MEDICO-CHIRURGICAL TRANSACTIONS.

PUBLISHED BY

THE ROYAL

MEDICAL AND CHIRURGICAL SOCIETY

OF

LONDON.

VOLUME THE SIXTY-THIRD.

LONDON:
LONGMANS, GREEN, READER, AND DYER,
PATERNOSTER ROW.

1880.

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L.—Librarian.	C.—Member of Council.

The figures succeeding the words *Trans.* and *Pro.* show the number of Papers which have been contributed to the Transactions or Proceedings by the Fellow to whose name they are annexed. *Referee, Sci. Com.*, and *Lib. Com.*, with the dates of office, are attached to the names of those who have served on the Committees of the Society.

OCTOBER, 1880.

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., Physician to the Cheltenham General Hospital; 13, Suffolk square, Cheltenham.

1877 Abercrombie, John, M.D., Hospital for Sick Children; 49, Great Ormond street.

1851 *Acland, Henry Wentworth, M.D., F.R.S., Honorary Physician to H.R.H. the Prince of Wales; Regius Professor of Medicine, and Clinical Professor in the University of Oxford.

1847 Acosta, Elisha, M.D., 24, Rue de Luxembourg, St. Honoré, Paris.

Elected

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, M.A. and M.D., F.R.S., Lecturer on the Practice of Physic at the Leeds School of Medicine, and Physician to the Leeds General Infirmary; 35, Park square, Leeds. *Trans.* 3.

1879 Allchin, William Henry, M.B., Physician to the Westminster Hospital; 94, Wimpole street, Cavendish square.

1863 Althaus, Julius, M.D., Senior Physician to the Hospital for Epilepsy and Paralysis, Regent's park; 36, Bryanston street, Portman square. *Trans.* 2.

1862 Andrew, Edwyn, M.D., Hardwick House, St. John's Hill, Shrewsbury.

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

1820 Andrews, Thomas, M.D., Norfolk, Virginia.

1878 Arnold, John, Medical Officer of Health; Trinidad.

1870 Arnott, Rev. Henry, Braeside, Beckenham.

1819 †Arnott, James Moncrieff, F.R.S., Chapel House, Lady Bank, Fifeshire, and 36, Sussex gardens, Hyde park. L. 1826-8. V.P. 1832-3. T. 1835-40. C. 1846, 1855-6. P. 1847-8. *Referee*, 1842-45, 1849-54, 1857-64. *Lib. Com.* 1836. *Trans.* 8.

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

Elected

1836 Baird, Andrew Wood, M.D., Physician to the Dover Hospital; 7, Camden crescent, Dover, Kent.

1851 *Baker, Alfred, Surgeon to the Birmingham General Hospital; 20a, Temple row, Birmingham.

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

1865 Baker, William Morrant, Assistant Surgeon to, and Lecturer on Physiology at, St. Bartholomew's Hospital; Surgeon to the Evelina Hospital for Sick Children; 26, Wimpole street, Cavendish square. C. 1878-9. *Lib. Com.* 1876-7. *Trans.* 5.

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

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

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 Scotland; Physician to Richmond, Whitworth, and Hardwicke Hospitals; Consulting Physician to the Coombe Hospital; Member of the Senate of the Queen's University in Ireland; 11, Merrion square east, Dublin.

1847 †Barclay, Andrew Whyte, M.D., Physician to, and Lecturer on Medicine at, St. George's Hospital; Medical Officer of Health for Chelsea; 23a, Bruton street, Berkeley square. S. 1857-60. L. 1861-2. C. 1865-6. V.P. 1872-3. *Referee*, 1863-4, 1870-1, 1877-80. *Trans.* 2.

1879 Barker, Arthur Edward James, Assistant Surgeon to, and Assistant Professor of Clinical Surgery at, University College Hospital; 87, Harley street, Cavendish square. *Trans.* 1.

1862 Barker, Edgar, 21, Hyde park street.

1833 †Barker, Thomas Alfred, M.D., Consulting Physician to St. Thomas's Hospital; 27, Wimpole street. C. 1844-5. V.P. 1853-4. T. 1860-2. *Referee*, 1848-51. *Trans.* 6.

Elected

1876 Barlow, Thomas, M.D. and B.S. Lond., Assistant Physician to University College Hospital, and to the Hospital for Sick Children, Great Ormond Street; 10, Montague street, Russell square.

1861 Barnes, Robert, M.D., Obstetric Physician to, and Lecturer on Midwifery at, St. George's Hospital; 15, Harley street, Cavendish square. C. 1877-8. *Referee*, 1867-76. *Lib. Com.* 1869-73 *Trans.* 4.

1864 Barratt, Joseph Gillman, M.D., 8, Cleveland gardens, Bayswater.

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; 32, George street, Hanover square. C. 1876-77. *Referee*, 1868-75, 1879-80. *Trans.* 7.

1868 Bastian, Henry Charlton, M.A., M.D., F.R.S., Professor of Pathological Anatomy in University College, London, and Physician to University College Hospital; 20, Queen Anne street, Cavendish square. *Trans.* 1.

1874 Baxter, Evan Buchanan, M.D., Professor of Materia Medica at King's College, London; Assistant Physician to King's College Hospital; Examiner in Materia Medica at the University of London; 28, Weymouth street, Portland place.

1875 Beach, Fletcher, M.B., Medical Superintendent, Metropolitan District Asylum, Darenth, near Dartford, Kent.

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. Camb., Oak Lea, Harrogate.

1856 Beardsley, Amos, F.L.S., Bay villa, Grange-over-Sands, Lancashire.

Elected

1871 Beck, Marcus, M.S., Assistant Surgeon to University College Hospital; 30, Wimpole street, Cavendish square.

1858 Begley, William Chapman, A.M., M.D., late of the Middlesex County Lunatic Asylum, Hanwell; 26, Saint Peter's square, Hammersmith. C. 1877–8.

1871 Bellamy, Edward, Surgeon to, and Lecturer on Anatomy at, Charing Cross Hospital; Professor of Anatomy in the Science and Art Department, South Kensington; 17, Wimpole street, Cavendish square. *Lib. Com.* 1879-80.

1847 Bennet, James Henry, M.D., The Ferns, Weybridge, and Mentone.

1877 Bennett, William Henry, Assistant Surgeon to St. George's Hospital; Surgeon to the Belgrave Hospital for Children; 5, Savile row, Burlington gardens.

1845 †Berry, Edward Unwin, 76, Gower street, Bedford 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.

1815 †Billing, Archibald, M.D., F.R.S., Member of the Senate of the University of London; 34, Park lane. C. 1825. V.P. 1828-9.

1878 Bindon, William John Vereker, M.D., 2, Elm Villas, Kilburn.

1854 Bird, Peter Hinckes, F.L.S., 1, Norfolk square, Sussex gardens, Hyde park.

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.

Elected

1851 †Birkett, John, F.L.S., *Vice-President*, 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; 59, Green street, Grosvenor square. L. 1856-7. S. 1863-5. C. 1867-8. T. 1870-78. V.P. 1879-80. *Referee*, 1851-5, 1866, 1869, *Sci. Com.* 1863. *Lib. Com.* 1852. *Trans.* 8.

1866 Bishop, Edward, M.D., Cintra park, Upper Norwood.

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.

1867 Bloxam, John Astley, Assistant-Surgeon to, and Teacher of Operative Surgery in, Charing Cross Hospital; Surgeon for Out-Patients to the Lock Hospital; Junior Surgeon to the West London Hospital; 8, George street, Hanover square.

1823 Bojanus, Louis Henry, M.D., Wilna.

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.]

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

1841 †Bowman, William, 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.

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

Elected

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.]

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

1876 Bridges, Robert, M.B., Casualty Physician to St. Bartholomew's Hospital; Assistant Physician to the Hospital for Sick Children; and Physician to the Great Northern Hospital; 52, Bedford square.

1867 Bridgewater, Thomas, M.B. Lond., 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; 34, Seymour street, Portman square. *Trans.* 3.

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, James Crichton, M.D., LL.D., F.R.S.Ed., Lord Chancellor's Visitor in Lunacy; 7, Cumberland Terrace, Regent's Park.

1880 Browne, James William, M.B., 8 Radnor place, Gloucester square, W.

1874 Bruce, John Mitchell, M.D., Assistant Physician to, and Lecturer on Materia Medica at, the Charing Cross Hospital; Assistant Physician to the Hospital for Consumption, Brompton; 60, Queen Anne street. *Trans.* 1.

Elected

1871 Brunton, Thomas Lauder, M.D., F.R.S., Assistant Physician to, and Lecturer on Materia Medica at, St. Bartholomew's Hospital; 50, Welbeck street, Cavendish square. *Referee*, 1880.

1860 Bryant, Thomas, Surgeon to, and Lecturer on Surgery at, Guy's Hospital; 53, Upper Brook street, Grosvenor square. C. 1873-4. *Sci. Com.* 1863. *Lib. Com.* 1868-71. *Trans.* 8. *Pro.* 1.

1855 Bryant, Walter John, M.R.C.P. Edinb.; Physician to the Home for Incurable Children, Maida vale; 23A, Sussex square, Hyde park gardens.

1823 Buchanan, B. Bartlet, M.D.

1864 Buchanan, George, M.D., Medical Officer of the Local Government Board; 24, Nottingham place, Marylebone road.

1864 Buckle, Fleetwood, M.D.

1876 Bucknill, John Charles, M.D., F.R.S.; 39, Wimpole street, Cavendish square.

1839 Budd, George, M.D., F.R.S., Consulting Physician to the Seamen's Hospital, Greenwich; Ashleigh, Barnstaple. C. 1846-7. V.P. 1857. *Referee*, 1843-48, 1851, 1854-6. *Lib. Com.* 1839. *Trans.* 5.

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, 1875-80. *Lib. Com.* 1836. *Trans.* 2.

1837 †Busk, George, F.R.S., F.L.S., Consulting Surgeon to the Seamen's Hospital, Greenwich; Member of the Senate of the University of London; 32, Harley street, Cavendish square. C. 1847-8. V.P. 1855. T. 1866. *Referee*, 1846-54, 1857-65. *Lib. Com.* 1847. *Trans.* 4.

Elected

1873 Butlin, Henry Trentham, Assistant Surgeon to, and Demonstrator of Practical Surgery at, St. Bartholomew's Hospital; Assistant Surgeon to the West London Hospital; 47, Queen Anne street, Cavendish square. *Trans.* 3.

1871 Butt, William F., 25, Park street, Park lane.

1868 Buzzard, Thomas, M.D., Physician to the National Hospital for the Paralysed and Epileptic; 56, Grosvenor street, Grosvenor square.

1851 *Cadge, William, Surgeon to the Norfolk and Norwich Hospital; 24, St. Giles's street, Norwich. *Trans.* 1.

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; 69, Wimpole street, Cavendish square, W. *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; 32, Old Burlington street.

1868 Cavafy, John, M.D., Senior Assistant-Physician to St. George's Hospital; 2, Upper Berkeley street, Portman square.

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; 58, Welbeck street, Cavendish square.

1845 †Chalk, William Oliver, 3, Nottingham terrace, York gate, Regent's park. C. 1872-3.

Elected

1844 †Chambers, Thomas King, M.D., Hon. Physician to H.R.H. the Prince of Wales; Consulting Physician to St. Mary's Hospital; Consulting Physician to the Lock Hospital; Shrubs Hill House, Sunningdale, Staines. 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., Medical Registrar, St. Bartholomew's Hospital; 11, Wyndham place, Bryanston square.

1859 Chance, Frank, M.D., Burleigh House, Sydenham Hill.

1849 Chapman, Frederick, Old Friars, Richmond Green, Surrey.

1877 Charles, T. Cranstoun, M.D., Lecturer on Practical Physiology at St. Thomas's Hospital; 61, West Cromwell Road, South Kensington.

1868 Cheadle, Walter Butler, M.D., Assistant-Physician to, and Lecturer on Pathology and on Skin Diseases at, St. Mary's Hospital; Assistant Physician to the Hospital for Sick Children; 2, Hyde park place, Cumberland gate.

1879 Cheyne, William Watson, M.B., Assistant Surgeon and Demonstrator of Surgery to King's College Hospital; 6, Old Cavendish street, W.

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

1865 Cholmeley, William, M.D., Physician to the Great Northern Hospital, and to the Margaret Street Infirmary for Consumption; 63, Grosvenor street, Grosvenor square. *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. *Referee* 1874-80.

Elected

1860 Clark, Andrew, M.D., Physician to, and Lecturer on 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*, 1859-80. *Lib. Com.* 1847. *Trans.* 5.

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

1866 Clarke, William Fairlie, M.D., M.A. Oxon., Southborough, Tunbridge Wells. *Trans.* 2.

1842 †Clayton, Oscar Moore Passey, 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.

1853 Clover, Joseph Thomas, 3, Cavendish place, Cavendish square. C. 1873. *Sci. Com.* 1863.

1879 Clutton, H. H., M.A., M.B., Assistant Surgeon to St. Thomas's Hospital; 16, Palace road, Albert embankment, S.E.

1857 Coates, Charles, M.D., 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; 13, Spring gardens, Charing cross. *Trans.* 2.

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

1843 †Cooper, William White, Surgeon-Oculist in Ordinary to H.M. the Queen; Consulting Ophthalmic Surgeon to St. Mary's Hospital; 19, Berkeley square. C. 1858-9. V.P. 1873-4. *Lib. Com.* 1847, 1856-7.

Elected

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., Surgeon to the Belfast General Dispensary; 146, Donegall Pass, Belfast.

1853 Cory, William Gillett, M.D., 1, Caledonia place, Clifton, Bristol.

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.

1877 Coupland, Sidney, M.D., Physician to, and Lecturer on Pathological Anatomy 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; 19, George street, Hanover square.

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

1868 Crawford, Thomas, M.D., Deputy Inspector-General of Hospitals (India); Umbalah, Punjaub.

1873 Creighton, Charles, M.B., Demonstrator of Anatomy, University of Cambridge, Anatomical Museum, Cambridge.

1869 *Cresswell, Pearson R., Dowlais, Merthyr Tydvil.

1874 Cripps, William Harrison, Surgical Registrar to St. Bartholomew's Hospital; Surgeon to the Great Northern Hospital; Assistant Surgeon to the Royal Free Hospital; 6, Stratford place, Oxford street. *Trans.* 1.

1847 †Critchett, George, Consulting Surgeon to the Royal London Ophthalmic Hospital, Moorfields; Ophthalmic Surgeon to the Middlesex Hospital; 21, Harley street, Cavendish square. C. 1865. V.P. 1872. *Referee*, 1867-71. *Trans.* 1.

Elected

1868 Croft, John, Surgeon to, and Lecturer on Operative Surgery at, St. Thomas's Hospital; 61, Brook street, Grosvenor square. *Lib. Com.* 1877-8.

1862 Crompton, Samuel, M.D., Physician to the Salford Royal Hospital and Dispensary; 24, St. Ann's square, Manchester.

1837 Crookes, John Farrar, Grimethorpe, Tunbridge Wells.

1860 Cross, Richard, M.D., Carlton House, Belmont road, Scarborough.

1872 Crosse, Thomas William, Surgeon to the Norfolk and Norwich Hospital; 22, St. Giles's street, Norwich.

1849 *Crowfoot, William Edward, Beccles, Suffolk.

1879 Cumberbatch, A. Elkin, Demonstrator of Anatomy at St. Bartholomew's Hospital; 17, Queen Anne street.

1846 Curling, Henry, Consulting Surgeon to the Margate Royal Sea-Bathing Infirmary, and the Ramsgate Seamen's Infirmary; Ramsgate, Kent.

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 at King's College, London, and Assistant Physician to King's College Hospital; Examiner in Anatomy at the University of London; 3, George street, Hanover square.

1847 Currey, John Edmund, M.D., Lismore, County Waterford.

1822 Cusack, Christopher John, Chateau d'Eu, France.

1872 Dalby, William Bartlett, M.B., Lecturer on Aural Surgery at St. George's Hospital; 18, Savile row. *Trans.* 2.

1836 *Daniel, James Stock, Ramsgate, Kent.

1877 Darbishire, Samuel Dukinfield, M.B., Physician to the Radcliffe Infirmary, Oxford.

Elected

1879 Darwin, Francis, M.B., Down, Kent.

1848 Daubeny, Henry, M.D., San Remo, Italy.

1874 Davidson, Alexander, M.D., Physician to the Liverpool Northern Hospital; 49, Rodney street, Liverpool.

1853 Davies, Robert Coker Nash, Rye, Sussex.

1852 Davies, William, M.D., 18, Gay street, Bath.

1876 Davies-Colley, J. Neville C., M.C., Assistant Surgeon to, and Lecturer on Anatomy at, Guy's Hospital; 36, Harley street, Cavendish square. *Trans.* 1.

1878 Davy, Richard, Surgeon to, and Lecturer on Surgery at, the Westminster Hospital; 33, Welbeck street, Cavendish square.

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 St. George's Hospital; 29, Chesham Street, Belgrave square.

1846 *Denton, Samuel Best, M.D., Ivy Lodge, Hornsea, Hull.

1859 †Dickinson, William Howship, M.D., Physician to, and Lecturer on Medicine at St. George's Hospital, and Senior Physician to the Hospital for Sick Children Examiner in Medicine at the University of London; 9, Chesterfield street, Mayfair. C. 1874-5. *Referee*, 1869-73. *Sci. Com.* 1867-79 *Trans.* 13.

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

1862 Dobell, Horace B., M.D., Consulting Physician to the Royal Hospital for Diseases of the Chest, City road; 84, Harley street. *Trans.* 2.

1845 Dodd, John.

1879 Donkin, Horatio, M.B., 60, Upper Berkeley street, Portman square.

Elected

1877 Doran, Alban Henry Griffiths, Surgeon to Out-Patients, Samaritan Hospital; Pathological Assistant to the Museum of the Royal College of Surgeons of England; 51, Seymour street, Portman square.

1863 Down, John Langdon Haydon, M.D., Physician to the London Hospital; 39, Welbeck street, Cavendish square. C. 1880. *Trans.* 2.

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

1879 Drewitt, F. G. Dawtrey, M.B. Ox., Hospital for Sick Children, Great Ormond street.

1853 Druitt, Robert, F.R.C.P. [8, Strathmore gardens, Kensington Mall.] *Trans.* 2.

1865 Drysdale, Charles Robert, M.D., Physician to the Farringdon Dispensary; Assistant-Physician to the Metropolitan Free Hospital; 17, Woburn place, Russell square.

1865 Duckworth, Dyce, M.D., Assistant-Physician to, and Lecturer on Skin Diseases at, St. Bartholomew's Hospital; 11, Grafton street, Bond street. *Trans.* 1.

1876 Dudley, William Lewis, M.D., Physician to the City Dispensary; 125, 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.

1874 Duka, Theodore, M.D., [Surgeon-Major, H.M.'s Bengal Army]; 38, Montagu square.

1871 Duke, Benjamin, 1, Cavendish terrace, Clapham Common.

1871 *Dukes, Clement, M.D. and B.S., Horton crescent, Rugby, Warwickshire.

1867 Dukes, M. Charles, M.D., Wellesley Villa, Wellesley road, Croydon.

1880 Dunbar, James John MacWhirter, M.B., 77, Ladbroke grove, Kensington park gardens, W.

Elected

1877 Duncan, James Matthews, M.D., LL.D. Ed., F.R.S. Ed., Obstetric Physician to, and Lecturer on Midwifery and Diseases of Women at, St. Bartholomew's Hospital; Examiner in Obstetric Medicine, University of London; 71, Brook street, Grosvenor square.

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

1874 Durham, Frederic, M.B., Surgical Registrar to Guy's Hospital; 38, Brook street, Grosvenor square.

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

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

1836 Earle, James William, late of Norwich.

1868 Eastes, George, M.B. Lond., Surgeon-Accoucheur to the Western General Dispensary; 69, Connaught street, Hyde park square.

1824 Edwards, George.

1823 Egerton, Charles Chandler, Kendall Lodge, Epping.

1869 Elam, Charles, M.D., Assistant-Physician to the National Hospital for the Paralysed and Epileptic; 75, Harley street, Cavendish square.

1861 *Elliot, Robert, M.D., Physician to the Fever Hospital and to the Dispensary, Carlisle; Coroner for Carlisle; 35, Lowther street, Carlisle.

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

Elected

1874 Evans, George Henry, M.D.

1879 Eve, Frederic S., Curator of the Museum, St. Bartholomew's Hospital; 14, Furnival's Inn, Holborn. *Trans.* 2.

1877 Ewart, William, M.B., Assistant Physician to the Hospital for Consumption, Brompton; Lecturer on Physiological Chemistry at St. George's Hospital; 33, Curzon street, Mayfair.

1875 *Fagan, John, Surgeon to the Belfast Hospital for Sick Children; 11, College square north, Belfast.

1864 Fagge, Charles Hilton, M.D., Senior Assistant Physician to, and Lecturer on Pathology at, Guy's Hospital; Examiner in Medicine at the University of London; 11, St. Thomas's street, Southwark. C. 1880. *Referee*, 1874-9. *Sci. Com.* 1879. *Lib. Com.* 1875-9. *Trans.* 6.

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

1858 Falconer, Randle Wilbraham, M.D., Consulting Physician to the Royal United Hospital, Bath; 22, Bennett street, Bath.

1862 Farquharson, Robert, M.D., M.P., 23, Brook street, Grosvenor square. *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.Ed., 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; Surgeon-General, late Bengal Medical Service; Examining Medical Officer to the Secretary of State for India in Council; President of the Indian Medical Board; 16, Granville place, Portman square.

1872 *Fenwick, John C. J., M.D., Physician to the Durham County Hospital; 16, Old Elvet, Durham.

1863 Fenwick, Samuel, M.D., Physician to the London Hospital; 29, Harley street, Cavendish square. C. 1880. *Trans.* 3.

Elected

1880 Ferrier, David, M.D., F.R.S., Professor of Forensic Medicine at King's College, and Assistant Physician to King's College Hospital; Physician for Out-patients to the National Hospital for the Paralysed and Epileptic; Examiner in Forensic Medicine at the University of London; 16, Upper Berkeley street, Portman square, W.

1852 *Field, Alfred George.

1849 Fincham, George Tupman, M.D., Physician to, and Joint Lecturer on Medicine at, the Westminster Hospital; 13, Belgrave road, Pimlico. C. 1871.

1879 Finlay, David White, M.D., Assistant Physician to the Middlesex Hospital; 21, Montagu street, Portman square.

1866 Fish, John Crockett, B.A., M.D. Camb., Assistant Physician to the West London Hospital; 92, Wimpole street, Cavendish square.

1860 Fitzgerald, Thomas George, Deputy Surgeon-General, [6, Whitehall yard.]

1866 Fitzpatrick, Thomas, M.D., M.A., Dublin; Physician to the Western General Dispensary; 30, Sussex gardens, Hyde park.

1842 Fletcher, Thomas Bell Elcock, M.D., Consulting Physician to the Birmingham General Hospital; 43, Clarendon square, Leamington. *Trans.* 1.

