarpi.	}					
					R. & L. primi internod.	}	...	...	Very defective		
77	E. Q., 12, M.	–/8/85	Congenital	All acts, slightly	R. pectoralis major.	...	...	Slight	...	...	
					Muscles of ball of R. thumb.	...	...	Slight	Normal		
					All muscles of R. arm.	...	...	Slight			
					1st R. D. I.	...	...	...	Defective		
78	W. W., 35, M., Butcher	18/3/78	1 year	All acts, both coarse and delicate	?	?	No	No	Normal	R. hand	
79	H. H., 58, M., Cashier	20/4/83	6 months	All delicate acts	...	...	...	...	...	...	
80	F. S. 23, M.	23/9/83	5 years	All fine acts	..	...	...	...	...	R hand	
81	D. L., 12, M.	15/8/82	Congenital	General clumsiness	...	...	...	...	...	...	
82	C. A. P., M., Clerk	16/10/84	A year	All fine acts	...	...	...	...	...	...	
83	C. S. V., 21, M., Student	23/1/84	?	...	...	...	...	...	...	...	
84	— R., 60, F.	8/5/85	...	All actions with R hand	...	...	...	...	...	...	
85	C. J. C., F., 46	22/9/86	10 months	Cannot crochet	...	...	...	...	...	R. hand, slight	
86	R. T., 27, M., Clerk	24/4/84	Since childhood	...	R. abd. and opp. poll.	...	...	...	Defective	R. hand	
					R. flex. brev. poll.	...	...	...	Defective		
					R. abd. and flex. brev. min. digit.	...	...	...	Defective		
					1st and 2nd R. D. I.	...	...	...	Some excess		
87	G. A. B., 21, M., Medical student	23/5/79	Always	...	...	...	...	...	...	...	
88	R. A., 44, M.	31/3/82	2 years	Lifting tea-cup to lips	R. flexor brev. poll.	...	...	...	Defective	...	

Paralytic (13 *cases*).

Nerve-tenderness.	Joint trouble.	Trophic change.	Supposed cause.	
one	...	...	?	Seen with Mr. Pye-Smith, of Sheffield. No lead line, but possibly due to lead poisoning. Holds pen with fore phalanx of thumb flexed at right angles, and the lead pencil is marked with notches made by the R. thumbnail. On faradising the extensors of thumb the metacarpal bone and near phalanx are extended, and the fore phalanx strongly flexed, and at the same time there is quivering of 1st R. D. I. Improved somewhat after 10 weeks' use of galvanic current.
Median. Ulnar. Musculo-spiral	...	Nails of left hand grow faster than those of right	Congenital fracture of clavicle causing pressure on R. brachial plexus	Mr. Barker's case (see 'Clin. Soc. Trans.,' 1886). The boy was operated upon. Within a few months he could write perfectly (the handwriting having previously been tremulous, and accomplished only by effort), the tenderness of nerve-trunks had disappeared, and loss of irritability of R. D. I. had disappeared also.
one	None	R. hand easily gets cold and blue	...	Has not been well since having smallpox seven years ago. Slight contracture (hemiplegic?) of right hand. Phalangeal joint of thumb collapses when he tries to write. Numbness of right radial region of hand. Tenderness over 3rd dorsal vertebra. Suffers from headache and depression. A sexual hypochondriac.
...	...	...	...	Hand constantly tends to flex. Voluntary interosseal movement very defective. Probably hemiplegic. Grasps pen very tightly.
Median, right	...	...	...	Tongue furred. Systolic basic murmur.
...	...	...	...	Clumsiness of right hand. Due to congenital right hemiplegia.
Median	...	...	...	Always clumsy at fine hand-work as a schoolboy. Tongue deviates to right. Slight contracture of right flexor tendons. Slight athetotic movements of right hand when extended. Heart normal. No history of fits. Had scarlet fever eight years ago.
Median	...	...	Infantile hemiplegia? blow on left temple? over-work	Severe illness as a baby. Faulty development of right upper limb. Right shoulder droops. Right clavicle $\frac{1}{2}''$ shorter than left. Right hand smaller than left. Right hand perspires when he writes, and electric sensibility is less in right than left hand, although irritability is equal at the two sides. Three years ago fell from his bicycle, struck his left temporal region, concussed his brain, and suffered from loss of memory for some months. Lately has been working (and writing) hard for an examination.
...	...	...	...	Drags right foot a little. Pain on left side of head. Hemiplegic.
Median, Musculo-spiral	...	...	...	Has been in India. Gouty history. Knee-jerk better marked on left side than right side. Holds pen very tight, and moves whole arm for stroke-making. Hemiplegic?
None	...	...	...	Some wasting, right pectoralis major. Hip disease as a child. Used crutches till he was 13. Now walks with a crutch-handled stick, which has caused bruising and subcuticular hæmorrhage on ulnar side of palm. Has a dread of writing in public. Cannot oppose right thumb to base of little finger without effort.
...	...	...	...	A left-handed man, strong, athletic, over 6 feet high. Measures from mid-sternum to tip of left coracoid process $8\frac{1}{8}''$, to tip of right coracoid process $7\frac{1}{8}''$. Left hand round base of fingers $8\frac{3}{8}''$, right hand $8\frac{1}{8}''$. Formerly a clerk, but gave it up because of writing difficulty. Has special difficulty with up-strokes, and always makes a six by means of two curved down-strokes, ( ). Broke his left arm when a boy, æt. 5. A left-handed bowler and "right-handed bat" at cricket. The 1st and 2nd R. D. I. are more irritable than 1st and 2nd L. D. I.
...	...	...	...	Left-handed, even for billiards. Left forearm measures in circumference $\frac{1}{2}''$ more than right forearm. Often hears a "click" on outer side of right forearm. Slopes his letters the wrong way, \.

GROUP 2.

No.	Name, age, sex, occupation.	Date.	Previous duration.	Loss of function other than writing.	Muscles.					Tremor.	P.
					Which affected.	Paralysis.	Spasm	Wasting.	Electric irritability.		
89	M., 71, M., Colonel	29/12/81	?	...	...	...	...	...	...	Both hands	
90	J. T., 67, M.	30/5/82	?	...	...	...	...	...	...	R. arm, R. leg	
91	G. T., 45, M., Warehouseman	13/9/82	27 years	...	...	...	...	...	...	Both hands	
92	P. W., 55, M., Business manager	24/5/83	"Some years"	Playing billiards	1st and 2nd R. D. I.	...	...	...	Lessened	R. hand	
93	J. B., 46, M., Banker	8/12/85	...	...	...	...	...	...	...	Both hands	
94	P. L., 33, M.	4/7/85	...	...	...	...	...	...	...	...	
95	T. I., 56, M., Manufacturer	...	...	...	...	...	...	...	...	...	
96	Mrs. S., 28, F.	26/4/82	?	Holding anything with finger and thumb	1st R. D. I.	...	...	...	Very deficient	None	
97	W. B., 46, M., Clerk	1882	3 months	All prolonged acts	...	...	...	...	...	Both hands	
98	F. E., 39, M., Clerk	7/6/79	?	...	...	...	...	...	...	None	A wr
99	A. D., 35, M., Banker	20/5/81	...	...	...	...	...	...	...	...	
100	H. H., 33, M., Ne'er-do-weel	24/9/78	Many years	?	...	...	...	...	...	Marked of both hands	
101	Lady L., F., 50	1884	...	All acts with the hand	...	...	...	...	...	...	
102	T. G., 42, M., Clerk	21/10/86	2 years	?	R. abd. and opp. poll.	...	...	...	Defective?	R. hand slight	In (fu ra

Degenerative (14 *cases*).

Nerve-tenderness.	Joint trouble.	Trophic change.	Supposed cause.	
...	...	...	...	Very gradual onset; due to senile tremor.
..	...	...	...	Paralysis agitans, which has been coming on for years. Optic discs small and white.
one	...	...	...	Colour-blind, very emotional, cries easily, temperate, optic discs normal.
one	...	...	...	Tongue foul; gouty; knee-jerk lessened. Occasional diplopia and lightning pains. Case of posterior sclerosis?
one	...	...	Overwork, worry	An old injury of left median nerve has caused wasting of muscles of ball of left thumb which renders comparison of electric irritability of no value. Optic discs are greyish in colour. Doubtful pre-systolic murmur. Father died, æt. 45, of paralysis. Is pale. Grasps pen very tightly, and the pen slips between index and mid-finger.
..	...	...	...	Difficulty in writing following a fall from his horse. When seen he was paraplegic with all the signs of lateral sclerosis affecting the right arm as well as the legs to some extent.
..	...	...	...	Gouty; writing difficult, caused by slight contraction of the palmar fascia.
No	R. 1st metacarpo-phalangeal	...	...	Pain over 1st R. D. I. when she holds anything. The right thumb creaks when flexed. Whole arm aches. The 1st R. D. I. is tender to touch, and a deep-seated thickened band can be felt in it. Ordered blister and mercurial ointment to rub in. Result?
one	...	...	...	Tremor of tongue; excessive knee-jerk on right side. For some months he has noticed that dipping the hands into *cold* water causes an immediate desire to evacuate the bowels. This does not occur with hot water, nor when the feet are dipped into cold or hot water.
one	Hips, shoulders, wrists, fingers	...	...	Rheumatic. Has had lumbago and muscular rheumatism. Tenderness over cervico-dorsal vertebræ. Writes well enough except for the pain. Completely cured by a blister over the tender vertebræ with saline purgatives and iodide of potassium.
...	...	...	Drink	Had been told he was suffering from "writers' cramp and disease of the spinal cord." Was treated for alcoholic dyspepsia and completely recovered.
...	Finger-joints often "crack" after writing	...	Drink	Gouty dyspepsia. Father a drunkard; mother nervous.
...	Finger-joints thickened	...	...	Rheumatic or gouty? Tender points between metacarpal bones. Her condition fluctuated, but she was much relieved by massage.
one	None	Congenital brown mark on inner side of R. arm	Writing?	Right side of chest smaller than left. Knee-jerk absent on both sides. Pale; has lost flesh rapidly. Memory impaired. Optic discs a dead white in centre. Has had much domestic worry. Separated from his wife.

GROUP 3.—*Neuro-muscular.* SUB-GROUP A.—*Neri*

No.	Name, age, sex, occupation.	Date.	Previous duration.	Loss of function other than writing.	Muscles.					Tremor.	Pain
					Which affected.	Paralysis.	Spasm	Wasting.	Electric irritability.		
103	J. V. C., 30, M., Clerk	9/2/86	13 months	?	...	...	...	...	Normal	Both hands	...
104	W. H., 26, M., Clerk	28/4/86	18 months	...	...	...	...	...	?	R. hand	...
105	S. P., F., 41	9/8/86	1 year	?	?	...	...	...	...	...	...
106	Miss W., F.	22/12/81	...	...	...	...	...	...	...	...	...
107	Miss B, F, 24, Artist	7/11/82	...	Delicate drawing and all work with R. hand	...	...	...	...	...	...	R. ar
108	Mrs. H. W., F., 38, Journalist	16/4/83	...	All acts with R. arm	...	...	...	...	...	...	...
109	W. B. O., 38, M, Govt. clerk	9/5/78	?	All acts requiring steadiness	...	...	...	...	...	R. hand	...
110	P. B. A., 33, M., Barrister	17/6/78	A few weeks	...	...	...	...	...	...	...	R. shoul and a afte writi
111	B. A., F., 38	10/4/84	6 months	...	...	...	...	...	...	R. hand	...
112	A. P. S., 32, M., Clerk	5/12/84	2 or 3 months	...	...	...	...	...	...	None	...
113	A. C., Major	-/11/83	Some weeks	...	...	...	...	...	...	...	...
114	M. O., 64, M., M.D.	16/9/85	1 month	...	...	...	...	...	...	...	...
115	W. L., 31, M., Merchant	24/11/83	9 months	...	...	...	...	...	...	...	...

GROUP 3.—*Neuro-muscular.* SUB-GROUP B.—*Alte*

116	J. H. A., 32, M., Solicitor and banker	6/6/83	2 months	...	1st R. D. I.	...	...	...	Defective	...	...
117	McN., 60, M., Captain	4/8/83	14 months	...	R. abductor opp. poll.	...	...	...	Very defective	...	...
					R. ext. primi internod. poll.	...	...	...	Very defective		
118	A. E. C., 27, M., Clerk	28/10/83	1 year	...	1st and 2nd R. D. I.	...	...	...	Very defective	None	...
					R. flex. long poll.	...	...	...	Very defective		
119	J. R., 37, M., Secretary	16/10/86	4 months	...	1st R. D. I.	...	...	Slight	Diminished	None	...
120	T. C. K., 35, M., Clerk	2/9/84	15 years	...	1st R. D. I.	...	...	...	Very defective	Both hands	Shoul Trice Arms

e lerness without altered muscular irritability (13 *cases*).

erve-te erness	Joint trouble.	Trophic change.	Supposed cause.	
R nedian	...	...	Writing	No marked difference of electric irritability. To wear capsicum plaster over tender nerves. To take arsenic.
R nedian	...	...	Writing	Was practising shorthand when difficulty commenced. The hand gets "cramped." The nerve is exquisitely tender. Ordered blister to median.
R nedian	...	...	...	Dyspeptic. Liable to at acks of "dumb hysteria." Father suffers from writing trouble. A sister liable to attacks of hemiplegia, which last 4 or 5 days.
R nedian ?	...	...	...	No objective change anywhere. Says she writes with difficulty, but writing is good. Has suffered from "nervous exhaustion." Has had rheumatism. Some very doubtful tenderness of right median nerve.
F nedian v tender	...	...	...	Some dyspepsia. Blister to tender nerve. Massage. Iron and quinine. Recovery complete. Has a sister who suffers in the same way.
A nerve-tnks of arm	...	...	...	Works hard "in society." Ordered blister, warm douche, &c. Result?
F nedian	...	...	Drink	Drinks too much. A brother died of general paralysis. Optic discs normal. Recovered completely after giving up alcohol and taking a holiday.
I nedian	...	...	Sedentary occupation	Uric acid in urine. Liver enlarged. Recovery complete after a course of saline purgatives and a holiday.
R nedian, R usculo-piral	...	...	...	Leucorrhœa, constipation, dyspepsia. Gouty? Blisters ordered for tender nerve-trunks.
Both edians	...	...	...	The hands get "cramped" after writing a few words. Very thin; liable to spasmodic asthma. Spots of eczema about body. Tongue clean. Urine normal. Family history negative.
I nedian	...	...	...	Military secretary. Capsicum plaster worn over tender nerve completely cured him.
I nedian	R. shoulder	...	...	Foul tongue. Rheumatic. Shoulder-joint creaks. Completely cured by saline purgatives.
?	...	...	...	No objective symptoms. Has been writing a book. Completely cured by massage.

scular irritability without nerve-tenderness (21 *cases*).

None	...	...	Lifting heavy weight and worry	A family tendency to "rheumatic fever."
...	R. phalangeal of thumb	...	...	Sudden onset with swelling of thumb and creaking of the joint. The phalangeal joint of thumb forms a re-entrant angle when writing and rolls towards index finger. Veins of both arms are big, but no evidence of pressure in mediastinum or elsewhere.
None	R. carpus	...	...	Sudden onset. There is a nodular swelling just above the wrist-joint. Patient states that "ten years ago a bone used to come out in the situation of the present swelling." Has worn a strap round the wrist ever since. A strong muscular man.
None	...	...	...	General health perfect. To have hand and arm shampooed.
...	R. shoulder	...	...	Writing painful. Dyspeptic. Always better in cold crisp weather than in warm damp weather. Much improved by wearing a capsicum plaster over shoulder, massage, arseniate of iron and a dinner pill.

No.	Name, age, sex, occupation.	Date.	Previous duration.	Loss of function other than writing.	Muscles.					Tremor.	I
					Which affected.	Paralysis.	Spasm.	Wasting.	Electric irritability.		
121	J. C., 40, Goldsmith	-/10/84	6 years	...	Abductor and opp. poll.	...	...	Yes	Deficient	Thumb	
122	R. C., 36, M., Accountant	27/6/85	13 years	Shaving and "scooping the meat out of an egg"	1st and 2nd R. D. I.	...	...	...	Very defective	R. middle finger	
123	C. G., 28, M., Banker	7/4/84	Years	...	1st R. D. I.	...	...	...	Very defective	R. hand	
124	A. McN., 33, M., Bank cashier	1/12/79	Some months	...	Flex. brev. min. digit.	...	...	...	Very defective	No	
					Right interossei	...	...	...	Some excess		
125	W. W., 31, M., Clerk	20/1/80	...	...	1st R. D. I., R. abd. opp. poll.	...	...	...	Defective	R. hand	
					4th R. D. I.	...	...	...	Defective	Triceps Biceps Pect. major	
					4th R. palmar I.	...	...	...	Very defective		
126	B., 51, M., Merchant	4/9/83	...	Playing violin	1st R. D. I.	...	...	...	Very defective	R. hand	
127	A. R., 32, M., Merchant	20/12/81	4 weeks	...	Ext. sec. internod. poll.	...	...	...	Diminished	R. hand	
128	R. B., 30, M., Shorthand-writer	7/12/86	2 months	?	Muscles of ball of R. thumb	...	...	...	Diminished	Both hands	
129	C. J. T., 31, M., Reporter	20/6/80	A few months	...	1st and 2nd R. D. I.	...	...	...	Defective ?	None	
					R. flex. brev. poll.	...	...	...	Excessive ?		
130	S. C., 35, M., Clerk	17/5/83	3 years	...	R. abduct. and opponens poll.	...	...	...	Very defective	R. hand when extended	
131	E. W., 38, M., Clerk	1879	2 or 3 years	...	1st R. D. I.	...	...	...	Markedly defective	...	
132	F. C. C., 32, M., Clerk	5/5/79	6 years	...	Muscles of ball of R. thumb	...	...	...	Excessive	...	
					R. abd. min. digit.	...	...	...	Defective		
133	T. S., 35, M., Government clerk	1878	3 years	Winding watch	1st and 2nd R. D. I.	No.	No.	No	Very defective	R. hand	
134	H. M., 32, M., Merchant	1878	Many years	?	1st and 2nd R. D. I.	No	No.	No	Defective ?	No	
135	C. F. M., 29, M., Clerk and agent	7/2/82	?	...	R. abductor and opponens	...	...	Yes	Very defective	...	Cer sl al wr wi h
136	W. M., 31, M., Clerk	3/6/82	Some years	All acts soon cause fatigue	R. abductor and opponens poll.	...	...	...	Defective	Both hands	R. and fi
					R. flex. long. poll.	...	...	...	Defective		

erve-lerness.	Joint trouble.	Trophic change.	Supposed cause.	
None	...	...	...	Occasional fibrillary tremor in ball of right thumb. Not improved by massage.
...	...	...	...	Writing, &c., bad. Cannot keep wrist on the desk. Is scored by the actual cautery on either side of the spine which was applied some years ago. Derived no benefit from a seven weeks' course of massage. Ordered to faradise 1st and 2nd R. D. I. systematically.
...	...	...	...	Some tenderness of cervico-dorsal vertebræ. Occasional jerking of the scapula? Ordered massage. Six months later was *in statu quo*.
None	Sprained wrist from a fall six years ago	...	A sprain and writing	Strokes made by moving whole hand. Worse when watched; nervous.
...	...	...	Writing	Has used a very fine pen.
...	...	...	Playing violin	Passionately fond of the violin and has fatigued 1st R. D. I. by holding the bow. Much improved by massage (September 29th, 1883).
...	...	...	...	Far phalanx of thumb flexes forcibly into the palm when he writes. Tremor of right hand during attempts to forcibly extend the right thumb. Faradising right ext. sec. internod. poll. causes great pain; flexion of far phalanx of thumb and tremor. This does not occur on the left side.
None	R. carpus R. thumb	Nail of R. forefinger pitted	...	Right metatarso-phalangeal joint of thumb enlarged, prominence over right carpus between bases of 1st and 2nd metacarpal bones. To paint the joints with iodine, hot sea baths, massage.
...	...	...	Over-writing	Hand weak. In writing "the thumb bends back and forefinger slips up the pen." Holds pen between thumb and mid-finger. Dyspeptic; nervous.
None	...	Crop of moles on R. arm and lichenous rash on R. forearm	Writing against time	—
...	...	...	Writing?	Holds pen between thumb and mid-finger. When writing forefinger becomes extended. Strokes are made by movement of pronation and supination.
...	...	...	...	When writing he supports his hand on the tip of the little finger, which he maintains rigidly abducted.
No	Near phalangeal of R. mid-finger R. shoulder creaks	None	Writing Fall from horse Sunstroke in 1869	Nervous and melancholic. Magazine writer.
No	Phalangeal enlarged	No	?	Never could write well. Forefingers of both hands crooked and everted towards radial border.
None	Carpus	...	...	Pain in cervical spine. Carpal bones prominent on the back of both wrists. Right forefinger slips up the penholder. Writes with the hand and the thumb cannot be abducted.
None	...	...	Excessive writing	In 1876 had to take eight months' rest on account of "nervous prostration." Right median nerve shows lessened irritability to faradism. Temperate in all things. Has pain in back and limbs.

GROUP 3.—*Neuro-muscular.* SUB-GROUP C.—*Nerve-tendern*

No.	Name, age, sex, occupation.	Date.	Previous duration.	Loss of function other than writing.	Muscles.					Tremor.	Pair
					Which affected.	Paralysis.	Spasm	Wasting.	Electric irritability.		
137	D. S., 35, M., Master printer	19/8/81	2 years	...	1st and 2nd R. D. I.	...	...	...	Very defective	R. hand	Bot arm
138	H., 45, M., M.D.	13/10/81	10 years	All prolonged acts	Muscles of R. hand, especially 2nd R. D. I.	...	...	...	Excessive	...	...
139	H. W., 30, M., Architect	1882	2 years	...	R. abductor and opponens poll.	...	...	...	Excessive	R. hand (occasionally when excited)	...
					1st R. D. I.	...	...	...	Diminished		
140	R., 33, M., M.D.	26/10/83	2 years	...	Extensors of thumb	...	...	Yes	Defective	...	...
					1st R. D. I. and R. abductor and opponens poll.	...	...	...	Defective		
					R. flexor long. poll.	...	...	...	Excessive		
141	G. B., 27, M., Clerk	4/1/84	3 years	Billiards, piano	R. serratus magnus	...	...	Yes	...	None	R. should
					R. pectoralis major	...	...	...	Defective		R. axi
											R. for arm
142	J. W. W., 40, F.	20/3/82	14 years	Tires after carrying anything	1st R. D. I.	...	...	...	} Very deficient	...	...
					R. abductor and opponens	...	...	Yes			
143	W. A. B., 34, M., Solicitor	13/5/86	15 months	?	1st R. D. I.	...	...	...	Diminished	...	...
144	J. R. V., Clerk	30/12/85	?	...	R. abductor and opponens	...	...	...	Increased	R. hand	...
					R. ext. 2nd internod.	...	...	...	Decreased		
145	A. M. S., 45, F.	6/26/85	9 years	...	All muscles of R. hand	...	...	...	Excessive	R. hand	Yes
146	Dr. B, 55, M.D.	28/10/85	3 years	?	1st and 2nd R. D. I.	...	...	...	Excessive	No	R. ha
147	C.T., 34, M., Reporter	1883	6 months	...	1st and 2nd R. D. I.	...	...	...	Defective	R. hand	In ar
148	W. H. H., 55, M.	11/11/84	25 years	...	1st R. D. I.	...	...	...	Excessive	R. hand	at nig ...
149	D. C., 23, M., Clerk	14/8/83	...	...	1st R. D. I.	...	...	...	Defective?	Both hands occasionally	...
					R. abductor and opp. poll.	...	...	...	Defective?		
150	R. B., 30, M.	5/10/80	2 years	Weakness when carrying a tray	R. extensors of thumb	...	...	...	Defective	Far phalanx of thumb	In ar
					R. abd. and opp. poll.						
151	H. E. P., 45, M., Indian civilian	27/4/81	8 years	...	Muscles of R. hand	...	...	...	Excess (slight)	R. hand	...
152	J. T., 47, M., Schoolmaster	20/7/81	...	...	R. & L. 1st and 2nd D. I.	...	...	...	Defective?	R. hand	...
					R. & L. 3rd and 4th D. I.	...	...	...	Excessive?		

l change of muscular irritability combined (32 *cases*).

erve-derness.	Joint trouble.	Trophic change.	Supposed cause.	
median	...	...	...	Onset sudden, with a "twitching of the fingers."
median	...	...	...	Worse when mentally agitated. Special difficulty with a small t at the beginning of a word.
median (very arked)	...	...	Excessive work at a competition drawing and domestic trouble	The right forefinger will not keep on the pen. When he writes with left hand the right hand is gradually extended and will not keep on the paper. Some loss of electric sensibility in skin of right hand. Right median nerve very irritable to faradism.
median	...	...	Injury	Never could write fast. When 14 had the hand smashed in a mangle. In writing phalangeal thumb-joint is forcibly flexed, and the forefinger slips up the penholder. Ordered to faradise the wasted extensors of thumb (the ext. secund. internod. is specially defective in irritability).
median	..	...	...	Writing good. Probably a case of neuritis affecting upper part of brachial plexus, and extending to long thoracic, anterior thoracic, and median nerves.
median, . ulnar, musculo-spiral	...	...	Dyspepsia. Came on after confinement	To blister tender nerves. Says the hand is "numb." Dyspeptic.
. ulnar	...	...	Writing	Pain and cramp on ulnar side of hand. Ulnar nerve very tender behind olecranon and above wrist. Cured by blistering the nerve and massage.
. median	...	R. thumb-nail much furrowed and brittle	Writing	Writing is stopped by forcible cramp-like contraction of the far phalanx of the thumb. Ordered to blister right median and employ massage.
median, slight	...	...	...	The application of faradism is painful in right hand but not in left. To blister right median and take arsenic.
median, musculo-spiral	...	...	Gout	Gouty. Had been undergoing a course of massage without benefit (hence the increased irritability of muscles). There is grating felt in the sheath of flexor tendons of middle finger close to head of metacarpal bone.
. median	...	...	Excessive writing	Says that "short hand" tires him more than long hand.
. median	...	...	...	Some defective sensation on right side. Very stout, breath alcoholic. Is troubled with "general pulsation" after meals. Veins over front of thorax are too full, but no other evidence of mediastinal growth or heart disease.
. median	...	...	Dyspepsia	The thumb "throbs" after writing. Tremor worse in hot weather, "when temperature is over 60° F." Dyspeptic. Improved by blister over median nerve. Massage was of no use.
. median	...	...	Over-writing	—
. median	...	...	...	When he writes there is involuntary contraction of mid-finger. Father was hypochondriacal. A brother epileptic.
. ulnar	...	...	Alcohol	Sleeps badly. Indulges freely in alcohol.