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

1877 Fonmartin, Henry de, M.D.

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

1852 †Forster, John Cooper, *Treasurer*, Surgeon to Guy's Hospital; Examiner in Surgery at the University of London; 29, Upper Grosvenor street. C. 1868-9. S. 1873-5. V.P. 1877-8. T. 1879-80. *Referee*, 1870-2, 1876, *Pro.* 1.

1877 *Fortescue, George, M.B., late Surgeon to the Sydney Infirmary; 6, Lyons terrace, Sydney, New South Wales.

Elected

1865 Foster, Balthazar Walter, M.D., Professor of Medicine at the Queen's College, Birmingham, and Physician to the Birmingham General Hospital; 16, Temple row, Birmingham.

1859 Fox, Edward Long, M.D., Consulting Physician to the Bristol Royal Infirmary, and Lecturer on Medicine at the Bristol School of Medicine; Church House, Clifton, Gloucestershire.

1858 Fox, Wilson, M.D., F.R.S., Physician-Extraordinary to H.M. the Queen; Physician in Ordinary to H.R.H. the Duke of Edinburgh; Holme Professor of Clinical Medicine in University College, London, and Physician to University College Hospital; 67, Grosvenor street. C. 1875-6. *Referee*, 1869-74. *Lib. Com.* 1866-70, 1874. *Trans.* 3.

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

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

1868 Freeman, William Henry, 21, St. George's square, South Belgravia.

1836 †French, John George, 10, Cunningham place, Maida hill. C. 1852-3.

1876 Furner, Willoughby, 111, King's road, Brighton.

1864 *Gairdner, William Tennant, M.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 Glasgow Royal Infirmary; 225, St. Vincent street, Glasgow.

1874 Galabin, Alfred Lewis, M.A., M.D., Assistant Obstetric Physician to, and Lecturer on Midwifery and the Diseases of Women at, Guy's Hospital; Assistant-Physician to the Hospital for Sick Children; 14, St. Thomas's street, Southwark. *Trans.* 2.

1865 Gant, Frederick James, Senior Surgeon to the Royal Free Hospital; 16, Connaught square, Hyde park. C. 1880. *Trans.* 3.

1867 Garland, Edward Charles, L.R.C.P. Edin., Yeovil, Somerset.

Elected

1867 Garlike, Thomas W., Malvern Villa, Churchfield road, Ealing.

1854 Garrod, Alfred Baring, M.D., F.R.S., *Vice-President*, Consulting Physician to King's College Hospital; 10, Harley street, Cavendish square. C. 1867. V.P. 1880. *Referee* 1855-65. *Trans.* 8.

1879 Garstang, Thomas Walter Harropp, Oakleigh, Dobcross, near Manchester.

1851 †Gaskoin, George, Surgeon to the British Hospital for Diseases of the Skin; 7, Westbourne park. C. 1875-6. *Trans.* 2.

1819 Gaulter, Henry.

1848 †Gay, John, Senior Surgeon to the Great Northern Hospital, and Consulting Surgeon to the Asylum for Idiots; 34, Finsbury place. C. 1874-5.

1866 Gee, Samuel Jones, M.D., Physician to, and Lecturer on Medicine at, St. Bartholomew's Hospital; Physician to the Hospital for Sick Children; 54, Harley street, Cavendish square. *Sci. Com.* 1879. *Lib. Com.* 1871-6. *Trans.* 1.

1878 Gervis, Henry, M.D., Obstetric Physician to, and Lecturer on Obstetric Medicine at, St. Thomas's Hospital; Examiner in Obstetric Medicine at the University of London; 40, Harley street, Cavendish square.

1880 Gibbons, Robert Alexander, M.D., Assistant Physician to St. John's Hospital for Skin Diseases; 80, Cadogan place.

1877 Godlee, Rickman John, Assistant-Surgeon to University College Hospital; and Demonstrator of Anatomy at University College; 22, Henrietta street, Cavendish square.

1870 Godson, Clement, M.D., Assistant-Physician-Accoucheur to St. Bartholomew's Hospital; 9, Grosvenor street, Grosvenor square.

1867 Goodeve, Edward, M.B., Hon. Physician to H.M. the Queen; late Surgeon-Major, H.M.'s Bengal Army; Drimagh, Stoke Bishop, near Bristol.

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.

1877 Gould, Alfred Pearce, M.S., Assistant Surgeon to, and Lecturer on Anatomy at, the Westminster Hospital; 16, Queen Anne street, Cavendish square.

1873 Gowers, William Richard, M.D., Assistant Professor of Clinical Medicine at University College, and Assistant-Physician to University College Hospital; 50, Queen Anne street. *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; Crawleydown park, Worth, 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.

1875 Greenfield, William Smith, M.D., Professor Superintendent at the Brown Institution; Assistant Physician to, and Lecturer on Morbid Anatomy at, St. Thomas's Hospital; 15, Palace road, Albert embankment. *Sci. Com.* 1879.

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. C. 1871-2. *Referee*, 1876-7. *Trans.* 1.

1860 Greenhow, Edward Headlam, M.D., F.R.S., Consulting Physician to the Middlesex Hospital; and Consulting Physician to the Western General Dispensary; 14A, Manchester square. C. 1876-7. *Referee*, 1870-5. *Trans.* 3.

1868 Grigg, William Chapman, M.D., Assistant Obstetric Physician to the Westminster Hospital; Physician to the In-Patients, Queen Charlotte's Lying-in-Hospital; Assistant-Physician to the Victoria Hospital for Children; 6, Curzon street, Mayfair.

Elected

1852 Grove, John, Westgate court, Canterbury.

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 the Queen; 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.

1837 Gully, James Manby, M.D.

1854 Habershon, Samuel Osborne, M.D., Physician to Guy's Hospital; 70, Brook street, Grosvenor square. S. 1867. C. 1869-70. *Referee*, 1862-6, 1868, 1871-80. *Trans.* 3.

1849 Hailey, Hammett, F.L.S., Tickford Lodge, Newport Pagnell, Bucks.

1870 Hamilton, Robert, Surgeon to the South 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.

1836 Harding, John Fosse, Ulverstone House, Uckfield, Sussex. C. 1858-9.

1856 Hare, Charles John, M.D., late Professor of Clinical Medicine in University College, London, and Physician to University College Hospital; 57, Brook street, Grosvenor square. C. 1873-4.

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 Clinical Medicine at, St. Thomas's Hospital; 39, Brook street, Grosvenor square. S. 1875-7. C. 1879-80. *Referee*, 1871-4. *Sci. Com.* 1879. *Trans.* 8.

1866 Harper, Philip H., 30, Cambridge street, Hyde park.

1859 Harris, Francis, M.D., F.L.S., 24, Cavendish square.

Elected

1880 Harris, Vincent Dormer, M.D., Casualty Physician to St. Bartholomew's Hospital, and Assistant Physician to the Victoria Park Hospital; 23, Upper Berkeley street, Portman square.

1872 Harris, William H., M.D., Professor of Midwifery and Diseases of Women and Children, Madras Medical College, Madras.

1870 Harrison, Reginald, Surgeon to the Liverpool Royal Infirmary, and Lecturer on Surgery at the School of Medicine; 38, Rodney street, Liverpool.

1854 Haviland, Alfred, Medical Officer of Health for the combined Districts of Northamptonshire; Northampton.

1870 Haward, J. Warrington, Surgeon to St. George's Hospital; Surgeon to the Hospital for Sick Children; 16, Savile row, Burlington gardens. *Trans.* 1.

1828 †Hawkins, Cæsar Henry, F.R.S., Sergeant-Surgeon to H.M. the Queen, and Consulting Surgeon to St. George's Hospital; 26, Grosvenor street. C. 1830-1, 1860. V.P. 1838-9. T. 1841-4. P. 1855-6. *Referee*, 1843-7, 1850-4, 1857-9, 1861-80. *Trans.* 12.

1838 †Hawkins, Charles, Inspector of Anatomical Schools in London; 27, Savile row, Burlington gardens. C. 1846-7. S. 1850. V.P. 1858. T. 1861-2. *Referee*, 1859-60. *Lib. Com.* 1843. *Trans.* 2.

1848 †Hawksley, Thomas, M.D., Consulting Physician to the Margaret street Dispensary for Consumption and Diseases of the Chest; 31, Grosvenor street.

1875 Hayes, Thomas Crawford, M.D., Assistant-Physician-Accoucheur and Assistant-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, Church House, Oldbury, Worcestershire.

Elected

1848 *Heale, James Newton, M.D., care of the Secretary of the Winchester Hospital, Winchester, Hants.

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.* 2.

1850 Heaton, George, M.D., Boston, U.S.

1821 Herberski, Vincent, M.D., Professor of Medicine in the University of Wilna.

1877 Herman, George Ernest, M.D., Assistant Obstetric Physician to the London Hospital; 7, West street, Finsbury circus.

1877 Heron, George Allan, M.D., Assistant Physician to the City of London Hospital for Diseases of the Chest, Victoria Park; Assistant Physician to the West London Hospital for the Paralysed and Epileptic; 40, Margaret street, Cavendish square.

1843 Hewett, Prescott Gardner, F.R.S., Serjeant-Surgeon-Extraordinary 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 Academy of Medicine, and of the "Société de Chirurgie," Paris; 1, Chesterfield street, Mayfair. C. 1859. V.P. 1866-7. *Referee*, 1850-8, 1860-5, 1868-80. *Sci. Com.* 1863. *Lib. Com.* 1846-7. *Trans.* 7.

1855 Hewitt, Graily, M.D., Professor of Midwifery in University College, London, and Obstetric Physician to University College Hospital; 36, Berkeley square. C. 1876. *Referee*, 1868-75, 1877-80. *Lib. Com.* 1868, 1874.

1872 Heyn, Julius Charles William, M.D., 42, Westbourne terrace, Hyde park.

1873 Higgens, Charles, Assistant Ophthalmic Surgeon to Guy's Hospital; 38, Brook street, Grosvenor square. *Trans.* 1.

1862 Hill, M. Berkeley, M.B. Lond., Professor of Clinical Surgery in University College, London, and Surgeon to University College Hospital; Surgeon to the Lock Hospital; 55, Wimpole street, Cavendish square. C. 1878-9.

Elected

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

1859 Hird, Francis, Senior Surgeon to the Charing Cross Hospital; 13, Old Burlington street.

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

1872 Hogg, Francis Roberts, M.D., Army Medical School, Netley, Southampton.

1843 †Holden, Luther, Surgeon to St. Bartholomew's Hospital; Consulting Surgeon to the Metropolitan Dispensary; Surgeon to the Foundling Hospital; 65, Gower street, Bedford square. C. 1859. L. 1865. V.P. 1874. *Referee*, 1866-7. *Lib. Com.* 1858.

1879 Holland, Philip Alexander, M.A., St. Bartholomew's Hospital.

1868 Hollis, William Ainslie, M.A., M.B., Camb., Assistant-Physician to the Sussex County Hospital; Park Gate, Preston road, Brighton.

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

1856 Holmes, Timothy, M.A. Camb., *Secretary*, Surgeon to, and Lecturer on Surgery at, St. George's Hospital; Surgeon in Chief to the Metropolitan Police Force; 18, Great Cumberland place, Hyde park. C. 1869-70. L. 1873-7. S. 1878-80. *Referee*, 1866-8, 1872. *Sci. Com.* 1867. *Lib. Com.* 1863-5. *Trans.* 6.

1846 †Holt, Barnard Wight, *Vice-President*, Consulting Surgeon to, and Lecturer on Clinical Surgery at, the Westminster Hospital; Medical Officer of Health for Westminster; 14, Savile row, Burlington gardens. C. 1862-3. V.P. 1879-80.

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

1878 Hood, Donald William Charles, M.B., 43, Green street, Park lane.

1879 Hood, Francis E. C.

Elected

1878 Houghton, Walter B., M.D., Assistant Physician to Charing Cross Hospital; 26, Cavendish square.

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

1865 Howard, Edward, M.D.

1874 Howse, Henry Greenway, M.S. Lond., Surgeon to, and Lecturer on Anatomy at, Guy's Hospital; Surgeon to the Evelina Hospital for Sick Children; 10, St. Thomas's street, Southwark. *Sci. Com.* 1879. *Trans.* 2.

1877 *Hudson, Robert Samuel, M.D., 58, West-end, Redruth, Cornwall.

1857 Hulke, John Whitaker, F.R.S., *Librarian*, Surgeon to, and Lecturer on Surgery at, the Middlesex Hospital; Surgeon to the Royal London Ophthalmic Hospital, Moorfields; 10, Old Burlington street. C. 1871-2. S. 1876-7. L. 1879-80. *Sci. Com.* 1867. *Lib. Com.* 1864-8. *Trans.* 8.

1857 Hulme, Edward Charles, Woodbridge road, Guildford. *Trans.* 1.

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 Human Anatomy and Physiology in the Cambridge University Medical School; Cambridge. *Trans.* 6.

1873 Hunter, William Guyer, M.D., Principal of, and Professor of Medicine in, Grant Medical College, Bombay; Surgeon-Major, Bombay Army, Bombay.

1849 Hussey, Edward Law, Senior Surgeon to the Radcliffe Infirmary, and Consulting Surgeon to the County Lunatic Asylum and the Warneford Asylum; 8, St. Aldate's, Oxford. *Trans.* 1.

Elected

1856 Hutchinson, Jonathan, Senior Surgeon to 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. *Referee*, 1876-80. *Lib. Com.* 1864-5. *Trans.* 9. *Pro.* 2.

1820 Hutchinson, William, M.D.

1840 †Hutton, Charles, M.D., Consulting Physician to the General Lying-in Hospital; 26, Lowndes street, Belgrave square. C. 1858-9.

1866 Iles, Francis Henry Wilson, M.D., Watford, Herts.

1847 Image, William Edmund, Consulting Surgeon to the Suffolk General Hospital; Bury St. Edmund's, Suffolk. *Trans.* 1.

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

1876 Irvine, James Pearson, M.D., Assistant Physician to, and Lecturer on Forensic Medicine at, the Charing Cross Hospital; 3, Mansfield street, Portland place [deceased].

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.

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

1863 Jackson, Thomas Vincent, Surgeon to the South Staffordshire General Hospital; Darlington st., Wolverhampton.

1841 Jacobovics, Maximilian Moritz, M.D., Vienna.

1825 James, John B., M.D.

1840 *Jenks, George Samuel, M.D., 18, Circus, Bath.

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.

Elected

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., *Librarian*, 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. L. 1878-80. *Referee*, 1853-61, 1864-9. *Lib. Com.* 1860-1. *Trans.* 10.

1868 JOHNSTON, WILLIAM, M.D., 44, Princes square, Hyde park.

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

1876 JONES, LESLIE, M.D., Medical Officer of Health for Blackpool; 3, Brighton Parade, Blackpool, Lancashire.

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.]

1837 †JONES, THOMAS WILLIAM, M.D., Bylocks, Enfield Highway. C. 1858.

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

1865 JORDAN, FURNEAUX, Surgeon to the Queen's Hospital, and Professor of Surgery at the Queen's College, Birmingham; 22, Colmore row, Birmingham.

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

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

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

1877 *KHORY, RUSTOMJEE NASERWANJEE, M.D. Brussels; Physician to the Parell Dispensary, Bombay; Lecturer to Native Midwives, Grant Medical College, Bombay.

Elected

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

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

1855 LANE, JAMES ROBERT, Surgeon to, and Lecturer on Surgery at, St. Mary's Hospital; Surgeon to the Lock Hospital; 49, Norfolk square, Hyde park. C. 1870. *Referee*, 1869, 1877-80. *Lib. Com.* 1869. *Trans.* 1.

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.

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

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.

1841 *LASHMAR, CHARLES, M.D., 83, North End, Croydon, Surrey.

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.

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.* 13. *Pro.* 2.

1877 LEESON, ARTHUR EDMUND, M.A., M.D., 45, Devonshire street, Portland Place.

Elected

1869 LEGG, JOHN WICKHAM, M.D., Assistant Physician to, and Lecturer on Pathological Anatomy at, St. Bartholomew's Hospital; 47, Green street, Park lane. *Lib. Com.* 1878-80. *Trans.* 2.

1836 LEIGHTON, FREDERICK, M.D.

1872 LIEBREICH, RICHARD, Consulting Ophthalmic Surgeon to St. Thomas's Hospital; 16, Albemarle street, Piccadilly.

1806 LIND, JOHN, M.D.

1878 LISTER, JOSEPH, 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., Surgeon to the Royal Eye Hospital Manchester; 21, St. John's street, Manchester.

1871 LITTLE, LOUIS STROMEYER, Shanghai, China.

1870 LIVINGSTON, JOHN, M.D., New Barnet, Hertfordshire.

1819 LLOYD, ROBERT, M.D.

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

1860 LONGMORE, 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.

1877 LOWNE, BENJAMIN THOMPSON, Lecturer on Physiology, Middlesex Hospital Medical School; 65, Cambridge gardens, Notting hill, W.

1852 LUKE, JAMES, F.R.S., Consulting Surgeon to the London Hospital; Fingest Grove, High Wycombe, Bucks. C. 1858. *Referee*, 1854-7, 1859-64. *Trans.* 4.

Elected

1879 Lyell, Robert W., Assistant Surgeon to the Middlesex Hospital; Assistant Surgeon to the Royal London Ophthalmic Hospital; 26, Harley street, Cavendish square. *Trans.* 1.

1857 Lyon, Felix William, M.D., 49, Albany street, Leith, near Edinburgh.

1867 Maberly, George Frederick, 98, Collins street east, Melbourne, Victoria.

1873 MacCarthy, Jeremiah, M.A., Surgeon to, and Lecturer on Physiology at, the London Hospital; 15, Finsbury square.

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

1862 *M'Donnell, Robert, M.D., F.R.S., Surgeon to Steevens' Hospital; 14, Lower Pembroke street, Dublin. *Trans.* 2.

1846 M'Ewen, William, M.D., Surgeon to Chester Castle; 27, Nicholas street, Chester.

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

1823 †Macilwain, George, Consulting Surgeon to the Finsbury Dispensary, Matching, Harlow, Essex. C. 1829-30. V.P. 1848. *Referee*, 1849-50. *Lib. Com.* 1838. *Trans.* 1.

1822 Macintosh, Richard, M.D.

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

1873 MacKellar, Alexander Oberlin, M.S.I., Assistant Surgeon, St. Thomas's Hospital; Albert Embankment, Westminster Bridge.

1876 Mackey, Edward, M.D., 123, Western road, Brighton.

1854 *Mackinder, Draper, M.D., Consulting Surgeon to the Dispensary, Gainsborough, Lincolnshire.

1879 Maclagan, Thomas John, M.D., 9, Cadogan place, Belgrave square.

1860 Maclean, John, M.D., 24, Portman street, Portman square.

Elected

1876 MACNAMARA, CHARLES, Surgeon to the Westminster Hospital; Surgeon Major Bengal Medical Service; Fellow of the Calcutta University; 13, Grosvenor street.

1842 MACNAUGHT, JOHN, M.D., 74, Huskisson street, Liverpool.

1880 MADDICK, EDMUND D., 184, Brixton road.

1876 MALLAM, BENJAMIN, Meadow Side, Leacroft road, Staines.

1855 MARCET, WILLIAM, M.D., F.R.S.; Villa Bianca, Cannes, and 39, Grosvenor street. C. 1871. *Referee*, 1866-70. *Sci. Com.* 1863. *Lib. Com.* 1866-8. *Trans.* 3.

1867 MARSH, F. HOWARD, Assistant-Surgeon to St. Bartholomew's Hospital; Assistant Surgeon to the Hospital for Sick Children, Great Ormond street; 36, Bruton street, Berkeley square. *Lib. Com.* 1880. *Trans.* 2.

1838 MARSH, THOMAS PARR, M.D.

1851 MARSHALL, JOHN, F.R.S., Professor of Anatomy to the Royal Academy of Arts; Professor of Surgery in University College, London, and Surgeon to University College Hospital; 10, Savile row, Burlington gardens. C. 1866. V.P. 1875-6. *Referee*, 1867, 1871-4, 1877-80. *Trans.* 2.

1864 MASON, FRANCIS, Surgeon to, and Lecturer on Anatomy at, St. Thomas's Hospital; 5, Brook street, Grosvenor square. C. 1880. *Trans.* 1.

1839 MEADE, RICHARD HENRY, Consulting Surgeon to the Bradford Infirmary; Bradford, Yorkshire. *Trans.* 1.

1870 MEADOWS, ALFRED, M.D., Physician-Accoucheur to, and Lecturer on Midwifery at, St. Mary's Hospital; 27, George street, Hanover square. *Lib. Com.* 1875-7.

1865 MEDWIN, AARON GEORGE, M.D., Dental Surgeon to the Royal Kent Dispensary, 11, Montpellier row, Blackheath, Kent, and 34, Bruton street, Berkeley square.

1880 MEREDITH, WILLIAM APPLETON, M.B., C.M., Assistant Surgeon to the Samaritan Free Hospital for Women and Children; 14, Old Burlington street.

Elected

1867 Meredyth, Colomiati, M.D., 10, George street, Hanover 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; 19, Temple row, Birmingham.

1854 Middleship, Edward Archibald.

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

1844 †Montefiore, Nathaniel, 18, Portman square.

1836 Moore, George, M.D., Hastings.

1873 Moore, Norman, M.D., Warden of the College and Lecturer on Comparative Anatomy, Demonstrator of Morbid Anatomy, St. Bartholomew's Hospital; the College, St. Bartholomew's Hospital.

1861 Morehead, Charles, M.D., Hon. Surgeon to H.M. the Queen; Deputy-Inspector General of Hospitals; 11, North manor place, Edinburgh. *Referee*, 1862-4.

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

1861 Morgan, John Edward, M.D., Physician to the Manchester Royal Infirmary, and Professor of Medicine in the Owens College, Manchester; 1, St. Peter's square, Manchester.

1878 Morgan, John Hammond, M.A., Assistant Surgeon to the Hospital for Sick Children, Great Ormond street, and to the West London Hospital; 12, Chapel street, Park lane.

1874 Morris, Henry, M.A. Lond., Surgeon to, and Lecturer on Clinical Surgery at, the Middlesex Hospital; 2, Mansfield street, Portland place. *Trans.* 4.

1879 Morris, Malcolm Alexander, Lecturer on Skin Diseases at St. Mary's Hospital; 63, Montagu square.

1851 Mouat, Frederic John, M.D., Deputy Inspector-General of Hospitals; Medical Inspector to the Local Government Board; and Member of the Senate of the University of Calcutta; 12, Durham villas, Kensington.

Elected

1868 Moxon, Walter, M.D., F.L.S., Physician to, and Lecturer on Clinical Medicine at, Guy's Hospital; 6, Finsbury Circus. *Referee*, 1879-80. *Trans.* 1.

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

1875 Murphy, William Kirkpatrick, M.A., M.D., 29, Queen Anne street, Cavendish square.

1873 Murray, Ivor, M.D., F.R.S. Ed., 8, Huntress Row, Scarborough.

1863 Myers, Arthur Bowen Richards, Surgeon to the 1st Battalion, Coldstream Guards; Hospital, Vincent square, Westminster. C. 1878-9. *Lib. Com.* 1877.

1876 Napier, William Donald, 22, George street, Hanover square.

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; 4, Wimpole street, Cavendish square.

1843 †Newton, Edward, 85, Gloucester terrace, Hyde Park. C. 1863-4.

1868 Nicholls, James, M.D., Duke street, Chelmsford, Essex.

1849 Norman, Henry Burford, Portland Lodge, Southsea, Hants. *Lib. Com.* 1857.

1847 *Nourse, William Edward Charles, late Surgeon to the Brighton Children's Hospital; Bouverie House, Mount Radford, Exeter.

1864 Nunn, Thomas William, Consulting Surgeon to the Middlesex Hospital; 8, Stratford place, Oxford street.

1870 Nunneley, Frederick Barham, M.D. *Trans.* 2.

1847 O'Connor, Thomas, March, Cambridgeshire.

Elected

1858 Ogle, John William, M.D., Consulting-Physician to St. George's Hospital; 30, Cavendish square. C. 1873. *Referee*, 1864-72. *Trans.* 4.

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

1860 Ogle, William, M.D., Superintendent of Statistics in the Registrar General's Department, Somerset House; 10, Gordon street, Gordon square. S. 1868-70. C. 1876-7. *Lib. Com.* 1871-5. *Trans.* 4.

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

1871 *O'Neill, William, M.D., Physician to the Lincoln Lunatic Hospital, Lincoln.

1873 Ord, William Miller, M.D., Physician to, and Lecturer on Medicine at, St. Thomas's Hospital; 7, Brook street, Hanover square. *Trans.* 6.

1877 Ormerod, Joseph Arderne, M.B., Casualty Physician to St. Bartholomew's Hospital; 25, Upper Wimpole street.

1875 Osborn, Samuel, 17, Gresham park, Brixton, and 10, Maddox street, Regent street.

1879 Owen, Edmund, Assistant Surgeon to St. Mary's Hospital; 49, Seymour street, Portman square.

1874 Page, Herbert William, M.A., M.C. Cantab., Assistant Surgeon to, and Lecturer on Operative and Practical Surgery at, St. Mary's Hospital; 28, New Cavendish street. *Trans.* 1.

1847 *Page, William Bousfield, Consulting Surgeon to the Cumberland Infirmary, Carlisle. *Trans.* 2.

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; Member of the Senate of the University of London; 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.* 11.

Elected

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

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

1873 Parker, Robert William, Assistant-Surgeon to the East London Children's Hospital; 8, Old Cavendish-street. *Trans.* 2.

1841 Parkin, John, M.D., 5, Codrington place, Brighton.

1865 Pavy, Frederick William, M.D., F.R.S., Physician to, and Lecturer on Medicine at, Guy's Hospital; 35, Grosvenor street. *Referee*, 1871-80. *Trans.* 1.

1869 Payne, Joseph Frank, M.D., Assistant-Physician to, and Lecturer on Pathological Anatomy at, St. Thomas's Hospital; 78, Wimpole street, Cavendish square. *Sci. Com.* 1879. *Lib. Com.* 1878-80.

1845 †Peacock, Thomas Bevill, M.D., Consulting Physician to St. Thomas's Hospital; Consulting Physician to the City of London Hospital for Diseases of the Chest, Victoria Park; 20, Finsbury circus. S. 1855-6. V.P. 1867. C. 1869. *Referee*, 1857-66. *Lib. Com.* 1855. *Trans.* 2.

1879 Peel, Robert, L.K.Q.C.P.I., 130, Collins street east, Melbourne, Victoria.

1856 Peirce, Richard King, 94, Addison road, Kensington.

1830 Pelechin, Charles P., M.D., St. Petersburg.

1855 *Pemberton, Oliver, 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.

1870 Perrin, John Beswick, Medical Tutor and late Demonstrator of Practical and Surgical Anatomy, Owen's College; The Homestead, Leigh, Lancashire.

1879 *Pesikaka, Hormasji Dosabhai, L.R.C.P. Glasg., Marine Lines, Bombay.

1878 *Philipson, George Hare, M.D., M.A. Cantab., Professor of Medicine at Durham University; Senior Physician to the Newcastle-upon-Tyne Infirmary; 7, Eldon square, Newcastle-upon-Tyne.

Elected

1852 PHILLIPS, RICHARD, 27, Leinster square, Bayswater. C. 1877.

1846 PHILP, FRANCIS RICHARD, M.D., Pendogget, Timsbury, Somersetshire.

1867 PICK, THOMAS PICKERING, Surgeon to, and Lecturer on Anatomy at, St. George's Hospital; 13, South Eaton place, Eaton square. *Sci. Com.* 1870. *Lib. Com.* 1879-80.

1841 †PITMAN, HENRY ALFRED, M.D., Consulting Physician to St. George's Hospital, and to the Royal General Dispensary, St. Pancras; 28, Gordon square. L. 1851-3. C. 1861-2. T. 1863-8. V.P. 1870-1. *Referee,* 1849-50. *Lib. Com.* 1847.

1871 POLLOCK, ARTHUR JULIUS, M.D., 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, 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. *Referee,* 1858, 1864-9, 1877-80. *Trans.* 4.

1865 POLLOCK, JAMES EDWARD, M.D., Physician to the Hospital for Consumption, Brompton; 52, Upper Brook street, Grosvenor square. *Referee,* 1872-80.

1871 POORE, GEORGE VIVIAN, M.D., Professor of Medical Jurisprudence in University College; Assistant-Physician to University College Hospital; Physician to the Royal Infirmary for Children and Women, Waterloo road; 30, Wimpole street. *Trans.* 1.

1846 POTTER, JEPHSON, M.D., F.L.S.

1842 POWELL, JAMES, M.D.

1867 POWELL, RICHARD DOUGLAS, M.D., Physician to the Hospital for Consumption and Diseases of the Chest, Brompton; Assistant-Physician to the Middlesex Hospital; 15, Henrietta street, Cavendish sq. *Referee,* 1879-80. *Trans.* 2.

Elected

1867 Power, Henry, Ophthalmic Surgeon to, and Lecturer on Ophthalmic Surgery at, St. Bartholomew's Hospital; 37A, Gt. Cumberland place, Hyde park. *Referee*, 1870-80. *Sci. Com.* 1870. *Lib. Com.* 1872-8.

1857 Priestley, William Overend, M.D., Consulting Physician-Accoucheur to King's College Hospital, and to the St. Marylebone Infirmary; 17, Hertford street, Mayfair. C. 1874-5. *Referee*, 1867-73, 1877-80. *Sci. Com.* 1863.

1874 Purves, William Laidlaw, Aural Surgeon to Guy's Hospital; 6, Stratford place, Oxford street. *Trans.* 2.

1878 Pye, Walter, Assistant Surgeon to St. Mary's Hospital; 4, Sackville street, Piccadilly.

1877 Pye-Smith, Philip Henry, M.D., Assistant-Physician to, and Lecturer on Physiology at, Guy's Hospital; Examiner in Physiology at the University of London; 56, Harley street, Cavendish square.

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.

1835 †Quain, Richard, F.R.S., Surgeon-Extraordinary to H.M. the Queen; Emeritus Professor of Clinical Surgery, University College, London, and Consulting Surgeon to University College Hospital; 32, Cavendish square. C. 1838-9. L. 1846-8. T. 1851-3. V.P. 1856-7. *Referee*, 1845-6, 1848, 1858-9. *Lib. Com.* 1846. *Trans.* 1. *Pro.* 2.

1852 Radcliffe, Charles Bland, M.D., *Vice-President*, 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. *Referee*, 1862-6, 1870-8.

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

Elected

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, 57, Gloucester road [11, Petersham terrace], Queen's gate, South Kensington.

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., Consulting Physician to Guy's Hospital; 26, Albemarle street, Piccadilly. C. 1873. *Referee*, 1860-72, 1875-80. *Trans.* 1.

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. *Referee*, 1867-9.

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

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

1845 †Ridge, Benjamin, M.D., 8, Mount street, Grosvenor square.

1863 Ringer, Sydney, M.D., Professor of the Principles and Practice of Medicine in University College, London, and Physician to University College Hospital; Examiner in Materia Medica in the University of London; 15, Cavendish place, Cavendish square. *Referee*, 1873-80. *Trans.* 4.

1871 Rivington, Walter, M.S., Surgeon to, and Lecturer on Anatomy at, the London Hospital; 22, Finsbury square. *Trans.* 2.

1871 *Roberts, David Lloyd, M.D., Physician to St. Mary's Hospital, Manchester; 23, St. John's street, Deansgate, Manchester.

1878 Roberts, Frederick Thomas, M.D., Professor of Materia Medica and Therapeutics in University College, London; and Physician to University College Hospital; 53, Harley street, Cavendish square, W.

Elected

1857 Robertson, John Charles George, Medical Superintendent of the Cavan District Lunatic Asylum; Monaghan, Ireland.

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

1843 Roden, William, M.D., Morningside, Kidderminster, Worcestershire.

1850 Roper, George, M.D., Physician to the Eastern Division of the Royal Maternity Charity; Physician to the Royal Infirmary for Children and Women, Waterloo Bridge road; 6, West street, Finsbury circus. C. 1879-80.

1857 Rose, Henry Cooper, M.D., F.L.S., Surgeon to the Hampstead Dispensary; Penrose House, Hampstead. *Trans.* 1.

1849 †Routh, Charles Henry Felix, M.D., 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., Surgeon to the Royal Sea-Bathing Infirmary; Cecil street, Margate, Kent.

1845 Russell, James, M.D., Physician to the Birmingham General Hospital; 91, New Hall street, Birmingham.

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

1856 Salter, S. James A., M.B., F.R.S., F.L.S., Lecturer on Dental Surgery at Guy's Hospital; 44, New Broad street, City. 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., Professor of Physiology at the University of London; Jodrell Professor of Human Physiology and Histology at University College, London; 26, Gordon square. C. 1869-70. *Referee*, 1867-8, 1876-80. *Sci. Com.* 1862, 1870. *Lib. Com.* 1876-80. *Trans.* 2.

Elected

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

1879 Sangster, Alfred, B.A., M.B., Lecturer on Skin Diseases at the Charing Cross Hospital; 7, Old Burlington street. *Trans.* 1.

1847 †Sankey, William Henry Octavius, M.D., Sandywell park, Andoversford, Cheltenham.

1869 Sansom, Arthur Ernest, M.D., Assistant-Physician to the London Hospital; 30, Devonshire street, Portland place. *Trans.* 2.

1845 †Saunders, Edwin, Surgeon-Dentist to H.M. the Queen, and to H.R.H. the Prince of Wales; 13A, George street, Hanover square. C. 1872-3.

1834 Sauvan, Ludwig V., M.D., Warsaw.

1879 Savage, George Henry, M.D., 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; Examiner in Surgery at the University of London; 66, Brook street, Grosvenor square. C. 1871-2. L. 1878. *Referee*, 1865-70, 1873-77, 1879-80. *Sci. Com.* 1862, 1867, 1870. *Lib. Com.* 1866-8. *Trans.* 5.

1873 Scott, John Moore Johnston, M.D., Lurgan, County Armagh.

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

1863 Sedgwick, William, 12, Park place, Upper Baker street. *Trans.* 2.

1877 Semon, Felix, M.D., 59, Welbeck street, Cavendish square.

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.

1840 Sharp, William, M.D., F.R.S., Horton House, Rugby. *Trans.* 1.

Elected

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.

1859 Sibley, Septimus William, 7, Harley street, Cavendish square. *Sci. Com.* 1863. *Trans.* 4.

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

1871 Silver, Alexander, M.D., Physician to, and Lecturer on Clinical Medicine at, Charing Cross Hospital; 2, Stafford street, Bond street.

1842 †Simon, John, 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, 1866-80. *Trans.* 1.

1865 Sims, J. Marion, M.D., Surgeon to the New York State Women's Hospital; 267, Madison Avenue, New York.

1857 Siordet, James Lewis, M.B., Villa Preti, Mentone, Nice.

1879 Smith, E. Noble, 24, Queen Anne street, Cavendish square.

1872 Smith, Gilbart, M.A., M.B., 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.

1866 Smith, Heywood, M.A. M.D. Oxon., Physician to the Hospital for Women; Physician to the British Lying-in Hospital; 2, Portugal street, Grosvenor square.

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, Surgeon to, and Lecturer on Clinical Surgery at, St. Bartholomew's Hospital; Surgeon to the Hospital for Sick Children; 5, Stratford place, Oxford street. S. 1870-2. C. 1875-76. *Referee*, 1873-4, 1880. *Sci. Com.* 1867. *Trans.* 3.

1864 *Smith, Thomas Heckstall, Rowlands, St. Mary Cray, Kent.

1847 Smith, William J., M.D., Consulting Physician to the Weymouth Infirmary; Greenhill, Weymouth, Dorsetshire.

1873 Smith, W. Johnson, Surgeon to the Seamen's Hospital, Greenwich.

1874 *Smith, William Robert, M.D., Physician to the Dispensary, Cheltenham; 15, Imperial square, Cheltenham.

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

1865 Southey, Reginald, M.D., Physician to, and Lecturer on Forensic Medicine at, St. Bartholomew's Hospital; 6, Harley street, Cavendish square. *Referee*, 1873-80. *Trans* 1.

1844 Spackman, Frederick R., M.D., Harpenden, St. Alban's.

1874 Sparks, Edward Isaac, M.B., Mentone. *Trans.* 2 [deceased].

1851 Spitta, Robert John, M.D. Lond., Clapham Common, Surrey. C. 1878-9. *Trans.* 1.

1875 Spitta, Edmund J., Ivy House, Clapham Common, Surrey.

1854 Stevens, Henry, M.D., Inspector, Medical Department, Local Government Board; Greenford House, Sutton, Surrey.

1342 †Stewart, Alexander Patrick, M.D., Consulting Physician to the Middlesex Hospital; 75, Grosvenor street. C. 1856-7. L. 1863-8. V.P. 1871-2.

1859 Stewart, William Edward, 16, Harley street, Cavendish square.

1879 Stirling, Edward Charles, Lecturer on Physiology at St. George's Hospital; 60, Great Cumberland place, Hyde park.

Elected

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

1865 Stokes, William, M.D., Examiner in Surgery, Queen's University, Ireland, and Surgeon to the Richmond Surgical Hospital; 5, Merrion square north, Dublin. *Trans.* 1.

1843 Storks, Robert Reeve, Paris.

1858 †Streatfeild, John Fremlyn, Surgeon to the Royal London Ophthalmic Hospital, Moorfields; Ophthalmic Surgeon to University College Hospital; 15, Upper Brook street, Grosvenor square. C. 1874-5. *Lib. Com.* 1867-8.

1871 Strong, Henry John, M.D., 64, North End, Croydon.

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

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

1860 Sutro, Sigismund, M.D., Senior Physician to the German Hospital; 37A, Finsbury square.

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

1855 Sutton, John Maule, M.D., Medical Officer of Health, Oldham; 244, Great Clowes street, Broughton, Manchester.

1861 *Sweeting, George Bacon, King's Lynn, Norfolk.

1854 *Symonds, Frederick, Surgeon to the Radcliffe Infirmary, and Consulting Surgeon to the Oxford Dispensary; 35, Beaumont street, Oxford.

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, Great Charles street, Birmingham. *Trans.* 4.

Elected

1864 Taussig, Gabriel, M.D., 70, Piazza Barberini, Rome.

1875 Tay, Waren, Surgeon to the London Hospital and Surgeon to the North Eastern Hospital for Children and the Hospital for Skin Diseases, Blackfriars; 4, Finsbury square.

1873 Taylor, Frederick, M.D., Assistant-Physician to Guy's Hospital; 15, St. Thomas's street, Southwark. *Trans.* 1.

1852 Taylor, Robert, 7, Lower Seymour street, Portman square.

1845 †Taylor, Thomas, Warwick House, 1, Warwick place, Grove End road, St. John's wood.

1859 Tegart, Edward, 49, Jermyn street, St. James's.

1874 Thin, George, M.D., 22, Queen Anne street, Cavendish square. *Trans.* 6.

1862 Thompson, Edmund Symes, M.D., 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.

1857 Thompson, Henry, M.D., Consulting Physician to the Middlesex Hospital; 53, Queen Anne street, Cavendish square.

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; 35, Wimpole street, Cavendish square. C. 1869. *Trans.* 5.

1862 Thompson, Reginald Edward, M.D., *Secretary*, Physician to the Hospital for Consumption, Brompton; 9, Cranley place, South Kensington. C. 1879. S. 1880. *Referee*, 1873-8. *Sci. Com.* 1867. *Trans.* 2.

1876 Thornton, John Knowsley, M.B., C.M., Surgeon to the Samaritan Free Hospital for Women and Children; 83, Park street, Grosvenor square. *Trans.* 1.

1875 Tibbits, Herbert, F.R.C.P. Ed., 30, New Cavendish street.

Elected

1848 †Tilt, Edward John, M.D., Consulting Physician to the Farringdon General Dispensary and Lying-in Charity; 27, Seymour street, Portman square. *Referee*, 1874-80.

1880 Tivy, William James, 1, Tottenham place, Clifton, Bristol.

1872 Tomes, Charles S., B.A., F.R.S., Lecturer on Anatomy and Physiology at the Dental Hospital; 37, Cavendish square. *Lib. Com.* 1879.

1867 Tonge, Morris, M.D., Harrow-on-the-Hill, Middlesex.

1871 *Trend, Theophilus W., M.R.C.P. Edinb., Raeberry Lodge, Southampton.

1879 Treves, Frederick, Assistant Surgeon to the London Hospital; 18, Gordon square.

1867 Trotter, John William, Surgeon-Major, Coldstream Guards; Bossall Vicarage, York.

1859 Truman, Edwin Thomas, Surgeon-Dentist in Ordinary to Her Majesty's Household; 23, Old Burlington street.

1864 Tufnell, Thomas Jolliffe, Consulting Surgeon to the City of Dublin Hospital; 58, Lower Mount street, Merrion square, Dublin. *Trans.* 1.

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 London Hospital; 15, Finsbury square.

1873 Turner, George Brown, M.D., San Remo, Italy.

1876 Venn, Albert John, M.D., Obstetric Physician to the Metropolitan Free Hospital; Assistant Physician to the Victoria Hospital for Children; 8, Upper Brook street, Grosvenor square.

1870 Venning, Edgcombe, late Surgeon, 1st Life Guards; 87, Sloane street.

1865 Vernon, Bowater John, Ophthalmic Surgeon to St. Bartholomew's Hospital and to the West London Hospital; 33, Curzon street, Mayfair.

1867 Vintras, Achille, M.D., Physician to the French Embassy and to the French Hospital, Lisle street, Leicester square; 141, Regent street.

Elected

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, and Lecturer on Clinical Medicine at, St. George's Hospital; 14, Park lane.

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

1868 *WALKER, ROBERT, L.R.C.P. Edinb., Surgeon to the Carlisle Dispensary; 2, Portland square, Carlisle.

1867 *WALLIS, GEORGE, Corpus Buildings, Cambridge.

1873 WALSHAM, WILLIAM JOHNSON, C.M., Demonstrator of Anatomy and Operative Surgery at St. Bartholomew's Hospital; Surgeon to the Metropolitan Free Hospital and to the Royal Hospital for Diseases of the Chest, City Road; 27, Weymouth street, Portland place. *Trans.* 2.

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; 37, Queen Anne street, Cavendish square. C. 1872. *Trans.* 1.

1851 WALTON, HAYNES, Senior Surgeon to St. Mary's Hospital, and to the Ophthalmic Department; 1, Brook street, Grosvenor square. *Trans.* 1. *Pro.* 1.

1852 WANE, DANIEL, M.D., 20, Grafton street, Berkeley square.

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

1858 WARDELL, JOHN RICHARD, M.D., Calverley park, Tunbridge Wells.

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

1818 WARE, JOHN, Clifton Down, near Bristol.

1866 WARING, EDWARD JOHN, M.D., 49, Clifton gardens, Maida vale.

Elected

1877 WARNER, FRANCIS, M.D., Assistant Physician to the London Hospital and to the East London Hospital for Children; 24, Harley street, Cavendish square.

1861 WATERS, A. T. HOUGHTON, M.D., Physician to the Royal Infirmary, and Lecturer on the Principles and Practice of Medicine, in the Liverpool Royal Infirmary School of Medicine; 69, Bedford street, Liverpool. *Trans.* 3.

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

1878 WATNEY, HERBERT, M.D., Assistant Physician to St. George's Hospital; 1, Wilton crescent, Belgrave square.

1837 †WATSON, SIR THOMAS, Bart., M.D., D.C.L., F.R.S., Physician-in-Ordinary to H.M. the Queen; Consulting Physician to King's College Hospital; 16, Henrietta street, Cavendish square. C. 1840-1, 1852. V.P. 1845-6. *Referee*, 1842-5, 1847-9.

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. *Trans.* 1.

1879 WATTEVILLE, ARMAND DE, M.B., B.S., 13, Old Cavendish street.

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

1840 WEBB, WILLIAM WOODHAM, M.D.

1842 †WEBER, FREDERIC, M.D., 44, Green street, Park lane. C. 1857. V.P. 1865.

1857 WEBER, HERMANN, M.D., Physician to the German Hospital; 10, Grosvenor street, Grosvenor square. C. 1874-5. *Referee*, 1869-73, 1878-80. *Lib. Com.* 1864-73. *Trans.* 6.

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

1878 WEISS, HUBERT FOVEAUX, House Surgeon, Lock Hospital, Dean street, Soho.

Elected

1874 WELLS, HARRY, M.D., British Vice-Consulate, Gualeguay-chu, Entre Rios, Argentine Confederation.

1854 WELLS, THOMAS SPENCER, Surgeon-in-Ordinary to H.M.'s Household; Surgeon to the Samaritan Free Hospital for Women and Children; 3, Upper Grosvenor street. C. 1870. *Trans.* 10. *Pro.* 1.

1842 †WEST, CHARLES, M.D., Corresponding Member of the Academy of Medicine of Paris; Asham House, Bournemouth. 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.B., Casualty Physician and Medical Tutor at St. Bartholomew's Hospital; Assistant Physician to the City of London Hospital for Diseases of the Chest, Victoria Park; 15, Wimpole street, Cavendish square.

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

1828 WHATLEY, JOHN, M.D.

1875 WHIPHAM, THOMAS TILLYER, M.B., Physician to, and Lecturer on Clinical Medicine at, St. George's Hospital; 37, Green street, Grosvenor square.

1849 WHITE, JOHN.

1877 WHITMORE, WILLIAM TICKLE, 7, Arlington street, Piccadilly.

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

1870 *WILKIN, JOHN F., M.D. and M.C., New Beckenham, Kent.

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

1863 WILKS, SAMUEL, M.D., F.R.S., Physician to, and Lecturer on Medicine at, Guy's Hospital; Physician in Ordinary to their Royal Highnesses the Duke and Duchess of Connaught; 77, Grosvenor street, Grosvenor square. *Referee*, 1872-80. *Sci. Com.* 1.