No.	Name, age, sex, occupation.	Date.	Previous duration.	Loss of function other than writing.	Muscles.					Tremor.	Pain.
					Which affected	Paralysis.	Spasm	Wasting.	Electric irritability.		
153	F. C. T., 19, M., Med. student	1/2/78	Some years	Dissecting	Flex. brev. poll.	?	No	?	Defective	No	Yes, after using hand
154	E. W., 44, M., Actuary	28/11/78	3 years	...	1st R. D. I. R. flex. brev. min. digit.	... ...	... ...	... ...	Markedly defective Defective	R. hand	...
155	J. G., 35, M., Law writer	2/4/82	...	...	R. flexor brev. poll.	...	...	...	Very defective	Both hands, but most of right	...
156	J. B., 45, M.	3/5/82	2 years	Weakness of thumb for all acts	R. flexor brev. poll.	...	...	...	Defective	R. thumb	...
157	C., 31, M., M.D.	17/4/84	8 or 9 years	...	R. flexor long. poll. R. extensors of thumb	...	...	...	Very defective	...	...
158	R. W. R., 32, M., Secretary	1/8/84	2 years	All work	1st R. D. I.	...	...	...	Very defective	R. hand	...
159	A. K. H., 36, Secretary	1885	Some months	...	1st R. D. I. Flex. brev. min. digit.	...	...	...	Defective Defective	...	...
160	E. J. W., 40, Bank manager	-/8/86	3 months	...	Ball of R. thumb	...	...	...	Slight excess	R. thumb	...
161	L. V., 34, M., Surgeon R.N.	5/6/82	3 years	Playing piano, lifting wineglass	1st R. D. I.	...	...	...	Excessive	...	...
162	T. N., 32, M.	25/7/82	3 years	...	1st R. D. I.	...	...	...	Very defective	R. hand	...
163	E. F. B., 27, M., Bank clerk	29/8/82	18 months	...	1st R. D. I.	...	...	...	Very defective	R. hand	...
164	G. W. B., 46, M., Law clerk	21/9/82	13 years	Grasp of R. hand defective	1st R. D. I.	...	...	...	Very defective	...	...
165	W. L., 35, M., Bank clerk	26/2/83	2 years	...	2nd R. D. I.	...	...	...	Excessive	...	...
166	S. C. R., 55, M., Jeweller's traveller	12/3/83	Some years	Lifting cases, carving, winding watch, and tucking in the ends of paper parcels	R. supinator longus R. supinator brevis Flexor carp. ulnaris	... ...	... ...	... ...	Defective Defective Defective	R. hand	...
167	W. R. Q., 37, M., Accountant	7/4/83	2 months	...	1st R. D. I?	...	...	...	Defective?	...	...
168	C. L., 59, M., U.S.A.	7/4/83	20 years	Both hands soon tire	1st & 2nd R. D. I.	...	...	...	Defective	...	...

Nerve-derness.	Joint trouble.	Trophic change.	Supposed cause.	
median	Wrist	None	Sprain of wrist. Fall from horse when 9 years old	Was not allowed to throw the cricket ball at school because of liability to strain the wrist. Has pain in front of wrist irrespective of work. Thinks the pain is in one of the tendons.
median	...	...	...	Writes with left hand.
median	...	...	15 hours engrossing per diem	Writing very difficult and tremulous.
median very tender	...	...	...	Very thin. Holds pen between fore- and mid-fingers with thumb flexed into palm. There is an abrasion on radial side of metacarpo-phalangeal joint of right forefinger from pressure of the penholder. Is worse when fatigued and overdone. To blister median nerve.
median	...	...	...	Sleepless. Dyspeptic. Began when he was much worried.
median	R. shoulder	...	Writing	Creaking of right shoulder. Urine phosphatic. Much improved in two months by hot baths, Donovan's solution, and a blister to the tender median nerve.
median	...	...	...	In writing the hand is supinated and the right ring finger is flexed. Greatly improved by massage, which caused marked increase in the size of the arm.
median	R. shoulder creaks	...	...	Writing is stopped by an internal rotation of the thumb. A very nervous subject; dyspeptic.
median	...	...	...	Jerking of hand. Served eight years in tropics. Three attacks of intermittent fever. Dyspeptic. Gouty?
median	...	...	Fall from horse on to R. hand?	Urethral stricture. Foul tongue. Voluntary interosseal movement of right hand very defective. Ordered to blister median nerve and regulate the bowels. Was much improved when seen again on October 10th, 1882.
median	...	...	...	When holding pen phalangeal joint of right thumb is re-entrant. Faradisation of flexor long. poll. causes tremor of hand, and of 1st R. D. I. causes more movement of neighbouring muscles than of the muscle faradised. Tongue furred and tremulous. To blister median and treat dyspepsia. Much improved on October 7th, 1882.
median	...	...	...	History of syphilis.
median very tender	...	...	...	Is obliged to raise his elbow before he can write. Ordered to blister right median nerve.
median	...	...	...	The electric irritability of right musculo-spiral and ulnar nerves diminished and of right median increased. Ordered to blister median nerve and to take Donovan's solution.
median	...	...	...	Dyspeptic. Gouty. A "free liver." To blister median and take saline purgatives. Great improvement.
median	...	...	...	Of studious habits. Has suffered from "nervous exhaustion." Ill-defined weakness of legs as well as arms. To blister median nerve and use galvanic current. Great improvement.

A CASE OF INTUSSUSCEPTION

OF THE

UPPER END OF THE RECTUM DUE TO OBSTRUCTION BY A NEW GROWTH.

EXCISION OF THE INTUSSUSCEPTION; SUTURE OF THE REMAINING BOWEL; COMPLETE RECOVERY.

BY

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Received December 14th, 1886—Read May 10th, 1887.

The interest in every form of intestinal obstruction appears to increase with the improvements in our methods of dealing with them. And though the condition met with in the following case is one of great rarity, and not likely often in a lifetime to come under the notice of any one man, the case would be nevertheless of much interest if it only offered one more illustration of what may be undertaken and achieved with the help of strict antiseptic precautions. I believe that without the latter this operation would not have been justifiable or successful; with them it was one which, albeit difficult, offered no complications, and was followed by a rapid and perfect recovery.

L. E. S—, æt. 28, was admitted into University College Hospital on June 7th, 1886, complaining of great constipation and the discharge from the rectum of much offensive purulent matter and clots of blood. All her life she has been subject to constipation, and more so since her marriage five years ago. She has been in the habit of allowing her bowels to be confined for two or three days, or even as long as a week. Otherwise she has been a healthy woman, and is so now to all appearance. Her third and last confinement was four months ago. The labour was difficult and painful, and forceps were necessary. Her family history is good, except that her father died of phthisis.

The trouble in the rectum commenced sixteen months ago with the appearance of small streaks of blood. Eight or ten months ago she became conscious of some foreign body in the bowel which began to prolapse about two or three months later at each stool. At this time blood was very forcibly expelled each time just before the prolapse, which had to be returned before the fæces could be passed.

She was treated with astringent injections at this time, with some benefit, but her medical man discontinued this treatment on account of her pregnancy. There has never been any diarrhœa alternating with constipation. The motions consist of hard lumps varying in size from that of a bantam egg to a marble, more commonly the first. A large, soft, irregular mass can be felt by the finger in the rectum. Under an anæsthetic it was drawn down and made to project from the anus, and being carefully examined was found to consist of an intussusception with a mass of new growth at its apex. This mass nearly surrounded the bowel with nodular projections, and occupied about two inches of its length. The mucous membrane for a short space on the right side of the bowel was not invaded. The infolding of the mucous membrane was felt about three inches from the anus, so that the position of the growth, with the intussusception reduced,

would be about six or seven inches from the anus. The mass could be sufficiently drawn through the anus to expose about an inch of healthy bowel above the edge of the growth. No swelling could be detected in the left iliac fossa, and no infiltration of the intestinal wall beyond the immediate seat of the growth.

Through the kindness of my colleague, Mr. Christopher Heath, this patient was placed under my care, and I removed the whole of the intussuscepted portion of intestine, including the growth.

Operation June 9th: Ether. The patient was laid upon the left side with the thigh well flexed. The rectum was then thoroughly washed out, a quantity of fæculent and purulent matter coming away. Then the intussusception was pulled well out of the anus and washed, within and without, with 5 per cent. carbolic acid solution, which was also made to flush the space between the intussusceptum and intussuscipiens, until the whole area of operation was thoroughly cleansed. Holding the two layers of the intussusceptum well together between the left index finger in the bowel, and the thumb on the latter beyond the growth, I passed a needle on a handle, threaded near the eye, with silk, through its double wall from without towards the lumen. This instrument, however, did not answer, and straight round sewing needles were substituted for it through the rest of the operation. With these a row of stitches was made to encircle the bowel, uniting the two layers of the intussusceptum firmly to one another, well above the new growth. Each thread was passed from the surface into the lumen and then back, and included from one sixth to one quarter of an inch of the circumference of the bowel in its loop. The intervals between the stitches were guarded by a second row similarly introduced about half an inch higher up the bowel. The growth was then cut away with scissors close below the lowest ring of stitches; but before the section was completed a few stitches were introduced above a spot at which the first row appeared to run too

close to the growth, and the section was carried at this spot above the first row. After this section had been completed, and the mass of the intussusceptum removed as a continuous ring, only one small vessel bled. This was tied with silk. The whole cut surface was then dried, and the bowel was returned, a pad of iodoform wool being placed over the rectum.

The mass removed consisted of the growth and about two thirds of an inch of healthy bowel above it. The peritoneal covering was quite healthy, and the growth well limited by the operation. The lobules of the neoplasm presented all the appearances of an adenoid tumour on the surface. There was not, to the naked eye, any infiltration of the wall of the bowel underneath. Each lobule was more or less pedunculated, and on microscopic section showed a fibrous centre with strands radiating in all directions, forming fine papillæ about half an inch long. Each of the latter was formed of delicate connective tissue covered with columnar epithelial cells. In the central pedicle normal tubular glands were seen cut at different angles to their axes. At the base of the growth there was a suspicious irregularity in the arrangement of the cells, islands of which were seen separated from one another by strands of connective tissue of varying thickness; but whether the neoplasm was actually malignant or only adenomatous it is difficult to say from microscopic examination.

There was considerable shock after operation, but this soon yielded to the usual remedies, but no brandy was given. The patient was sick soon after her return to bed, and during the night, but not subsequently. The next morning (10th) the pulse was good, 116, temp. 100.° There was no distension of the abdomen, and only a feeling of being somewhat "bruised" in the left iliac region. She was put on spoon diet with ice to suck. Flatus was passed in the evening.

11th.—Patient better, no abdominal tenderness or any kind of pain. Takes beef-tea. The pad of wool over the

anus was changed; there was very little discharge on it.

12th.—Patient is quite free from any pain; pulse 104, temp. 101·2°.

13th.—The temperature fell to normal to-day, and remained below 100° from this up to the end of convalescence.

14th.—To-day the threads which hung out of the anus were cut short. A large, well-formed motion, without any scybala, was passed, and with no sign of pus or blood; one ligature came away with it. Altogether three motions were passed on this day, the first causing some pain.

17th.—Quite comfortable, and longing for solid food. A large stool, partly solid and partly liquid, was passed for the first time since the 14th.

On the 21st she required Conf. Sennæ, and an enema, and had three motions as a consequence. None of the threads ever appeared since the first. Fish was allowed to-day.

On the 25th a saline aperient was ordered, as there had been a great accumulation of fæces in the colon before the operation, and all had not come away yet. This draught and an enema produced a free evacuation.

On the 29th I made a digital examination of the rectum, and could feel the ridge produced by stitching the cut edges together. There was no trace of stricture. The threads were not felt.

A few days later the patient left for her home, feeling and looking perfectly well. I have since heard that she remains so.

Remarks.—Intussusception of the rectum appears from published statistics to be one of the rarest forms of intestinal invagination. The relative frequency of the various forms is thus given by Lichtenstein, quoted by Treves. Out of 100 cases, 44 were ileo-cæcal, 30 enteric, 18 colic (including rectal), and 8 ileo-colic. If one comes to look

into the matter more closely, it is found that the number of actual rectal intussusceptions among the 18 per cent. set down under the heading colic is very small.

It is also extremely rare to find intussusception of any kind caused by *malignant* growths in the wall of any part of the bowel. Polypi are a common cause of the condition, but non-pedunculated new growths very rarely so. I have been surprised at the small number it is possible to collect from home and foreign sources. Mr. Treves, in his work on 'Intestinal Obstruction,' only speaks of having seen two. One of these, treated by my colleague Prof. Heath, is in our museum at University College, the other in the museum of the Royal College of Surgeons. After some trouble in searching English and Continental literature, I am only able to collect eight authentic cases in addition to the two just referred to, and the case which is the subject of this paper, making eleven in all. Lichtenstein mentions four, but rejects one of them (Cruveilhier's). Another of his references (Quain's case) I have been unable to verify after search through the whole of the 'Transactions of the Pathological Society,' in which he states it is to be found. Of the remaining two, one was seen by himself, the other by Knauss. Then there is a case of adenoid new growth of the sigmoid flexure with intussusception recorded by Mr. Durham in 'Path. Trans.,' vol. xxiii, p. 116, and another of adenoid cancer of the cæcum with intussusception by Dr. Greenhow, Ibid., vol. xviii, p. 114.

The fact that these last two are the only cases of the kind to be found throughout the whole series of the 'Path. Trans.,' further attests the rarity of the conjunction of *non-polypoid* growths with intussusception. The next case I find recorded by Fleiner, in Virchow's 'Archiv.' The mass was excised by abdominal section by Czerny, of Heidelberg, and turned out to be an intussusception due to an "adenoid carcinoma" of the cæcum. Then two cases are recorded in the 'Progres Médical,' No. 8, 1886, by Verneuil, in which the patients, old women, suffered

from intussusception of the rectum due to adenoid epithelioma. Quite recently Kulenkampff has described a similar case in the 'Centralblatt für Chirurgie,' 1886, No. 47, and my own case makes the eleventh. In six of these the sigmoid flexure, or upper part of the rectum, was the seat of the disease, in the other cases the latter was found in or near the cæcum.

Of the six rectal intussusceptions, four were removed through the anus, the two remaining being left untouched. Verneuil appears to have been the first to attempt this. In his first case it was accomplished with the écraseur, but the peritoneal cavity was opened in the operation, and the patient died in a couple of days. In his second case he first performed linear rectotomy to reach the mass, then placed Dupuytren's enterotome above the growth to provoke adhesion, and, at the end of a few days, further secured the walls of the bowel with six sutures before cutting away the intussusception. The patient died two days later of peritonitis, the result of fæcal extravasation, in spite of the stitches. Kulenkampff drew down the tumour in his case, and cut it away, stitching apparently as he went. At the last cut he was aware that he had wounded a knuckle of bowel adherent to the peritoneal surface of the rectum. This, however, he sutured at once through the wound in the rectum. When the latter had been stitched, the stump was only replaced within the anus. The patient recovered from the operation, but with complete obstruction of the bowels, so that on the tenth day the colon was opened in the left groin, and an artificial anus was formed. Later, the rectum became pervious, and the artificial opening appears to have closed, the patient making a good recovery. In my own operation the large number of stitches used at short intervals prevented any gaping of the peritoneal wound, and thus saved the peritoneum from the contamination which took place in Verneuil's second case. I suppose that in all cases operated on in this way there will be always a risk of opening an adherent loop of small intestine, but by careful

palpation the presence of such a loop may be made out, and if a doubt still exists the rectum might be carefully incised above the intussusception before being encircled by stitches.

NOTE.—Writing in July, 1887, this patient's medical man assures me that she remained perfectly well in every way and had given birth to a healthy child just a year after operation.

(For report of the discussion on this paper, see 'Proceedings of the Royal Medical and Chirurgical Society,' New Series, vol. ii, p. 257.)

RESULTS OF AN EXPERIMENTAL INQUIRY

AS TO THE

BEST METHOD OF RESTORING THE CANAL AFTER REMOVING PORTIONS OF THE SMALL INTESTINE.

BY

E. STANMORE BISHOP, F.R.C.S.

(COMMUNICATED BY SIR SPENCER WELLS.)

Received February 5th—Read May 10th, 1887.

I DESIRE, in the first place, to express my satisfaction at being, by the courtesy of Sir Spencer Wells and your kind permission, enabled to bring my experiments and their results forward this evening, and, in the next place, to explain as far as I can the object with which these experiments have been performed and the results so far obtained.

And to clear the way I start with four propositions.

1. Union of the intestine is peculiar in this, that in no other tissue is it so imperative that absolute and perfect apposition should be obtained and maintained until organic union has taken place in every part. Anything less than this may result in a leakage, septic peritonitis, and death.

(Blood-vessels are of course excepted, but I am not aware that any serious attempt has ever been made to suture these with a view of restoring their lumen.)

2. Our ideal object in operations on the intestine is to restore the *status quo ante*, that is, to reproduce a tube of normal calibre, with no contractions at the point of suture, and free on its serous surface from any adhesion to parts around which may restrict its normal movements or act as a band under which another loop may become strangulated.

3. Nothing traceable to the surgeon himself should be left behind when the operation is finished which may render the patient liable to a second interference to obviate or remedy its effects.

4. It is necessary in order to obtain rapid and firm union that the two serous surfaces should be applied face to face, in other words, the coats must be turned inwards. A valvular, ring-like projection must thereby necessarily result, surrounding the entire lumen, and narrowing by so much and for so long as it exists the calibre of the gut.

Premising the above, it follows that the ideal suture for enteroraphy should accurately approximate and hold in position every part of the apposed surfaces of the gut, not only at the time of suture but also until firm organic union has taken place between the parts. It should not tend to contract the intestine, should not produce permanent adhesions, should leave nothing in the bowel but what may easily pass through any part of it, and, lastly, should remove as it comes away the valvular ring as soon as its purpose has been secured.

Can so much be said of any suture at present proposed or in use for this purpose?

Of sutures for the intestine there exist already thirty-four different kinds. But it is not necessary to describe them all in detail, nor should I venture to weary this Society by attempting it. Those members who care to examine them in full I venture to refer to a small paper of mine which appeared in the 'Medical Chronicle,' September,

1885. These sutures fall naturally into groups, which may either be at once set aside as useless or their respective merits and demerits compared.

Inasmuch as the first group does not attempt to comply with the primary necessity, that of approximation of serous surfaces, it will be sufficient merely to mention the names of their proposers, and pass at once to those methods which observe this law. The first group contains those proposed and described by John Bell, Reybard, Le Dran, Larrey, Ritch, Sabatier, Ramdohr, Louis, Chopart and Desault, Benjamin Bell, Amussat, Beclard, Henroz, Assaki and Duplay, Heister, Garengeot, Petit, Richter, Loffler, Palfin, and the *sutura quatuor magistrorum*.

The next group contains but two—the methods of Baudens and Denans. These both require the use of internal metal rings. Not only are these difficult to introduce but—and this is the chief objection to them—the rings are left behind in the interior of the intestine, and are foreign bodies which when loose will make their way towards the anus, and may at any time form the nucleus of an obstruction which will necessitate a fresh operation for its relief; moreover, if these methods are practised upon the small intestine how are these rings to pass the ileo-cæcal valve, and are they not almost certain to set up irritation, even ulceration, at that point?

Of the two remaining groups it may be said that they contain all the methods at present in use, and it is needful, therefore, to devote more attention to them. Fortunately, however, the main points of each class are the same in each modification, and the advantages and disadvantages depend so much upon these main points, that it is not necessary to take each individual method separately.

The first of these, or the third group in order, comprises the methods of Dupuytren, Vesien, Gely, Appolito, and Bouisson. All these agree in the use in various ways of a continuous thread.

The fourth group comprises the methods of Lembert, Czerny, Jobert, Briedenbach, and Gussenbauer. All these

are interrupted stitches, isolated from one another and placed vertically to the plane of the intestinal lumen.

The use of a continuous thread has certain advantages; by it the entire surfaces are firmly brought into apposition at the time of suture, and if applied with mathematical exactness the thread is capable of producing absolutely correct apposition, no point being left unguarded; but it has a capital disadvantage, which, I submit, renders it absolutely untrustworthy. All sutures tend to work their way loose into the lumen of the gut. All parts of the bowel do not throw off the suture at one and the same time. However firmly the suture may be applied, however thoroughly it may bring the apposed surfaces together at the time, it is only firm and efficient as long as every part of it is so; when, therefore, it has become loose by the natural freeing of one part, any other portion of the divided surface not yet firmly and organically united will leak and the whole aim of the suture be defeated. To put it in another way, no patient or surgeon will feel safe until the time required for the loosening of the whole thread has elapsed; until that time there will always be the possibility of sudden leakage, peritonitis, and death.

The advantages of interrupted sutures are these. They are comparatively easily and quickly used; they allow of fairly easy adaptation of one bowel-end to another, and each stitch is independent of its fellows, so that the fall of one does not tend to loosen the grip of its neighbour. But the drawbacks are serious. Placed as they all are, vertically to the plane of the intestine, they command absolutely only that line of intestinal wall within their grip; between each stitch and its fellow is a portion of wall which is free, and these spaces tend, as Travers said, "to form apertures, the diameter of which is as the distance between the stitches." The danger of such possible apertures need scarcely be pointed out. Besides, these stitches can have no influence upon the after behaviour of the valvular ring, which may hereafter act as an obstruction. It may be said that this ring will

probably flatten down in time, but I am not aware of any observations which prove this. Certainly in themselves these sutures have nothing likely to secure such a result.

Therefore the case stands thus: the continuous sutures have disadvantages where the interrupted have advantages, the interrupted drawbacks, which the continuous avoid; neither of them have any after effect upon the valvular ring, nor do I see how they can have.

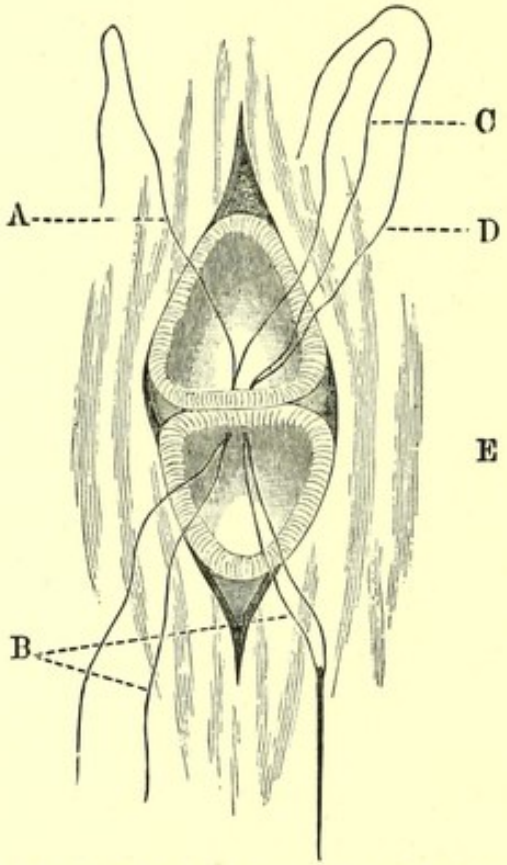
If then a suture were devised, which, whilst interrupted so that each stitch were perfectly independent, should yet obtain all the advantages which a continuous has to offer, in that it accurately approximated every point of the two surfaces to be united, and permitted no space to remain unguarded until firm organic union had taken place, would it not be allowed that a distinct advance had been made towards a perfect suture for this purpose? And if, when this was done, it could be shown that the disposition of the suture resulted in that as it came away it carried with it the whole of the "valvular ring" left behind at the time of operation, surely it would be admitted that this method most thoroughly fulfils all the requirements of the ideal suture described above.