1865 WILLETT, ALFRED, Surgeon to St. Bartholomew's Hospital; Surgeon to St. Luke's Hospital; 36, Wimpole street, Cavendish square. C. 1880. *Trans.* 1.

Elected

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]. 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; 9, Prince of Wales road, Norwich.

1866 WILLIAMS, CHARLES THEODORE, M.D., Physician to the Hospital for Consumption, Brompton; 47, Upper Brook street, Grosvenor square. *Lib. Com.* 1880. *Trans.* 3.

1872 WILLIAMS, JOHN, M.D., Assistant Obstetric Physician to University College Hospital; 28, Harley street, Cavendish square. *Referee*, 1878-80. *Lib. Com.* 1876-80.

1859 WILLIAMS, JOSEPH, M.D. Holmhurst, Cambridge park, Twickenham.

1868 WILLIAMS, WILLIAM RHYS, M.D., Commissioner in Lunacy; 19, Whitehall place.

1839 †WILSON, ERASMUS, F.R.S., late Professor of Dermatology, Royal College of Surgeons of England; 17, Henrietta street, Cavendish square. C. 1877. *Lib. Com.* 1845. *Trans.* 2.

1863 WILSON, ROBERT JAMES, F.R.C.P. Edin., 7, Warrior square, St. Leonard's-on-Sea, Sussex.

1850 *WISE, ROBERT STANTON, M.D., Consulting Physician to the Southam Eye and Ear Infirmary; Banbury, Oxfordshire.

1825 WISE, THOMAS ALEXANDER, M.D., Thornton, Beulah hill, Upper Norwood, Surrey.

1879 WOAKES, EDWARD, M.D., 57, Harley street, Cavendish square.

1841 WOOD, GEORGE LEIGHTON, 28, Green park, Bath.

Elected

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

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.

1879 Woodward, G. P. M., M.D., Deputy Surgeon-General, Puckeridge, near Ware, Hertfordshire.

1842 Worthington, William Collins, London road, Lowestoft, Suffolk. *Trans.* 3.

1865 Wotton, Henry, M.D., 62, Bedford gardens, Kensington.

1878 Yeo, Gerald F., M.D., M.Ch., Professor of Physiology in King's College, London; King's College, Strand.

[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

1853 Brodie, Sir Benjamin Collins, Bart., M.A., D.C.L., F.R.S., Brockham Warren, Reigate.

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

1873 Christison, Sir Robert, Bart., M.D., D.C.L., LL.D., Physician-in-Ordinary to H M. the Queen in Scotland; 40, Moray place, Edinburgh.

1868 Darwin, Charles, M.A., F.R.S., Corresponding Member of the Academies of Sciences of Berlin, Stockholm, Dresden, &c.; Down, Bromley, Kent.

1857 Farr, William, M.D., C.B., D.C.L., F.R.S., 78, Portsdown road, Maida hill.

1868 Hooker, Sir Joseph Dalton, M.D., C.B., 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; Royal Gardens, Kew.

1868 Huxley, Thomas Henry, LL.D., F.R.S., Professor of Natural History in the Royal School of Mines; Secretary to the Royal Society; Corresponding Member of the Academies of Sciences of St. Petersburg, Berlin, Dresden, &c.; 4, Marlborough place, St. John's wood.

Elected

1878 Lubbock, Sir John, Bart., F.R.S., High Elms, Bromley, Kent.

1847 Owen, Richard, C.B., D.C.L., LL.D., F.R.S., Superintendent of the Natural History Departments in the British Museum; Foreign Associate of the Academy of Sciences of the Institute of France; Sheen Lodge, Mortlake.

1873 Stokes, George Gabriel, M.A., D.C.L., LL.D., Lucasian Professor of Mathematics in the University of Cambridge; Secretary to the Royal Society, &c.; Lensfield Cottage, Cambridge.

1868 Tyndall, John, D.C.L., LL.D., F.R.S., Professor of Natural Philosophy in the Royal Institution; Corresponding Member of the Academies and Societies of Sciences of Göttingen, Haarlem, Geneva, &c.; Royal Institution, Albemarle street, Piccadilly.

FOREIGN HONORARY FELLOWS.

(Limited to Twenty.)

Elected

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

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

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

1875 Draper, John William, M.A., LL.D., Emeritus Professor of Chemistry and Physiology in the University of New York; 13, University Buildings, Washington square, New York.

1876 Edwards, H. Milne, M.D., Member of the Institute of France, and of the Academy of Medicine; Dean of the Faculty of Sciences and Professor at the Museum of Natural History of Paris; 57, Rue Cuvier, Paris.

1835 Ekströmer, Carl Johan, M.D., C.M., K.P.S., and W., Physician to the King of Sweden; President of the College of Health, and Director-General of Hospitals; Stockholm.

1868 Gross, Samuel D., M.D., F.C.P. Philad., D.C.L. Oxon., LL.D. Camb., Professor of Surgery in the Jefferson Medical College of Philadelphia.

1878 Gueneau de Mussy, Noel, M.D., Member of the Academy of Medicine; Physician to the "Hôtel Dieu;" 4, Rue St. Arnaud, Paris.

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

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

1859 Henle, J., M.D., Professor of Anatomy at Göttingen.

Elected

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

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

1856 Langenbeck, Bernhard, M.D., Professor of Surgery in the University of Berlin.

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.

1862 Pirogoff, Nikolaus, M.D., Professor of Surgery to the Medico-Chirurgical Academy in St. Petersburg, and Director of the Anatomical Institute; Consulting Physician to the Hospitals Obuchow, Peter-Paul, and Maria Magdalena; St. Petersburg.

1878 Scanzoni, Friedreich Wilhelm von, Royal Bavarian Privy Councillor, and Professor of Medicine in the University of Würzburg.

1878 Schwann, Theodor, M.D., Professor at the Royal University of Liege, Belgium.

1856 Virchow, Rudolph, M.D., Professor of Pathological Anatomy in the University of Berlin; Corresponding Member of the Academy of Sciences of the Institute of France; Berlin.

LIST OF RESIDENT FELLOWS

ARRANGED ACCORDING TO

DATE OF ELECTION.

1815 Archibald Billing, M.D.
1819 Jas. M. Arnott, F.R.S.
1823 George Macilwain.
1828 Cæsar H. Hawkins, F.R.S.
1833 Sir George Burrows, M.D., F.R.S.
Thomas A. Barker, M.D.
1835 Richard Quain, F.R.S.
Thomas A. Nelson, M.D.
1836 Alexander Shaw.
J. George French.
1837 Sir Thomas Watson, M.D., F.R.S.
Thomas Blizard Curling, F.R.S.
George Busk, F.R.S.
Thomas William Jones, M.D.
1838 Charles Hawkins.
Henry Spencer Smith.
1839 T. Graham Balfour, M.D., F.R.S.
W. J. Erasmus Wilson, F.R.S.
Fred. Le Gros Clark, F.R.S.
James Dixon.
1840 Chas. J. B. Williams, M.D., F.R.S.
Charles Hutton, M.D.
Samuel A. Lane.
Sir James Paget, F.R.S.
1841 Henry A. Pitman, M.D.
William Bowman, F.R.S.
John Parkin, M.D.
Paul Jackson.
1842 Charles West, M.D.
Frederic Weber, M.D.
Alex. P. Stewart, M.D.
John Simon, C.B., F.R.S.
John Erichsen, F.R.S.
Oscar M. P. Clayton.
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The Abstracts of the papers read will be furnished to the Journals as heretofore.

AN ACCOUNT

OF A

LARGE DERMOID CYST FOUND IN THE ABDOMEN OF A MAN.

BY

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AND

CHARLES BRODIE SEWELL, M.D.

(Received October 11th—Read October 28th, 1879.)

The tumour now exhibited was removed post mortem from the abdomen of a gentleman who was during life under the care of Dr. Sewell. On March 8th, 1879, Dr. Sewell received from him a note saying that he had a carbuncular boil "on the old spot," and would be glad to be seen. The patient was a strong and vigorous man, aged 28, greatly given to hunting. The "old spot" was the buttock, and the inconvenience in riding produced by a boil in such a place was, in fact, the main reason for his seeking medical aid, the boil having been much irritated by a hard day's hunting. Dr. Sewell found the patient in bed, and being struck by a change in his appearance—for he was well known to him—and par-

ticularly by some puffiness of the face, he made a general examination, in the course of which he found the abdomen large and fluctuating, evidently containing fluid. The urine was normal, the fæces hard and light in colour.

It seemed to Dr. Sewell a strange circumstance that this young man, so devoted to hunting that subsequently during an operation he said, "Go on, don't mind me, I'll think about hunting, and I shan't feel," who up to the very day of his sending for the doctor was active and cheerful, and who only expected that he had a boil, should thus keep his bed, and should not seem to have a wish to leave it, without having had one word of warning of danger, or having been put under any depressing treatment. Dr. Sewell considered it a curious indication of the effect of gradually advancing disease upon a generally healthy constitution; and the end, as Dr. Sewell remarked, coincided strangely with the beginning, for he was patient and even cheerful to the last, excepting when in unbearable agony.

The distribution of the fluid was not quite clear, for although the general appearance was that of ascites, there was permanent resonance in the left flank and across the abdomen above the umbilicus, with dulness above, these points not changing with change of position of the body. The recti muscles were widely separated along the median line, and the liver was much pushed up, the patient being conscious of some shortness of breathing. This, indeed, he remembered to have felt of late, and it also occurred to him that he had become a little unwieldy on horseback, but he had simply thought that he was getting too stout and was out of condition. There was no œdema of the legs. The separation of the recti was considerable, and in view of the prevention of its increase a consultation was held between Dr. Sewell, Mr. Henry Smith, and Dr. Ord. It was then resolved that the abdomen should be tapped, and a trocar of moderate size was entered in the median line a little below the umbilicus, a point at which the absence of intestine had been ascertained.

About two pints of fluid, looking like very well made pea soup, were drawn off, but with difficulty, the stuff becoming much inspissated and finally ceasing to flow, although the swelling was scarcely at all reduced. The fluid speedily solidified to the consistence of a pomatum, but without odour. It had an acid reaction. When melted and examined under the microscope it showed only hairs and soft epithelial scales, very few of which had nuclei. There were no hooklets of hydatids and no crystals.

Within three hours after the operation symptoms of acute peritonitis set in, and the patient died on the fifth day.

An examination of the body was made on the following day, when a large cyst was found, now chiefly filled with solid matter, occupying a great part of the abdominal cavity. The cyst lay in the back of the abdomen, and appeared from its position to be retro-peritoneal in its origin. It was firmly united to the bladder and rectum, which seemed incorporated in its walls, and also by old adhesions to bands of both large and small intestines at various parts of its anterior surface. Besides this, general peritonitis existed, and had produced numerous fresh adhesions, particularly in the neighbourhood of the track of the trocar. The abdominal and thoracic viscera, apart from the adhesions mentioned, were normal in their structure, and the external genital organs were natural. The cyst was removed entire with the rectum, bladder, ureters, kidneys, and portions of intestine attached. When these were as far as possible removed, the bladder and part of rectum being left, it weighed 14½ pounds. In shape it resembled a flattened pear, or a uterus with rounded angles, the smaller end directed downwards into the space between bladder and rectum. Its length was 13 inches, its breadth 10 at the greatest, 4½ at the lower end, and its depth from behind forwards from 4 to 8 inches. The genito-urinary organs were, much adherent to it; they were also in various ways

pushed out of their proper positions, but no abnormalities could be discovered. The orifice made by the trocar was visible and patulous. When the tumour was placed under water for examination fine pale hairs were seen projecting through the orifice from within. The contents of the cyst were then evacuated and had a strong rancid odour, which adhered most obstinately to the hands for many hours. They were in part a pulpy fat, mixed with abundance of hairs, in part a solid mass of the consistence of soap, having hairs imbedded. Specimens of the hair and of the fat are exhibited. The walls of the cyst were about the sixteenth of an inch in thickness, and were not elastic. Over a large area at one end of the tumour they presented, after removal of the adhering tissues, just the appearance of the corium of skin when viewed from its inner surface; they were mottled with gland-like patches, and reticulated with vessels and lymphatics. When subsequently the cyst was carefully emptied, cleansed, and distended, abundance of hairs were seen projecting from the wall into the cavity. Microscopic sections of the cyst-wall showed a structure identical with that of skin; that is to say, there could be seen epidermic and dermic layers, the former lining the cavity, hair sacs, in some cases containing hairs, and very large sebaceous glands, but no traces of sudoriparous glands. These were all much flattened or oblique to the surface. There were no regular papillæ. Regular papillæ could not be expected to exist in an envelope so enormously distended as was the wall of this cyst. But the sections for the microscope are irregularly studded with projections of the derm into the epidermis, having the appearance of distorted papillæ.

The contents of the cyst were examined by Dr. Bernays, who found them to consist almost entirely of fat—namely, of 99·75 per cent. of fatty acids. The rancid odour had led us to suppose that the constituents of sweat must enter into the composition of this matter. But Dr. Bernays cannot detect a trace of butyric acid or sweat

products. This observation accords with the absence of sudoriparous glands from the walls of the sac.[1]

The specimen is very remarkable and rare in respect of its being a dermoid cyst developed in the abdomen of a man, in close structural relation with the bladder and rectum, and as regards its size it is probable unique.

One instance of a dermoid cyst occupying such a position is recorded by Dr. Wilks and Mr. Curling. In the 'Pathological Transactions,' vol. xiii, p. 148, Dr Wilks describes "some fat removed during life from a cyst near the bladder." The patient was a man, aged 21, who had experienced for twelve months increasing difficulty in urination. Mr. Pretty, of Fressingfield, having found a fluctuating tumour projecting into the rectum and pressing on the bladder punctured it through the rectum and evacuated two pints of oily matter, which became solid in cooling. Dr. Wilks, examining the fat, referred it to "one of those dermoid cysts which are occasionally found in various parts of the body as congenital growths. In the 20th volume of the same 'Transactions,' at page 238, Mr. Curling relates how the same man came under his care

[1] A report of a further examination of the fat by Dr. Bernays is appended:

"Brownish soft mass, with odour of perspiration, containing hairs. Very little soluble in water.

"Free acid smelling of impure butyric acid . . 0·06 per cent.

"Actual gravity at 38° C. . . . . 0·8855 „

"It saponifies with the greatest ease, showing presence of free fatty acids, which amount to no less than 98·4 per cent.

"The fatty acids are chiefly oleic and palmitic, although some olein or palmitin is distinctly present, as the actual glycerin obtainable was 0·45 per cent.

"Total oleic acid, as calculated from lead salt, 56·64 per cent.

"Trace of salt.

"Details.—Melting point at	32° C.
"Sp. gr. at 38° C.	0·8855
"Fixed fatty acids	98·4
"Volatile	0·06
	98·46
"Oleic acid	56·64
"Glycerin	0·45"

eight years later, with symptoms of stone in the bladder, how he removed by lithotomy an oval flat stone composed of mixed phosphates, and how this stone on section proved to be formed round a nucleus of short dark hairs, corresponding under the microscope with human hairs. Mr. Curling also detected in front of the rectum, and rather to the right side, an induration, which he supposed was the remains of the contracted cyst.

Lebert ('Anatomie Pathologique Générale,' vol. i, p. 256, 1857—61); gives ten cases in which non-ovarian dermoid cysts containing fat and hair were found "dans les parties profondes du corps." This occurs among 188 dermoid cysts, 59 non-ovarian, 129 ovarian. Of the 10, 2 were in thorax, 2 near liver, 2 in mesentery, 2 near uterus, 1 between uterus and bladder, 1 between uterus and rectum. The sex is mentioned in 8 cases, only one of which was a male.

It is obvious, therefore, that the occurrence of such cysts in the male, though exceedingly rare, is not unknown. The position between the bladder and rectum appears to be not uncommon in women, for besides Lebert's cases, Blackman, in the 'American Journal of Medical Science' for January, 1869, records some, and is quoted by Mr. Curling in the paper above referred to. So far we have been dealing only with cysts containing fat and hair, but cysts containing teeth are more abundant in the body. Lebert quotes 11 cases of the kind. And, again, the non-ovarian cysts, in which other tissues and organs, or even nearly all the anatomical elements of a fœtus, are developed, must be ranked with piliferous cysts when we are endeavouring to trace their origin.

When such dermoid cysts are formed in the ovary it is not difficult to understand that the reproductive power resident under certain circumstances in Graafian follicles and ovum may have been set prematurely into operation. Mr. Savory, indeed, suggests that this is an extreme instance of the phenomena called parthenogenesis, exhibited regularly among invertebrata, as, for instance, in the Aphis.

According to this view the impregnation which has resulted in the formation of one individual leaves developmental force in excess, producing a second ovum within that individual, which may go one or many degrees toward the formation of another. Lebert's theory of "hétérotopie plastique" can be readily accommodated to Mr. Savory's suggestions, for it expresses the result in terms which do not explain the cause. "Beaucoup de tissus simples ou composés, et des organes plus complexes même, peuvent se former de toutes pièces dans les parties du corps où à l'état normal on ne les rencontre point." But it must be remembered that parthenogenesis is an orderly and regularly recurrent phenomenon in certain invertebrates, while the productions consistent with it in man are very rare. Putting aside, therefore, the idea of straying development in a Graafian follicle as inapplicable to the explanation of bud-growths in the human male, we may examine one or two modes in which they may come about.

1. The idea of "fœtal inclusion"—of a second less perfect individual being embraced within the developmental area of a perfect individual. This would apply very plausibly to Velpeau's case, in which a congenital cyst containing a small fœtus was removed from the scrotum of an adult.

2. The idea of "developmental inclusion," where during development parts belonging to one surface or apparatus become by some accident separated from their proper belongings and implanted in a wrong position. The somewhat common occurrence of dermoid cysts about the head and neck is most probably attributable to a process of this kind. In these parts, central raphæ and lateral fissures are often imperfectly closed; the fusion of opposing inverted surfaces is imperfectly performed. Then a fusion at the surface, associated with failure of fusion beneath, causes the formation of a cavity lined by cutaneous elements, which, growing and distended by the products of its walls, becomes a dermoid cyst. Sir James Paget has, as we all know, given much attention

to the results following from imperfect closure of branchial fissures associated with errors of development in the structures related with them. Remak, in 1854, suggested the idea of the origin of cysts in the neck from faulty branchial sutures. Cresset more recently had in a small but very suggestive treatise, 'Sur l'appareil branchial des vertébrés,' worked out this idea as applicable to dermoid cysts in the head and neck. And in last year's 'Pathological Transactions,' Mr. Wagstaffe has collected a large number of instances of dermoid cysts developing not only at the outer angle of the orbit or beneath the tongue, but at other points where fissures run. Of these he enumerates twenty-four, and describes in particular dermoid cysts distinctly connected by pedicles with the fronto-maxillary, lacrymo-maxillary, and fronto-malar clefts respectively.

3. The idea of aberrant development. There are deformities to which the body is subject arising out of the persistence of embryonic structures which should in the natural way disappear or undergo complete remodelling. Such deformities are most common in the genito-urinary organs, where out of a number of parts seen in early embryonic life certain groups grow to constitute the organs of one or other sex, while others disappear or remain rudimentary. Occasionally, we find that organs which should have disappeared persist, sometimes in association with perfect, sometimes with imperfect, development of other parts or organs; a male apparatus simulates a female or the reverse. Applying this idea to a dermoid cyst growing in the abdomen of a man in propinquity to the posterior aspect of the neck of the bladder, we may be reminded that the ovary and testes arise in a common blastema, though each as it grows is brought into communication with a different excretory tube. If any part of this blastema fail during development to be brought into its proper system by means of the proper communication—as occasionally we may see happen to the entire testis—the part so left out, having no

relations to guide it, may be readily conceived as proceeding to irregular development, and in this case its destiny being either to become ovary or testis may take on ovarian ways and develop dermoid cyst as an ovary might.

If in such a case as the present "developmental inclusion were the cause, we should have expected the tumour to be related with the abdominal walls. But it is clearly related with the genito-urinary organs, and we cannot see our way to suggesting how any developmental inclusion could fix a cyst in such a position. On the other hand, we have been unable to find any structural aberration in the genito-urinary organs. We are, therefore, left between fœtal inclusion and aberrant development, with an inclination to the latter, founded upon consideration of corresponding ovarian tumours.

DESCRIPTION

OF

MALFORMATIONS OF THE GENITAL ORGANS OF A MAN

ASSOCIATED WITH PERSISTENCE OF ONE OF THE DUCTS OF MÜLLER.

BY

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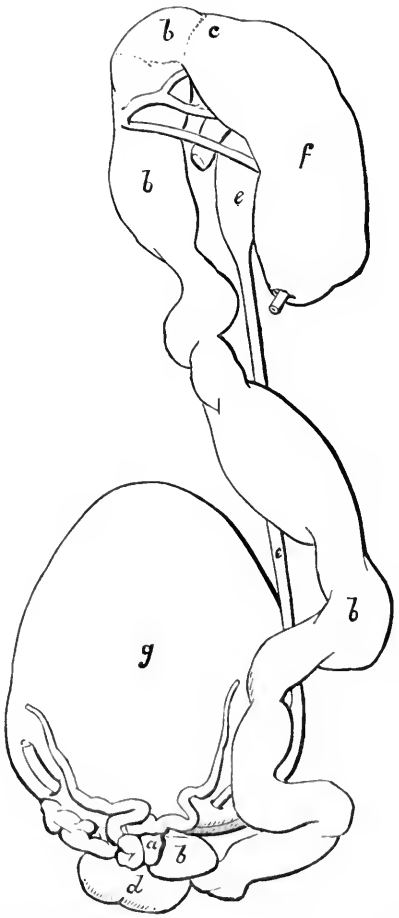
(Received October 11th—Read October 28th, 1879.)

J. P—, æt. 36, painter's labourer, died, under my care, in St. Thomas's Hospital, on the 3rd of March of this year, of tuberculosis of the lungs and tubercular meningitis. During life some abnormity of the genital organs had been noticed; the right testis had not descended, a tumour in the groin being supposed to indicate the position of the missing gland. At the post-mortem examination the supposition was verified. The right testis was found in the lower end of the inguinal canal, sheathed by a large tunica vaginalis extending down into the scrotum. This extension was shut off from the peritoneal cavity at the internal abdominal ring. When the abdomen was opened and the internal genito-urinary organs exposed there was found on the right side a large

tube, extending from the kidney to the neck of the bladder. This was at first supposed to be a supernumerary ureter, but the skilful dissection made by my friend and colleague Mr. Stewart, has revealed its true character, and shown it to be a part of the genital organs. The very beautiful preparation on the table (see woodcut) allows of



a. Right vesicula seminalis.
b. Supposed duct of Müller.
c. Position of supposed remains of Wolffian body.
d. Prostate.
e. Ureter.
f. Kidney.
g. Bladder.

an examination of the tube in its whole length. It begins as a blind sac attached to the head and inner side of the right kidney; after contracting a little opposite the middle of the kidney it expands again into three successive

bulges, each of the calibre of distended small intestine, between that point and the under surface of the bladder. It then becomes tortuous, with an average diameter of $\frac{3}{4}$ inch, and after a last dilatation to nearly twice that calibre, ends abruptly in the median line of the floor of the prostatic urethra by a patulous orifice about equal in size to the section of a No. 6 catheter. This orifice is placed about one line above the puncta of the ejaculatory ducts, the right of which is a little lower down than the left. Both ureters and both ejaculatory ducts are present. The ureters end naturally, and so does the left vas deferens. But on the right the vas deferens is less tortuous, therefore shorter than natural; the vesicula seminalis is represented by a quadrate, scarcely lobed bag, forming a continuation, not a diverticulum, of the vas, and is again continued in a straight line by a short ejaculatory duct. The vesicula is pushed over to the left side by the dilated extremity of the tube, and with this intrusion the slightly altered entry of the duct into the urethra is related.