Permit me very shortly to describe its method of application.

The two divided ends of intestine being brought together so that their mesenteric borders lie in an exact plane, a fine round needle, No. 11, armed with a long double silk thread, is passed from the mucous surface of one, through the entire walls of both, to the mucous surface of the other. The needle, and with it the double thread, is drawn through until about five inches of the thread are left on the side from which it has been passed. The needle is then again passed in the reverse direction, at a distance of 2 to 3 mm. from the first puncture, and the threads drawn through until a double loop is left, having also a length of five inches. (Fig. 1.) One of the loops is cut through, the other is drawn up, and knotted with a reef-knot on the side started from. When

the knot is made the ends are cut off close. Thus one loop has been formed, uniting the two bowel walls by their serous surfaces. On the far side of the loop a long single thread is left, passing through the same opening as that passed through by the distal limb of the loop. (This thread is required later in finishing the last loop, and is useful all through the operation as a means whereby the

FIG. 1.

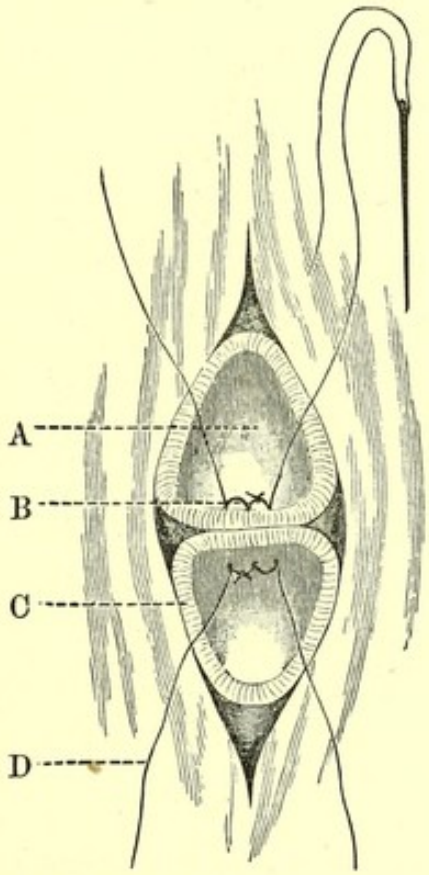


A. First thread left by dividing one of the first two loops.
B. Threads which drawn up and tied will form the first loop.
C. First loop.
D. Left limit of second loop.
E. Abdominal wound.

bowel may be kept in position at the abdominal wound). On the near side of the loop is another thread attached to the needle, and also passing through the same opening as that which holds the near limb of the loop. Reversing the needle and carrying it again through the walls in the same direction as at first another loop is made, which in

its turn is knotted on the opposite side to the first knot, (Fig. 2), and by a repetition of the same acts a series of loops is formed all around the lumen of the intestine, each individual loop surrounding its own moiety of both walls, passing through the same openings as its fellows on either side, but perfectly independent of them, lying transversely to the line of union, and parallel to the plane of the

FIG. 2.



A. Lumen of intestine.
B. Knots of suture inside intestine.
C. Everted mucous membrane.
D. First thread left to unite with last to form last loop.

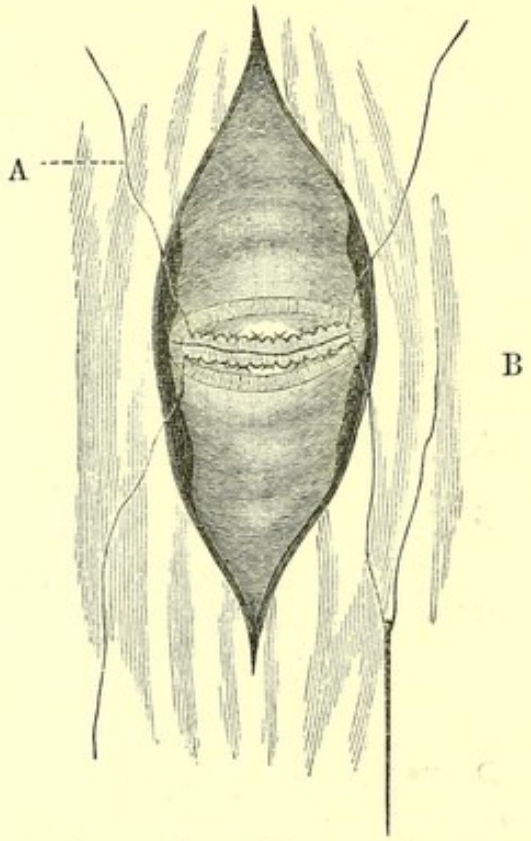
intestine, not so tightly tied as at once to strangulate the tissue enclosed, but certain, as it ulcerates out, to carry with it that portion of the valvular ring. The knots, and nearly the whole of the loops, are, moreover, inside the re-formed canal. (Fig. 3.)

No needle is used with a cutting edge. All the stitches

are made with a small round needle, which pierces, but does not cut the tissues.

The finest silk is used, as it is not desired that the material of which the threads are made should be absorbed, but that it should be cast off into the lumen of the canal; on the other hand, fine catgut is used for uniting the mesentery for the opposite reason.

FIG. 3.



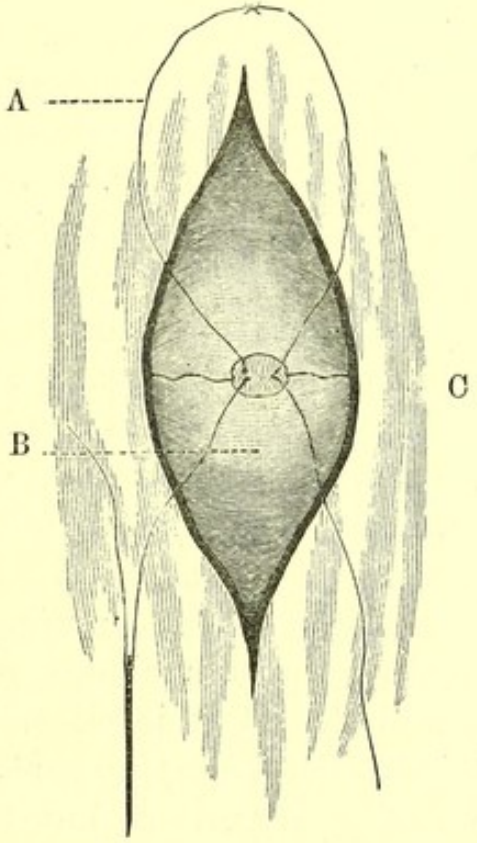
A. First thread left to unite with last to form loop.
B. Abdominal wound with bowel at level of the skin. Half the circumference is united, and the resulting valvular ridge is seen with knots of suture on both sides.

As the stitching goes on the two ends of the bowel become turned in, so that at last the suture lies entirely within the intestine, being separated from the peritoneal cavity by a space equal to the thickness of the wall of the gut.

As each loop is tied it will be seen that if, instead of cutting off only the ends of the thread engaged in making

that knot the needle is cut away entirely, one long thread will be left passing through the last opening made. When the last loop comes to be tied, this is done, and the loop is formed by tying one end of this thread to the corresponding end of the first, which, it will be remembered, was left on beginning the suture (Fig 4). The loop thus formed is drawn up, and knotted securely on the opposite side, thus closing the bowel.

FIG. 4.



A. First thread united with last to form the last loop.
B. Reunited intestine presenting at level of abdominal wall.
C. Abdominal wound.

It will be seen that it is possible to finish the suture entirely in this way, leaving almost no point of the stitch in sight; but in practice it is often more satisfactory, after the last loop is tied, to introduce one Lembert suture, so as more evenly to close the final opening. This stitch is made with gut, as its use is merely temporary, and absorption of it is desired.

The results which, *a priori*, are expected from this method are the following:—The specimens will show whether or not they have been obtained. The advantages of both interrupted and continuous sutures are obtained, and their drawbacks consequently avoided. Leakage, except from the giving way of the knots, which should never happen if the silk is reliable and reef-knots used, is impossible. The serous surfaces are accurately applied to one another. Each suture affects its own moiety of tissue and no other. The sutures and knots being contained in the lumen of the intestine from the first, make their way most easily in the direction shown by Travers, B. Bell, Dupuytren, &c., to be the one always followed by intestinal sutures, namely, towards the interior.

From the same cause, *i.e.* the disposition of the threads and knots, no constantly irritating foreign body is left in contact with the peritoneum covering surrounding parts. Such bodies, even if perfectly aseptic, will develop, and tend to render permanent the results of plastic peritonitis, results such as adhesions between the sutured intestine and neighbouring coils, the wall of the abdomen, and, but far more constantly, with the omentum. Such adhesions cannot fail to be sources of future danger. Some adhesion of the omentum appears almost always to follow the infliction of a wound, but it is claimed for this stitch that, no *constant* irritation being present, such adhesion does not become firmly organised, but tends to become again loosened, and between the second and third month becomes again perfectly free, and in its normal relation to the wounded part.

No after-contraction takes place in the part sutured. The valvular ring necessarily produced in the first instance by any stitch capable of properly uniting the intestine, is by this suture again removed, and a smooth internal surface, covered by villi, is left. This process commences during the first fortnight, and is complete usually about the third month.

To prove and illustrate the foregoing statements, I beg

to submit to your notice the following macro- and microscopical specimens:

You will notice that the valvular ring, which is well marked in specimens A. and B., is less so in all the following specimens; in C. and D. it is breaking down, and the sutures coming away, and this in exact proportion the one to the other. In E. only one suture remains, whilst in F. and H. no trace of suture or valvular ring can be found. The omental adhesion is complete in the first five specimens, yet much less strong in specimen E., whilst in specimen G. it exists only as two small threads. In specimens F. and H. it has ceased to exist altogether.

Specimen G. shows that these changes do not proceed as quickly in some cases as in others, but that they are always of the same kind.

The microscopical specimens show that connective tissue is formed between the serous surfaces, glueing them together; that as the inner ring is cut away the superfluous tissue, like callus around bones, is absorbed, that the muscular coat, as we should expect, is not reformed, but that the ends are united by connective tissue, and that as it contracts it draws the ends of that coat very firmly together, and that the mucous membrane is so closely united that villi and even Lieberkuhn's follicles appear to exist over the internal face of the scar itself. Further details are supplied by the cards accompanying each preparation.

In conclusion, I may say that all these animals were operated upon in Owens College, Manchester, with one exception, and I beg to tender my thanks to Professors Lund, Stirling, and Dreschfeld, of that college, for their very valuable and kindly help and advice; also to Dr. Haslam, of that college, who made all the microscopical sections, and who with Dr. Hunton, of the Ancoats Hospital, assisted me in the experiments themselves. All the animals, except the first two, were killed at definite times, being in first-rate health and condition at the time, and having given us no cause for anxiety after the fourth day. They were all treated in much the same manner, being

allowed absolutely nothing for three days, after which they were fed with Brand's Essence of Beef and water for two days: for the next week with soft milk food, and then were allowed the usual diet of dog biscuits, meat, bones, bread, &c.

The specimens referred to in this paper are now in the Museum of the Royal College of Surgeons.

(For report of the discussion on this paper, see 'Proceedings of the Royal Medical and Chirurgical Society,' New Series, vol. ii, p. 262.)

THE RELATION

OF A

CERTAIN FORM OF HEADACHE TO THE EXCRETION OF URIC ACID.

BY

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Received February 14th—Read May 24th, 1887.

In a paper which appeared in the 'Practitioner'[1] in 1884, on the "Influence of Diet on Headache," I made some observations on the great improvement that occurred in a case of paroxysmal headache on the total abstention from butcher's meat, beer, wine, &c. I considered that the history of the case and its treatment tended to show "that the headache and other phenomena are the result of a poison circulating in the blood, that that poison is a product of the process of digestion of certain foods, especially butcher's meat, &c.," and this opinion I have found no reason to alter, but, on the contrary, my further investigations have greatly strengthened it.

With regard to the exact chemical nature of the poison, however, I was apparently not so fortunate; for I first of

[1] 'Practitioner,' 1884, vol. xxxiii, No. 2.

all suggested that it might be an alkaloid produced, by bacteria in the intestines, out of nitrogenous food taken in excess of the individual powers of digestion; but a subsequent careful investigation of the history and symptoms brought out such a strong relationship to certain modified cases of gout that in a second paper to the 'Practitioner,'[1] I put forward this view of the subject, and at the same time I began to turn my attention to the nitrogenous excreta of the urine, as the question seemed to be almost certainly one of nitrogenous metabolism. It is the result of these urinary investigations that I now wish to bring to your notice.

The headache in question is a very well-defined one, and as a rule easily separable from the very numerous headaches due to other causes; it has been written about by Dr. Liveing,[2] Professors Latham[3] and Trousseau,[4] and has been noticed more or less shortly by other well-known writers, some of whose remarks are quoted in my second paper to the 'Practitioner.' It has been called megrim, sick headache, nervous headache, bilious headache, &c.

That the headache is not necessarily hemicranial is now, I believe, generally admitted, nor are special optical phenomena a necessary part of the picture.

It may affect the whole head, but when at its worst there is generally one spot more affected than the rest, and in the case that forms the subject of this paper this spot is the centre of the occiput. The pain when severe is throbbing, a throb corresponding to each arterial pulsation; it is greatly increased by movement, or by stooping down.

Among concomitant symptoms are mental dulness and inability to fix the attention, extreme and persistent coldness of the extremities, and other vaso-motor phenomena, as slight rigors, and irregular local perspirations; appetite is uncertain but not altogether lost, the bowels are often

[1] Ibid., March, 1886.

[2] 'Megrim and Sick Headache,' Churchill, 1873.

[3] 'On Nervous or Sick Headache,' Cambridge, 1873.

[4] 'Lectures on Clinical Med.,' New Syd. Soc., vol. iv.

irregular, and there may be a feeling of pain, heat, or fulness in the right hypochondrium; the tongue is clean, pulse slow and of high tension and the temperature normal or slightly subnormal, these latter symptoms serving to distinguish it from the frontal headache of constipation. There may be sensory phenomena, as the well-known optical ones, or I have sometimes noticed the hearing altered so that sounds seemed greatly intensified; there is often a family history of headache, or gout, or both (see my second paper to the 'Practitioner').

It is brought on by any relative excess in the meat food, especially in butcher's meat, and I have seen numerous cases both in private and among out-patients where, though meat seemed to be taken in great moderation, the headache has been markedly better when it has been entirely left off; and in the case which forms the subject of these investigations the headaches were reduced by diet treatment from one in a week to one in a month, and gradually down to one in eighteen months, and as I shall show, can be brought back at any time by a change of diet.

Excessive exercise is a predisposing cause of headache, and it must be remembered that it (exercise) has as its result a large excretion of urea and uric acid.

After trying various methods of estimating urea I finally settled down to use the hypobromite process with the precautions and corrections recommended by Dr. Noel Paton;[1] with regard to uric acid, there was not much choice, and Professor Haycraft's method, also recommended by Dr. Paton, was adopted. These two processes I used throughout my experiment and have every reason to be satisfied with the apparent accuracy of the results obtained.

It will save further explanation if I say at once that I am myself a sufferer from this form of headache and its concomitant symptoms, and that these experiments were made on my own urine. They were therefore carried out with a regularity and continuity which it would, I think, have been very difficult to attain to with even the most

[1] 'Practitioner,' March, 1886.

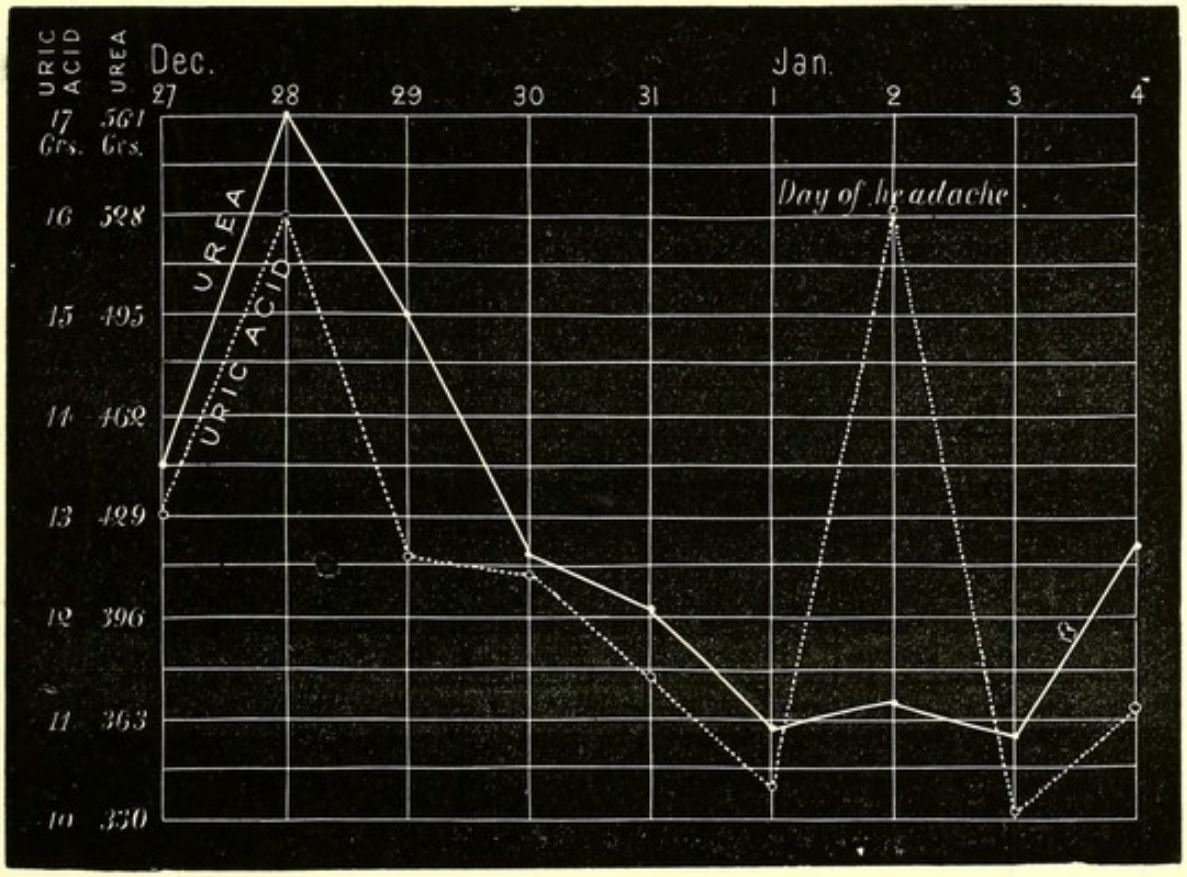
obliging of patients. Thus from the 24th of October last to the beginning of January my urine was collected, and the urea and uric acid, and sometimes other constituents, estimated almost without a break. I also made a daily note as to weather, exercise, food, drugs (if any), and health, especially the presence or absence of headache, or threatenings of it. I also weighed myself from time to time. I always took special care to write down my notes about health and headache before beginning the analysis of the urine for the twenty-four hours in question, so that the result of this could not influence my statements.

On the 2nd of November, I began, for the purpose of this experiment, to take butcher's meat and cheese, each once in the day, having previously (except when away from home) abstained from them for several years; and I continued to take them in very slowly increasing amounts (but only once a day) till the 2nd of January. At first they seemed to have little effect; but gradually the threatenings of headache got more frequent and severe till in the twenty-four hours ending 26th of December, I had a headache lasting more than eight hours, and on the 2nd of January (1887) I had a more severe headache than I had experienced since May, 1885; and that one was I believe due to my breaking through my diet rules with several too good dinners. On the 2nd of January I left off the meat and cheese again, and I have since (now more than four weeks) been practically free from headache, having on one occasion only a slight threatening.

I think, therefore, that there can be no reasonable doubt of the intimate relation of this form of headache to diet, at least in this particular case, and my observations lead me to believe that cases of it that are apparently similarly affected by diet are not by any means rare.

As to the relation of this headache to the excretion of uric acid, my results at first were very equivocal, and though it seemed to me that there was some relation between the two, I thought that the uric acid was, if anything, slightly diminished on the days on which

threatenings of headache occurred. When, however, on the 26th of December I got a decided headache, it occurred to me to separate the urine passed during the headache from that before and after, and I at once got definite results, which were not only exactly repeated in the more severe headache of the 2nd of January, but these latter



results showed clearly the diminished excretion before and after the headache, and so served to explain the equivocal results previously obtained. It would take too much space to give all the results of my urinary analysis, so that I shall confine myself to those from the 25th of December onwards, which include the more important of my positive results, and these I have entered in the accompanying table.

Date.	Twenty-four hours ending.	Amount of urine.	Sp. grav.	Total urea.	Total uric acid.	Relation of uric acid to urea.	Exercise.	Food.	Drugs.	Health and remarks. Weight 126—127 lbs. (without clothes).
1886. Dec. 25	9.50 a.m.	1620 cc.	1022	Grs. 499	Grs. 12·5	1—39	Minus; a very little walking	Ordinary, plus Once meat, Once cheese	None	Slight headache on afternoon of 24th; bowels open.
„ 26	a.m. p.m. hrs. min. 9.50–6.40 = 8 50 on 25th	Not taken	„	186	5·7	1—32	—	—	„	Headache lasting most of the time.
„ 26	p.m. a.m. 6.40–9.50 = 15 10 25th 26th	„	„	318	6·9	1—46	—	—	„	Headache passed off.
				504	12·6					
„ 26	9.50 a.m.	1450 cc.	1030	514	12·7	1—40	Minus	Once meat, Once cheese, Christmas puddings	„	Headache as above. This was a separate analysis of the two parts mixed together.
„ 27	9.50 a.m.	1590 cc.	1025	441	13·1	1—33	Wet day; no exercise	As on 26th	„	Threatening of headache off and on during day. Flatus, and bowels imperfectly open.
„ 28	9.50 a.m.	1820 cc.	1024	561	16·0	1—35	Exercise plus, viz. 2—3 hours' work in the snow	Twice meat, Once cheese	Sodæ Salicyl., 2—3 doses of gr. iij	Headache continued to threaten. Sodæ Sal. greatly improved matters.
„ 29	6.45 p.m.	1540 cc.	1025	499	12·7	1—39	Exercise minus; 5 hours' out-patient's work	Once meat, Once cheese	Sodæ Sal., gr. iij or iv	Well and bowels open. Some rheumatic pains in fingers.
„ 30	6,45 p.m.	1500 cc.	1022	415	12·4	1—33	Exercise minus	„	Sodæ Sal., gr. iij—iv; Calomel,	Slight sore-throat. Threatening of headache afternoon of 30th.

									gr. $\frac{1}{5}$th; Pot. Cit., Sp. Am. Aromat, and Sp. Æth. Nit., 2—3 doses	From morning of 31st. No headache.
1887. Jan.	1	6.45 p.m.	1680 cc.	1020	362	10·4	1—34	Exercise minus; 5—6 hours' out-patient's work	„	Calomel, gr. $\frac{1}{10}$th; Sodæ Sal., gr. iv—v	Sore-throat better. Stiff-neck getting better. Severe headache after 5 p.m. on the 1st.
„	2	p.m. a.m. 6.45–8.30 1st 2nd	990 cc.	1023	213	9·2	1—23	Exercise little or none	No meat or cheese	Sodæ Sal., first in small doses; 8.30–6.45, gr. xx in 2—3 doses	Headache very bad evening of 1st and at night. Not gone in the morning, but better and worse on and off through the day of 2nd.
„	2	a.m. p.m. 8.30–6·45 2nd 2nd	480 cc.	1029	155	6·9	1—22	„	„		*Note.*—Only the above two analyses were made on the 2nd.
			1470 cc.		368	16·1	1—22				
„	3	6.45 p.m.	1650 cc.	1020	356	10·2	1—34	Walking 2½ miles	„	A few grs. of Sodæ Sal.; Quin. Sulph., gr. ij	Headache bad on night of 2nd. Threatening during day, but better on the whole.
„	4	6.45 p.m.	1360 cc.	1024	418	11·2	1—37	3—4 hours' walking and standing	„	Quin. Sulph., gr. ij, evening of 3rd	Well and bright, especially latter part of day. *Note.*—There were three separate analyses on Dec. 26th, and two on Jan. 2nd.

N.B.—Brackets include those figures that refer to parts of days only.

The only results that require notice with regard to my previous analyses are (1) That the uric acid seemed to bear a smaller proportion to the urea on the days when there was partial or slight headache (and see also the proportion 1 to 39 and 1 to 40 on the 25th and 26th of December respectively), and (2) that the average excretion of uric acid, was for a long period about 12 gr. per day. With regard to the accuracy of the processes used, I would draw special attention to the results of the three analyses of the 26th of December, where it will be seen that the total of the two part analyses is in the case of the uric acid almost exactly the same as the result of the separate analysis of the whole, and in the case of the urea there is only a variation of 10 gr. in a total of over 500 gr.

On reference to the table then it will be seen that on the 26th of December, during a period of headache lasting eight hours fifty minutes, 5·7 gr. of uric acid were passed, this being at the rate of ·64 gr. per hour (and I have said above that the average is about 12 gr. per day, or ·5 gr. per hour), and further on the same day during fifteen hours ten minutes free from headache 6·9 gr. were passed or ·46 gr. per hour (*i. e.* below the average) ; on the other hand, the urea varied but little, being for the first period about 22 gr. per hour, and for the second about 21 gr. per hour ; the uric acid variation was little short of $\frac{1}{3}$, while the urea varied less than $\frac{1}{20}$; but I shall come back again to this relation of uric acid to urea. On the 2nd of January we find precisely the same relation, only here the headache lasted the whole twenty four hours, and we have the large uric acid excretion of 16·1 gr. or ·67 gr. per hour, against a total excretion on the previous day, when there was only about one and a half hours of headache, of 10·4 gr., while the urea only varied from 362 gr. on the 1st to 368 gr. on the 2nd ; the uric acid was here increased by more than $\frac{1}{3}$, while the urea only increased $\frac{1}{60}$. Then I purposely divided the urine of the 2nd of January into two parts corresponding as nearly

as I could get them to the two parts of the 26th of December.

In the first part, 6.45 p.m. to 8.30 a.m., viz. thirteen hours forty-five minutes, 9·2 gr. of uric acid were passed, or ·66 gr. per hour; and in the second part, ten hours fifteen minutes, 6·9 gr. or ·67 gr. per hour, so that on this day, when the headache lasted the whole twenty-four hours, the uric acid excretion remained much above normal the whole time.

It appears then that on both these days when there was headache present, the uric acid excretion was above ·6 gr. per hour; but when the headache was absent either before or after the paroxysm, it fell below the average as ·46 gr. on the 26th of December, ·43 gr. on the 1st of January, and ·42 gr. on the 3rd.

There is thus shown to be a very definite relation between the uric acid and the headache, viz. that it (uric acid) is diminished before and increased during the paroxysm, and what is more remarkable, the increase and diminution very nearly balance one another. Thus it may be seen from the table, or the diagram of curves, that the uric acid falls below the urea on the five or six days before the headache by rather more than 4 gr., while on the day of headache uric acid is above urea by about 5 gr.; the difference being due, I suppose, to some retention of uric acid on some of the days previous to the beginning of this diagram. That the uric acid is below the urea on the two days after the headache shows, I conclude, that there is again some retention going on.

In will be seen from the table that on the 28th of December, 16 gr. of uric acid were passed, though there was only a threatening of headache on that day, and in the analysis not given in this table I note, on the 29th of November, that 15·9 gr. were passed, with the note on health, "Quite well, bowels open;" and on the 26th of November 15·2 gr. without any note of a headache being present. What, then, is the explanation of this apparent contradiction? If we had nothing but the absolute amount

of the uric acid to go upon, its relation to the headache might be somewhat obscure; but a glance at column 7 of the table, showing the relation of uric acid to urea, gives us, I think, a clue to the reason why there is a headache on one day with a uric acid excretion of 16·1 gr., and not on another day with 16 gr. excretion.

Throughout the whole of these analyses I have been careful to note the relation of the uric acid to the urea every day, and the result gives me an average in health of about 1 to 33; and it will be seen that on the 28th of December, on which day 16 gr. of uric acid were passed with only a threatening of headache, the relation of uric acid to urea was 1 to 35, while on the 2nd of January, with 16·1 gr. uric acid and a very severe headache, the relation to urea was 1 to 22, a relation which has never been met with before. On the 29th and 26th of November, the days mentioned above, the relations were 1 to 31 and 1 to 33 respectively, which are either normal or close to it. It may be objected that on the 26th of December during the headache the relation was 1 to 32, or nearly normal; but I think that in all probability the urine of this headache was not sufficiently accurately separated from that *excreted* before and after it, so that we have an approximation here to the equivocal results I have before alluded to; and in any case the difference between a relation of 1 to 32 during, and 1 to 46 after the headache, is sufficiently well marked.

On the days I have mentioned above, where there was excessive excretion of both urea and uric acid in something like the normal relation to each other, this excess was the direct result of more than ordinarily severe exercise, a result which is perfectly in accord with those obtained by North[1] in his experiments "On the Influence of Bodily Labour on the Discharge of Nitrogen;" and as long as the relationship of the uric acid to the urea shows no great fluctuations there is no headache; but, as I have previously pointed out in my second paper to the 'Practitioner,'[2]

[1] 'Proc. Roy. Soc.,' 1885.

[2] 'Practitioner,' March, 1886, p. 182.

over-fatigue appears to precipitate attacks both of gout and megrim, and there is no doubt that during these large excretions of urea and uric acid, headache is not far off; and if from any cause the uric acid fluctuates considerably in its relation to the urea, headache will be present during its excess and absent during its diminution (especially the diminution that follows the excess), and the headache will vary in intensity directly with the excess of uric acid over the urea.

We must now inquire into the cause or causes which underlie this fluctuation in the excretion of uric acid. The cause, whatever it is, affects the uric acid, and not the urea, which is steadily excreted at about the same rate as if nothing had happened. There is probably no excess in the formation of uric acid, but merely an alteration in the time of its excretion.

What cause can we think of that affects the excretion of uric acid and not that of urea? It can hardly be due to anything wrong with the kidneys, for that would probably affect both urea and uric acid; besides the cause here is very transient in its effects, and therefore probably not organic. Moreover, we have proof that urates may accumulate while the kidneys are quite normal, for Ebstein[1] has called attention to the fact that extensive arthritis uratica may be found in the post-mortem room along with normal kidneys; and on the whole it appears to me that the best explanation that has yet been given, and the one which appears to best fulfil the above requirements is that of diminished alkalescence of the blood, which is no new theory, but in one form or another has existed for a long time. Thus Sir W. Roberts mentions it in some previous editions of his work, and in the last edition (the fourth) he says,[2] "It may be regarded as probable that the defective power of the kidneys to eliminate uric acid in gout arises from a diminished alkalescence of the blood;" and I think also that the researches of Dr. A. Auer-

[1] 'Die Natur der Gicht,' p. 18.

[2] 'On Urinary and Renal Diseases,' ed. iv, p. 73.

bach,[1] "Ueber die Saürewirkung der Fleischnahrung," are very much to the point here, as he shows that vegetable and animal feeders differ considerably in their power of preserving the alkalinity of their blood against acids formed in their bodies, or introduced from without; and that even where, as in animal feeders and man, this power is greatest, it may be gradually diminished and finally overcome by continued excess of acids. If we may suppose then that the gouty somewhat resemble vegetable feeders, in having less than the normal power of forming ammonia to resist acids, and prevent their taking alkali from the blood, then there will eventually come a time when, on flesh diet and beer, the alkalinity of their blood and tissue fluids will be so far overcome that urates will be far less easily soluble in them than in the normal condition. I think that we have here, in a nutshell, a key to a large part of the pathology of gout, explaining at one glance the great benefit noticed to follow on abstention from meat and beer, and the value of alkalies in treatment. The same connection has been noticed, and the same explanation no doubt applies to the influence of diet and drugs on this form of headache; and I would specially draw attention here to the action of beer, for the relation between fourpenny ale and gouty deposits is a matter of every-day observation. Now, beer and ale are strongly acid in reaction, and I found in a pint bottle of Ind, Coope's pale ale, estimated by the same method as the acidity of the urine, the equivalent of about 14 gr. of oxalic acid. Dr. Garrod[2] says that the average acidity of the day's urine is equal to about 30 gr. of oxalic acid, so that a pint of this beer represents a very considerable dose of acid, and beer has often appeared to me to precipitate a headache. A pint of fourpenny ale, estimated in the same way, yielded the equivalent of 18 gr. oxalic acid, and a pint of stout 25 gr.; so that it appears that the cheaper beers are even more strongly acid. Mr. Wynter Blyth says that the chief acid in beer is acetic.

[1] 'Virchow's Archiv,' 98, p. 512.

[2] 'Brit. Med. Journ.,' i, 1883, p. 705.

I would suppose then that in the case of this headache, a considerable excretion of urea and uric acid is going forward, as on the 25th of December (see table); suddenly from some cause, either internal formation (dyspepsia and acid fermentation, &c.) or unwary introduction from without, a dose of acid is brought in, and for a time diminishes the alkalescence of the blood, and causes retention of uric acid. It will be seen by looking at the table that though 12·5 gr. of uric acid were passed on the 25th it was diminished with reference to the urea; then comes the headache, and with it, on the 26th (first part), a plus excretion of uric acid having its source in the previous retention.

With regard to the theory of diminished alkalinity of the blood, and the way in which it enables us to explain the effect of the diet treatment of this headache, I will quote what Sir W. Roberts[1] says of the "alkaline tide:"—"If as is believed, the normal alkalescence of the blood is due to the preponderance of alkaline bases in all our ordinary articles of food, a meal is *pro tanto* a dose of alkali, and must necessarily for a time add to the alkalescence of the blood." If an ordinary meal is a dose of alkali, a somewhat vegetarian meal, from which butcher's meat and beer are absent, must surely be a large dose of alkali; and here we have ready to hand a fairly good explanation of the effects of somewhat vegetarian diet in this form of headache, for there can be little doubt that it acts by keeping up the alkalescence of the blood, and preventing retention of uric acid from occurring.

The relation I have now shown to exist between the excretion of uric acid and this headache has been to some extent anticipated by those who have noticed the intimate relation of this headache to gout, and in my paper in the 'Practitioner' of March, 1886 (page 181), thinking over these relations, and referring to Prof. Latham's 'Lectures on Headache' I ask, "May not excess of uric acid in the blood cause such vaso-motor irritation, the excess being

[1] 'On Urinary and Renal Diseases,' ed. iv, p. 56.

only occasional and existing for a short time, so long as the kidneys remain undegenerated"? I should not now endorse the latter part of this, as I think we have at hand a better explanation of retention than any degeneration of the kidneys could afford; though no doubt where the kidneys eventually undergo degeneration this does not improve matters. And Prof. Latham,[1] in his "Lectures on the Pathology of Rheumatism, Gout, and Diabetes," kindly notices my question quoted above, and in reply to it says, "Certainly; and in persons whose sympathetic ganglia have this sensitive constitution the causes which lead to the formation of uric acid will develop an attack."

In conclusion, I think that this relation between the headache and the excretion of uric acid (viz. that there is diminished excretion of uric acid before and increased excretion during the headache; but that taking the two periods together there is no absolute increase in the excretion of uric acid, or alteration in its normal relation to the urea) will be found to hold good for all cases of headache which are influenced by diet in the way I have described; and further that the fluctuations in excretion here noticed may help us to clear up several important points in the pathology of gout and other diseases connected with uric acid; more especially by showing the way in which temporary retention of uric acid may be brought about, and in strengthening the hypothesis that in gout there may be more or less permanent diminution of the alkalescence of the blood, resulting in a chronic or constantly recurring retention of uric acid.

I must express my regret that I can only bring forward the results in a single case; but these results are so definite and complete in themselves that I think I have good ground for the deductions I have made from them; while the difficulty in obtaining anything like the same facilities for examining the urine of other people is obvious; and though several friends and fellow-sufferers have kindly promised me samples of urine collected with

[1] 'Brit. Med. Journ.,' i, 1886, p. 629, *et seq.*

the necessary care, I may still have to wait some time before I can get results fairly comparable to those obtained in my own case.

(For report of the discussion on this paper, see 'Proceedings o the Royal Medical and Chirurgical Society,' New Series, vol. ii p. 269.)

THREE CASES

OF

ALCOHOLIC PARALYSIS

(MULTIPLE NEURITIS).

BY

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It is only comparatively recently that paralyses, formerly thought to be due to a central lesion, the proof of which could not be found, owing, it was supposed, to defective methods of pathological investigation, have been traced to their true source in the peripheral nervous system. Perhaps the most important factor, in this country at least, in the production of this form of disease, is the abuse of alcohol, and it is to the class of cases which own this ætiology that those belong which form the subject of the following paper. All three were admitted into the Middlesex Hospital during last year (1886), and I may say, to save repetition, that all were married females.

My first case, E. F—, æt. 28, came under observation on January 26th. There was nothing suggestive in her

family history, but she was stated by her former medical attendant to be a woman of loose morals and a chronic drunkard. She had had four children, three of whom were stated to have been stillborn; and she had suffered from two previous attacks of the same nature, apparently, as the present, occurring after confinements, and lasting for from five to six weeks each.

History of present illness.—She has complained of more or less weakness in the knees for nearly a year, the weakness being noticed particularly on going upstairs. In August and September of 1885 she had an attack of what was called "congestion of the liver," and complained then of being very weak. About the beginning of November she experienced some difficulty in walking, and this has gradually increased. About the end of the same month she began to complain of numbness in the fingers, and was said to have no feeling in the forearms when pinched. Soon after this double wrist-drop was noticed. Gradual wasting, most marked in the arms and legs, has been observed during the last three months, and for about four weeks she has been wandering in her mind.

Present condition.—Pulse 108, small, regular, compressible; resp. 24, laboured; temp. 102·4°. The tongue is red at tip and edges, dry and tremulous, coated on the dorsum with a thick, brownish-white fur; the skin is hot, dry, and harsh. She is much emaciated, has an apathetic expression, and lies helplessly almost on her back in bed, with the legs flexed, and resting on their right side. The face is pale, except on the cheeks, where there is an irregularly-shaped pink flush. The pupils are moderately dilated and equal, acting to light and accommodation. The lips are dry, teeth decayed, gums congested, and there is a dark red line on them close to the teeth. The voice is feeble and the speech rather thick. She has an inefficient paroxysmal cough unaccompanied by expectoration. The thorax is large and rounded; the respiratory movements are chiefly upper costal, and are increased in amount as regards elevation but not as regards expansion. During inspira-

tion there is recession of the epigastrium. Over the front of the chest the percussion note is somewhat increased in resonance ; breath-sounds are harsh, expiration prolonged, and abundant large and small râles are heard in the mammary and infraclavicular regions on both sides, chiefly during inspiration. Behind the percussion note is clear, but numerous dry and moist sounds are audible. The heart's maximum impulse is found in the third left interspace an inch within the nipple line ; it is feeble but well defined. The area of dulness extends from the upper border of the third cartilage to a point in the fourth left interspace an inch within the nipple. At both apex and base the sounds are shorter than usual, but there are no murmurs audible.

The abdomen is soft and flaccid, and marked by lineæ atrophicæ. It recedes during inspiration and is bulged during expiration. The liver reaches to about an inch below the costal margin in the nipple line. The area of splenic dulness is normal.

Patient has the delusion that there are several children in the bed with her ; she cries readily, as when she is being examined ; rambles in her talk and is forgetful. Muscular power in both arms is feeble, the muscles of both arm and forearm are wasted, and there is double wrist-drop, the hands hanging like flails. Extension causes pain and there is no power of supination. Superficial and deep reflexes are absent, but sensation is perfect everywhere. The right forearm at its thickest part measures $7\frac{3}{8}$ inches and the left 7. The interossei of the hands and the muscles of the ball of the thumb are greatly wasted. Round the middle of the biceps the right arm measures $7\frac{1}{4}''$, the left $8\frac{1}{8}''$. The legs are also much wasted, but sensation is perfect, and there is no œdema. She can draw the legs up and move them slightly, but there is no movement of the feet, which are " dropped " like the hands. There is no patellar reflex, no plantar reflex, and no ankle clonus, the attempt to produce which seems to cause considerable pain. There is also pain on passive movement

generally, particularly on movements of extension. She has no difficulty in swallowing and has complete control over bladder and rectum. On ophthalmoscopic examination the fundus of each eye appears quite normal. Examination of the throat shows nothing abnormal, and no nodes are found on the bones. There is no facial or ocular paralysis, and the facial muscles are not wasted although those of the neck are.

The urine is opaque and yellow in colour, acid, having a specific gravity of 1028, deposits an abundant sediment of lithates, and is free from albumen and sugar.

Electrical examination of the muscles of leg and arm by Dr. Pasteur, the Medical Registrar, to whom and to Dr. Pringle I am indebted for all the electrical investigations, gave the following results:

Left forearm.—No contraction front and back to *faradic* current.

Galvanism:

Supinator longus	A.C.C. (μ 7)	= C.C.C.
Ext. carpi ulnaris	A.C.C. (μ 5·5)	= C.C.C.
Ext. carpi radialis	A.C.C. (μ 6)	> C.C.C.

The response was immediate and the contraction voluminous and slow.

Left leg.—No contraction to *faradic* current of the following muscles: Tibialis anticus, extensor communis, peroneus longus, peroneus brevis, extensor brevis, gastrocnemius.

Galvanism:

Tibialis anticus	A.C.C. (μ 5) > C.C.C.
Extensor communis	A.C.C. (μ 5) > C.C.C.
Peroneus longus	A.C.C. (μ 8) = C.C.C. (mere flicks).
Peroneus brevis	A.C.C. (μ 8) = C.C.C. (decided).
Extensor brevis	No contraction.
Gastrocnemius	No contraction.

No contraction of intercostals is obtained either with faradism or galvanism.

No change in her condition was recorded until the evening of January 31st, when she had an attack of

syncope, for which a small quantity of brandy was given her. During the night she slept well and in the morning had rallied somewhat. The grasping power of the hands, however, was very feeble, and she passed her motions involuntarily.

On Feb. 9th it was noted that the heart's first sound was murmur-like and rough. Her general appearance was improved although the cough was still troublesome and the breathing laboured and rapid.

Next day (Feb. 10th) she was much worse; the breathing was still rapid (32) and shallow; she had a wild appearance with flushed cheeks, glistening eyes, lips dry, and covered with sordes; the tongue also was dry and fissured and coated with a thick yellow fur; pulse 126, small, feeble, and irregular. Over the chest generally mucous râles were audible both with inspiration and expiration.

During the succeeding night she was restless and sleepless, and at 3.45 a.m. she died rather suddenly from failure of the respiration.

The temperature was febrile throughout, being rarely below 100° F. and often as high as 103° F.; the respirations were double the normal rate, and the pulse averaged about 120.

At the post-mortem examination (made eight hours after death) the only gross pathological appearances were found in the liver and lungs. The former was large and fatty throughout. As to the lungs, the left was adherent to the chest wall; each had cavities at the apex surrounded by areas of caseation which were undergoing rapid softening. The rest of the upper lobes presented small caseous nodules of recent tubercular disease, and in the lower lobes there were recent tubercles extending down to the base. The middle lobe of the right lung also showed recent infiltration and a small cavity.

In the brain and its membranes there were no obvious changes. The membranes of the cord were normal. The cord itself was firm throughout. On section the grey

matter was so inconspicuous as to be scarcely distinguishable from the white; it appeared to be generally shrunken. It may be noted in passing that this appearance has been reported by Dr. Hadden in one of his cases.[1]

Microscopical examination.—The cord together with portions of the left median and musculo-spiral and of the right anterior crural and plantar nerves and extensor carpi radialis longior muscle were hardened in a 2 per cent. solution of bichromate of ammonium, and after being placed in spirit for some months were cut with a freezing microtome and stained with various reagents. The results as regards nerves and muscle are seen in the preparations now shown to the Society (see Plates VII and VIII).

The cord, a section of which is also shown, appears to be normal in all its divisions—cervical, dorsal, and lumbar, excepting, perhaps, that to the naked eye the grey matter does present in certain sections something of the shrunken look which was referred to in the report of the post-mortem examination. Under the microscope, however, there is nothing to distinguish it from a cord free from all suspicion of disease, in particular the large cells in the anterior horns appear everywhere healthy.

Right plantar nerve.—In transverse sections the perineurium is seen to be thickened, and there is an increase in the connective-tissue nuclei. The walls of the vessels are also thickened and infiltrated by numbers of round-cells (leucocytes). The nerve-bundles show an increase in the nuclei of the sheaths, and there is a great excess of fine fibrillated connective tissue in the endoneurium (fig. 1).

Under a high power these appearances are brought out with greater distinctness; the perineurium is infiltrated with round-cells in parts, some being also scattered throughout the endoneurium. There is scarcely a sound nerve-fibre to be seen, the axis-cylinders having for the most part disappeared, and the medullary substances

[1] 'Trans. Path. Soc.,' vol. xxxvi, p. 53.

being granular where the fibres themselves have not become altogether indistinguishable. A few axis-cylinders, however, still remain, some of which are much swollen (fig. 2).

In longitudinal sections, and in specimens where the nerve has been teased out merely in glycerine, the granular degeneration is well seen.

The myelin is segmented and the tubules irregular in their diameter, being empty and fined away to a thread-like prolongation where the myelin has disappeared, and bulged where it has accumulated in degenerate masses (fig. 4).

Right anterior crural nerve.—In this nerve similar conditions exist but the degenerate changes are less extreme. Many apparently sound fibres showing clearly the axis-cylinders occur in the transverse section; and in the longitudinal the segmentation and granular condition of the myelin are less marked; the perineurium is also less thickened.

Left median nerve.—In transverse section increase in the connective tissue of the endoneurium is well seen, and there are many atrophied nerve-fibres. Some of the fibres are rather swollen and granular showing no axis-cylinder. A considerable proportion, however, appear quite normal. The connective-tissue nuclei and leucocytes are abundant.

Left musculo-spiral nerve.—The same description applies to this as to the median, except that there appears in transverse section to be a larger relative number of sound fibres. In the longitudinal section, however, most are degenerated at some point, and the segmentation and aggregation in masses of the myelin are conspicuous.

Right extensor carpi radialis longior.—The section of this muscle shows an enormous increase in the nuclei of the connective tissue and of the sarcolemma, with numerous leucocytes. The muscle-fibres are diminished in number and size, and the bundles are widely separated; here and there the fibres are almost completely hidden by the masses of nuclei (fig. 5).

Areas of fat-cells occur in places between the muscular bundles, in which are embedded vessels with thickened walls ; and in one of the sections are seen two fine nerve-twigs cut across, which present the same degenerative appearances, with thickened perineurium and nuclear proliferation, as are seen in the larger nerves.

Spinal nerve-roots.—These are sound in all regions of the cord.

My second case, M. J. P—, was brought into the hospital on November 19th, 1886, unable to walk or even stand. Her age was stated to be 28, although she looked older. She had had three children, one of them stillborn ; had always previously enjoyed fair health, and had been a teetotaller until four years ago. Since then she had been in the habit of drinking heavily, gin being her favourite beverage. Latterly she had been almost continuously intoxicated. There was nothing of importance in the family history.

History of present illness.—Her illness was stated by her husband to have come on eight weeks before admission. She then began to feel weak, and to suffer from loss of appetite, losing power in her legs, and staggering in walking. A fortnight later, failure of power in the hands and wrist-drop were observed. She had also wandered in her talk and slept badly.

Present state.—On admission she is described as a corpulent woman with florid complexion, tremulous, and coated tongue and offensive breath, complaining of loss of power in legs and hands, with numbness in the legs ; of pains also in the limbs especially on movement. She lies in bed on the right side with the legs flexed, and is unable to turn or to extend the legs without assistance.

The examination of chest and abdomen shows nothing abnormal except that the heart's sounds are very feeble, and there is some enlargement of the liver. Her voice is feeble, her speech thick, and she has delusions. There is no facial or ocular paralysis and the pupils are normal. When

she lies on her back the arms are folded across the chest, and there is well-marked wrist-drop on both sides with some wasting of the muscles of forearm and hand. The fingers are flexed, and she cannot fully open the hand; there is no power of extension of the wrist, but she can move the arms so as to cross them over the chest or put them up to her face. Any manipulation of the arms causes pain. The grasp of the hands is exceedingly feeble. The legs below the knee are wasted and the muscles flabby. She cries out with pain when the muscles or nerve-trunks are pressed upon. She is unable to move either leg to any extent, and all passive movements cause pain. The feet are "dropped;" there is no œdema. Sensation of the skin of feet and legs is much impaired, and there is neither plantar nor patellar reflex, nor ankle clonus. The feet are cold. She has no difficulty in swallowing and no loss of power in the sphincters, or appearance of bedsore. The urine is acid, sp. gr. 1015, free from albumen and sugar.