The walls of the tube are about the thickness of those of the ureters in the upper part of their course, but below they resemble rather those of the vasa deferentia.

A section has been carried vertically through the upper fourth of the kidney and the sac adjoining. The sac is smooth-walled internally, except for a few puckerings at its very extremity, where it is in contact with the apex of the kidney. In the middle of these is a small orifice looking like the entry of a fine tube. But this ends blindly, injections failing to pass. The section of the kidney shows it to be capped by a small flattened glandular body, triangular as now viewed, with one surface looking to the sac, another continuously attached to the apex of the kidney but defined by a band of fibrous tissue, the third free. The wall of the tube where in contact with this is thin, about the $\frac{1}{16}$th of an inch; where in contact with the kidney at least $\frac{1}{6}$th an inch. The glandular body is renal in its appearance and minute

anatomy; that is to say, it contains numerous Malpighian bodies with thick walls and glomeruli. Both the capsules and the glomeruli are in various degrees of degeneration; both are encroached upon by connective tissue; a transparent plasma within the capsule often limits seriously the space for the glomerulus; and both structures have all the appearance of having undergone much contraction. The renal tubes related with them are arranged irregularly, are not differentiated at different parts, and have thicker walls than normal. The tube has fibrous walls lined with stratified squamous epithelium. In the outer part of the walls is seen muscular tissue not regularly arranged.

The bladder is enlarged and hypertrophied. The testis was not preserved, and no record of its condition was kept.

That this is a case of partially arrested development of the genital organs on the right side, with survival of certain fœtal structures, is very clear.

The Society will pardon me if I refer for a moment to the phenomena observed in the development of the internal genital organs. At an early period of fœtal life the common genital gland, destined to become testis or ovary, as the case may be, appears as a mass of blastema lying along the inner front of the pre-existing Wolffian body. About the same time a tube, closed above, opening below into the common urogenital sinus, is formed on the outer front of the Wolffian body, near, but structurally distinct from, the duct of that body. This tube is the duct of Müller. In the female these ducts persist as the Fallopian tubes, uterus and vagina. In the male their united lower ends persist as the vesicula prostatica, a small structure in man, but much larger and more organised in the lower mammalia, particularly in the beaver and the solipeds.[1] The middle tract on each side disappears; the blind upper end is supposed to remain as the hydatid

[1] See Leuckart on the "Vesicula Prostatica," 'Todd's Cyclopædia of Anatomy and Physiology,' vol. iv, p. 1415.

of Morgagni. In the male the Wolffian bodies certainly cease to exist as urinary structures. Indeed, according to older observations of J. Müller, and more recent statements of Cleland and Banks, they disappear almost completely, leaving only the organ of Giraldés to mark their original existence. Their ducts, however, become the convoluted tube of the epididymis and the vas deferens on each side. If they themselves survive at all, as they do in Kobelt's view, they become metamorphosed into the tubules of the epididymis, thus taking on, like their ducts, a new functional relation.

Taken in conjunction with the non-descent of the right testes, and imperfect evolution of the right vas deferens and vesicula seminalis, the tube running from the head of the kidney to the floor of the prostatic urethra would appear to be a surviving Müllerian duct. It is unconnected with the kidney, and indeed with all other structures, the prostate excepted. The small glandular structure capping the kidney, in contact with, though not structurally connected with the blind end of the tube, is probably a survival of the Wolffian body, arrested in its retrograde course at a time corresponding to the arrest of the similar course in the Müllerian duct. Although a renal structure persists in this piece of gland, no duct is found in relation with it, and there is no division into cortical and medullary regions.

As far as I know, the Müllerian ducts do not survive in their entirety in any male mammal. In some female mammalia, however, as in the pig, the ducts of the Wolffian bodies, which disappear in the human female, persist as the ducts of Gaertner. Such a survival of the Müllerian duct as we are dealing with may be compared with such persistence of the ducts of the Wolffian bodies in the animals mentioned. It is unfortunate that the testis was not more carefully examined, particularly with reference to the presence of the organ of Giraldés and the hydatid of Morgagni. The explanation is that when the organs were removed from the body the deformity was

supposed to be of the ureter, and when the identification was made by the skill of Mr. Stewart, opportunity had passed, the body having been buried.

The large orifice by which the duct opens into the urethra appears to be the opening of the vesicula prostatica. To avoid loss of a continuous presentation of the structure further section of the prostate has been avoided.

It may be added that the man was married and had several children, and that he suffered from dysuria, dependent on stricture, for some time before death.

The only similar case of which I have been able to find record is shortly reported in the 'British Medical Journal' of September 6th of this year, as having been examined by M. Rémy. The subject was a boy, aged 6; the malformation was unilateral; the persistent Müllerian tube took much the same course as in the present case, but its head was related with a number of cysts, supposed to be the remains of the Wolffian body. The hydatid of Morgagni and the organ of Giraldés were both present.

The drawing accompanying the specimen (see woodcut, p. 12) was made by Mr. Stewart, to whom all the credit of the identification and preparation is due.

CASE OF CHOLECYSTOTOMY

PERFORMED FOR

DROPSY OF THE GALL-BLADDER DUE TO IMPACTION OF A GALL-STONE.

BY

LAWSON TAIT, F.R.C.S., &c.,

SURGEON TO THE BIRMINGHAM HOSPITAL FOR WOMEN.

(Received October 14th—Read November 11th, 1879.)

The great success which has followed the performance of operations for ovarian tumours has led to the extended use of abdominal section in directions which could hardly have been hoped for till within the last few years. My own experience, of now nearly two hundred abdominal sections, has been such as to lead me to this conclusion, that no abdominal or pelvic tumour ought to be left unexplored, if it seems to affect seriously the life and comfort of the patient, unless it be incontestibly of a cancerous character.

Dr. Marion Sims attributes to Dr. Handfield Jones, and I think correctly, the merit of first suggesting that the liver and gall-bladder should be included within the field of surgical practice more fully than they had been up to that point, and particularly that surgical interference should be made in cases where death is threatened from the impaction of a gall-stone.

To Dr. Marion Sims himself must be given the credit of having followed out this suggestion with his usual

boldness and ability, and he himself points out that the case in which he did it was not successful, only because the operation was too long delayed.

To my good fortune it has fallen to be the first to follow out Dr. Handfield Jones' idea and Dr. Sims' plan successfully.

Elizabeth M—, æt. 40, was admitted to the hospital on August 18th, having been sent to me by Dr. Abraham Colles, of Bridgnorth, on account of an abdominal tumour.

She had been married eighteen years, had borne six children, her menstruation had always been normal, and she had enjoyed perfectly good health until the summer of 1878. At that time she began to suffer from severe spasmodic pains in the right side, these being always aggravated by walking or by lifting even slight weights. In September she noticed a swelling at the seat of pain, and this slowly increased. During last winter her pain became much more intense, her appetite failed, she lost strength and flesh rapidly, and on admission she presented an emaciated and almost cachectic appearance. She also suffered at that time from incessant headache and sickness, and obstinate constipation. The seat of pain was over the right kidney, where there was a heart-shaped tumour, firm and elastic, in which no fluctuation could be detected, and which was extremely tender to the touch. On examination under ether, this tumour was found to be perfectly movable towards each side, indeed, it could be pushed completely across the middle line to the left side. All round it a note of intestinal resonance could be produced. When pushed over to the left side, its heart-like shape became very apparent, and when it lay on the left side of the vertebral column, with its apex directed downwards and to the left, its base evidently retained a connection with the right side.

A careful examination of the urine gave only negative results, though she spoke vaguely of its having been occasionally dark in colour, muddy, and deficient in quantity.

At the consultation, held with my colleague Dr. Edginton, upon the case, a variety of suggestions were made for diagnosis, the chief of which were cystic enlargement of a floating kidney, a tumour of the head of the pancreas, and dropsy of the gall-bladder. But no decided diagnosis was attempted, and my proposal to open the abdomen, and thus ascertain the nature of the tumour, was agreed upon.

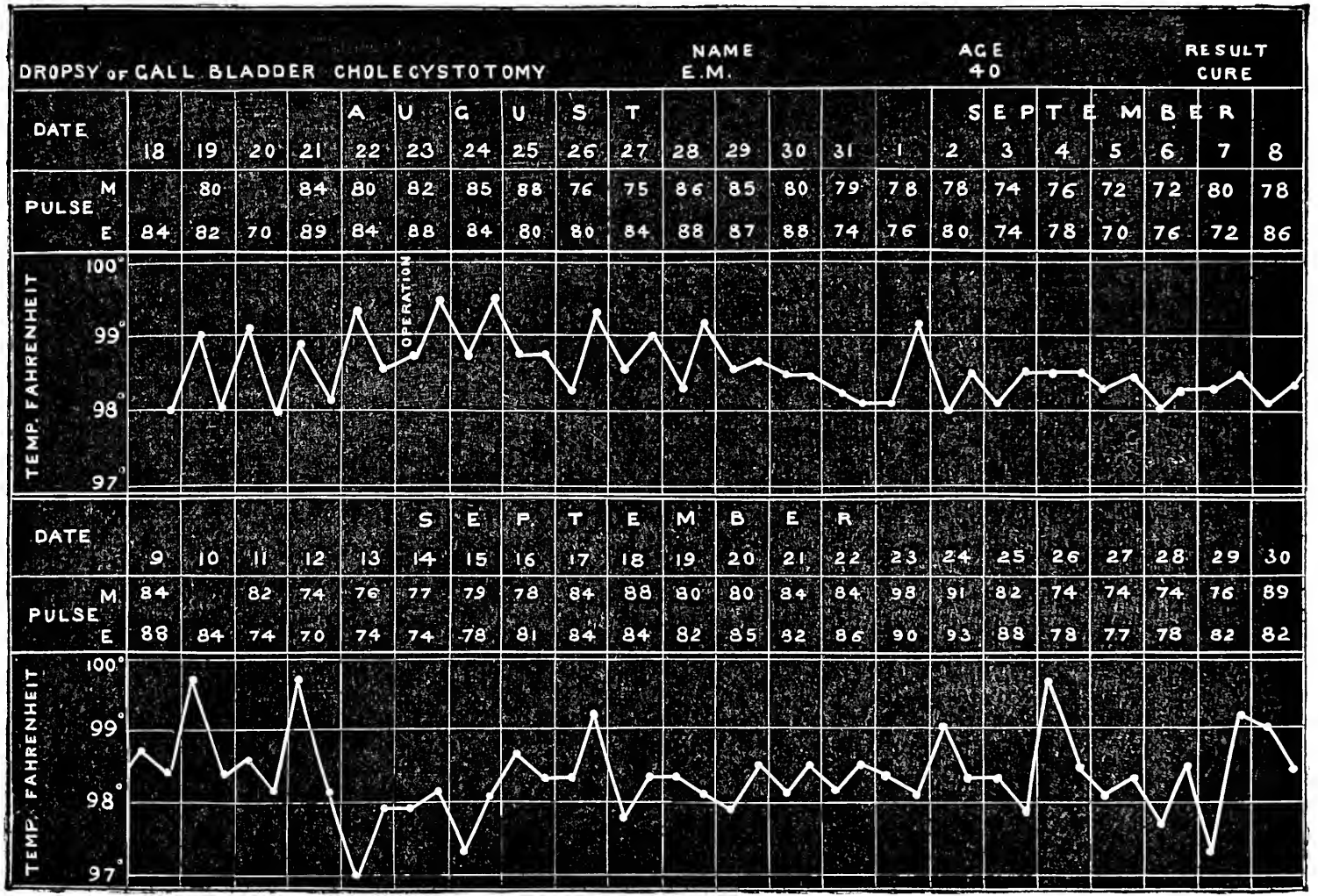
On August 23rd I opened the abdomen in the middle line, to the extent of four inches, the umbilicus forming the centre of the incision. It then became at once evident that the tumour was a distended gall-bladder. I passed the needle of an aspirator into the apex and drew off a quantity of white starchy-looking fluid, probably amounting to between twelve and fifteen ounces, but I cannot speak positively as to its amount, as it was unfortunately thrown away by a nurse immediately after the operation. I then opened the gall-bladder at the point of puncture, so as to admit my finger, and came at once upon a large round gall-stone lying loose in the cavity. This I easily removed, and on further search I found another of rather larger size, and probably of pear-shape, at the entrance of the duct, impacted in it, and evidently the cause of the dropsical distension of the gall-bladder. The removal of this stone was a matter of very great difficulty, in fact, it took a very much longer time to effect than all the other steps of the operation put together. From the long, narrow, funnel-like cavity in which it was lodged, and from the mobility of the bladder, it was very difficult to seize, and when at last I did get hold of it I found it adherent to the mucous surface. I had then to consider the extreme likelihood that in removing this impacted stone I might tear the walls to which it was attached, and thus certainly kill my patient. I therefore performed a very careful and protracted lithotrity, chipping little fragments off the stone regularly all over its exposed surface till I had the satisfaction of lifting out its nucleus. I then passed a blade of a fine

pair of forceps on each side of it, and by a gentle squeeze broke up the remainder, and was then enabled to lift it all out. The weight of the stone removed entire is 4·2 grammes, and that of the fragments I could gather of the broken stone is 2·9 grammes, but of the latter stone as much again must have been lost on the sponges which were packed into the wound during the process of crushing, and upon which I had constantly to wipe my instruments. I washed the cavity out repeatedly and took every precaution that I could to secure that no fragments were left. I then stitched the wound in the gall-bladder to the upper end of the wound in the abdominal walls by continuous sutures, leaving the aperture into the bladder quite open, and then I closed the rest of the abdominal opening in the usual way. The operation was performed with complete antiseptic precautions, and the anæsthetic employed was ether.

She rallied from the operation completely in a few hours. I dressed the wound antiseptically the same evening at 11 p.m., and found the dressings stained with healthy bile. In the further progress of the case there is very little to report, save that the flow of bile from the wound continued till the 3rd of September, when the dressings were discontinued and zinc ointment was used in their place. The stitches were removed and the wound was completely healed on September 9th, when she began to take solid food, up to that time her diet having been restricted to milk and beef tea. On the 14th she sat up for the first time, and on the 30th she went home quite restored to health, free from pain and all her former symptoms, and having gained at least a stone in weight.

The annexed temperature chart is the best indication of the evenness and rapidity of her recovery.

Looking back upon this case, I do not think that a more accurate diagnosis was possible, for there was an entire absence of those symptoms which usually characterise cases of gall-stone. After the operation she told us that one of her neighbours had said to her one


DROPSY OF GALL BLADDER CHOLECYSTOTOMY
NAME E.M.
AGE 40
RESULT CURE
DATE
AUGUST SEPTEMBER
18 19 20 21 22 23 24 25 26 27 28 29 30 31 1 2 3 4 5 6 7 8
PULSE M 80 84 80 82 85 88 76 75 86 85 80 79 78 78 74 76 72 72 80 78
PULSE E 84 82 70 89 84 88 84 80 80 84 88 87 88 74 76 80 74 78 70 76 72 86
TEMP. FAHRENHEIT
100°
99°
98°
97°
OPERATION
DATE
SEPTEMBER
9 10 11 12 13 14 15 16 17 18 19 20 21 22 23 24 25 26 27 28 29 30
PULSE M 84 82 74 76 77 79 78 84 88 80 80 84 84 98 91 82 74 74 74 76 89
PULSE E 88 84 74 70 74 74 78 81 84 84 82 85 82 86 90 93 88 78 77 78 82 82
TEMP. FAHRENHEIT
100°
99°
98°
97°

day that she thought the patient was jaundiced, but beyond this no history could be got at of any symptoms pointing clearly to the true nature of the case. The singular mobility of the tumour was also a most puzzling condition. Fortunately, our advanced practice in abdominal surgery makes our limited powers of diagnosis in such a case of less importance, and I thoroughly agree with Dr. Sims that we should not wait till the approach of almost fatal symptoms puts the diagnosis in unmistakable fashion, but that "we shall make an early exploratory incision, ascertain the true nature of the disease, and then carry out the surgical treatment that the necessities of the case may demand."

I have only one other point to speak of, and that is that the operation was done with rigid antiseptic precautions, as all my abdominal sections have for some time been. It would be foreign to my purpose here to enter into a discussion for which I do not think my own experience is as yet ripe, but the impression is growing upon me that my increasing success in these operations has little or nothing to do with the antiseptic system. I use it, however, with the utmost rigour, in order that my patients may have every chance for their lives, and because I have as yet had insufficient evidence that it does them any harm beyond delaying the healing of wounds, as it almost invariably does.

ON

TREPHINING FOR TRAUMATIC EPILEPSY,

WITH AN ILLUSTRATIVE CASE.

BY

JAMES F. WEST, F.R.C.S.,

SENIOR SURGEON TO THE QUEEN'S HOSPITAL, BIRMINGHAM.

(Received October 14th—Read November 25th, 1879.)

The value of the operation of trephining has been, and still is, a vexed question in the surgical world; at one time its merits have been extolled to the skies, at another the operation has been regarded as almost certainly fatal, and therefore only to be resorted to in very dangerous cases or where death would otherwise be inevitable. Between these extreme views the golden mean of practice will probably be found. The trephine is not to be used without good grounds for its employment, but in traumatic epilepsy and paralysis it has often proved of the greatest utility. With a view of helping to illustrate the treatment of epilepsy by the operation of trephining, I venture to bring forward this case, in which its good effects are certainly beyond dispute, the girl, from being in a state approaching coma and passing her urine and fæces involuntarily, having been restored to a state of comparative health; her speech and reason returned a few days after the trephine was

used, and she has been rescued by means of the *operation* and *the operation alone* from idiocy, if not from death. I think, under these circumstances, I am fully justified in putting the case on record.

I thank your highly esteemed President personally for giving me the opportunity of bringing this subject before the Medical and Chirurgical Society, and I trust that in the discussion which will ensue, the opinions of many members who have given attention to this question, and who are far more entitled to speak on it than I am, may be elicited, and that thereby some of the difficulties in this branch of surgery may be cleared away.

Eleanor Holden, æt. 14, residing at 19, Belgrave Street, Birmingham was admitted into the Queen's Hospital on November 21st, 1878.

She was first placed under the care of Dr. Heslop, and then transferred for surgical treatment to Mr. West's charge. During early childhood and until she was seven years old she was sharp and intelligent. She went to school and was able to read and write a little. In the spring of 1871, she was struck on the forehead by a stone, thrown by a boy in the street; she fell to the ground unconscious, but rallied in a few minutes, so as to be able to walk home. The mother described the wound as being "a hole so that you could see right into her head." She was attended to by a chemist, had no grave symptoms, and was considered to be quite well in three weeks. From the date of the accident the mother, however, noticed that she was drowsy, forgetful, less intelligent, and that she sooner grew tired than the other children. She frequently complained of "funny feelings in her head," which were worse after excitement, or after alcoholic stimulants. During the last three years, these phenomena have been more pronounced. She sometimes falls asleep over her food. During the current year she has had fits, which are described by her mother as follows:—"Suddenly she goes in a helpless way; she may be busying about, and instantly she seems 'to go bad;' she falls

down gradually and gives a mournful cry, saying, 'Oh, my hand,' 'Oh, my arm;' sometimes one, sometimes the other." She complains most of the left side. Since the first of these epileptic fits she has been affected in her speech, sometimes entirely losing it, and she has passed her water involuntarily. She only vomited during the first attack, but never since. These fits occur paroxysmally, and they are becoming of more frequent occurrence, and longer in duration, and she now loses her speech for three or four days together; she has never had any hemiplegia or impairment of vision or hearing.

Present attack.—She had a fit four days before admission, and she has not spoken since. She has passed urine and fæces involuntarily, this being the first time of involuntary defecation. She is able to take nourishment.

Symptoms on admission.—Patient is small for her age; she has well-marked notches in her teeth; is fairly nourished; is unable to stand; she lies "all in a heap" in her mother's lap. Her expression is vacant and staring—quite idiotic. Pupils somewhat dilated and possibly unequal; pulse 97. She neither answers nor attempts to answer questions, nor will she open her mouth or protrude the tongue. There is twitching of the orbicularis oris muscle. There is no paralysis of the extremities present. A firm white cicatrix, as large as a fourpenny-piece, and depressed below the surface of the skin, to the extent of an eighth of an inch, is found upon the right side of the forehead, two inches above the eyebrow, and just to the right of the median line. All medical treatment having failed, and the patient's condition being apparently irremediable by other measures, Mr. West determined to trephine, and the operation was performed on November 25th, 1878, as follows:—The patient having been anæsthetised by ether, a crucial incision extending down to the bone was made over the depressed cicatrix. Mr. West then separated the pericranium with a raspatory, and having reflected the raised flaps removed with a small trephine two circles of bone, the one immediately

over the depression in the outer table and the other a little to the right of and adjoining it. The former fragment showed that there had been depression of the outer table and compression of the diploë, although the inner table bore no evidence of having been fractured. The membranes were not injured in the operation, the spicula being washed away with a stream of tepid carbolised water. The pericranium was brought together by four catgut and the skin by four silver sutures. The operation was performed with antiseptic precautions, and dressed after Lister's method, except that no drainage tube was employed. The patient vomited immediately after the operation, but had no symptoms of any kind during the day. The pulse and temperature, which in the morning were 92° and 97° respectively, rose at night to 198° and 98·5°.

25th.—She passed a comfortable night, and has no symptoms; she answers questions slightly; wound dressed; parts normal. Three sutures removed.