A note made on November 23rd states that she is in much the same condition as on admission physically, but that her mental state is more confused. She says she wishes to go home, and that she is quite able to get up and walk. She is restless and sleepless at night and very noisy. She requires to be fed, and has occasionally slight difficulty in swallowing. Two days later there were sordes on the lips and teeth. On the same day it is also noted that there are frequent involuntary movements and twitchings. The chief respiratory movements are thoracic, but even these are limited. The movements of the diaphragm are reversed, the epigastrium bulging during expiration and receding during inspiration. She has a short cough, the tongue is dry and coated with a brown fur, and she now passes her urine and motions in bed. She is still restless and noisy at night, crying out for beer. The electrical examination was made on this and the next day, the results being as follow:

Left arm.—Triceps extensor reacts to moderate, and

supinator longus to strong, *faradic* currents. The muscles in front of the forearm react to weaker currents. There is no reaction to strong *faradic* currents in the extensor communis digitorum or in the extensor minimi digiti.

Galvanism :

Extensor communis digitorum A.C.C. (μ 3) > C.C.C.
Extensor minimi digiti C.C.C. (μ 2) > A.C.C.

Legs.—Faradism.—Strong secondary currents produce no contraction of the gastrocnemius, peronei, tibiales, or extensors of foot, either over the muscles or from motor points.

Galvanism.—With a current of five milliamperes there is both anodal and kathodal contraction of the extensor longus digitorum of right leg ; the contraction is slow and wave-like (A.C.C. = C.C.C.), and other factors of modified degenerative reaction are present ; the amount of current required for contraction being subnormal, and contraction produced not being that of health. No other group of muscles was examined on account of the impossibility of moving patient's legs without causing pain, but these reactions probably give a correct index of the condition of other paralysed muscles.

After this date she got rapidly worse, the wrist-drop and foot-drop became more marked, and there was much tenderness in the calves of the legs ; her incoherence continued and she spoke with weak voice and a snuffling tone ; the tongue was dry and protruded with difficulty. The pulse became more rapid and feeble, the extremities cold, and the breathing shallower. She had occasional twitchings of the arms the day before her death, which took place on November 29th, at 3.45 a.m.

The temperature was only on three occasions over 100°, and was below that figure on the evening before her death ; it was generally about normal. The pulse, 72 at first, increased to 160 during the ten days she was in the hospital, and her respirations rose from 20 to 32.

At the post-mortem examination the lungs were found to be congested and extremely œdematous, the liver very

fatty and of a pale fawn colour; the kidneys large, with swollen cortex and showing fatty striæ.

In this case also the brain and its membranes presented no obvious appearance of disease. The spinal cord on section seemed somewhat atrophied in the central grey matter, but no other changes were noted.

The cord, together with portions of several nerves from the right side of the body, and of the right extensor communis digitorum, were hardened in solution of bichromate of ammonium for five weeks, and examined in the same way as were those of the previous case.

Microscopic examination.—The cord here too may be described as normal, although it does not present such a typically healthy appearance as was found in the other. The ganglion cells are fewer in parts, most of them are shrunken, numbers have dropped out altogether, leaving holes in the section; and of those which remain, the branching processes are less clearly defined. These appearances, so far as they deviate from the strictly normal type, are probably due to the preparation of the cord for cutting having been less successful than in the other case. It should also be noted that the blood-vessels are larger and more numerous than usual.

Right anterior crural nerve.—The chief noteworthy appearance in transverse section is the large number of the nuclei of the nerve sheaths, and of large round-cells scattered through the endoneurium, and infiltrating the walls of the vessels. Roughly speaking, about half of the nerve-fibres appear normal; the rest present a cloudy or granular appearance of the nerve substance. There is no increase in the connective tissue.

In longitudinal section some segmentation of the myelin is apparent, but it is not a strongly marked feature.

Right anterior tibial nerve.—Here, on the other hand, the segmentation of the myelin is the most striking feature; there is not a sound fibre to be seen in the specimens examined (fig. 6).

Right musculo-spiral nerve.—In transverse section some

of the nerve-fibres are swollen and granular, others shrunken, a large number apparently normal, showing well the axis-cylinder. In longitudinal section also the fibres appear granular; the myelin in many is segmented, and collected in round or oval masses.

Right median nerve.—The appearances here are similar in kind to the above; in degree they are more marked. In addition the nuclear proliferation before referred to is conspicuous in endo- and perineurium, and round the vessels.

Right ulnar nerve exhibits precisely the same appearances as those found in the last mentioned nerve, and in about the same degree.

Right phrenic nerve shows no appreciable appearance of pathological change.

Right pneumogastric nerve.—A few of the fibres appear degenerated, and there is here and there slight segmentation of the myelin.

The *spinal nerve roots* are sound, both anterior and posterior.

Extensor communis digitorum.—The striation of some of the muscle-fibres is indistinct, and some are decidedly granular. There is, perhaps, slight increase in the nuclei of the sarcolemma.

The *liver* shows early cirrhosis and fatty change.

My third case, E. C—, a cook, æt. 43, is still under treatment, so that the ultimate test in her case cannot be applied; but if regard be had to the history and symptoms it would scarcely be too much to say that her pathological state, in kind if not in degree, could be predicated with tolerable certainty.

She came under my observation on November 5th, 1886. There was a history of rheumatism in two members of her family. She herself had suffered occasionally from winter cough, and two or three years ago had an attack of hæmatemesis. She had had two miscarriages in the first two years after her marriage, and one living child subse-

quently, the latter being now a healthy woman of twenty-two years of age. There was no history of syphilis on the side of either the patient or her husband. Her present illness came on about three weeks before admission with pains about the joints, which were thought to be rheumatic; these were first noticed in the knee-joints, and then in the hands and ankles.

She was observed, on admission, to be a sparely nourished woman, peevish in manner, with dilated facial capillaries, having pain in the joints and a short cough. Her chest showed fair elevation and expansion, and was normally resonant; breathing was weak and accompanied here and there by scattered sonorosibilant sounds. The heart's apex was in the normal position, impulse feeble, sounds fairly normal. In the abdomen the liver extended to near the level of the umbilicus, measuring eight inches in the vertical nipple line. Its margin was sharp, and the surface smooth; it was free from tenderness, and was distinctly felt descending with deep inspiration. There was no dulness in the flanks, and no jaundice. Both knee-joints and ankles were tender, and the former were thought to contain a little fluid; there was no redness, and they did not feel hot.

She had all the appearance of a person of alcoholic habit, but what with the family history of rheumatism, the apparent localisation of pain in the joints, and slight effusion into the knees, I was disposed to pass over her case very lightly as one of slight rheumatism, but on examining her again a few days later (Nov. 10th) I observed that she had double wrist-drop. The hands could be held out in straight line with the forearm but there was no power of further extension. The extensor muscles were wasted, and there was tenderness on pressure over nerve-trunks, and on grasping the arm or forearm. As to the legs, it was found that the ankle-joints were painful on movement, and also the muscles of the calves when handled. The muscles of the legs generally were wasted and flabby, the feet dropped, and without

power of dorsal flexion. Patellar and plantar reflexes were completely absent; the feet and hands were neither shiny nor œdematous.

On renewing inquiry into her history it was elicited from her friends that during the last eight and a half years she had taken spirits and beer very freely, and that on the occurrence of her present illness the joint pains were followed by weakness of the legs which rapidly became more marked, so that she was unable to walk. She had no numbness or tingling anywhere, but for a year had suffered from a feeling of coldness in the feet.

In the hospital she was restless and slept badly at night. After the lapse of little more than a week from the time when her paralytic symptoms were noted she complained of a feeling of numbness in the legs. Power of grasp in both hands was very feeble. Her urine was acid, sp. gr. 1030, depositing lithates but free from albumen and sugar. She took food well and began to sleep better, but she occasionally passed her motions in bed while retaining her urine. She was apathetic and rambled in her talk. The following is a note of the electrical reaction of her muscles.

All the muscles on back of forearm react well to *faradic* current.

Galvanism:

Ext. carpi ulnaris C.C.C. (μ ·5) > A.C.C.
Ext. com. digitorum C.C.C. (μ 1·5) > A.C.C.

Marked wasting of muscles. No tremors, coarse or fine.

Muscles in front of forearm react to weakest faradic current.

Galvanism:

Flex. sublimis digitorum C.C.C. (μ ·5) > A.C.C.
Supinator longus C.C.C. (μ 3·5) > A.C.C.
Flex. carpi ulnaris C.C.C. (μ ·5) > A.C.C.

Difference between A.C.C. and C.C.C. extremely slight, in many cases the two are practically equal.

Legs.—All the muscles of both legs act to a rather

strong, primary *faradic* current, except the peronei of left side, which do not react at all.

Galvanism.—Right leg :

Tibiales, Extensors, Gastrocnemius	A.C.C. (μ 2) = C.C.C. Action slow and wave-like.
Peronei	A.C.C. (μ 6) > C.C.C.

Left leg :

Tibiales, Extensors, Gastrocnemius	A.C.C. (μ 2) = C.C.C.
Peronei	A.C.C. (μ 4) > C.C.C.

A few days later (Nov. 29th) it was noticed that cutaneous sensibility both in arms and legs was much impaired and she was becoming more apathetic and incoherent. She could not grasp anything in order to feed herself. She could move the legs slowly but not the feet, and there was scarcely any sensation on pricking the skin of arms, legs, chest, or abdomen. The respiratory movements were almost wholly thoracic and the breathing shallow ; she had occasional twitching of the arms but not of the legs.

The following notes record the further progress of the case.

Dec. 2nd.—Patient lies on her back with her knees drawn up. She is in a more stupid and drowsy condition, will scarcely answer when spoken to, and does not protrude the tongue. The cheeks are flaccid and are blown out in expiration ; the skin is dry and harsh. Movements of the chest are very limited, and there is scarcely any respiratory movement of the abdomen. There is no jaundice. Resonance of front of chest is fairly good ; breath-sounds weak and accompanied by faint sonoro-sibilant sounds. Posteriorly resonance is normal, breath-sounds scarcely audible ; no adventitious sounds are heard. Heart's maximum impulse well felt in fourth interspace, three quarters of an inch internal to nipple ; sounds normal and well pronounced. Legs and arms are rather

more wasted. Ophthalmoscopic examination of each fundus shows nothing abnormal.

7th.—Patient is much brighter to-day although she rambles in her talk and her speech is rather thick; the tongue is clean and moist; the diaphragm acts in respiration tolerably well, as evidenced by epigastric bulging and descent of the liver during inspiration. There is now but little complaint of pain when the limbs are manipulated, but the dropping of the hands and feet is as well marked as ever. She complains of no discomfort and says she feels better. Urine and fæces are sometimes passed in bed. She has the delusion that she gets up every day and walks in the ward.

11th.—Mentally she is clearer now. Sensation of pain on pinching the skin of the legs is acute. There is some rigidity of the adductor muscles of the thighs; she lies with the knees pressed together. The pupils are moderately dilated and act sluggishly to light.

15th.—The extensors of the forearm are stronger. She is able to bring the hand above the horizontal line of the forearm when the hands are held out, and she can slowly pronate and supinate both hands. She can turn in bed without assistance, and does not now pass urine or motions in bed.

17th.—Electrical examination of right leg made to-day shows that the peroneal and sural groups of muscles all act to moderately strong *faradism*.

Galvanism:

Extensor longus	A.C.C. (μ 4)	= C.C.C.
Gastrocnemius	A.C.C. (μ 6)	> C.C.C.
Peronei	A.C.C. (μ 5)	= C.C.C.

In all cases the contraction is feeble, slow, and wave-like; the degenerative reaction is less marked than on first examination. The examination and necessary manipulation of limbs now cause no pain. There is a red line on the gums close to the margins of the teeth; the tongue is clean and moist, but of a deep red colour, and somewhat glazed.

Up to this point there was evidence of some improvement, but on December 30th and two following days she had a sharp attack of diarrhœa, which pulled her down a little. She still, however, showed the possession of more power in the extensors. Her delusions continued. It should have been mentioned before that she had no spinal pain or tenderness, and that she could distinguish heat and cold perfectly over the feet and legs below the knees. There was some power of dorsal flexion of the foot, and a slight amount of glossiness of the skin of the fingers was noted.

She has lately (January 7th) become more restless at night, and has been trying to get out of bed. The wasting of muscles, especially those of the legs, is still more marked; the interossei of the hands are much wasted also; reflexes remain absent; cutaneous sensibility of feet and legs is perfect, except that she does not feel when a light touch is applied to the outer and upper part of the left ankle.

January 11th.—Electrical examination. *Right arm.*—All the muscles of arm, forearm, and hand react to moderate faradism, and the galvanic reactions are quantitatively and qualitatively normal. *Right leg.*—The muscles act as before to faradism; their action to galvanism requires a more powerful current than formerly (eight to ten milliamperes). A.C.C. still equals C.C.C. About this time the patient was got out of bed, and allowed to sit up for an hour in the evenings. It was noted that the feet did not become œdematous when in the dependent position.

18th.—She gets up daily for an hour, and is decidedly stronger. She is unable yet to stand, however: she is clearer in her mind. There is much impairment of cutaneous sensibility over the backs of the hands and over the outer and posterior aspect of the forearms; the sensation of pain when pricked with a pin is retarded for the most part in hand and arm, where it is not absolutely abolished. As to the legs, the sensation of pain is decidedly retarded, and in the sole of the foot is absent altogether. Reflexes are still absent, and wasting is very marked. She is able

to lift the legs from the bed, and to move them laterally, crossing one over the other.

28th.—Patient complains that she does not feel so well, and this she attributes to going out and falling on the street. (She has not been out of the ward since her admission to the hospital.) She moves her limbs more freely, however, and has no pain.

February 14th.—Yesterday, for the first time, she was able to feed herself; to-day, she complains of abdominal pain, chiefly in the region of the lower border of the liver. There is but little, if any, tenderness, her tongue is clean and moist, and she does not feel sick. She can raise herself in bed, and has much greater power of extension in the arms, and of dorsal flexion of the feet. There is no difference as regards wasting of muscles, and no return of reflexes.

19th.—She is now able to walk a little round the ward, supported on either side, and can stand with very little assistance. In walking she seems to have most difficulty in getting under way, the ankles bending under her when she first stands up, but after being fairly started she moves her legs pretty well. The toes are turned in, however, and she tends to walk on the outside of the foot; the toes are rather scraped along the floor. She walks best when looking down at her feet. When sitting, she can cross one leg over the other with tolerable ease.

28th.—There is a marked improvement now in her mental state. Her memory is clearer, and she has not the delusions about getting up and going out for a walk which she used to have. She can walk now with a little support on one side.

In about ten days after this she could walk with the help of a stick only, and was able to dress herself, and a week later the stick was discarded, and she walked without support of any kind. It was noticed, however, that after being out of bed the greater part of the day her feet and legs became œdematous; but this condition has gradually improved, her progress has latterly been uninterrup-

ted, and she may now be pronounced convalescent. She has steadily gained weight (from 7st. 8¾ lbs. on March 11th, to 9 st. 4 lbs. on May 6th), and she eats and sleeps well. The patellar and plantar reflexes are still absent.

Remarks.—I have brought these cases under the notice of the Society in the hope that they would form a contribution of some interest to the clinical history and pathology of alcoholic paralysis, especially since no cases accompanied by full pathological details are to be found in its published 'Transactions.' In saying this I am not unmindful of the papers contributed by Dr. Reginald Thompson and Dr. Broadbent in 1868 and 1884 respectively, but these dealt with the disease chiefly from the clinical standpoint.

Until a few years ago the symptoms in cases such as those I have narrated were attributed to some change in the spinal cord, and this view was held by both British and foreign writers. Dr. Wilks, for instance, wrote of alcoholic[1] paraplegia as a "spinal affection" from the influence of alcohol exerted on the spinal cord. Dr. Broadbent's communication, also just referred to, bore the title "On a Form of Alcoholic Spinal Paralysis," and in the discussion which followed the reading of his paper at the Society's meeting on February 12th, 1884, the opinion seemed to be generally held that the disease owed its symptoms to some change, either organic or dynamic, in the cord.[2] And it may be said that the writings of both German and French observers pointed for the most part to similar conclusions, till Lancereaux[3] published cases in which the peripheral nerves were described as manifestly altered, a large number of the fibres of the nerves examined showing the myelin to be segmented in rounded granular masses of unequal size. The

[1] 'Lancet,' March 9th, 1872.

[2] Dr. Buzzard, however, referred to the observations of Lancereaux and others as suggesting its peripheral origin.

[3] 'Gazette Hebdomadaire,' No. 8, Feb. 25th, 1881.

same author also published further observations in an article on the general subject of Toxic Paralyses[1], in which he repeated his previous conclusions that Alcoholic Paralysis is a disease of peripheral origin, having definite symptoms, course, and pathology.

Since this direction has been given to pathological investigation many cases have been recorded both in this country and abroad,[2] and there has been a general agreement that inflammatory or degenerative changes have always been present in some part of the peripheral nervous system. The view that the essence of the disease is an inflammation has found expression in the name "multiple neuritis;" but it has been suggested that the morbid condition found in the nerves is rather a degeneration similar to what takes place in the peripheral part of a nerve after division, and that the term "neuritis" is a misnomer. With reference to this, I think it may be said that my fatal cases, especially the first, would seem to favour the former view. The masses of leucocytes seen in the perineurium, as well as the numbers of similar cells scattered through the endoneurium, together with the appearance of the walls of the vessels, induce the conclusion that there at least the lesion is of the nature of an inflammation. May it not be, as suggested by Dr. Ross, of Manchester, in a paper recently published,[3] that different pathological conditions may exist at different levels of a diseased nerve, the degeneration being found below the point at which a perineuritis has been established? It seems to me *a priori* reasonable to expect changes of an inflammatory nature as the result of prolonged alcoholic excess, and I should look upon the condition as comparable to what is found in the liver in cirrhosis.

Regarding the diagnosis of my three cases, I think there can be no doubt. The first case certainly died of

[1] 'Gazette des Hôpitaux,' No. 46, April 21st, 1883.

[2] See Appendix.

[3] 'British Medical Journal,' January 1st, 1887.

acute phthisis, but the symptoms of alcoholic paralysis were even better marked than those referable to the lungs, and the morbid appearances at least equally so. Both fatal cases had cirrhotic and fatty livers, and the liver of the third case appears to be in the same condition. The history in each case pointed entirely in the same direction. There was nothing to favour a suspicion of syphilis, gout, lead-poisoning, or diphtheria, or of any other disease likely to give rise to a nerve-lesion; and in all there was the marked and prolonged history of alcoholic indulgence, as well as the presence of morbid conditions other than those found in the nerves, likely to result from such a habit.

I should say that the paralysis of the diaphragm and intercostal muscles, which was well marked in both the fatal cases, had much to do in determining the fact of death at the particular time it occurred.

This consideration applies most strongly, however, to the second case, since in it there was no efficient and immediate cause of death found except œdema of the lungs.

As to other symptoms, the muscular pains, the mental derangement, the absence of bedsores, and of paralysis of the sphincters, and the electrical reactions of the paralysed muscles, are sufficient to establish the diagnosis.

Tubercular disease of the lungs has very often been associated with alcoholic paralysis, and this is an interesting fact, which has not yet received, so far as I am aware, any special explanation. It seems to me that here, as often elsewhere, the simplest explanation may be the true one, that both the disease of nervous system and of lungs are due to the one common cause. Abuse of alcohol is not unlikely to determine the appearance of phthisis in those who have any hereditary predisposition to the disease, as well as to lead to other insanitary conditions tending in the same direction.

In connection with this point it should be mentioned that M.M. Pitres and Vaillard have lately published[1] the

[1] 'Revue de Médecine,' March, 1886, p. 193.

results of investigations into the condition of the peripheral nerves in the subjects of pulmonary tuberculosis, being led to this by observation of the troubles of sensation and motion, and the trophic changes which they have met with in that disease. The position laid down by them in the paper referred to is that tuberculosis acts like diphtheria, variola, typhus, and enteric fever in occasionally producing a change in the peripheral nervous system, such change consisting of a parenchymatous neuritis, evidenced by segmentation of the myelin, proliferation of the nuclei of the inter-annular segments, disappearance of the axis-cylinders, and atrophy, more or less complete, of the nerve-fibres. They quote a number of cases in detail, collected from various sources, in some of which there were no marked clinical symptoms of nerve disease,—nothing beyond vague and passing pains. In others muscular wasting was a prominent feature; in a third group sensory troubles, such as pain and cutaneous hyperæsthesia or anæsthesia, predominated. In all the cases examined the pathological appearances above enumerated were found in peripheral nerves, and the brain and spinal cord were healthy. In some the clinical features were precisely those of alcoholic paralysis, excepting the mental derangement; but it is worthy of note that in two at least there was a decided history of alcoholism as well. Their conclusions are set forth in the following terms:

"In the course of tuberculosis, just as in the course of other infectious maladies, it is not uncommon for the peripheral nerves to become the seat of parenchymatous alterations, presenting the histological characters of inflammations called degenerative.

"2. These depend on an inflammation in the nerve itself, and not on a pre-existing lesion of the brain or spinal cord, and are found in subjects whose nervous centres and spinal roots are in a condition of complete integrity.

"3. They are indifferently found in sensory, motor, and mixed nerves. They are equally seated in the cranial

nerves (including the optic and oculo-motor nerves) in the pneumogastric, phrenic, &c.

"4. Their symptoms, very complex and very variable, are as yet incompletely understood. However, in comparing the observations published up to the present time, they may be divided into three groups.

"The first comprises those cases where the symptoms of neuritis shown at the autopsy have passed unnoticed amid the severe troubles attendant upon the evolution of the tuberculosis (latent neuritis).

"In the second are placed those in which muscular atrophies, localised or diffuse, have constituted the predominant symptom (amyotrophic neuritis).

"In the third may be included those cases in which the nerve inflammations have produced during life the more or less serious troubles of sensation, hyperæsthesias, anæsthesias, neuralgias, &c. (painful or anæsthetic neuritis.)

"5. The frequency of peripheral neuritis in the subjects of tuberculosis, the variability of its distribution, and consequently of its symptoms, explain the existence and the many clinical forms of most of the nervous troubles which supervene in the course of tubercular disease."

The considerations advanced by these authors open up an important field for observation regarding the changes to be found in the peripheral nerves in the subjects of phthisis, which it may be hoped will soon be more completely occupied. They have at least established a case for further inquiry. It must be said, however, that in none of their cases is alcoholic influence expressly excluded, and this is a fact of some importance when the difficulty of obtaining such a history from patients is borne in mind, and when it is further remembered that varying degrees of alcoholic indulgence produce different effects on different individuals. And, as already mentioned, two of the cases quoted in their paper (one of Strümpell's and one of Müller's) were alcoholic.

As to treatment, my first and second cases were so rapidly fatal as to make any discussion on this point in

relation to them of no value. In the case of the third patient, iodide of potassium did not appear to do any good, and bromide of potassium and chloral, instead of making her quieter and producing sleep, seemed to have the opposite effect. Further than this, she has had strychnia in gradually increasing doses for many weeks, to the extent of ten minims of liquor strychniæ every six hours, and latterly cod-liver oil, with faradism and rubbing to the wasted muscles. Probably the benefit she has received has resulted more from the withdrawal of alcohol than from any more direct element of treatment, and it has been a favorable point in her case that she has always been able to take food well. At the same time, I have no doubt that the faradism and rubbing, as well as the strychnia and cod-liver oil, have played a considerable part in her restoration to health.

APPENDIX.

A very full bibliographic index, brought down to 1885, will be found at the end of the work first mentioned below. Those references which follow complete the list to date, so far as I have been able to find.

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Hun. Alcoholic Paralysis, American Journal of the Medical Sciences, April, 1885, p. 372.

Schulz. Beitrag zur Lehre der Multiple Neuritis bei Potatoren, Neurolog. Centralblatt, 1885, pp. 433, 462, and 482.

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Buzzard. On some Forms of Paralysis from Peripheral Neuritis of Gouty, Alcoholic, Diphtheritic, and other Origin (J. & A. Churchill), 1886.

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Gowers. Multiple Neuritis, in Manual of Diseases of the Nervous System, vol. i, pp. 91—102 (J. & A. Churchill), 1886.

Kast. Primäre degenerative Neuritis, Deutsches Archiv für Klin. Med., vol. xl, part 1, p. 51.

Bernhardt. Ueber die Multiple Neuritis der Alkoholisten, Zeitschrift für Klin. Med., vol. xi, part 4, 1886.

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Ormerod. Illustrations of Peripheral Nerve Disease, St. Barth. Hosp. Rep., vol. xxii, pp. 181—185.

Duckworth. Three Cases of Multiple Peripheral (Alcoholic) Neuritis in Women, ibid., p. 253.

Seguin. Alcoholic Multiple Neuritis, Journ. of Mental and Nervous Disease, New York, March, 1887, p. 206.

(For report of the discussion on this paper, see 'Proceedings of the Royal Medical and Chirurgical Society,' New Series, vol. ii, p. 275.)

DESCRIPTION OF PLATES VII AND VIII.

(Three Cases of Alcoholic Paralysis (Multiple Neuritis), by DAVID W. FINLAY, B.A., M.D., F.R.C.P.)

PLATE VII.

(From drawings by Dr. FINLAY.)

Fig. 1 (E. F—, Case 1).—Transverse section of plantar nerve, stained with logwood and mounted in Canada balsam, showing thickening of the perineurium and of the walls of the blood-vessels, with proliferation of nuclei and leucocytal infiltration. × 175 (about).

Fig. 2.—Part of the same section more highly magnified. The appearances of degeneration in the parenchyma of the nerve are better seen; the small dark spots are remaining axis-cylinders, some of the larger are probably swollen axis-cylinders; the others are nuclei of the nerve-sheaths and leucocytes. The comparatively hazy-looking large round bodies are nerve-tubules with granular contents from which the axis-cylinders have disappeared. × 450.

Fig. 3.—Normal nerve, transverse section, for comparison with Fig. 2, and prepared in the same way. × 450.

PLATE VIII.

(From drawings by Dr. FINLAY.)

Fig. 4 (E. F—, Case 1).—Plantar nerve: fibres teased out, treated with osmic acid and mounted in glycerine, showing degeneration and segmentation of the myelin. × 450.

Fig. 5.—Extensor carpi radialis longior. Transverse section stained with logwood and mounted in Canada balsam. Many of the muscle-fibres are atrophied, and the whole section is crowded with nuclei and leucocytes. × 175 (about).

Fig. 6 (M. J. P—, Case 2).—Anterior tibial nerve, longitudinal section, stained with osmic acid and carmine, and mounted in Canada balsam. The appearances are entirely similar to those found in Case 1 (Fig. 4). × 450.

Fig 1.

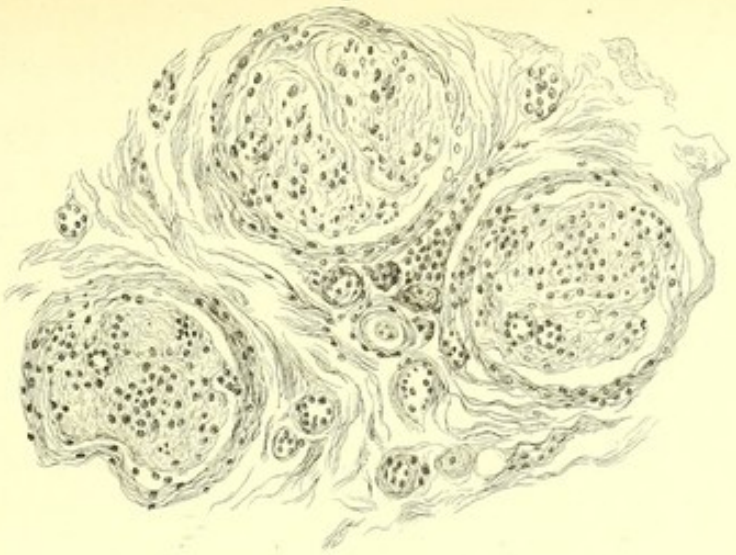


Fig. 2.

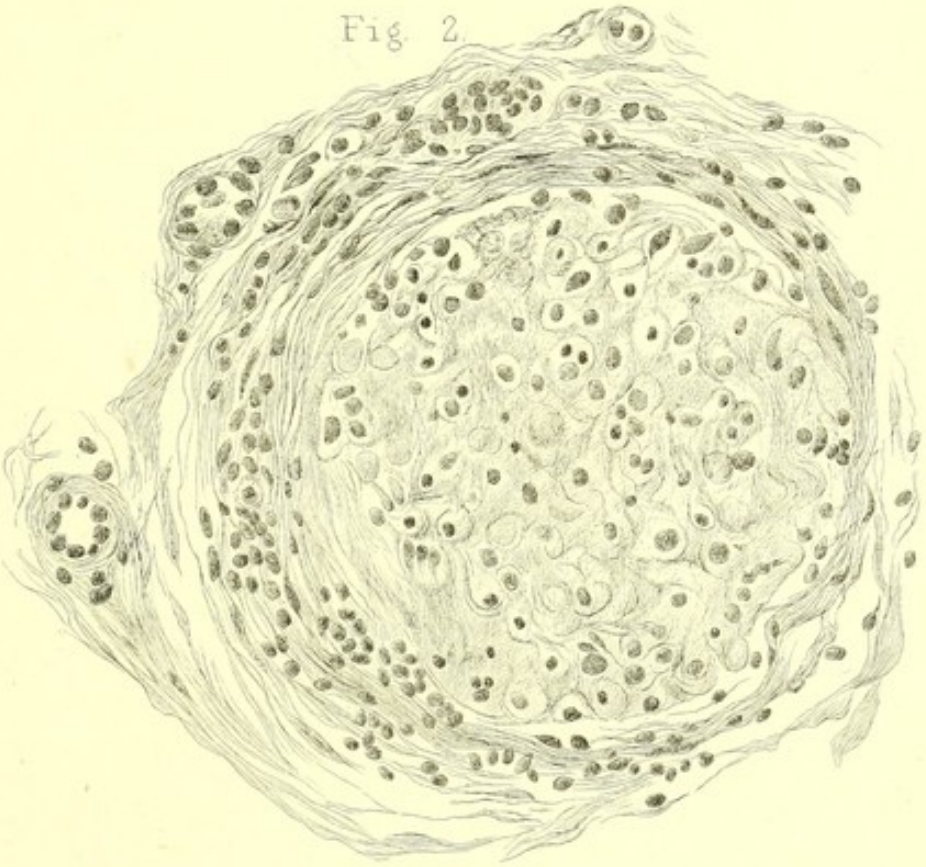
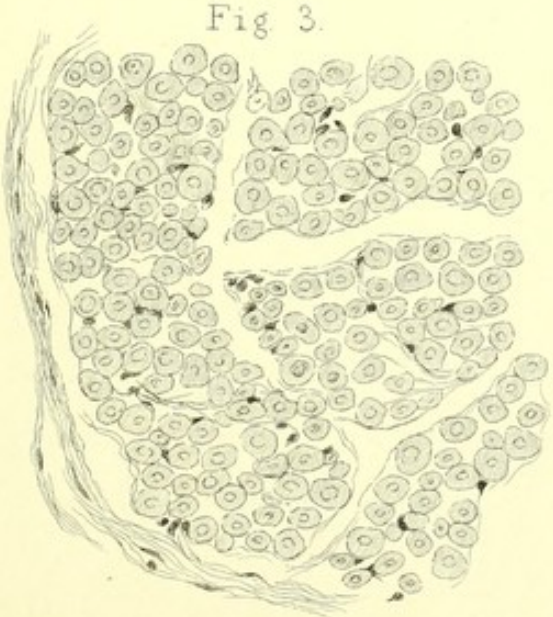


Fig 3.



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Fig. 4.

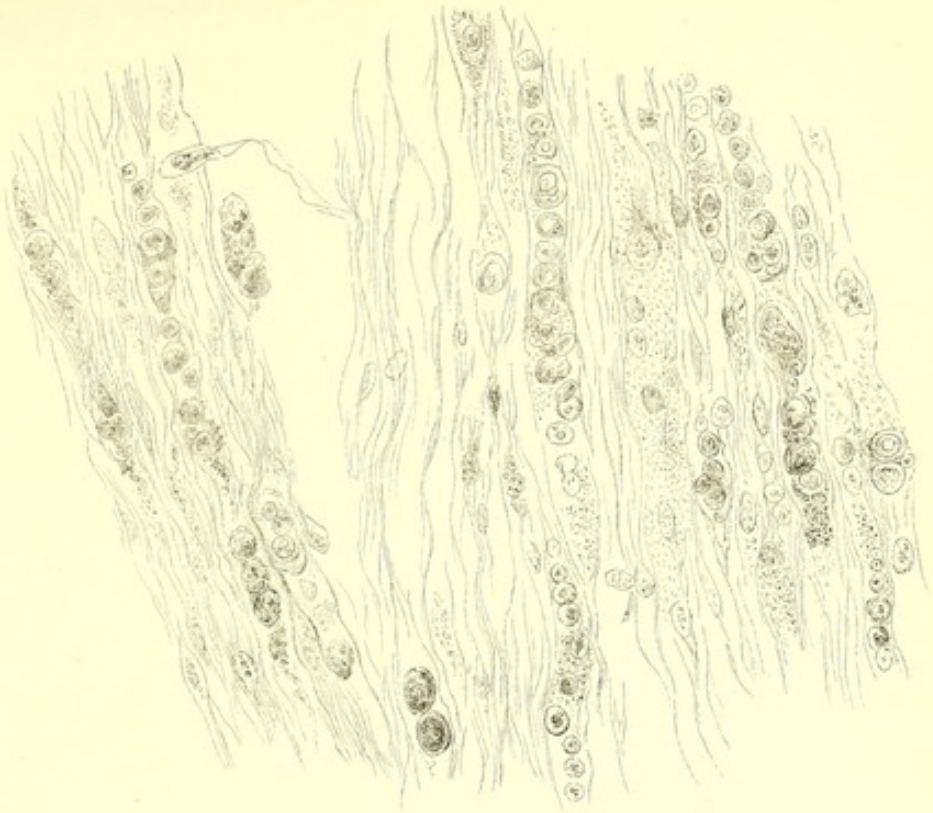


Fig. 5.

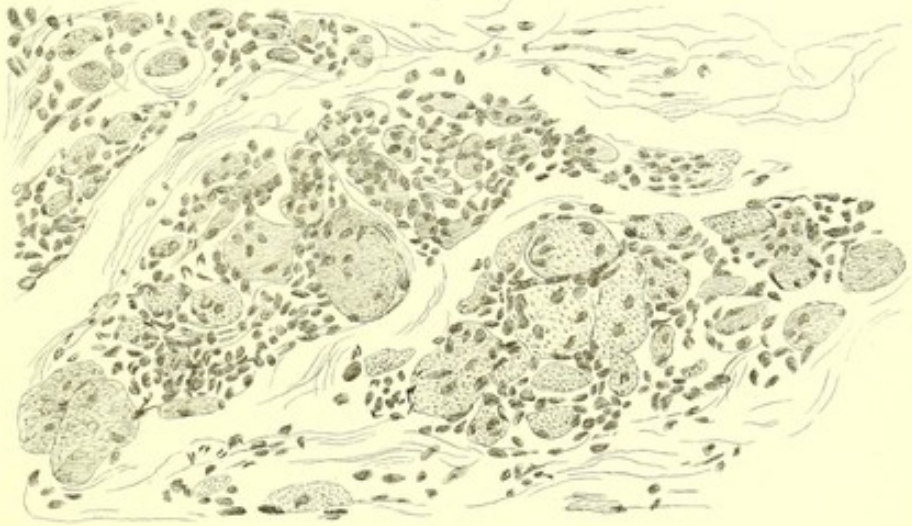
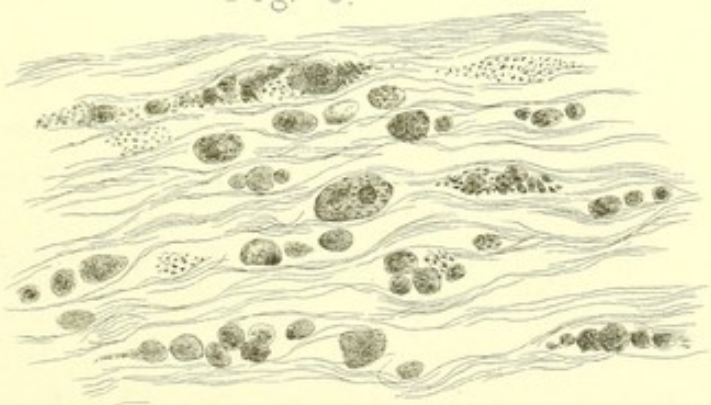


Fig. 6.



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MULTIPLE GANGRENE OF THE SKIN IN INFANTS

AND ITS CAUSES.

BY

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Received March 8th—Read June 14th, 1887.

THE occurrence of multiple gangrenous lesions of the skin in children has been noted from time to time by various authors, but our knowledge of the significance of most of these cases is chiefly due to Mr. Jonathan Hutchinson, who showed that gangrenous ulceration was a rare complication of varicella, and, subsequently, that it might still more rarely, also follow vaccination.

I hope to carry the matter further by the cases I am about to relate, and demonstrate that even this is not the whole truth, but that other lesions, not in any way of a specific character, may also give rise to this serious condition.

Secondly, that there are various grades of the process after varicella, some of them so mild as to be only slightly ulcerative. In these, the eruption comes out in crops and may keep up the disease for a considerable time. These cases are generally attended with severe

itching, and appear to me to correspond with the cases called by Mr. Hutchinson "varicella prurigo."

Thirdly, a precisely analogous condition may follow vaccinia.

Fourthly, I would suggest that the condition is of microbic origin and may follow any pustular eruption in which the soil is suitable. The predisposing conditions are *tuberculosis*, as was first pointed out by Dr. Barlow, while *congenital syphilis* and *rickets* are probably also etiological factors in a certain number.

Class 1.—*No Antecedent Varicella.*

Case 1.—Ellen A. B—, æt. 1, was first brought to University College Hospital as an out-patient on July 7th, 1886.

The family history was good except that one maternal aunt died of phthisis, ascribed to hard work at the wash-tub, but the child was suffering from well-marked rickets, had some bronchitis and profuse sweating. Three weeks previously, an eruption had commenced on the neck and spread all over the body, and when seen, every part except the face, hands, and feet was more or less covered, most thickly on the chest, scapula and forearms, with red papules, from a pin's head to a hemp-seed in size, most of them flat, angular, and shining, but some, chiefly on the thighs near the knees, were round and convex. On the largest and deepest coloured of these last was a minute pustular centre. There was very little, if any, itching. The child was shown to the Dermatological Society on July 14th as a case of lichen planus infantum with miliaria rubra on the thighs, and the diagnosis was not disputed.

It was in these miliaria lesions near the knees, that the more serious lesions commenced on July 16th and 17th. At the same time some more mattery heads appeared upon the buttocks, which broke and became sores by the 18th. She was brought again to the hospital on the 21st. There

were then many small ulcers on the buttock and lower part of the abdomen, two on the forearm, one about one third of an inch in diameter in front of the thigh (the first to be formed), and two above the knee on the left side.

The child was taken into the hospital on July 24th: she was fairly well nourished but had a good deal of bronchitis, nearly all over her chest.

Distribution.—The lichen papules on the upper part of the body had not entirely gone. There was nothing on the front of the scalp, but on the left side, on a level with the ear, were some small superficial excoriations covered with crusts, and a few also on the right side. There was also an irregular group of ulcers, from one eighth to three quarters of an inch in diameter, scattered irregularly over the back of the head in the left mastoid region and immediate neighbourhood, and a few also on the occipital region.

On the right side of the face were about half a dozen superficial ulcers about one eighth of an inch across. On the right arm were six or eight crusted excoriations, scattered irregularly over both surfaces. All but two were quite superficial sores; of these, one was a pustule one sixteenth of an inch across, with red areola, and the other a lesion with a dark central crust one eighth of an inch across, slightly depressed below the surface, also with a red areola. On the left arm were two deep ulcers, two thirds of an inch in diameter, on the flexor aspect, and two about a quarter of an inch in diameter. On the wrist there were two smaller ones, one on the upper arm, three on the outer surface, and two superficial on the elbow tip; the arms were free.

There were a few superficial sores on the chest and on the right clavicle, and on the lower ribs on the left side, was an irregular group of five, not very deep, and from an eighth to a quarter of an inch in diameter. All these were covered with a reddish-brown scab, sunk below the surface, and one of them had a pustular margin and

resembled a vaccine vesicle. Immediately below the ribs, was a flat pustule a quarter of an inch across, with a narrow red areola. On the abdomen only three or four lesions were present above the umbilicus, but there were a large number scattered below it, most of them scabbed, and some with pustular margins, but the scabs were simply dried secretion. There were also a few pustules from a pin's head to a hemp-seed in size, some flat, some convex, and on the right flank was an excavated ulcer three quarters of an inch across. On the anterior surface of the thighs were a few small lesions on the upper part, but on the left near the knee were two sharp-cut ulcers half an inch across and a quarter of an inch deep in the centre. On the right thigh, rather higher up, was a somewhat larger and deeper ulcer and a smaller one near it. On the outer side of the thighs the ulcers were very deep and numerous, thickly aggregated in the upper two thirds, and varying from an eighth to three quarters of an inch in diameter.

They were less numerous on the left than on the right side ; the left leg below the knee was free, but the right, had two on the front of the knee and about half a dozen lower down. On the back were a few comparatively superficial and small lesions at the neck, and over the scapulæ, and three or four in other parts scattered irregularly. There were two large deep ulcers on the loins, but the greater number were on the buttocks and upper two thirds of the thighs, especially the right. On the buttocks they were so thickly placed that they coalesced in one place on the left buttock into an ulcer an inch and a half long, and half an inch wide, the edges were sharply cut, sinuous and undermined, and the base deep and irregular. These ulcers, having been kept moist with the napkin, were not covered with scabs, but none of these were granulating, and there were more on the left than the right buttock, but the deepest were on the right side. The anus and its immediate neighbourhood were quite free. With the exception of two ulcers just below the left calf, the back

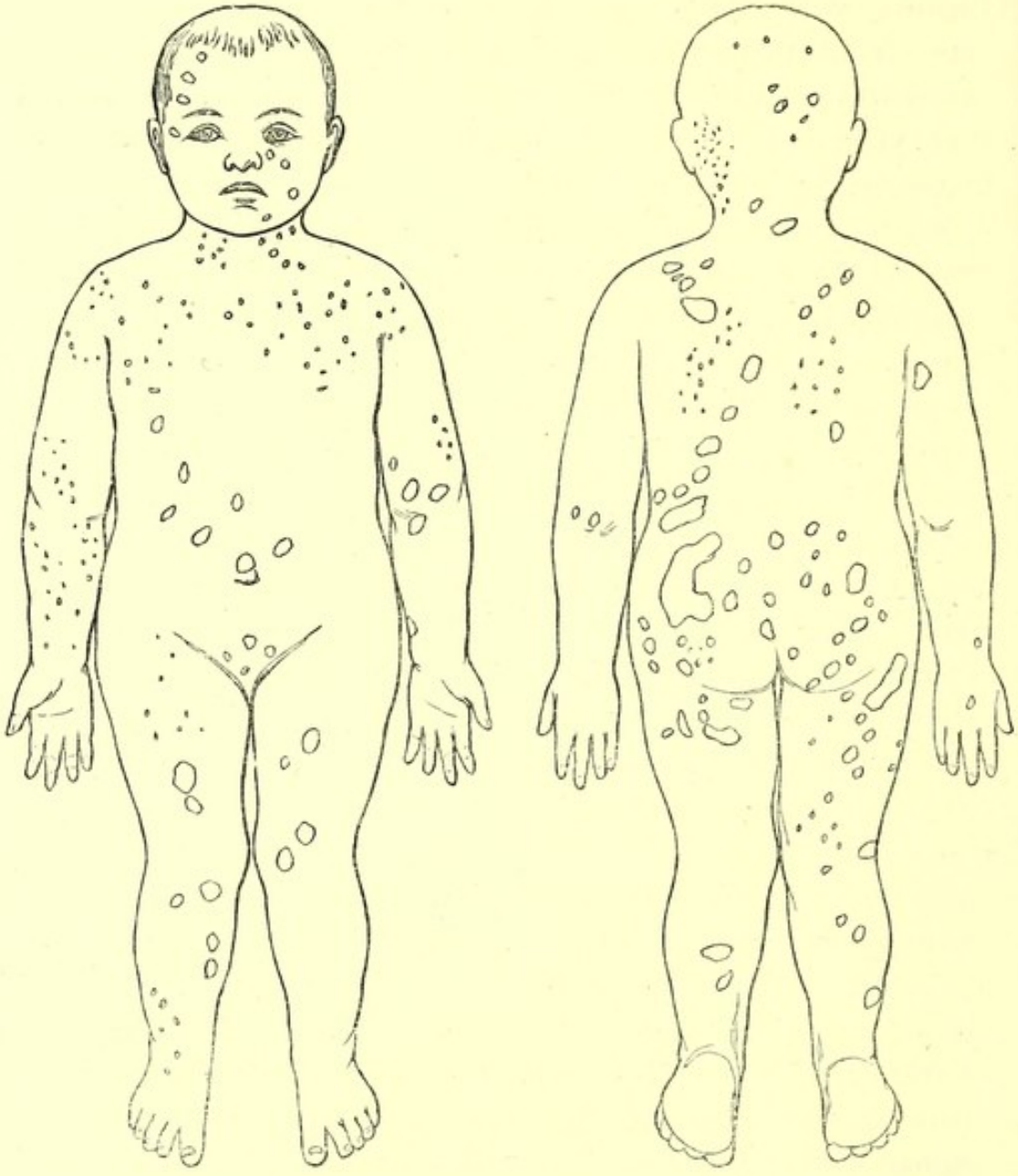
of the left leg was free from the upper third of the thigh downwards, while they were sparsely scattered all over the right leg and thigh. The palms and soles were free.

Observation showed that the lesions were produced in the following way :—A pustule about a sixteenth of an inch formed, with red areola, enlarged to an eighth of an inch, then dried into a scab, surrounded by a ring of pus; ulceration proceeded laterally and vertically below the scab to a varying extent, and a slough was formed, the centre becoming quite black. When this slough was thrown off, it left a sharply-cut roundish or oval ulcer with slightly irregular margins, sometimes undermined, sometimes going abruptly down to the deepest part of the ulcer, at others sloping obliquely down towards the centre. The ulcers never spread after the separation of the slough, and they were of all depths and sizes, from a superficial excoriation to a very deep ulcer. The largest single ulcer was about three quarters of an inch in diameter, and about a third of an inch at its deepest part, but there were some compound ones larger than this.

Wet boracic lint was ordered for the buttocks, but as the patient did not seem comfortable with this, iodoform wool was applied, and iodo-vaseline for the rest of the body. The wool, however, adhered too closely, and gave so much pain on removal that iodoform powder at first, and afterwards iodol sprinkled on were substituted. A few fresh pustules appeared at intervals and ran the course already described. On August 9th there was diarrhœa; a few fresh lesions had formed, but most of the sloughs had separated, and on the 11th it was noted that while the ulcers on the buttocks were deeper, there were no fresh lesions. The diarrhœa continued until death on August 19th, there having been no fresh lesions since the 11th. The temperature chart ranged from 99° to 104°. I was away for the vacation during the last ten days of life, and the post-mortem was made fifteen hours after death by Mr. Campbell Williams in Dr. Barlow's presence.

Post-mortem.—Body much emaciated, rigor mortis pre-

sent. On the thorax there were some signs of repair in the ulcers, but those on the buttocks were very deep, clean cut, and undermined. On the scalp they were not numerous, but two of them, the size of a florin and



half a crown respectively, in the occipital and left parieto-occipital regions, went down to the pericranium. There was no attempt at symmetrical distribution anywhere except on the thorax.

On opening the body, the pleuræ were healthy, the lungs were consolidated at both bases, especially on the right side, and much congested, and there were one or two tubercles in both lungs. The bronchial glands were about the size of an almond and caseous. The liver was enlarged and fatty, the mesenteric glands were large and infiltrated with tubercular deposit ; all the other viscera were normal.

As the above account is somewhat meagre, Dr. Barlow has been kind enough to supplement it from a few brief notes of his own.

"Some of the lesions on the scalp had ulcerated so deeply that there was a thin horny floor which was firmly adherent to the subjacent bone, but the bone was not actually bare."

"In the thorax, the bronchial glands were caseous and enlarged, the lungs were emphysematous in front, and the lower lobes at the back showed extensive areas of bronchopneumonia which had become partially caseous. On the under surface of the upper lobes were some groups of undoubted miliary tubercle. There was a small vomica almost certainly due in part to softening of one of the bronchial glands which penetrated into the root of the lung at the lower lobe on one side, and close to it was another caseous gland. The mesenteric glands were enlarged and caseous, and in the intestines some of the Peyer's patches showed small deposits of tubercle with very early commencing ulceration."

I have four other cases in which there is no reason to believe that varicella had any connection with the ulceration. In one of these (Case 3) the eruption was bullous and may be considered to belong to a different category, but at all events there was deep ulceration and no sign of varicella, and I therefore record it here. In Case 4 the evidence against a varicella connection is very strong, I should say conclusive, and in Case 5, a severe and fatal one, not only did it begin on the lower extremities, but the mother averred that she was acquainted with chicken-

pox eruption, and that the antecedent lesions did not at all resemble it.