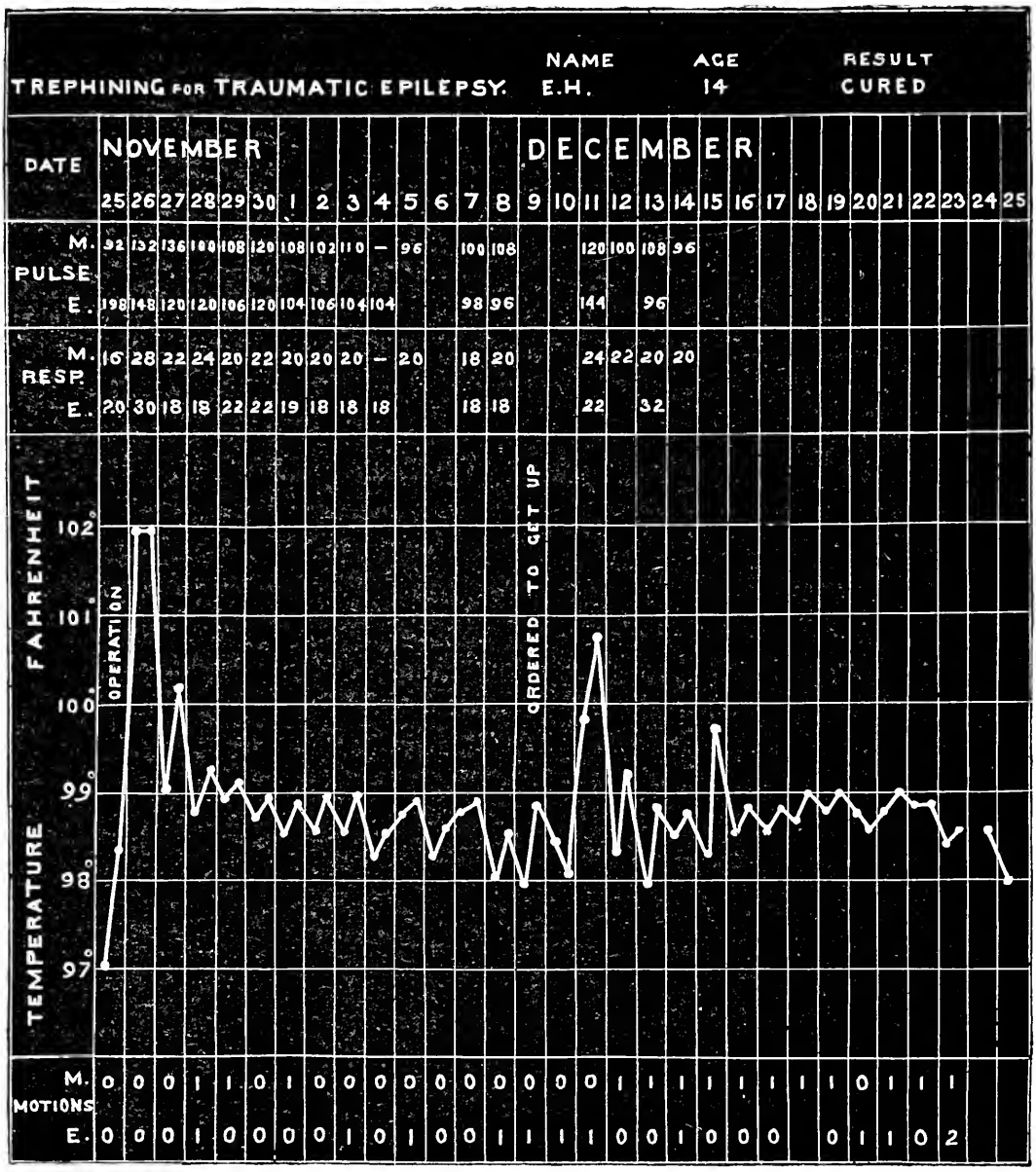
27th.—She had a good night, and is now able to answer questions readily. She has retained her urine and fæces since the operation; she protrudes the tongue when asked to do so.

28th.—She now can talk quite naturally; wound when dressed appeared solid, except at the upper part of the crucial incision. The remaining suture was removed.

30th.—She is quite intelligent. Bowels regular; tongue moist and clean. The condition of the temperature and pulse from the date of the operation will be seen on examination of the accompanying chart.

December 3rd.—Antiseptics discontinued. On December 9th she sat up, and from that time till December 28th, when she left the hospital, she was walking about the wards, and was quite cheerful and happy. A gutta-percha cap, well padded and moulded to the shape of the head, was worn with comfort.

February 17th, 1879.—She was readmitted to-day, looking very poorly. She had been working very hard at home, and had been taken ill quite suddenly. By means


TREPHINING FOR TRAUMATIC EPILEPSY.
NAME E.H.
AGE 14
RESULT CURED
DATE
NOVEMBER
DECEMBER
25 26 27 28 29 30 1 2 3 4 5 6 7 8 9 10 11 12 13 14 15 16 17 18 19 20 21 22 23 24 25
PULSE
M. 92 132 136 100 108 120 108 102 110 – 96 100 108 120 100 108 96
E. 198 148 120 120 106 120 104 106 104 104 98 96 144 96
RESP.
M. 16 28 22 24 20 22 20 20 20 – 20 18 20 24 22 20 20
E. 20 30 18 18 22 22 19 18 18 18 18 18 22 32
TEMPERATURE FAHRENHEIT
102°
101°
100°
99°
98°
97°
OPERATION
ORDERED TO GET UP
MOTIONS
M. 0 0 0 1 1 0 1 0 0 0 0 0 0 0 0 0 1 1 1 1 1 1 1 0 1 1 1
E. 0 0 0 1 0 0 0 0 1 0 1 0 0 1 1 1 1 0 0 1 0 0 0 0 1 1 0 2

of free purgatives and absolute quiet she became quite intelligent again in ten or twelve days, and on March 17th she left the hospital as well as before, having had no epileptic attacks during her stay in the hospital.

May 5th, 1879.—She was readmitted with well-marked hemiplegia of the left side, but with no impairment of the intelligence; her speech was thick and her tongue, when protruded, turned to the left side; pupils equal.

Absolute quiet and purgatives put matters right in the course of ten days, so that no sign of paralysis remained.

On May 16th she was again able to go home. Since that time she has occasionally visited the hospital as an out-patient, but has never required to stay; she has taken iron and quinine, and has several times passed a few weeks in the country.

Her mental faculties have become more acute, and her temper better since the operation. She is, however soon tired, and requires great kindness and consideration from all those about her.

In bringing this case forward, I do not propose to go into the general question of the use of the trephine in injuries of the skull, but I intend to confine my observations as far as possible to those cases of epilepsy and paralysis in which it has been used, and from the consideration of recent writings on the subject to endeavour to show the value of the operation in certain suitable cases. I think it desirable to quote some authors and to give some statistics, but I trust I may be pardoned if I have omitted to mention many whose opinions are entitled to be heard, and that the omission may be attributed to my anxiety not to embarrass my paper with a too elaborate citation of authorities.

That epilepsy is not unfrequently due to injuries of the skull is abundantly proved in the history of surgery, but it is less widely known that relief of the symptoms has again and again followed the discharge of some portion of exfoliated bone, or the elevation of a portion of some of the cranial bones by the trephine.

An interesting example of this disease where benefit was afforded by the keeping open an issue in the head, by which exfoliated fragments of the skull could be discharged, is given in Dr. Graves's 'Clinical Lectures,' p. 409.

Mr. South, in dealing with this subject, says ('Chelius's System of Surgery,' vol. i, p. 415) :—"Trepanning may be necessary in continued severe headache with convulsions, or epileptic symptoms which decidedly arise from the place of the earlier injury," and he quotes four cases : two from the Museum of St. Thomas's Hospital, in which the operation was unsuccessful, and two from the practice of the elder Cline, in which trephining was followed by perfect recovery.

One of the most recondite and valuable memoirs in the English language, on 'Trephining for the Relief of Epileptiform Attacks occurring after Injury to the Head,' was written by my friend Dr. James Russell, Senior Physician of the Birmingham General Hospital, and appeared in the 'British Medical Journal' of 1865, and in it the whole subject is thoroughly discussed.

After giving the particulars of a case which occurred in his own practice, which was operated on by Mr. Bolton, and which unfortunately terminated unsuccessfully, he gives a *resumé* of 80 cases of epilepsy arising from injury, in 50 of which trephining was performed; of these, 44 recovered from the operation; progressive amendment and recovery ensued in all of them except in 5 cases. Speaking of the operation of trephining in epilepsy, Prof. Gross, in his 'System of Surgery' (vol. ii, p. 117) says, that the first case was by La Motte in 1795, and was partially successful; and he also mentions that in 1820 Dr. Dudley, in vol. i of 'Transylvania Journal of Medicine,' published 5 cases, of which 3 were successful.

Prof. Gross has himself had 4 cases, 1 cure, and 3 deaths, while he has witnessed 3 others, which ended fatally. Dr. J. S. Billings ('Cincinnati Lancet,' June,

1867) has given an analysis of 72 cases of epilepsy subjected to trephining, of which 42 were cured, 4 were unchanged, 16 proved fatal, and the rest were improved but not entirely relieved. In the 'Surg. Hist. of the War of the Rebellion,' "15 cases of gunshot fracture of skull are reported in which trephining was used, in which the patients recovered sufficiently to resume their duties and 4 sufficiently to return to modified duty, while in 6 the men recovered and were unchanged, paroled or furloughed;" 35 recovered after trephining with different degrees of physical disability.

It is, however, only right to state, in contrast to these favorable results of operative treatment, that the most recent English statistics of trephining, as given by Mr. Bryant in his 'Practice of Surgery' (vol. i, p. 232, 3rd edition, 1879) are not encouraging. He says: "At Guy's Hospital trephining and elevation of bone for head injuries have been performed in 51 cases during seven years, and of these only 12 recovered. At St. Bartholomew's Hospital it was recorded by Callender, in 1867, that the operation had not been performed for six years. At University College Erichsen gives 6 cases of recovery out of 17." On the other hand, the experience of the American Surgeons during the War of the Rebellion ('Surgical Hist.', table 8, vol. 1, p. 319) is far more satisfactory, 95 having recovered out of 220 cases, *i.e.* a rate of mortality of 56·6; while Dr. Echeverria ('Archives de Médecine,' December, 1876) gives a table of 145 epileptics who were trephined, from which it appears that 93 were cured, 18 improved, 5 in which no change occurred, 1 aggravated, and 28 died—a mortality of 17·5 per cent.

Striking testimony to the value of the trephine in depressed fractures of the skull is given by Dr. Robert Hudson of Redruth ('British Medical Journal,' July 21st, 1877). No more enthusiastic admiration of the operation could be expressed than is given in his summary. "It is believed by those surgeons (*i.e.* in Redruth and Camborne)

that no danger whatever attaches to the operation; and recovery is the rule after trephining operations." [1]

The most valuable addition to our knowledge of this question is given in a recent French work, 'La Trépanation guidée par les localisations cérébrales,' by Dr. Le Just Championnière, Paris, 1878. In 1874 Dr. Le Just Championnière had under his care a man who was picked up insensible in the street with a superficial scalp wound over the left parietal eminence and some ecchymosis of the eyelids. He was found to have incomplete paralysis of the right upper limbs, and four days afterwards he was attacked with convulsions affecting the right shoulder and left arm, the lower limbs also being seat of regular spasms.

No improvement taking place and after due deliberation, Dr. Championnière explored the scalp wound, detected a fracture in the left parietal bone, and, to make a long story short, had the satisfaction of seeing his patient recover, not only from the epilepsy, but also from the paralysis; some aphasia, which persisted for a longer period, eventually disappeared. Dr. Championnière expresses the greatest faith in the operation of trephining, and asserts that it may be undertaken with a confidence formerly unknown, since the labours of Broca, Ferrier, Charcot, and others, have established the doctrine of cerebral localisation; "etant donnés certains troubles moteurs consecutifs à des traumatismes du crâne on peut determiner par leur observation assez nettement la region de l'écorce cérébrale

[1] A case showing the good effects of the trephine in epilepsy, which was under the care of Mr. Cooper Forster and Dr. Wilks, is reported in the 'Lancet' for August 30th, 1873. The patient was a boatman, æt. 39, who suffered from epileptic fits produced by "a blow on the top of the head, which stunned him and produced a slight swelling" which remained four months after the accident. Iodide of potassium in large doses, and free incision down to the bone, which liberated a small quantity of pus, not having put a stop to the fits, Mr. Forster "trephined to the right of the median line and removed a piece of bone, which was rough and excavated on the outer surface, of great density of structure, and of increased thickness, being three eighths of an inch thick in one place and a quarter of an inch thick in another." There was not "the least sign of a fit" after the operation.

compromisé, pour guider avec securité la main du chirurgien qui se propose de libérer par la trepanation du crâne."

Dr. Championnière gives to M. Broca the credit of being the first to initiate this method of trephining, guided by the phenomena of cerebral localisation. M. Broca's first case was unsuccessful, but in 1866 he brought before the Surgical Society of Paris the case of a child on whom the operation had been performed by him with perfect success for epileptiform convulsions coming on after fracture of the skull. In the discussion which followed M. Legouest, the eminent professor of military surgery, spoke in favour of the operation, and cited a case of fracture of the left parietal bone with paralysis of the right arm, in which a fatal result was in all probability avoided by the use of the trephine. He thus formulates his opinion of trephining in doubtful cases :—" Enfin pour exprimer toute ma pensée, je dirai, si singulière que puisse paraitre ma formule, qu'il y a probablement indication de trépaner toutes les fois qu'on doute qu'il y a lieu de le faire."

The subject of trephining has recently been the subject of important memoirs by MM. Larrey and Sédillot; the former, though not very strongly in favour of the operation, lays down a series of plain directions as to when and in what cases it should be done, and when it is contra-indicated. These would be too lengthy to quote in full, but, as bearing on the subject of the trephine in epilepsy and paralysis, he recommends it " in cases where there are grave cerebral complications, prolonged hemiplegia with supposed extravasation, which have resisted all other methods of treatment." M. Sédillot, in a communication to the Society of Medicine of Strasburg in 1869, expressed himself strongly in favour of trephining, and stated that now antiseptic treatment was employed the operation might be undertaken with far less risk than formerly, adding that, while at the Strasburg Hospital trephining had never before been successful, under the new method of procedure the operation had been followed by good

results. In M. Sédillot's opinion as to the value of the antiseptic method of dressing after this operation I fully concur. In my case it answered admirably, the wound healing rapidly, and the patient being throughout entirely free from constitutional excitement, as is shown by reference to the accompanying temperature charts. In 1876, M. Sédillot again advocated trephining in a paper presented to the Académie des Sciences, which contains the result of 106 observations made by him. In Germany trephining has not been a favourite operation; Stromeyer was strongly opposed to it, while Nussbaum, Esmarch, Beck, and Bergmann only perform it in exceptional cases, and their views may be taken as typical of the present state of opinion on this subject in the various schools of German surgery.

In France, on the other hand, trephining is becoming a popular operation, and one which daily seems to find fresh advocates.

The operation has been placed on a different basis since the existence of certain zones on the surface of the brain in the fronto-parietal region, which if irritated give rise to convulsive movements on the opposite side of the body, or if removed or destroyed, to paralysis of those parts, has been determined by the observations of Hitzig and Ferrier. It remained now only to determine what are the points on the surface of the skull which correspond with those parts of the brain. This difficult question Dr. Lucas Championnière proposes to solve by means of biauricular cartoons; he thinks it possible, by their means, to establish so correct a diagnosis of the site of the lesion that he could undertake the operation of trephining with precision in cases of paralysis of one set of muscles when other symptoms are absent, or where there are convulsions of the opposite half of the body to that which has been the seat of the injury.

The directions laid down by him, and by M. Broca, for finding the fissure of Rolando, around which most of the motor centres are situated, are very elaborate, and their

enumeration would occupy too much time. Attention to them is no doubt of the highest importance to anyone called upon to diagnose the region of the brain which is the seat of injury. Further research in this direction will no doubt add largely to our knowledge of the indications which are to be derived from the theory of cerebral localisation. In the meantime we may be thankful to those, like Turner and Ferrier in this country, and Broca and Charcot in France, who have done so much for the department of physiological anatomy, denominated cranio-cerebral topography.

It seems to me in conclusion that in all cases of brain injury followed by epilepsy or paralysis which call for operative interference we have three great questions to determine :

1st. Is the theory of cerebral localisation sufficiently established to enable us to act upon it ?

2nd. Are our indications in any given case, either of paralysis or epilepsy, sufficiently precise and well marked to warrant us in recommending the use of the trephine at a particular point of the skull ?

3rd, and lastly, have we a better chance of serving our patient by immediate, by intermediary, or by secondary trephining.

The best answer I can give to these three questions may be thus summarised :

The first point must, I think, be conceded, after the recent experience of surgeons whom I have quoted both in France, Germany, and America.

The second point will be a long time before it is definitely settled, but cases such as those of Broca, Championnière, and the one recorded in this paper, gives us encouragement, and reference to them may aid us in determining to what part of the skull the trephine should be applied.

The third point must depend on the nature of the accident, and the extent of the lesion of the brain and its

membranes, the constitution of, and the circumstances surrounding, the patient; but, as a general principle, it may be taken that secondary operations are less fatal than either primary or intermediary, and that when undertaken, the antiseptic treatment of the wound should always be employed.

ON TWO CASES

OF

CARCINOMA OF THE BREAST,

PRECEDED BY SO-CALLED

ECZEMA OF THE NIPPLE AND AREOLA.

BY

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(Received October 15th.—Read December 10th, 1879.)

Since the appearance of a short paper by Sir James Paget in the 'St. Bartholomew's Hospital Reports' for 1874, "On Diseases of the Mammary Areola preceding Cancer of the Mammary Gland," two contributions on the same subject by Mr. H. T. Butlin have been made to the 'Transactions of the Royal Medical and Chirurgical Society.' Mr. Butlin's first paper, contained in the 59th volume of the 'Transactions,' gives a description of the minute anatomy of two breasts, the areolæ of which had been the seat of long standing eczema, and which were removed complete on account of the disease of the areola, the breasts themselves being unaffected with cancer. The second paper, contained in the 60th volume, gives a description of two breasts in which carcinoma had actually occurred, preceded by eczema of the nipple and areola.

During the discussion which followed the reading of this latter paper, I exhibited a drawing of the breast of a woman who came under my observation in the summer of 1876, the nipple and areola of which were affected in a similar manner to, but more severely than those described by Mr. Butlin. The deeper seated changes in this breast were such that several competent microscopists who examined

them hesitated to pronounce them cancerous. This I ventured to state in my remarks at that time. Malignant, however, the disease proved to be, and in eighteen months from the time of the removal of the breast, the woman died of widely disseminated secondary cancer. In the early part of the present year another very similar case came under my care. This patient also died having secondary cancer in various internal organs.

These two cases I have thought worthy of being brought under the notice of this Society for three reasons. *First.* The supervention of cancer upon eczema of the nipple and areola is, I believe, at present unknown except in London, though it is now more than five years since Sir James Paget's paper appeared. The cases mentioned by Sir James Paget and Mr. Butlin are, so far as I know, the only ones as yet published.[1] *Second.* In no previously reported case have the complete clinical history and the post-mortem conditions been described. *Third.* Both these cases differ in some important clinical points from Sir James Paget's description of the cases seen by him, and as the association between so-called eczema of the nipple and cancer of the breast is as yet imperfectly understood, and but little recognised, any marked differences in the general characters of the affection deserve to be made known. Further, both my cases have furnished Dr. Thin with material for working out his views on cancerous growths generally, which he has explained in a paper read before this Society.

CASE 1.—*So-called eczema of the nipple and areola followed by ulceration of the nipple and carcinoma of the mamma. —Recurrence in the peritoneum, liver, and cicatrix.— Death.*

Dinah L—, æt. 40, applied at the cancer out-patient

[1] Since this paper was sent in to this Society, my colleague, Mr. Lawson, at a meeting of the Clinical Society, on October 24th, 1879, read the report of a somewhat similar case.

department of the Middlesex Hospital on July 20th, 1876, for disease of the left breast.

A needlewoman by occupation, she had been married fifteen years, and was the mother of seven children, the youngest of whom was two years old. She had also had two miscarriages. Her catamenia had ceased to be regular since the previous December. She had suckled five of the children with both breasts, but the last two with the right breast only. She had weaned her last child five months before coming under observation, but there was still some milk in the left gland. There was no history of cancer in her family; both father and mother had lived to a good old age, being seventy-three and eighty-four respectively, and her brothers and sisters were all in good health. She herself had suffered from small-pox when seven years old, rheumatic fever when twenty, and an abscess in the patella bursa six months ago.

For six years the nipple of the left breast had been sore. It commenced with a small blister on the summit of this nipple, which, after a time disappeared; then the nipple became red and moist until it gradually melted away, leaving a hole or depression in its place. All this had happened about four years ago, but there was no increase in the size of the hole till six months before, when it began to enlarge and was surrounded with a raised edge; during the whole of the four years there was a weeping eczematous condition of the surface around the hole which had become much worse of late.

When I first saw her there was in the site of the nipple a depressed and foul ulceration bathed in a thin and rather glutinous pus, and having an irregular sinuous border. The upper portion of the border was raised and everted, and consisted of hard, coarse, pale, granulating tissue, the hardness not extending much beyond the edge. At the lower and outer side the margin was neither raised nor indurated.

Beyond the ulceration, and spreading over the skin of the breast in an annular manner for about one inch and a

quarter in all directions, was a purplish-red and weeping surface quite soft to the touch, and showing at places a disposition to be covered with new cuticle.

In the substance of the breast beneath this ulceration, there was some ill-defined hardness, but the ulcer was not fixed or tied down to the underlying tissues. A small warty growth existed a little way from the ulcer. One of the axillary glands was slightly enlarged. The right breast was marked by the scar of an abscess which burst five months ago, and the areola of this gland was a good deal retracted.

She was admitted into the hospital on August 21st, and by this time the ulceration had extended both in area and in depth; the edges were more raised, and some slight hæmorrhage frequently occurred from its surface. The eczematous condition had spread to the skin beyond the areola.

On August 30th, the left breast and the enlarged axillary gland were removed through one wound; on the third day afterwards the sutures were removed, and on the nineteenth day the whole of the wound, except a small spot in the axilla, was soundly healed. She left the hospital well on October 3rd.

From this date till January 27th, 1878, a period of nearly one year and four months, I heard nothing of her. I was then asked to visit her as she was extremely ill, when she gave the following acount of herself:

Shortly after leaving the hospital the catamenia, which had ceased for eight months, reappeared; a little later and she became pregnant, and in August 1877, she was delivered of a healthy-looking child. Two months afterwards she was taken ill with sickness, giddiness, and faintness, quickly followed by a severe pain in the pit of the stomach. Three days afterwards she felt a lump in the belly, near to the pit of the stomach, from which sharp penetrating pains shot across the body and through to her back. The lump increased rapidly in size; she frequently vomited a greenish fluid, occasionally tinged with blood;

and had repeated attacks of diarrhœa, attended with a bearing-down pain in the rectum. During the last week both the severity of the pain and the size of the tumour had much increased. The general surface of her body was slightly jaundiced—the face and conjunctiva still more so. As she lay on her back a large tumour, intensely hard to the touch, was seen extending across the right hypochondriac and the umbilical regions, the border of which projected very prominently against the abdominal walls. Some nodules of bony hardness could be felt over other parts of the abdomen, and a wave of fluid was distinctly displaced by the fingers from between the abdominal wall and the surface of the enlarged liver. The cutaneous veins of the abdomen were much enlarged. A flat, indurated growth, the size of a crown piece, occupied the middle of the scar in the left mammary region.

The axillary glands were unaffected. Large internal hæmorrhoids projected beyond the sphincter ani. She was again admitted into the hospital, and died seven days afterwards.

At the post-mortem examination, which was made by Dr. Coupland, the peritoneum was found extensively studded with firm cancerous nodules. Much bile-stained fluid in the peritoneal sac. The liver was enormously enlarged, especially its right lobe, and weighed 108 ounces; its surface was uneven and nodulated, and its capsule thick, rough, and opaque. On section the tissue was extremely firm, of the consistence of gristle, and was pervaded by tough, gristle-like nodules, whitish, ill-defined, and mottled with patches of orange-coloured pigment. Spreading out in all directions around the nodules similar material infiltrated the hepatic tissue, the lobulation of which was very ill-defined. There were some hard, whitish, flattened nodules in the falciform ligament.

The various diseased parts, both those removed at the time of the operation, and subsequently at the post mortem, were examined microscopically. The following is a report of the same made by Dr. Goodhart:

"In the region of the nipple was a rather deep excoriated or ragged-looking ulcer with but little thickening of the edges or evidence of any new growth. At another spot, not far off, on the free surface of the skin, was a small warty projection about the size of a pea, and 2‴ high. The rest of the skin had a peculiar milky-white marking over it, as if there had been a good deal of superficial ulceration now healed.

"On section the ulcer described does not go deeply, and the vertical tubes constituting the nipple look uninjured as they run in the fat. The wart-like projection is quite cuticular, and no tendency to infiltration can be seen anywhere. The breast itself is much atrophied, being merely represented by straggling fibrous bands in the substance of the fat. These are usually mere fibres, but in some part they attain considerable thickness.

"The skin appears to me everywhere of rather unusual thickness.

"Sections of the warty growth of the skin; of the tubuli recti of the nipple; of the ulcer; and other deeper parts of the breast, were examined microscopically, and with the following results:—The wart had a rather alveolar structure, and was composed of large mononucleated cells in places tending towards angularity of outline, and of glandular or epithelial appearance. They closely corresponded with those found elsewhere in the deeper layers of the skin. Underneath the ulcer the whole thickness of the skin (Plate II, Fig. 1) was densely infiltrated with cells, but elsewhere, though all the sections that were examined were considerably affected in the same way, the distribution was more partial, and it was in these places that the mode of extension of the disease was seen. Small groups of cells were seen, generally speaking, round gland tubes (Figs. 3, 4, 6), but occasionally round small vessels, and in all cases having a suspiciously close resemblance to the epithelial type. In only one or two places did these foci extend beyond the true skin, but, as will be seen in the drawings (Fig. 2), they extended to its very limit.

"The gland-tubes themselves (Pl. II, figs. 4 and 5), were crowded with epithelium in an abnormal manner, but this is an appearance so apt to be produced in preparation of the slides, that though it may have been morbid, we shall not insist upon it. The adipose tissue, which to a casual examination looked healthy, was in all probability also diseased, as cells, sometimes single, sometimes in twos and threes, of appearance similar to those before described, were seen in many places. It seems probable that they would have proceeded to the formation of foci as others had done in the skin, and this makes me think that ere long there would have been a wide-spread infiltrating growth throughout the entire gland.

"The appearances described appear to me to indicate that some chronic overgrowth of the glandular epithelium was occurring; that the cells thus produced had appropriated tracts in the cellular tissue outside their proper tubular confines, had made that tissue their home, and were thus spreading without limit. This and the fact that the interstitial cells are not small like those of a chronic inflammation, but large and angular, make it clear that if wanting at present in the aggressive character of a malignant growth, though this may well be doubted, the microscopical features show it to be really of that nature.

"I have subsequently had an opportunity of examining parts of the liver and the scar from the breast of this patient. The liver contained firm white nodules of ordinary carcinoma, and of the appearances under the microscope it will be enough to say that there is a wide-spread alveolar growth, the stroma of which, composed of hyaline bands of moderate thickness, forms spaces of regular size filled by clusters of small round cells. The portal canals are evidently the route by which it spreads into the liver. The earliest stage is not at all unlike an ordinary cirrhosis; the fibrous tissue of the portal canal being thick and studded with small round cells. There is also in many places a formation of wide spaces (? lymphatic or cavernous) at the circumference of the portal canals,

and thence the hepatic lobules are attacked. The liver cells appear to atrophy rather than to take any active part in the growth. The bile ducts are not easy to detect, but they do not appear to participate in the disease. With regard to the scar in the breast it is densely infiltrated with small round cells."

CASE 2.—*So-called eczema of the nipple and areola, followed by cancer of the mamma and lymphatic glands.—Recurrence in lungs, bronchial glands, liver, and suprarenal capsules.—Broncho-pneumonia.—Death.*

Emma H—, æt. 33, first applied to me on February 20th, 1879, for treatment of a small inflammatory swelling at the junction of the third left costal cartilage with the sternum. Whilst examining this swelling, which was trivial in character and disappeared altogether in a few days, I was struck with an eczematous condition of the areola of the left breast. To this I paid the most attention, much to the surprise and disappointment of the patient, who remarked that the nipple had been a little sore between four and five years,[1] but she thought nothing of that. Nor was she aware of the presence in the breast itself of an indurated nodule the size of a hazel-nut. She was a single woman, engaged as an ironer, but she had had two children, one when seventeen years, and the other when twenty-five years old. She had suckled both the children at both breasts without any complication; both nipples being then prominent. She had always had good health, and there was nothing in either her previous or family history of any importance except that she admitted being a free drinker, and her aspect confirmed this.

Between four and five years ago a little scale appeared on the summit of the nipple, which after a time either fell

[1] She had been seen at the hospital on July 26, 1877, and there is a note in the hospital case-book of that date to the effect that she had then chronic eczema of the nipple and areola; but no other mammary affection.

off, or was picked off, leaving exposed a moist surface. The scale reformed and was shed again and again, and the nipple became more and more flattened up to two years ago, when what remained of it was on a level with the areola. At this stage, on one occasion, she poulticed the breast on account of the discharge from it, and a "core" came away as from a gathering. This left a hole in the place of the nipple, which gradually healed up, but since then the slightly weeping eczematous area has gone on spreading circumferentially until the whole of the areola was involved. A depression still marks the immediate site of the nipple, and forms nearly the centre of a circle, one and a half inches in diameter, of superficial chronic eczematous inflammation, the base of which is thickened and a little indurated, so that the whole forms a slightly elevated disc about the thickness of an old-fashioned penny. The colour is mostly brownish-red, but violet-red in some parts. The margin is somewhat sinuous. The circumferential part of the area is partly moist and superficially abraded, and partly covered with very thin yellowish scabs. The central, which is also the violet-coloured part, is covered in a rather irregular manner with a new epithelium, which here and there extends in patches towards the circumference. The whole area is perfectly flat without any granulation on the surface.

The discharge from it is very scanty. It is but little or not at all tender, and free manipulation causes no pain. The rest of the skin of the breast is quite natural, but there is a small cord-like projection below the xiphoid cartilage, situated immediately beneath the skin but quite unconnected with the breast, which has been present for several months. In the breast tissue, besides a small hard nodule above and to the inner side of the nipple, there is beneath and below the areola another nodule, the size of a cob-nut, continuous with the under surface of the eczematous disc; and between the upper nodule and the disc there is a linear tract of condensed tissue in the gland. The nodules are not sharply defined, and the

breast generally has a firmer feel than the opposite mamma. There is an enlarged gland in the axilla, of which the patient has been aware, eight or nine months; it has never caused much pain.

The woman was admitted into the hospital, and on March 1st I removed the breast, but left the gland in the axilla untouched. On section the breast was seen to contain a number of nodules which, to the naked eye, in no way appeared to be of a scirrhous character.

On the 11th of March the wound was united.

On the 24th of March three or four slightly enlarged glands were felt in the left axilla, to which some absorbent ointment was applied. At the end of another week these glands had considerably increased in size, and on March 31st the patient was again anæsthetised, and a cluster of glands, the largest the size of a filbert, was removed. Most of them, on section, to the naked eye appeared soft, yellow, and succulent, but the largest had some firmer white growth in its substance.

On April 2nd the sutures and the drainage tube were removed; there was no suppuration and the wound was nearly healed, but the next day she had shivering, and the temperature went up to 100·6°; yet there was no other constitutional disturbance.

On April 6th she had another rigor, her pulse went up to 118 and her temperature to 100·6°; yet the wound was quite healed except where the drainage tube had been, and there was no pent-up pus or inflammatory thickening anywhere.

On April 23rd the cicatrices were quite sound; but there was some slight fulness of the glands above the clavicle. She complained of getting out of breath on the most trivial exertion: and the question naturally arose, are the mediastinal glands, or the lungs becoming secondarily affected.

On May 2nd she left the hospital for a convalescent home. During the last week of her stay in the hospital she suffered pain in the epigastrium and right hypochon-

drium, at first slight, and attributed by her to catching cold whilst sitting in the garden, but gradually increasing in intensity.

There was tenderness in these regions and the liver was considerably enlarged, more especially in the epigastric portion. There was dulness in the interscapular groove of the back to the right of the spine. The supra-clavicular glands on both sides were enlarged, more especially on the left, where a diffused swelling was visible. The dyspnœa on exertion continued.

She remained at Eastbourne a week, and on her return went to live at Peter Street, Westminster, from which she removed to St. George's Hospital on May 15th, where she died on May 23rd from broncho-pneumonia. I am indebted to my friend Mr. Marshall for the notes of the post mortem examination.

Both lungs were emphysematous and œdematous in their upper lobes. The lower lobe on both sides was semi-solid from broncho-pneumonia of a very diffuse type. The small bronchi contained puriform secretion. It was chiefly in these lobes that the cancerous infiltration was noticed. It occurred in two situations mainly. 1st, along the bronchi, which were thereby irregularly thickened. 2nd, as cancerous thickenings of the lymphatic channels at the surface of the lung; and as thin, flat expansions under the pleura. The lung being rather irregularly congested, the white lines and deposits of cancer gave to the organ a peculiar variegated aspect.

In addition, minute white nodules were here and there encountered in the lung tissue.

The bronchial glands contained deposits.

The liver weighed 5 lb. 10 oz. Left lobe very coarsely granular at the surface. The organ was firm and waxy on section, and showed extensive, very intimate, and very irregular cancerous infiltration, the colour of the cut surface varied through many shades of red and brown into yellow; of the yellow patches some were due to fatty degeneration of the surviving liver tissue, while others

resulted from decay of the softer cancerous deposits. Cancerous glands were formed on the porta.

The kidneys weighed $8\frac{1}{2}$ oz., their capsules were thickened and partly adherent, and their surfaces very irregular. Both the supra-renal capsules contained cancerous deposits; that in the left was as large as a marble. The parts removed by operation were handed over to Dr. Thin for microscopical examination, and he reports that "the nodule in the mammary gland consists of an epithelial growth presenting the characteristic appearances of cancer extending and burrowing amongst indurated connective tissue. Transverse sections through the remains of the nipple show that some of the ducts are plugged with epithelial cells of various sizes, the cells being therefore of new growth and pathological in character. The skin around the nipple, which in life had the moist appearances of some forms of eczema, showed the following alterations:—The epidermis was almost completely destroyed, the ragged remnants of the rete mucosum consisting of epithelial cells in process of decay; the papillary layer of the cutis was the seat of congestion and was filled with exudation cells; the pars reticularis cutis was mostly healthy. The affection of the skin seems therefore to be a superficial chronic destructive inflammation. The enlarged glands subsequently removed from the patient contain growths of epithelial cancer similar to those in the mamma."

The differences above referred to between Sir James Paget's very concise and lucid description of his cases, and the two now reported are as follows:

1. In one of my cases the eczematous eruption extended beyond the areola, and in both it passed into a somewhat deeper ulceration of the skin.

2. Sir James Paget says cancer has followed in every case within at the most two years, and usually within one year. In one of my cases the eruption had commenced nearly six years, and in the other between four and five years before cancer was suspected. How long the can-

cerous changes had been going on it was impossible to say, but at the longest it is improbable that any such growth had existed for more than one year, if so long; so that, in each instance, the eczema had preceded the cancer by some four or five years at least.

3. Sir James Paget says that in the cases he has seen, there was always a clear interval of apparently healthy tissue between the diseased part of the skin and the cancer in the substance of the breast; but in one of the above cases there was a well-marked cord of indurated tissue, and in the other, some ill-defined hardness between them. Moreover, the microscope points to a direct chain of communication, not only within, but also around the galactophorous ducts, between the surface ulceration and the deep-seated nodules.

4. As to the age of the patients in Sir James Paget's cases, it varied from forty to sixty or more years, but in one of the above the age was thirty-three years and in the other forty years at the time when cancer was first suspected; and if we deduct the number of years during which the eczema had existed, it would make one of the patients twenty-nine years and the other thirty-five years of age at the most, when the local disease commenced.

During the years from 1872 to 1878 inclusive, I have seen in the cancer out-patient department of the Middlesex Hospital, 585 cases of cancer or imputed cancer, in 305 of which the seat of disease was the mamma. The above cases are the only instances in which eczema of the nipple and areola has been associated with cancer of the breast. Nor have I learnt from the subjects of the mammary cancer that they had previously suffered from either eczema or psoriasis of the nipple. In eight instances there was eczema of the nipple, but no cancer; one of these patients was only eighteen years old, and was suffering from eczema of the skin of the greater part of the mamma. She had been told, so she stated, at another metropolitan Hospital, that there was a cancer, to use her expression, in the "mouth part of the breast," but I could

not find any condition which gave colour to this opinion. This case was interesting from the age of the patient, and from the fact that she was neither pregnant nor suckling, and that the eczema was confined entirely to the mamma.

DESCRIPTION OF PLATES I AND II.

Eczema of Nipple and Areola (Henry Morris, M.A.).

PLATE I. Eczema of nipple and areola. Case of Dinah L—.

PLATE II. Ditto. Case of Dinah L—. Microscopic drawings of sections, showing the changes which followed it in the sublying cancer.

FIG. 1. Vertical section of the superficial parts of the true skin in the neighbourhood of the ulcer.

a. Epithelial layer.

b. Fibro-cellular tissue of corium. The intervening part is closely crowded with cells of new formation. 1 in.

FIG. 2. Vertical section of the skin from the ulcerated spot.

a. Sections of stained tubes full of cells.

bb. Sections of glands with epithelial cells arranged irregularly.

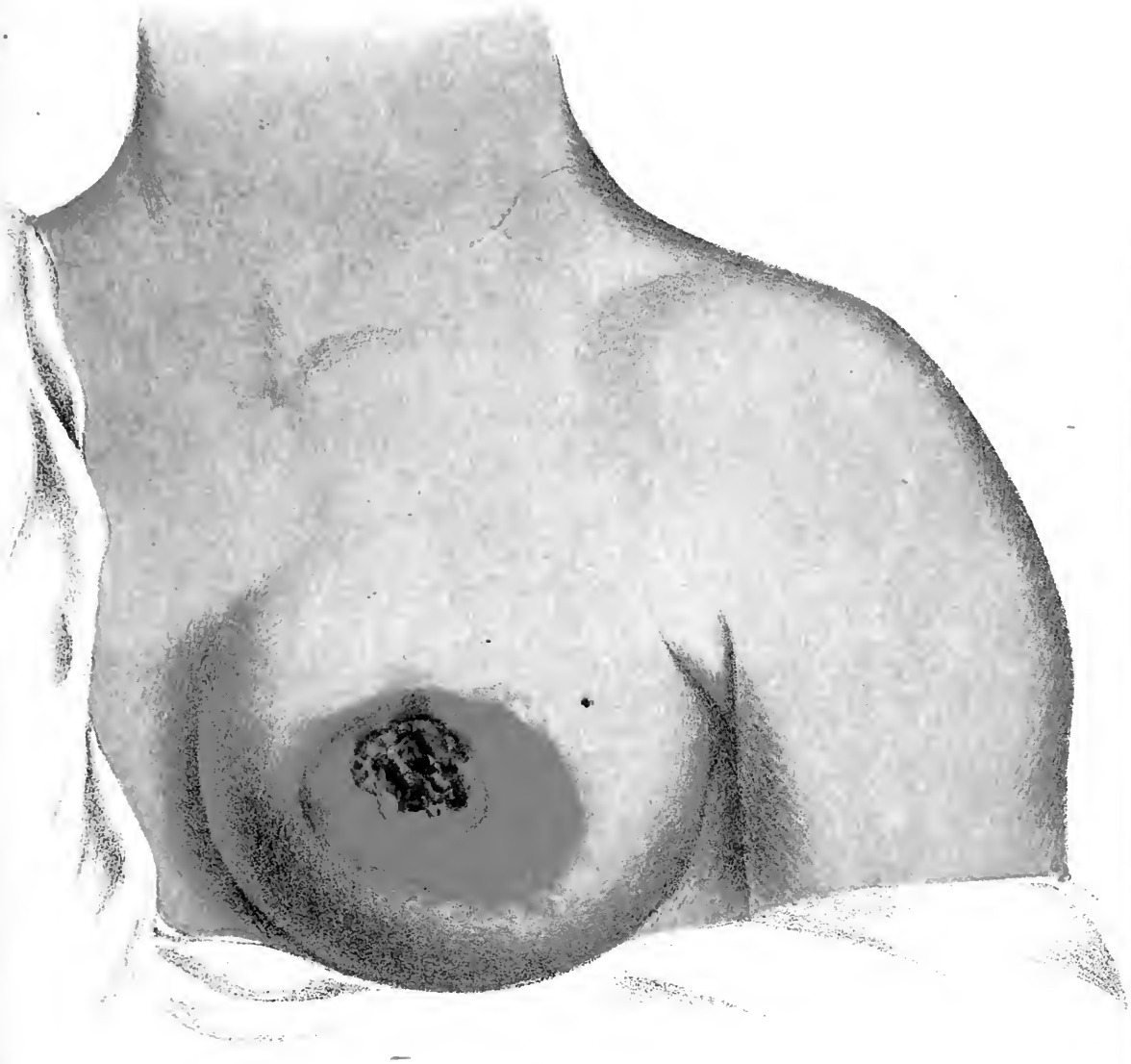
ccc. Foci of new growth in various parts of the fibro-cellular tissue of the skin.

d. Subcutaneous fat. 1 in.

FIG. 3. Section of a small nodule of new growth in the deepest layer of the corium. It shows transverse sections of tubes with a close infiltration of cells in the fat and fibro-cellular tissue outside. 1 in.

FIGS. 4, 5, 6, show under a high power the form of the cells, their arrangement in the gland ducts, and their spreading in the tissues beyond. $\frac{1}{8}$ in.

So called eczema of Nipple and Areola



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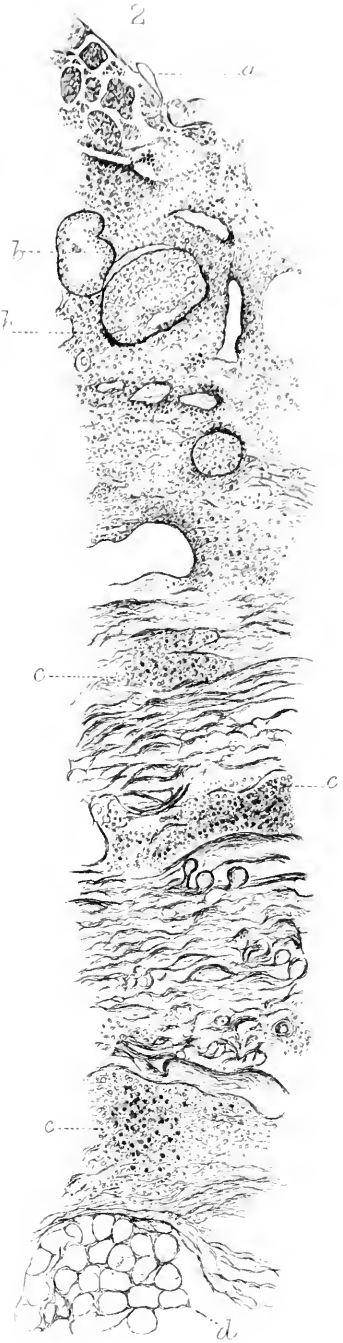

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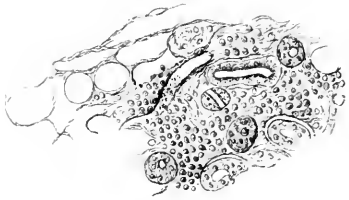

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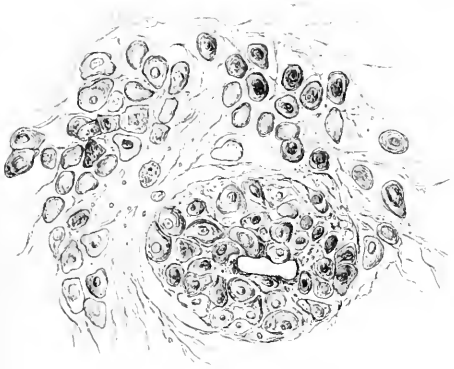


5.

6.

DESCRIPTION OF TWO CASES

OF

DIRECT DISLOCATION BACKWARDS OF THE FEMUR

WITH

FRACTURE OF THE RIM OF THE ACETABULUM,

WITH REMARKS ON THE MODE OF PRODUCTION OF DISLOCATIONS BACKWARDS.

BY

FREDERIC S. EVE, F.R.C.S.,

CURATOR OF THE MUSEUM, ST. BARTHOLOMEW'S HOSPITAL.

(Received December 1st, 1879—Read January 13th, 1880.)

Case 1.—Wm. H—, æt. 48, was brought into St. Bartholomew's Hospital dead. He had thrown himself out of a window at a considerable height from the ground.

In addition to a dislocation of the right femur, the post-mortem examination revealed a fracture of the base of the skull.