Case 2.—Mary A. C—, æt. 6 months, was admitted as an out-patient at the Shadwell Hospital on February 27th, 1882. She was vaccinated when two weeks old; there was no evidence of varicella, there were no physical signs in the chest; she had been suckled but was much emaciated. The eruption began in the small of the back, two weeks before she came to the hospital; it was limited to the trunk, except one small ulcer outside of the left leg below the knee. It was abundant on the buttocks and reached half way up the back, where it was to some extent symmetrical. In the front of the body, on the right side only, there were five ulcers and a few scars; there were one or two on the head, and many scars from previous lesions in various parts. Those in front were scabbed superficial ulcers half an inch in diameter with a bright red areola; there were a few pustules like small boils which began as minute pustules on an indurated inflamed base, the whole being the size of a No. 3 shot.

The treatment consisted of sulphide of calcium internally, and diluted ammonio-chloride of mercury ointment applied to the sores. On March 27th all the ulcers were healed and the child was thriving, but an abscess having formed on the back she was admitted into the hospital, where she died on May 7th, but I have been unable to obtain particulars of the post-mortem.

Case 3.—Adelaide S—, æt. 3 years, was admitted as an out-patient at the Children's Hospital June 25th, 1883. The disease began nine days previously with a bulla half an inch across, then others formed and began to ulcerate. When first seen, there were bullæ half an inch across, containing clear serum and without any areola; there were pustules from an eighth to a quarter of an inch across with an areola; superficially ulcerated patches covered with a layer of pus; deep ulcerations, one very deep, on the site of the first bulla, three quarters

by a third of an inch in area, nearly all of the lesions being confined to the right side of the thigh and leg. During the previous two days several minute pustules had formed, a few on the inner side of the left thigh and on the right side of the head. The child was slightly rickety, fairly well nourished, but she had been pulled down by a continual diarrhœa which had only been stopped for a fortnight. The mother had lost four children out of eleven, three of whom died of "consumptive bowels;" "they seemed to drop off at three months and waste, but they none of them had any rash." The case was diagnosed at the time as an "ulcerating pemphigus."

Case 4.—Louisa M—, æt. 3 years, was admitted as an out-patient to the Shadwell Children's Hospital on May 12th, 1884. The child had had "glass-pock" eight months previously and whooping-cough had been present for a month. One week before she came to the hospital a white patch formed on the occiput with blood in the middle following two pea-sized vesicles full of water; others followed on the scalp and on the right second finger. On the right parietal region when first seen was a white patch about a square inch in area slightly depressed below the surface, looking as if the skin was undermined with the border slightly raised and vesicular. Near it, was a millet-sized vesicle with a faint red line round it and there was also an areola round the large one. Near the white patch in the middle of a half inch vesicle was a scab, and there was also on the occiput another scabbed patch with blood-stained crust, about an inch and a half in diameter; there were several smaller lesions with black sloughy centres about the scalp, and a deeply excavated ulcer an inch in diameter over the right ear, and the lips were scabbed and had superficial ulcers upon them.

Case 5.—Ellen A. H—, æt. 16 months, came first to University College Hospital on February 17th, 1880. The father was consumptive, the mother well, but lived poorly. She had had nine children, eight of whom were living,

and three miscarriages, but not consecutive ; the eldest, a boy, æt. 17, had had some cervical abscesses, but the others were healthy. The child before the patient, died when twenty months old, and had similar lesions on the skin for four months before death ; it had improved under my colleague Mr. Barker's treatment, although it subsequently died of bronchitis. The spots first came on the right leg, and then spread upwards. The following notes were taken on March 17th :—The patient was fairly nourished, but suffered from enlarged cervical glands. She was vaccinated when six months old, and the eighth or ninth day afterwards, small red pimples came out over the whole body, which " felt like a nutmeg-grater," but this did not last longer than a month. A fortnight before she came to the hospital a few spots came on the right leg, preceded by fretfulness, then others came in a corresponding position on the left leg, fresh ones continued to come out, extending from below upwards to the buttocks, and then over the body, ulcerating soon after their formation. When first seen the bulk of the eruption was papular, while pustules existed round the hair-follicles of the legs. There was an eczematous patch about an inch in diameter on the head ; there had been some eczema behind the ears, and otorrhœa.

The distribution of the lesions was as follows :—There were the remains of a good many on the scalp, but they had not been of a severe kind, and they were dry and nearly well when the child was seen ; there were about a couple of dozen on the back of the forearms, and a few on the front, and on the upper arm, but the palms and soles were free. The upper two thirds of the trunk were almost free, but there was some desquamation on the lower third. They were comparatively numerous, increasing in number and size from above downwards, being pretty freely distributed over the buttocks and thighs, and densely crowded from below the knees to the feet, the lesions as a whole being roughly symmetrical. The course of the eruption is as follows :—It begins as slightly red, pin's-head sized, flattish

papule, with only an indication of suppuration at the apex. As it enlarges, the pustulation becomes more distinct, but the lesion is still flat at the top. When it is about an eighth of an inch in diameter the pus dries into a scab ; most of the lesions gradually get well ; others, however, especially on the feet and legs, have a pustular border formed round the scab, and this again has an areola, the whole somewhat resembling a vaccinia lesion. This soon ulcerates beneath the scab, forming a sharply punched-out flat-bottomed ulcer, with a ring of inflammation round. Others again are much deeper, a black slough having formed which, after separation, leaves a clean-surfaced but deep ulcer ; or the smaller ulcers may extend peripherally and vertically, and then are irregular at the base, though the outline is generally roundish or oval, unless neighbouring lesions have coalesced. There were no ulcerations on the head and face, a few superficial ulcers on the forearms, and one or two on the trunk. On the thigh and buttocks were several of large size, but the ulcerative process was most marked below the knees, those on the feet being the deepest and largest, while the lower third of the leg was fairly riddled with ulcers, varying from a quarter to more than an inch in diameter, and some of them nearly a third of an inch deep. The child was not seen again, having died a few days later in convulsions. No post-mortem could be obtained.

I have next a series of six cases of varying grades of severity all accompanying or following undoubted varicella. The first one was of the most virulent kind.

Class 2.—*Antecedent Varicella.*

Case 1.—Georgina S—, æt. 7 months. Both father and mother were syphilitic. The child was always weakly, and snuffled, but presented no other evidence of syphilis. On October 31st she was unwell ; on November 1st a vesicular rash appeared on the head and back which was evi-

dently varicella; in a few days it dried into scabs. On November 3rd a black blister formed on the inner side of the right knee, which afterwards became a large gangrenous ulcer; the slough was one inch by three quarters in size. On the evening of November 3rd, and the morning of the 4th, the mother noticed that all the vesicles were becoming black blisters. She was admitted into University College Hospital on November 8th. The child was small, feeble looking, and very drowsy. On the right knee, as already described, was a deep ulcer, another on the flexor aspect of the left knee, several on the buttocks, and a small one on the left ankle. Just above the pubes was a patch consisting of a central scab surrounded by a vesicle, and this by a red areola. It began on November 5th. On the left side was a patch formed on November 6th; in this only a faint areola was present, the vesicular part was more distinct, and the scab was smaller and lighter coloured.

On the buttock several small red points showed signs of vesiculation. On November 14th the temperature was 99·8°, there were no new sores, and the old ones had not spread. On the 15th the temperature reached 105·2° at 7 a.m.; it fell to 97·6° at 7 p.m., and the child was weak and listless. On the 16th the temperature rose again to 104° at 2 a.m., and the child died at 7 a.m.

Post-mortem.—The upper lobe of the right lung was filled with minute infarcts the size of radish seeds, most of them breaking down in the middle. In the left lung there was broncho-pneumonia at the base. The liver was fatty. The skull-cap was thick, rough, and soft at the anterior part of the vertex, evidently due to syphilis. The brain was normal. There was no evidence of tubercle anywhere. A closely analogous case to this is recorded by Mr. Warrington Haward in the 'British Medical Journal' for 1883, in which the child died with pyæmic lesions in the lungs.

In the next case the ulcers appeared when the varicella

was nearly well, and only two of them were deep and gangrenous.

Case 2.—Ruth B—, æt. 3 years. No history of phthisis in the family. The varicella broke out three weeks before I saw her, and when the pustules were nearly well sores appeared on the face and on the back of the ears.

On the abdomen and lower rib margin and over the left rectus were two dark, sloughy spots with a red areola, one of them rather deep; there were several red marks on the head and face from the previous eruption.

In the third and fourth cases all the lesions were superficial and developed on the pock pustule.

Case 3.—William N—, æt. 7 months, came as an out-patient to Shadwell on October 31st, 1881. There was no phthisis in the family, nor any evidence of it in the patient. The eruption had been out for three weeks. It began as little spots all over like "glass-pock" on the head and back, and the sores began a week before admission.

The pocks never got well, but sores came where the scabs had been commencing on the head. When first seen, the lesions were in the following positions:—On the temples, there was an ulcer on each side, the largest about a third of an inch across, and of conical shape. There were a few varicella scars on the cheeks, and on the scalp were many pustules the size of those of varicella, and a few ulcers a third of an inch across. There were two or three scars on the forehead, and one pustule. On the left arm was an ulcer half an inch across and a quarter of an inch deep, the slough having separated, and there was a corresponding lesion on the right arm. The back was covered with varicella scars, and there were two or three papules with a tendency to pus formation on the top; there were also some fresh papules on the right cheek and leg. The eruption was attended with considerable itching. Quinine internally and iodoform externally were

prescribed, and when seen again on November 4th the old lesions were healing, but some fresh ones had appeared on the forehead, face, and arm, with a few on the trunk and legs, the number being greatest on the extensor surfaces.

The new eruption was in pale red papules a pin's head in size and upwards, some vesicular and dried into scabs, a few a quarter to half an inch across and beginning to ulcerate. The legs and arms were rather thickly covered with small papules ; the larger ones came out at once and did not form from small ones ; they all itched violently. The elder brother, who had had varicella when he was seven months old, was always kissing the patient, and several papules had appeared on his cheeks and chin, which increased in size the following week, but did not ulcerate. The patient went on very well until November 8th, when catarrhal pneumonia supervened, probably from exposure in his journey to the hospital, and he died on December 2nd. There was no autopsy.

I have met with two cases in connection with vaccination corresponding with the milder cases of ulcerating varicella, although in one place the ulcer was large and deep, which would remove doubts, if any existed, as to the similar character of the eruption.

Class 3.—*Ulcerating Eruptions after Vaccination.*

Case 1.—Henry B. H—, æt. 10 months, was brought to the Shadwell Children's Hospital on January 23rd, 1882. The mother was dying of phthisis. The patient was thin and delicate. Three weeks after vaccination, the ears and one nostril began to discharge, and the right eyelid became inflamed. The eruption began on the head a week later as red pimples, the size of a small pea or less, then enlarged, and pustules formed on the top one quarter to three quarters of an inch across ; then the head became

covered with scabs a quarter of an inch across. When first seen, the head was covered with scabs from a quarter to one inch across, but with little or no inflammation round, and numerous scars of previous eruption were present. There was one inflamed pustule on the left forefinger, and a deep ulcer three quarters of an inch by half an inch over the left ischial tuberosity. There were a few on the back and legs, one just coming, consisting of a flat pustule one eighth of an inch across, raised on a red base, and the toes had been affected. There were many scars, but no attempt at symmetry. On January 30th the child was brought again, and nearly all the ulcers, except the large one over the hip, had healed.

I have some very imperfect notes of the other case, in which a red pimply eruption came out one week after vaccination, and lasted a week; it was followed by a vesicular rash, which came first on the shoulders, and then down the arms and legs, feet, palms, and soles. There were also some on the back of the head, and a few down the back. The vesicles became pustules with a red areola, were from a millet seed to a pea in size, and accompanied by intense itching.

There are thus a dozen cases of varying degrees of severity, some so mild and chronic, that were it not for intermediate links we should hesitate to class them with those gangrenous cases at the other end of the chain, which are fatal in a few days.

From a consideration of these and twenty-one other cases published by Mr. Hutchinson and other authors, we may draw up the following general account of the disease.

The place of onset, and mode of development varies according to whether it appears early or late in the course of the varicella, or is independent of that disease.

If it begins while the varicella lesions are still present, it commences on the head or upper part of the body, and instead of the scab being thrown off from the pock, ulce-

ration takes place beneath it, and often a pustular border with a red areola is formed, the whole resembling a vaccination pustule, the process extending both in depth and peripherally; a black slough is formed from a quarter to an inch or more in diameter, the smaller ones, still with a pustular border and areola: after attaining a certain size, varying much, the process of separation sets in, and when completed, a sharp-edged, roundish or oval conical ulcer is formed deep or shallow, in proportion to the diameter of the slough, some of the largest being quite three quarters of an inch in the centre. Extension of the ulcer seldom takes place after the separation of the slough has commenced. When the lesions are closely aggregated coalescence will probably ensue and then very large ulcers, irregular both in contour and floor, are produced. If any fresh crops are formed, or when it commences after most, if not all, of the varicella lesions have cleared off, perhaps a fortnight or more from the onset, or in cases following vaccination, or otherwise unconnected with varicella, the ulcerative lesions usually commence on the lower half of the body. They especially affect the buttocks and thighs, as a pin's-head sized papulo-pustule, which extends to the size of a pea or larger, ruptures, and, except on the buttocks or wherever it is kept moist, dries in the centre to a scab, with the pustular border and red areola like vaccinia, and from this point follows the same course as those which started in a varicella pustule. In some cases the buttocks and parts in contact with the napkin, and sometimes the legs and the thighs, are fairly riddled with ulcers of all sizes, shapes, and depths. On the trunk and rest of the body they are not usually numerous, and though some may be very large and deep the majority are comparatively superficial.

Where the lesions are numerous and deep there is naturally much constitutional disturbance, the temperature ranging up to 104° or even higher. Lung complications, tubercular, pyæmic, or inflammatory, are very frequent, and determine or hurry on the fatal issue. Should the

child survive it is surprising how rapidly the lesions cicatrize, of course leaving deep and indelible scars.

There are all grades of the disease. In the mildest form ulceration may be quite superficial, the lesions reaching to the vaccinia-like stage and then drying up, often accompanied by pruritus, and lasting by successive crops for a considerable period; there may also be simultaneously observed mere excoriation up to pretty deep ulceration, with or without a few lesions going on to gangrenous sloughs; while in the most extreme cases hæmorrhage occurs into all the vesicles, which become rapidly gangrenous and lead to the death of the child in a few days, either with a general tuberculosis, or, pyæmic lesions. When less severe than this the contents of the vesicles are not hæmorrhagic, but a large proportion of them become gangrenous, and death is very likely to ensue but may be deferred for some time, and is usually due to a secondary complication.

As regards *etiology*, all the cases hitherto recorded have occurred in infants or young children; an analysis of my own and eleven of other authors, in which the age is stated, shows that by far the majority occur under one year, the figures being fourteen not exceeding one year, six not exceeding two years, and three under three years of age; the youngest was three months old.

My colleague Mr. R. W. Parker, had a case of a girl, æt. 12, in whom a hydroa was aggravated by the administration of iodide of potassium, into hæmorrhagic bullæ which then discharged and gave rise to extensive ulceration and sloughing lesions very suggestive of the disease under consideration. By far the majority occur in girls: fifteen out of the twenty-one cases where the sex is mentioned, and of my own cases ten out of twelve were females. With regard to the diseases antecedent to it my own cases are alone available for reference, most of the other reporters of cases having accepted Mr. Hutchinson's dictum that they were all consequent on varicella or vaccinia. In one after vaccination, a mild case, and in

five others there was not the slightest evidence of varicella, and in one the child had been under close observation for lichen planus infantum, and the ulcerative lesions appeared to develop on miliaria rubra pustules. These facts suggest that under certain circumstances any eruption of isolated pustules may be the starting-point of the ulcers. Among predisposing causes tuberculosis has been present in so many that, as Dr. Barlow first pointed out, it must be more than a mere coincidence. In one of my fatal cases *congenital syphilis* was present, in another *rickets*, while a few were apparently quite healthy. Gangrenous ulcers of probably similar character occur sometimes as a complication of variola in adults, as well as in children.

The diagnosis is not difficult, with or without a history of varicella; the occurrence of numerous gangrenous ulcers in a young child, or even of deep ulceration beginning as a pustule, enlarging, drying into a scab in the centre, and then ulcerating, form a group of symptoms quite unmistakable.

The prognosis is serious in proportion to the tender age of the infant, the number, depth and extent of the lesions, the amount of constitutional disturbance, the presence of tuberculosis, pyæmic, or inflammatory lung symptoms.

The treatment must be general and local; quinine in one- or two-grain doses in milk every four hours is often serviceable. In some of my cases sulpho-carbolate of soda in five-grain doses every three hours has been apparently beneficial. All complications must be treated as they arise.

Wet boracic lint under oiled-silk until the sloughs have separated, and subsequently iodoform or iodol vaseline will keep the ulcers antiseptic. Freshly-made iodide of starch paste, painted on, is another convenient application, and Pasteur, of London, found a warm solution of chlorinated lime on lint give most relief. These measures and the administration of concentrated, or, in young infants,

partially digested foods, and putting the patient in the best hygienic conditions, offer most chance of success.

I desire now (by way of appendix) to call attention to certain local gangrenes of the skin in children, especially of the genitalia and neighbourhood, of which the attending phenomena present some analogies to the cases hitherto considered.

Class 4.—*Local Gangrenes of the Skin.*

Case 1.—Elizabeth H—, æt. 2½ years, out-patient at Shadwell, June 23rd, 1882. She was quite well up to the 17th, when "gatherings as if she was chapped" appeared on the ears, and then the vulva was attacked. When first seen there was superficial ulceration behind and above both ears, and on the upper part of the auricle itself, the right side being worse than the left. There were some isolated pustules over the inferior maxilla, one of which was ulcerating. The whole vulva was in a state of extreme inflammation, swollen, dusky red, especially at the lower part, where there was superficial ulceration, and also between the labia; there was also a scanty, thick, purulent discharge from the vulva. The child was fairly nourished but looked ill, and her temperature was 101°. She was admitted into the hospital and recovered without any further ulceration.

It may be objected to this case that it is not in the same category as those in which there is sloughing, but I think she only just escaped this by timely treatment.

Case 2.—Florence B—, æt. 1 year and 6 months, out-patient at Shadwell, admitted October 19th, 1883. She had measles three months previously; she got over it all right, but subsequently her chest was affected, and she was treated for "bronchitis and inflammation," and when I saw her she had sonorous and sibilant râles all over the

chest, but no dulness. Two weeks before admission pimples like little blisters appeared round the anus, then spread forwards to the top of the privates; these broke, ulcerated, and fresh ones appeared daily for four days until they were so crowded that they coalesced. On admission there was a deep ulcer two inches by three quarters on the left buttock, and at the side of the anus. Further forward, and separated by a narrow interval of healthy skin, was another irregularly bordered ulcer, which reached up to an immensely swollen vulva. The lower two-thirds of the left labium was in an ulcerating and sloughing condition, and on the right buttock was another smaller ulcer the size of a shilling, and there were two more on the right labium; the parts round were of a deep dusky red and much swollen. The child looked very ill and she was taken into the hospital under Mr. Reeves. He made incisions into the tissues where there was most tension, and applied iodoform dressings, but the child died of asphyxia on October 26th, and no post-mortem could be obtained.

Case 3.—George R—, æt. 2 years, out-patient at the East London Hospital for Children, admitted December 15th, 1884. He had had measles five weeks previously, and got on well up to two weeks ago, when little white spots like blisters, but containing "mattery-looking stuff," with the skin round them reddened, came in the groin. They burst and became open sores, were poulticed, and the ulcers spread rapidly; the flesh looked black, and had become much worse the last two days. When seen there was, in the left inguinal region, at the bottom of the abdomen, a deeply excavated and undermined ulcer about three quarters by half an inch. In the left groin there was a deep slonghing ulcer three inches long by three quarters of an inch wide, and quite half an inch deep in some parts; it extended on to the scrotum, but was more superficial there; on the inner side of the leg it had gone down to the subcutaneous tissue. Half an inch below the large one was an oval ulcer half an inch long, very deep, and

with undermined edges which represented an earlier stage of the process, the large one having been formed by the coalescence of four or five "blisters" the size of a pea, and without any special arrangement with regard to each other. At the root of the penis in front was a superficial excoriation produced by scratching, but the child seemed to be in no pain. The temperature was 100·4° in the rectum. There were some subcrepitant râles at the bases, but no dulness. The mother said he had quite recovered after the measles, ate well, and ran about until these ulcers appeared. His subsequent history is unknown.

Case 4.—Emma L—, æt. 14 months, admitted on November 29th, 1886, as out-patient at Shadwell. Ten days previously two little white spots the size of a pea appeared in the groin; they were at first full of water but soon burst, and the mother applied fuller's earth and poulticed them. On admission there was on the thigh an ulcer the size of a hemp seed, and a red areola round it; it had begun the day before as a red spot, and was like the others were at first. On the lower border of the groin, extending down to the vulva, was a chain of ulcers with yellowish sloughs and inflamed base; the largest was about one inch long and a quarter of an inch wide, and the next largest three quarters by half an inch, and lower down still the ulcers were roundish and from a quarter to half an inch in diameter. The right labium was swollen and red, and so was the abdomen for a short distance between the ulcers, but the child seemed lively and well and not in pain until the thigh and groin ulcerated. A sister, æt. 16 years, was attending at the City Road Hospital for consumption, and this child had a bad cough, but there were no physical signs in the chest. On December 1st a small pustule with red areola appeared on the left cheek exactly similar to those in the groin, which were healing. The subsequent history is unknown.

The next one differs in many ways from the preceding

cases and is possibly of tropho-neurotic origin ; it is, however, worthy of record as a local phagedænic ulceration.

Case 5.—William M—, æt. 9 years, admitted as out-patient at Shadwell on April 30th, 1883. His general health had always been good, and he had had no previous illness up to a week before Christmas, when he was exposed to a chill ; he seemed languid, and then, without pain preceding it, a "blister" the size of a farthing appeared one inch below the middle of the clavicle, which broke and left an open sore, and this spread to a wound three and a half inches by two and three quarters in extent and of irregular shape. Just above this was a superficial oval ulcer about an inch in its long diameter, and below there was a serpentine ulceration, beginning to the right of the middle line and extending irregularly for about an inch and a half to the left of it, but nowhere more than a quarter of an inch broad.

Since the ulceration his head has been in the position of right torticollis and he seems languid but is otherwise tolerably well. The ulcer took nearly six weeks to attain its full size and did not begin to heal for some time after that, but had been well for some weeks when seen by me.

The small patch under the right clavicle was still livid red, and without sensation. The large patch was also deficient in sensibility, a pin-prick being felt as something sticking in, but there was no pain. He was put on iron and cod-liver oil, and gradually recovered, but it was four months before he was restored to health, and the skin of the scar was slowly regaining its natural colour, but there was even then defective sensibility in the cicatrix.

Hilbert[1] records two cases of spontaneous gangrene of the eyelids in female infants under one year old. A small pustule with yellow scab first formed without apparent cause on the upper lid, rapidly enlarged, the part beneath became gangrenous, and when the slough separated a circular ulcer nearly an inch in diameter was left, which

[1] 'Viertelj. f. Derm. u. Syph.,' vol. xi, 1884, p. 117.

healed rapidly. Both children were healthy and well nourished.

It will occur to everyone that the first four of this set of cases are referable to noma, especially as two of them came on after measles, but granting that this is so it will be noticed that they all began as vesicles or pustules, and from that went on to ulceration and sloughing. So far they therefore corroborate my statement that vesicular eruptions, other than those of varicella and vaccinia, may be the starting-point of gangrenous ulcers.

In a case of Dr. Barlow's which he was kind enough to show me recently, besides the deep punched-out ulcers of the vulva and groin, there were some scabbed spots like dried-up pustules sparsely scattered about the trunk and limbs, and although no history of varicella could be obtained Mr. Hutchinson considered that varicella was at the bottom of it. Even if that were so in Dr. Barlow's case (which I do not believe) it certainly was not in all of mine. On the other hand, there was a strong suspicion of tuberculosis in three of the cases. There are other analogies with cancrum oris, moreover, which may start from quite a superficial ulcerative stomatitis, and this, too, is more frequent in girls than boys, and rarely begins after the sixth year. "Broncho-pneumonia is very common, and pyæmic abscesses have been found in the lungs" (Eustace Smith), and the pneumonia preceded or followed the gangrene. Meigs and Pepper and others have drawn attention to the predisposing influences to it of the tubercular diathesis, while Barthez and Rilliet describe it as beginning with ulceration, aphthæ, or phlyctenæ of the mucous membrane and only rarely with œdema. I do not of course ignore the fact that vesicles or bullæ are often the first sign of a gangrenous inflammation, but these are of different characters, and I am equally convinced that even when arising from otherwise harmless causes they may be the starting-point of gangrene in tubercular or otherwise predisposed subjects.

The conclusions I would draw from the consideration of these cases are :—

1. That infants and children under three years old are liable to multiple gangrenous ulcers.

2. That while the gangrenous lesions most frequently follow those of varicella and vaccinia they may also ensue from other pustular and non-specific lesions under certain conditions.

3. That when once the gangrenous process has started the fresh lesions do not necessarily arise from a pre-existing pustule but may come independently.

4. While the original eruption varies much, the gangrenous lesions are very similar in their course and development, viz. of vesicle, pustule, scab and slough surrounded by a red areola closely resembling, before the slough has formed, a vaccine lesion.

5. That the general conditions which coexist are not only specific fevers, such as varicella, vaccinia, and, for the local forms, measles and scarlatina, but tuberculosis and, perhaps, rickets, which are probably related etiologically to the gangrenous lesions.

6. The one feature that these diseases have in common is the febrile state, but while it is very probable that antecedent fever may be a predisposing cause we are not yet in a position to say that it is essential or that other factors are not required for the production of the gangrene.

Finally, I have only to express my regret that the record of some of the cases is less complete than could be desired, but they were nearly all out-patients, and only seen once or twice, and in the high pressure at which that sort of work has to be done, details which would find a place in a more leisurely record are apt to be omitted.

(For report of the discussion on this paper, see ' Proceedings of the Royal Medical and Chirurgical Society,' New Series, vol. ii, p. 282.)

ON A FORM

OF

CHRONIC INFLAMMATION OF THE LIPS AND MOUTH,

WHICH SOMETIMES ENDS FATALLY, AND IS USUALLY ATTENDED BY DISEASE OF THE SKIN AND NAILS.

BY

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My attention has been strongly drawn, during the last ten years, to certain cases, of which previously I had no knowledge, in which a severe form of ulcerative stomatitis is followed by disease of the skin, and which sometimes, after a duration of some months, end fatally. I have not been able to find any published description of this disease, or rather of this combination of symptoms. The cases are important on account of their severity, from their close resemblance in some instances to syphilis, and also, perhaps, because they are curable for the most part by one remedy only. On this latter point I must speak carefully, as the evidence is not complete. There appears, however, considerable reason for believing that small doses of opium, frequently repeated, will usually cure the malady, which resists all other drugs, and tends slowly but surely to end in death. I am not prepared to advance any suggestion as to the cause of the conditions which I am about to describe. In all the best-marked examples of

them the patients have been men, and in most of them men past middle life, living in the country, enjoying, until the beginning of the attack, excellent health, and in positions which enabled them to secure all the comforts of life. Two of them were master tanners, one a clergyman, one a farmer, and one a gentleman of no occupation. All these were country residents, but in some less well-marked cases the patient resided in London. It seems not improable that the condition of health which gives rise to this form of stomatitis may be allied to that which originates certain forms of skin disease which are attended with severe constitutional symptoms, and in which occasionally the mouth is affected. Of these pemphigus is the best example, but not the only one. In it, however, I believe that the affection of the skin always precedes the affection of the mouth, whereas in the cases which, with the permission of the Society, I am about to describe, the reverse is the case.

I must ask the indulgence of the Society in the somewhat difficult task of bringing forward, in some detail, the particulars of a number of different cases which more or less resemble each other. It will be, I am afraid, impossible to avoid being tedious, and at the same time by no means easy so to state the cases as to prevent their being confused with each other. I will begin with a narration in a first group of those which were most severe, and most closely conformed to what may be considered the type of the disease. I will afterwards mention some which were less severe and concerning which there may be some question whether they are exactly of the same nature.

It will be a mistake, I am convinced, if we attempt to define the malady very closely, since it would lead to the omission of many collateral facts from which we are quite as likely to derive aid in discovering its true relationships as from the more typical cases. I will describe the cases almost in the order in which they have come under my own observation.

The first of them was a patient who was sent to me by

my colleague Dr. Ramskill, to whom he had been recommended by Dr. George Jones, of Framlingham. Dr. Jones had taken great interest in the case, and was kind enough after the man's death to supply me with notes. My narrative of his condition whilst in the London Hospital, and of the autopsy, will be taken from the notes kept by Mr. Hingston, who was at that time my dresser, and a very able and diligent student. Briefly I may summarise this case by stating that the man was a master currier of fifty-eight years of age, that he began to be ill at Christmas, 1879, and died in the beginning of May, 1880. His illness commenced with sore mouth which was quickly followed by swollen hands and feet "like chilblains." Later on a vesicular eruption occurred on his hand, with inflammation of the nails, and he had spontaneous ptyalism with sore throat and sore lips. Next a bullous eruption extended over his whole body, and his nails fell off. Lastly, large granulation masses developed on the sores left by the bullæ, and some of these became pedunculated.

The man died apparently from exhaustion. He had long been in a most miserable condition, but although he had lost much flesh he had not become extremely emaciated. The post-mortem showed some recent broncho-pneumonia with pleurisy on the left side, and it revealed also the existence of a large lympho-sarcomatous tumour developed in front of the spine, and embedding the pancreas and large vessels. This mass weighed three pounds and a half. Nothing had occurred during life to lead us to suspect its existence, but as the man's skin was covered with excoriations we had not made any manual examination of the abdomen. Probably this tumour had but little to do with the cause of death, and I do not as yet see any probable connection between it and the rest of the symptoms. There was quite enough in the state of his skin and mouth to account for the fatal event; and whilst we must not forget the existence of the tumour, I incline for the present to set it aside as having been possibly an accidental concomitant of the disease.

In my next case, which was almost an exact parallel to the preceding, ending like it fatally, and running its course in five months, there was no tumour.

The patient in this case was a gentleman residing at Dorking, under the care of Mr. C. W. Chaldicott. His illness began whilst he was staying at the seaside in the winter of 1882, and on February 10th he came under Mr. Chaldicott's care, "with a very severe form of inflammation of the mucous membrane of the mouth." "The whole interior of the mouth was intensely, vividly red, excepting that in a few spots on the arch of the palate, and on the tonsils and pharynx, there were patches of very delicate pellicular whiteness, as if produced by the application of caustic. He had already been under the care of Mr. Coulson and Dr. Morell Mackenzie, and was taking the iodide of potassium. Mr. Chaldicott gave him a single mercurial pill, and two days later there were all the appearances of profuse ptyalism. Shortly after this a patch of moist papillary growth appeared in the cleft between the scrotum and the left thigh. At this time I first saw the patient, who was now confined to bed with severe inflammation of the whole interior of the mouth, including the tongue and lips. The pharynx was also inflamed, and he had great difficulty in swallowing. There was a remote and obscure history of syphilis in this case, and at first I thought it best to advise that the iodide of potassium should be freely used. In the course of a week or two, however, I became convinced that there was no reason to suspect that the condition was due to syphilis. A large papillary patch appeared in the opposite groin, and all the toe-nails of the right foot inflamed. A little later all the toe-nails of the left foot passed into a similar condition and some of them were exfoliated. Later still small bullæ like those of pemphigus appeared on the feet and legs first, but afterwards on the trunk and arms. At this time he was taking fluid food freely, and a considerable quantity of stimulant. He was able to take exercise in his grounds, and on one occasion bore a journey to London

with but little fatigue. As soon as the skin disease commenced we began the use of arsenic in combination with quinine; it had, however, no influence in arresting the progress of the disease, and after a little time the whole skin with exception of the head and face was covered with sores. When the bullæ broke, papillary excrescences sprouted up from their base. During the whole of this time the mouth remained sore. There were ulcers in the cheeks, and on the tongue, and a condition of constant ptyalism. At the end of exactly five months from the beginning of the illness, the patient, who had become greatly emaciated and very weak, sank from exhaustion. I had seen him with Mr. Chaldicott several times, and we had done our best to discover any cause for our patient's malady. The house in which he lived appeared to be quite healthy, and his wife, children, and servants, all remained well. In the endeavour to discover a cause, and also to devise a successful plan of treatment, we had the benefit of consultation with Sir James Paget and Dr. Bristowe. Nothing, however, which was done had any obvious effect upon the disease. During the last ten days of life small doses of opium were given, but it was possibly begun too late.

To recapitulate the principal features in this case we have an inflamed mouth, which after a few weeks took on a condition resembling ptyalism. Next, papillary growths in the flexures; next, suppurative inflammation of all the nails; next, a pemphigoid eruption over the whole body; and lastly, debility, emaciation, and death. The autopsy did not reveal any structural disease excepting recent hepatization of the lower lobe of the right lung.

These two cases are the only ones under my own care in which the disease has ended fatally. But in several of those about to be related, the inflammation of the mouth resisted treatment so long and the failure of health was such that we much feared for the result.

At the same time that this last case was under care, I had under observation two others. In both of these the

stomatitis was severe, but the skin disease never became formidable. The subject of one of these was a master tanner, a patient of Mr. Cordwent's, at Milverton, near Taunton. He was about fifty years of age, and had enjoyed very good health until his sore mouth began. He did not believe that he had been exposed to any injurious influence in connection with his trade, and he said that he did not himself handle hides. When he first came to me he had sores on his lips, in the pouches of his cheeks, and on his palate. They were large ulcerated surfaces, with red margins, and in most parts covered by a distinct pellicle. If this pellicle was removed they bled easily. The tongue was but very slightly affected. During the first part of the case there was no eruption on the skin, but after the sore mouth had lasted for about three months there appeared some patches of a sort of papillary psoriasis on the backs of his hands, and his toe-nails inflamed, and several of them were lost. I had the advantage of a consultation with Sir James Paget in this case, as well as in the preceding one; I subsequently admitted the man into the London Hospital, and he was there seen by several of my colleagues, his case exciting great interest. The treatment which was first tried was arsenic internally, and the application of caustics to the ulcers. He also took quinine and used various mouth-washes. The treatment under which at length the ulcers healed and recovery took place, was the administration of Liquor Opii Sedativus in ten-minim doses three times a day. Mr. M— had been under my observation from March 25th to June 30th, and during the greater part of this time he had been in the hospital. When he left us both mouth and feet were perfectly well. When the mouth was at its worst he was very weak and had lost much flesh.

A year later, in answer to a letter of inquiry, Mr. M— told me that he remained in fairly good health, but was still liable to have his lower lip chap and peel. He came to town to show himself two years later still. He was in good health and had shown no tendency to relapse.

My next case is almost an exact repetition of the one just given, and it is scarcely worth while to repeat the details. A healthy clergyman, aged 46, became the subject of a sore mouth, which at first nothing would cure. The sores began on his tongue, but spread to his gums, pouches of the cheeks, and the lining of the lips. The prolabia were but little inflamed. Before Mr. E— came under my care he had had the advice both of Sir William Jenner and of Sir James Paget, and had tried internally both arsenic and iodide of potassium. He had left the country and come to reside in London, but neither the change of residence nor any drugs which had been used had effected anything for his advantage. He had lost flesh and strength and become very anxious. It was four months after the beginning of his complaint that I first saw him. There was then much fœtor from his mouth, and numerous ulcers, with dusky, liver-coloured edges. On some of the ulcers there was a distinct tendency to the production of a grey pellicle. In this case there was no skin disease, with the exception of a lichenoid eczema on the left side of the abdomen, to which he had been liable for some years. I cannot claim any share in the cure of this patient. He was disappointed with the result of a single application of caustic to the sores, and fortunately for himself left me, and consulted Mr. Pollock, who at once put him under the influence of opium and with the very pleasing result that a cure quickly followed.

So far as I know the cure has been permanent. Mr. Pollock has mentioned this case in a paper, "On the Use of Opium as an aid to Surgery," 'British Medical Journal,' 1884, vol. i, p. 801.

The last case which I have to relate is one which has been exceedingly well characterised, so far as the mouth is concerned, but has been unattended by any inflammation of the skin, the extremities, or, in fact, of any part excepting by extension from the mouth. Although for a considerable time the disease appeared to resist all treatment and to be likely to end fatally, a most satisfactory

cure has at length resulted under opium. The subject of this case is a farmer from near Rugby, named S—, about 66 years of age. He had always been a remarkably strong, healthy man, until his sore mouth set in. He was sent to me by Dr. Clement Dukes, of Rugby, in the beginning of December, 1885. He had at that time suffered from sore mouth for about six weeks, and it had resisted various methods of treatment, which had been perseveringly used. The inflammation involved both lips, the pouches of his cheeks, his tongue, and his palate. There was swelling, congestion, and large excoriated patches in various places. He was rapidly losing flesh, and could swallow only fluid food. There was no disease of the skin in any part, and his nails were quite sound. His breath was fœtid from the secretion from the ulcers. He had lost many teeth, and those which remained were covered with greenish, glutinous secretion, as his mouth was too painful to allow of his using a toothbrush. There was no material ptyalism, and his teeth were all quite firmly fixed. I began the treatment by prescribing quinine in two-grain doses, with three minims of Battey's solution of opium. A fortnight later the dose of the latter was increased to five minims, and at the same time he was made to wash his mouth very freely indeed with weak Condy's Fluid.

On January 29th, after six weeks' treatment, he was not much better, excepting that his palate was less inflamed. His lips and tongue were still much swollen and raw. From his lower lip the inflammation had extended upon the skin to the chin, and the whole of this region was much swollen, raw, and granular. He reported that he took plenty of fluid food, but he slept badly, and felt very weak. He had got so much thinner that all his relatives thought he was going to die. The opium had caused him much constipation, but at this date I increased it to eight minims, giving at the same time aperients. A fortnight later I had the pleasure of finding his tongue almost well, and his lips and mouth

generally much improved. There could I think be but little doubt that the improvement was consequent on the increased dose of opium. He was now able to swallow more food, and was feeling stronger.

After this the dose of opium was still further increased, with the result that the recovery was in the course of a few months complete. Mr. S— is I believe at the present date, that is, one year after the treatment, in good health and his mouth quite sound.

With this case it will be convenient, I think, to conclude this group. I have, however, seen one other case in the person of a gentleman under the care of Mr. Charles Macnamara, who was kind enough to send him for my inspection. In him a very sore mouth had been coincident with an eruption on the skin, and with steady failure of health, and I believe that death followed a few weeks after I saw the patient. In this case the patient had lost his health after his return from India; he suffered from diarrhœa for some time. Opium was tried during the last few weeks, but I do not know to what extent it was pushed. The soreness of the mouth, lasting for weeks, and the peculiar eruption, were the marked features of the case, and it was on account of these symptoms that the patient was referred to me.

If I may count Mr. Macnamara's case as one of the same group—and there seems no reason for omitting it—we have six examples of the same combination of symptoms, in three of which death occurred.

In two the patient is known to have recovered under the opium treatment, and to have remained without any definite relapse up to the present time. All the six patients were men, and none of them under forty-five years of age. All had been in good health until the first symptom of sore mouth occurred. In all the three fatal cases the inflammatiou of the skin was extensive and severe, whilst in the three which recovered it was but slight, in two, indeed, being almost absent. In one of these three, although the eruption was but very limited in

extent, affecting only the hands and feet, there was the peculiar feature of inflammation of the toe-nails; and from four of the toes the nails were shed. In none of the fatal cases had the opiate treatment been tried until within a very short period of death.

In my next group I have to mention cases in which we still encounter the combination of a sore mouth and an eruption on the skin; and with the soreness of the mouth taking precedence in point of time over the skin disease. In these, however, the disease showed itself much more amenable, and in fact on several occasions got well without any definite treatment. It also showed a very definite tendency to recurrence after a few years' interval. Two of the patients in this group, which comprises only three, were comparatively young. One was a lady of thirty-five, and the two others were men, aged respectively forty-six and thirty-six. In one of these, which before I saw it had long been treated as syphilis, arsenic effected a definite cure on more than one occasion. In two of them the influence of opium seemed to be very definite, in one a rapid cure taking place under its influence. In none of these three cases was there any approach to the failure of health which occurred in those of the first group. It may possibly be the fact that the age of the patient has something to do with determining the severity of the disease.

My next case is one in which sores in the mouth and on the tongue occurred repeatedly in association with an eruption on the skin, and in which the diagnosis presented much difficulty.

Mrs. P—, a lady æt. 35, living in the country, came under my care on March 2nd, 1880. The surgeon who attended her told me that she had been under his treatment for ulcer in the mouth and eruption on the skin for sixteen months. A distinguished physician had been consulted and the case had been pronounced syphilis, but a very long treatment with specifics had failed to cure.

I found the tongue, cheeks, and lips covered with white-

edged ulcers, which certainly looked very suspicious. She had at this time no skin eruption excepting on the elbows and knees, but I was told that there had formerly been patches on the palms and soles and between the toes. The eruption had first shown itself at Christmas, 1878, and the sore mouth came soon after.

I had some doubt as to whether the symptoms really were specific although I could give no other diagnosis; and we agreed to leave off both mercury and iodides, to use soothing remedies to the mouth, and to give arsenic. I did not see her again for several months but was informed that the mouth soon got much better. Possibly it had been made worse by mercury.

In the beginning of the following June, however, Mrs. P— came to me again with a single sore in the mouth, and on June 27th, in spite of my treatment, several small florid ulcers had formed in the mucous membrane of the lips, and there was general inflammation of lips, cheeks, and palate. At this time there were no sores on the tongue. She had now a general eruption of dry, scaly, superficial patches on various parts of the body, on the waist, where the strings of her dress pressed, and on her knees and thighs. On the mucous membrane of the labia were some abruptly margined sores just like those on her lips. I examined her mouth and found that she had no artificial teeth and none that were stopped. She considered that her relapses were due to catching cold, and excepting a peculiar proneness to catch cold she considered herself in good health. She had been taking arsenic for a month and we continued it in increased doses and combined with it the chlorate of potash. Two weeks later the sores on the genitals had healed but the mouth remained very sore and many abrasions had now appeared on her tongue. The eruption was fading. About six weeks later she came to me again, her mouth being almost well and the eruption having made a considerable change in type. She now had a copious rash of erythema multiforme on her hands. On her fingers the eruption looked

much like flat chilblains, and there were red fissures in the finger clefts. She told me that on a former occasion her toes had been affected in a somewhat similar manner. The sores which had healed in her mouth had left white scars.

From the last date, the end of 1880, I saw nothing of Mrs. P— until March, 1883. She told me that she had in the interval been free from symptoms with the exception of being now and then threatened with relapse both in the mouth and on the skin after taking cold. During the last three weeks she had experienced a relapse more severe than usual and a number of pellicular ulcers were again present inside her cheeks and lips. Leucomata from the old ones were still visible. On this occasion she had no eruption on the hands, but during a relapse last autumn a few spots had appeared there. She considered that she had always prevented the attacks from developing by using the arsenic which I had formerly prescribed. She was easily influenced by it and often had her eyelids made puffy.

Reviewing this case now in the light of a three years' narrative, I feel able, in the first place, to dismiss conclusively the suspicion that it was syphilis. In the first instance I may confess that I was almost inclined to share in the diagnosis which had been previously given. We may admit that the combination of conditions, sores on the genitals, between the toes and in the mouth, with an eruption on the skin, was such as might easily lead astray in this direction. The results of treatment leave us, however, in no doubt. The eruption always disappeared under arsenic and the stomatitis yielded to chlorate of potash in combination with it. We have then a case of stomatitis clearly not of local origin, and attended by numerous ulcers on the tongue, lips, and cheeks, and associated with skin eruption of very varying type. That which had been first called eczema was subsequently dry and scaly, next looked like lichen planus, and next like bad chilblains or erythema multiforme. It affected the

hands and feet especially, though it was not confined to them. I do not think that we need fear to recognise an essential similarity between this and the other cases which I have narrated, and it may perhaps help us to a right conclusion as to their nature. It is, possibly, a modified form of lichen planus. In that disease, as I have already shown, an eruption appears on the tongue and on the cheeks. The difference is here that these patches ulcerate and are attended by general stomatitis; both are curable by arsenic, if curable at all, with, however, the reservation that the arsenic may cure the skin disease and not cure the mouth. This drug has, I think, but little influence on the mouth, and when pemphigus causes bullæ in the mouth it usually fails to do good. If we accept the suggestion of alliance with lichen planus then, inasmuch as the latter is after all only a variety of psoriasis, we succeed in placing them all together as modifications of one and the same diathesis.

A very interesting example of the disease was brought to me by Dr. Whistler. Its subject was a gentleman, aged about forty-six, and the attack of inflammation of the mouth for which I saw him was the third from which he had suffered, but it was the first in which any eruption had appeared on the skin. His first attack began in May, 1880, and lasted till August. On this occasion after having resisted many remedies his mouth finally got well very quickly when he went to the seaside. Another attack occurred in November, 1885, and again the cure was effected by a visit to the sea in December. In neither of these attacks was his skin in any way affected, and during the intervals his mouth got perfectly well.

The one for which I saw him had begun about two weeks before Dr. Whistler brought him to me. There was a distant and doubtful history of syphilis, but he was married and had a healthy family, and nothing had occurred of recent years to indicate that any taint persisted. His mouth was exceedingly sore and the conditions at first sight certainly suggested specific disease.

If it were of that nature, however, it must have been of recent acquisition, for the stomatitis was acute and symmetrical. It was quite out of the question that there had been any recent syphilis, indeed the history of the two previous attacks and of complete recovery between them, may, I think, be considered to put this suspicion entirely aside. Nor on careful examination did the state of his mouth resemble anything that occurs in syphilis.[1] There were large patches inside his lips and cheeks which were well margined and covered by a thick pellicular membrane. When this membrane was peeled off the surface easily bled. This tendency to bleed was, I may remark, noticed in most of the other cases. The tongue and palate were not affected. The skin eruption, which had been developed quite independently of any drug treatment, consisted in the formation, on the hands and feet, of patches of erythema as large as sixpences, which rapidly vesicated, but without producing bullæ. There were not more than about half a dozen of these on each of the extremities, and none on other parts. No suggestion could be made as to the cause of the attack. Mr. T— appeared to be perfectly well, but was beginning to feel somewhat low, and to lose flesh, in consequence of the difficulty in taking food. He got quickly well under small doses of opium.

In conclusion, it may be remarked that none of the cases seem to afford any light as to the real cause of the malady. It may, however, be plausibly conjectured that it is allied to some of the severe forms of skin disease which are attended by failure of health and tendency, unless cured, to death. Examples of this we have in pemphigus, of which the natural termination is death in six months unless cured by arsenic, and in which as a further feature of resemblance the mouth is in some cases affected as well as the skin. Nor is common pemphigus the only instance, for pityriasis rubra and certain rare forms of psoriasis and lichen ruber and planus, especially when occurring in the aged, show a tendency to

[1] In this my opinion coincided with Dr. Whistler's.

end in death by exhaustion. In lichen planus, as I have shown some years ago (and as was, indeed, first noticed by Dr. Sparks), there is an eruption on the tongue and in the cheeks. The precise form of the skin eruption does not seem to be of much consequence, for whatever it is, if it become very extensive, the patient's strength may fail and death may result. It is a very remarkable fact that these diseases, like the one which I have been describing, almost always begin in those who up to the time of the attack have enjoyed excellent health. The failure of strength always appears to be due to the extension of the local disease. The feature in which my cases differ from those to which I have just referred is in that the mouth inflamed before the skin was affected, and suffered throughout with preponderating severity. The disease of the skin was, as I remarked in the beginning of my paper, very different in the several cases. In one it appeared to be a direct extension from the lips to the skin of the chin, which became greatly thickened and covered with papillary outgrowths. In others it was bullous like pemphigus. In several, papillary outgrowths at the flexures occurred in great abundance, whilst in two a few eczematous patches on the hands and abdomen constituted all the skin affection that ever showed itself. In all the severe cases the nails inflamed.

It is impossible not to be struck with the apparently specific efficacy of opium, and the uselessness of other drugs. I think it may be fairly asserted that in all the cases in which opium was given early enough it cured the patient, and it not only removed the local symptoms but restored perfect health. In the first case it was not given at all, in the second only a week or two before death, and in Mr. Macnamara's patient the state was so far advanced that recovery was almost hopeless.

I do not doubt that this observation of the usefulness of opium may with advantage be extended to many chronic maladies affecting the skin in elderly persons.

I will ask permission to cite by way of illustration a

case which has recently been under my care which places the value of opium in a very strong light.

The patient (whose hands were represented in a portrait shown to the Society) was a lady of sixty-one, who had enjoyed excellent health until her hands began to inflame in the manner depicted. It was a sort of exfoliative dermatitis which developed with exact symmetry on hands and feet, and gradually spread. All the nails were inflamed, and when at length it attacked the scalp all the hair fell out. The lady had had much and very skilful treatment before she came to me, but the disease had steadily advanced. I had her under care for four months before I thought of trying opium, and during this period various local appliances were used, and arsenic and phosphorus and other internal remedies had a very fair trial. So far from improving, the state of things got worse and worse, and the general health began to fail very definitely. The patient had lost much flesh, she was bald, and her hands were quite disabled by swelling when I first prescribed opium. From this time improvement set in, and after about four months' use of the drug we could boast that her hands were quite well, that her nails and hair had grown again, and that she was in excellent health. The drug has now been laid aside for three months, and there has been no relapse.[1] The case differs chiefly from the others mentioned in this paper in that there never was any sore mouth.

[1] Since this paper was read I have seen this patient again with some symptoms of relapse, but they were quite slight.

(For report of the discussion on this paper, see 'Proceedings of the Royal Medical and Chirurgical Society,' New Series, vol. ii, p. 286.)

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