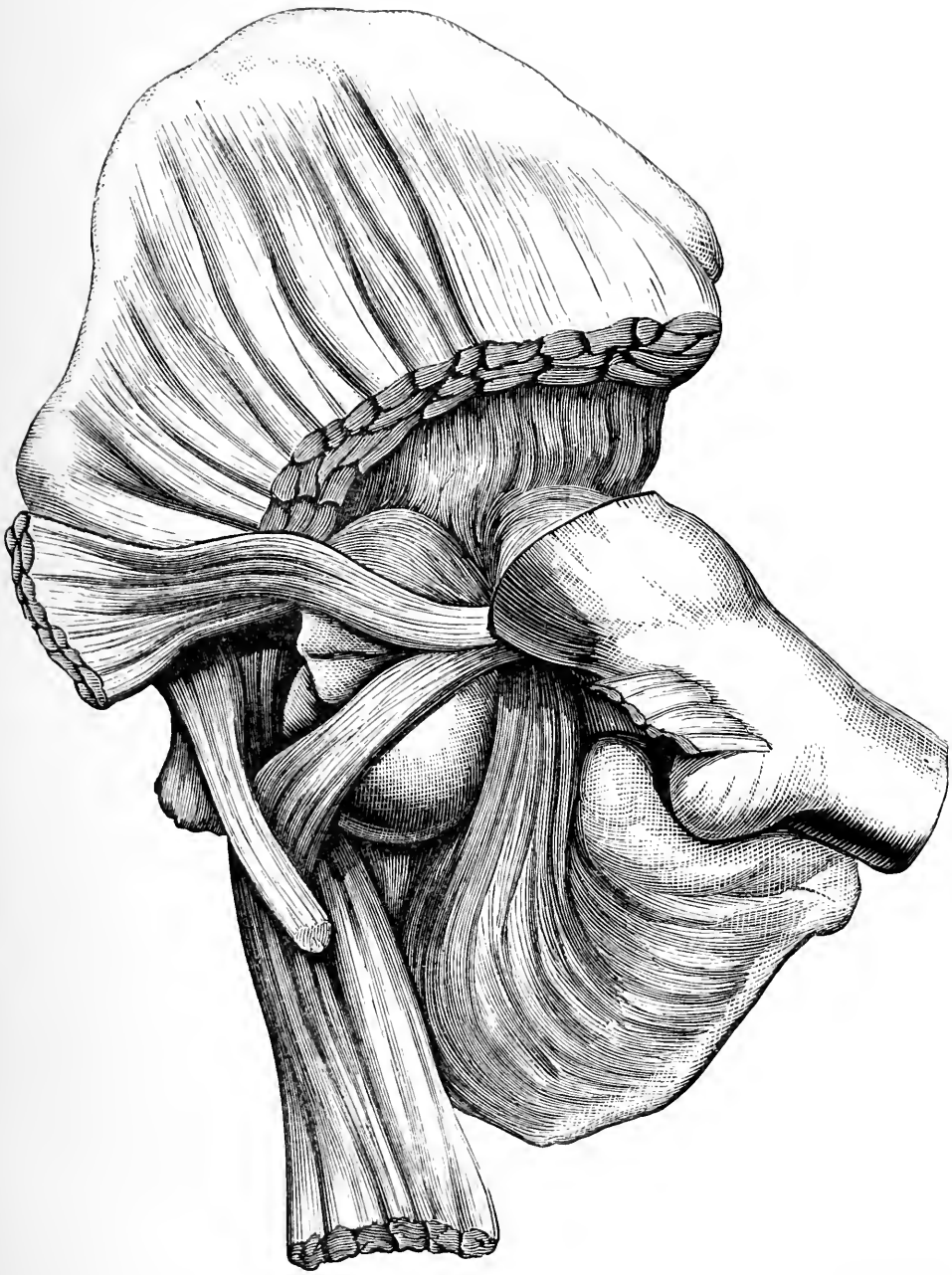
The right thigh was adducted and rotated inwards. The knee was slightly flexed and rested on the lower part of the left femur, just above the patella; the right great toe rested on the base of the left. There was some fulness in the gluteal region. The limb was remarkably rigid.

The condition of the parts found on dissection and shown in the specimen[1] is as follows (see Woodcut, Fig. 1) :—The head of the femur is seen lying on the ischium and margin of the acetabulum on a level with the lesser sciatic notch and immediately above the tuberosity of the ischium.

The sciatic nerve passes over the most prominent part of the exposed articular surface. The head of the femur is firmly bound down by the tendon of the obturator internus, which stretches tightly across it just above the level of the horizontal diameter. The posterior and lower portion of the rim of the acetabulum is broken off and pushed up above the femur, carrying with it the pyriformis, which is thus put on the stretch, but not lacerated. The rough fractured surface of the ischium is partly covered by the head of the femur, which rests upon it, and partly exposed to view. The gemellus inferior is torn across ; with this exception, all the muscles are intact. The quadratus femoris is reflected, exposing the obturator externus, which tightly embraces the neck of the femur below. The acetabular attachment of the posterior portion of the capsule is torn to a limited extent ; the upper and posterior part is intact and attached to the upturned margin of the acetabulum ; the inferior portion of the capsule covering the cotyloid notch is also untorn. The psoas and iliacus muscles were removed to expose the ilio-femoral ligament, which is not lacerated. No attempt whatever to reduce the dislocation was made until the dissection was completed. The head of the femur was found rigidly fixed by the ilio-femoral ligament, and the obturator internus, the latter of which as described above, passed across the articular surface ; both were tightly stretched. On flexing the femur the head readily glided from beneath the obturator internus, under the obturator externus to the cotyloid notch ; there was then no obstacle to reduction.

[1] Preserved in the Museum of St. Bartholomew's Hospital, Series III, No. 157.

FIG. 1.



T. Godart del.

Direct dislocation of hip backwards on to ischium; fracture of posterior margin of acetabulum, unreduced.

Case 2.—Wm. J—, æt. 55, was admitted to St. Bartholomew's Hospital under care of Mr. Willett, to whose kindness I am indebted for permission to communicate the case.

Whilst at work excavating, a large fall of earth took place upon him.

On admission he was much collapsed and complained greatly of pain in the abdomen. The left hip presented the ordinary appearances of sciatic dislocation. No attempt at reduction was made on the day of the accident, owing to the collapsed condition of the patient, but the following day he was placed under an anæsthetic and an attempt made to effect reduction by the ordinary manipulation. This failing, Mr. Willett slightly flexed and adducted the thigh, and on applying slight traction the head at once returned to the socket. From this circumstance Mr. Willett inferred that it was a case of direct dislocation backwards, probably with fracture of the margin of the acetabulum. Symptoms of peritonitis appeared, and the patient died the next day.

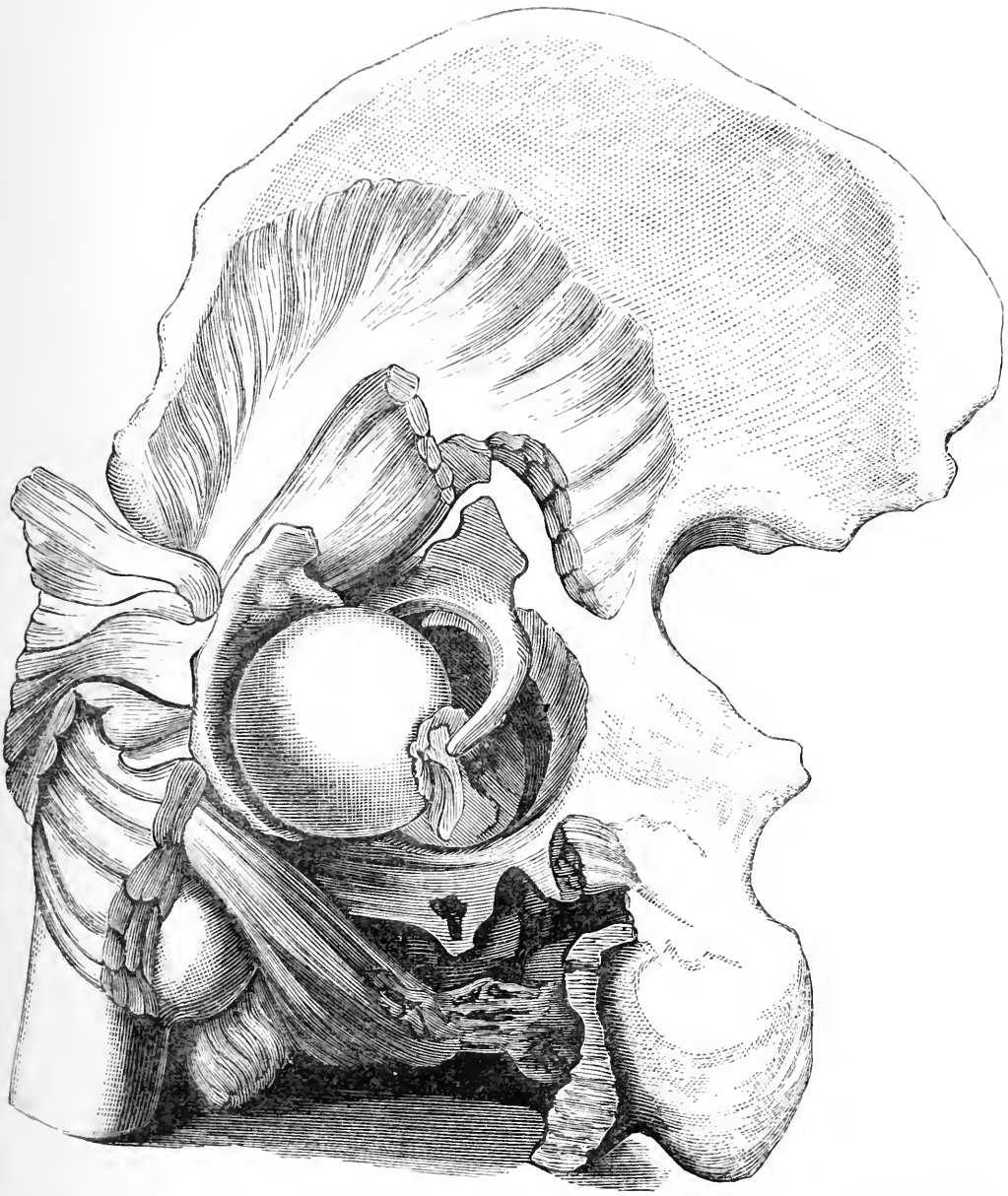
The post mortem examination revealed intense general peritonitis due to the rupture of a portion of intestine, which lay apparently in the right inguinal canal at the time of the accident. The hip was handed over to me for dissection.

The following is a description of the preparation [1] shown. (See Woodcut, Fig. 2.)

The gluteus maximus and medius have been removed; the lower part of the gluteus medius is reflected. The external rotator muscles are cut across and reflected. The posterior portion of the rim of the acetabulum is broken off and upturned. There is a corresponding rent in the capsule, which has been torn through at its acetabular insertion. The rent extends downwards to within one inch of the cotyloid notch, here it passes upwards and forwards towards the femoral attachment, leaving a narrow tongue-shaped portion of capsule still adherent to the inferior

[1] In the Museum of St. Bartholomew's Hospital, Series III, No. 158.

FIG. 2.



T. Godart del.

Direct dislocation of hip backwards into ischiatic notch, with fracture of posterior margin of acetabulum and laceration of obturator externus.

margin of the acetabulum and transverse ligament. The cartilage covering the lower and anterior portion of the head of the femur is irregularly ground off. The obturator externus muscle is slightly lacerated at its insertion ;[1] the pyriformis and gemellus superior are also slightly torn, but no other muscles were damaged. There were considerable extravasations of blood around the upper part of the femoral vein, between the gluteus medius and minimus, and into the substance of the obturator externus. From the position of the fracture of the rim of the acetabulum and the rent in the capsule, it is evident that the femur was dislocated directly backwards as in the previous case. In addition the periosteum was torn off the ischium on a level with the great sciatic notch, indicating that the head of the femur was thrown on to this portion of the bone, probably between the pyriformis and obturator internus muscles.

From the mode of occurrence of the injury, it may be assumed that the man was stooping, using his shovel or mattock, when the fall of earth took place upon his back, throwing him upon the knee, and by the sudden wrench and force of the blow, driving the head of the femur through the posterior portion of the capsule.

Remarks.—I communicate the details of these cases of direct dorsal dislocation of the hip under the impression that they will be of interest and value in themselves, but they gain additional interest from the bearing they have on a paper on dislocation of the hip, in vol. lx of these 'Transactions,' by Mr. Henry Morris. The cases related support to some extent Mr. Morris's view that "direct dorsal dislocations are *always associated* with fracture of the acetabulum, or head of the femur, or both."

In order to ascertain the absolute correctness of this statement, which, as the author avows was founded on experiments, I tabulated, after careful examination, the features of *recent dislocations* backwards in the London

[1] N.B. It has since been accidentally increased.

museums, with special reference to the following points under discussion :—whether direct or indirect, and if direct whether associated with fracture ; the condition of the inferior portion of the capsule and the relation of the tendon of the obturator internus to the head of the femur. The longest period intervening between the date of the dislocation and of death in the cases referred to is three weeks. (See Table, p. 58.)

Of the *nine* specimens tabulated *eight are direct* dislocations, only *one is indirect*. Following Mr. Morris a dislocation is described as *direct* when the head of the femur is thrown directly backwards through the posterior portion of the capsule ; *indirect*, when it escapes by abduction through the inferior portion of the capsule covering the cotyloid notch, and is then forced backwards by inversion of the limb.

The great preponderance of direct over indirect dislocations in the table is absolutely at variance with the experimental deductions made by Mr. Morris, who states, in the commencement of his paper, that he "proposes to to give reasons for believing that abduction is the position in which all dislocations of the thigh happen." The disproportion between the cases of direct and indirect dislocation cannot, I think, be to any great extent due to accidental circumstances, which led to the specimens passing into the museums.

Some allowance should, however, be made for this, as the violence must have been great in order to cause by a complication the death of the patient, as it did in most of the cases. The cause of death was, however, an accidental complication, and in nearly every case the mode of production of the injury was not of such a nature as would inevitably cause death.

Again, of the eight direct dislocations, three occurred without fracture of the posterior portion of the acetabulum, or of the head of the femur ; the possibility of which, as stated above, has been absolutely denied by Mr. Morris.

In order to substantiate the fact that these three speci-

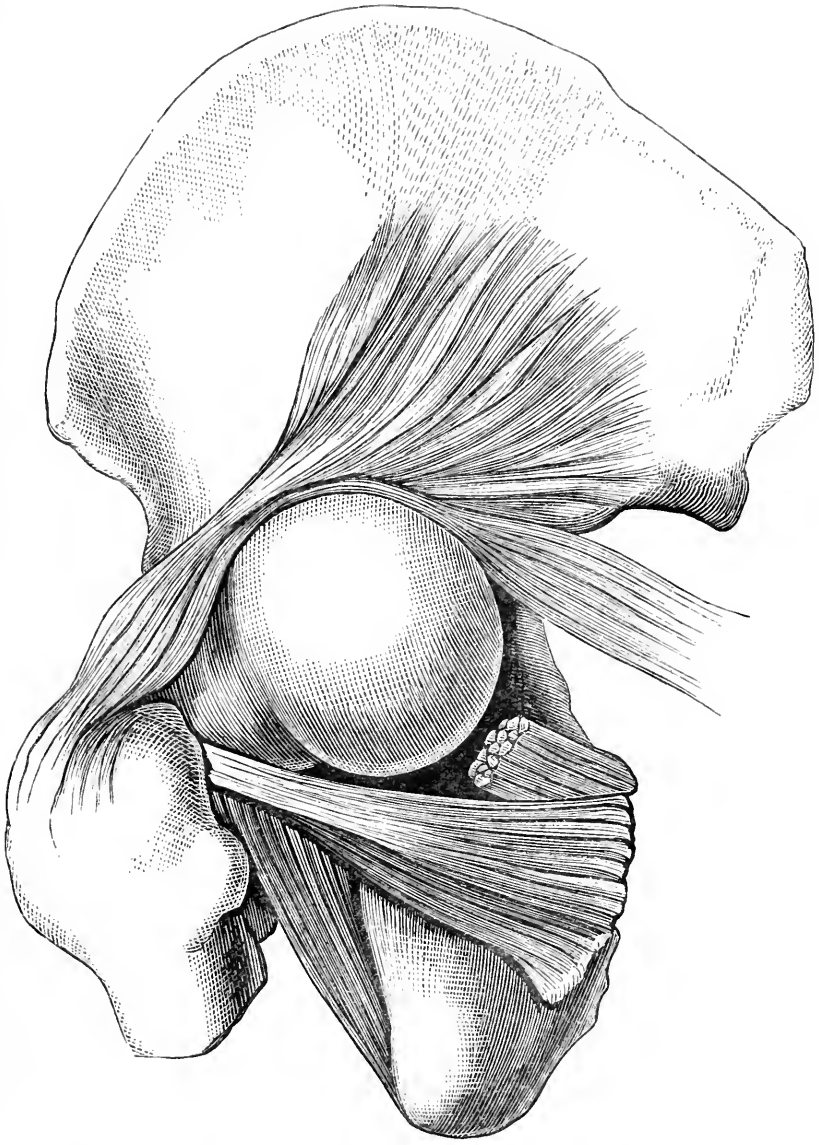
Table of Specimens of recent Dislocations of the Femur backwards in the London Museums.

Museum.	Form of dislocation.	Presence or absence of fracture.	Condition of capsule.	Relation of tendon of obturator internus to head of femur.	Reduced or not.	Cause.	Remarks.
St. Thomas's	Direct	None	Inferior portion of capsule intact	Tendon below head embracing posterior surface of neck	Unreduced	Fall of heavy casting on hip	See description by Mr. MacCormac, 'St. Thomas's Hosp. Rep.,' vol. ii, p. 143.
St. George's	,,	None	Ditto, small opening in posterior part	Tendon removed	Ditto	Fall from height	This specimen shows clearly that the inferior portion of the capsule was not lacerated, and its present condition agrees with the description of the dissection in the catalogue.
St. Bartholomew's, S. 3, No. 56	,,	A fracture through the ascending ramus of ischium and base of pubes, but not influencing the dislocation	Inferior portion of capsule intact	Tendon torn from muscle	Ditto	Ditto	I have taken this specimen out of the bottle and carefully examined the capsule. Case related by Mr. Wormald.*
Ditto, S. 3, No. 20	,,	Fracture of posterior third of acetabulum	Ditto	—	Reduced	Not stated	Ditto. Death three weeks after injury.
Ditto,* S. 3, No. 68	,,	Fracture of posterior portion of rim of acetabulum	Inferior portion not visible, posterior portion lacerated	—	Ditto	Ditto	The position of the fracture and of the laceration of the capsule indicate that it was a direct dislocation.
Case 1, related	,,	Ditto	Portion over cotyloid notch intact	Tendon stretched over head	Unreduced	Fall from height	—
Case 2	,,	Ditto	Inferior portion intact	—	Reduced	Fall of earth on back	—
Guy's	,,	Fracture of head of femur	Ditto	Tendon below head	Unreduced	Fall from height	Related by Mr. Birkett, 'Med.-Chir. Trans.,' vol. lii, p. 133.
Hunterian	Indirect	Fracture of acetabulum	Posterior portion intact, inf. torn	In front of head	Ditto	Run over	Related by Mr. Morris, 'Med.-Chir. Trans.,' vol. lx, p. 179.

* 'Lond. Med. Gazette,' 1837.

mens are instances of direct dislocation, it will be necessary to go briefly into the evidence. Mr. Mac Cormac's case in the St. Thomas's Hospital Museum is the most

Fig. 3.



Copied from 'St. Thomas's Hospital Reports,' vol. ii, p. 143.

perfect, and the description is in every way satisfactory. (See Woodcut, Fig. 3.) The position of the tendon of the obturator internus muscle below the head of the

femur, and tightly grasping the posterior surface of the neck, with the distinct statement that the inferior portion of the capsule was not lacerated, is sufficient evidence, without going into the mode of production, which is also confirmatory. Specimen, Ser. 3, No. 56,[1] in the St. Bartholomew's Hospital Museum I have re-examined, and find that the inferior portion of the capsule is not torn. The fracture of the os innominatum in this specimen is anterior to the acetabulum, just skirting the anterior margin, and therefore does not affect the dislocation.

In the specimen at St. George's the inferior portion of the capsule, extending over the cotyloid notch and backwards to a level with the posterior portion of the tuberosity of the ischium, is certainly not torn; and, although it is a dry preparation and might be objected to for that reason, yet the present condition agrees with the description made at the time of dissection.

Cases of direct dislocation with fracture of the margin of the acetabulum appear to be comparatively common; there are four out of eight cases in the table. Bigelow mentions six cases in which this lesion was discovered on dissection, and Hamilton as many more. Probably this form of dislocation is more common than is generally supposed, because it cannot usually be diagnosed during life.

Various considerations render it probable that *indirect* dorsal dislocations by no means so greatly preponderate over *direct* dislocations as Mr. Morris maintains. In order that the head of the femur may be thrust through the inferior portion of the capsule, over the cotyloid notch, the thigh must be abducted in the extended position.[2] If, therefore, the *indirect* dislocation is the rule, the majority of dislocations backwards occur when the thigh is extended, or very nearly so. This is totally at variance

[1] Case III in the Table.

[2] The head of the femur may be forced through the inferior portion of the capsule by forcible inversion of the thigh flexed at a right angle with the trunk, but abduction, rather than assisting the manipulation, drives the head against the ilio-femoral ligament.

with the opinions of Sir Astley Cooper, Hamilton, and Bigelow, who teach that flexion and adduction is the position in which dislocations backwards occur; and reference to the description of cases clearly shows that this is the position in which a large number, probably the majority, of backward luxations take place. Further, we must assume a peculiar alternation of movements which will cause forcible inversion to follow abduction of the limb, in order that the head of the femur, after leaving the socket, may be driven upwards and backwards.

These movements, as Mr. Morris explains, occurred in the case of indirect dislocation he describes, but they are complicated and not likely to happen frequently.

The association of inversion with abduction appears less likely to be of frequent occurrence as the latter movement is normally associated with eversion. For example, it will be found impossible to straddle the legs widely apart, that is, abduct them, while the feet are inverted; and if the limb were inverted while abducted and extended, the head of the bone would be driven against the posterior and inferior portion of the capsule, and not against the thin portion covering the cotyloid notch.

These considerations lead me to infer that abduction would at least as frequently produce dislocation forwards as dislocation backwards, since abduction is more commonly associated with eversion in ordinary circumstances.

Mr. Morris states that in the reduction of direct dislocations, the manipulation, so successful in indirect dislocations, would be difficult and destructive "because more of the capsule and the small rotator muscles would have to be broken down before the head could be guided round the acetabular rim to the cotyloid notch." As the result of experiment I find, however, that it is not necessary in order to reduce a direct dislocation above the tendon, that the head of the femur should pass down to the cotyloid notch.

If the head of the bone is thrust through a limited incision at the acetabular attachment of the capsule, above the tendon of the obturator internus, it can readily be

returned by flexion and circumduction through the opening in the capsule by which it escaped; the thigh must, however, be abducted widely. By this manipulation the ilio-femoral ligament is rendered tense, and the head of the femur thus drawn into the socket.

In such an experiment if the obturator internus be divided the capsule can, as Bigelow describes, be lacerated with slight force by circumduction, and the head then returned to the acetabulum through the cotyloid notch. Therefore, in direct dislocation below the tendon, the untorn inferior portion of the capsule would not obstruct reduction through the cotyloid notch.

Because a direct dislocation backwards cannot be produced for experiment without division of the capsule, it is assumed that this form is of the rarest possible occurrence.

I have also found it impossible to produce a direct dislocation backwards either by the blows of a heavy sledge hammer, directed on the extremity or outer side of the lower end of the flexed and adducted femur; or by forcible adduction and flexion. The cause of failure is, I think, the impossibility of applying sufficient force in the right manner, since the displacement is, in by far the larger majority of cases, occasioned by a wrench, rather than a blow directly driving out the head of the femur. The mode of production of this variety of luxation appears to be as follows:—A man, for instance, falls with some violence on the outer side of his knee, the thigh is forcibly adducted and driven upwards and backwards; at the same time, the whole weight of the falling body is thrown in the opposite direction, and consequently a great strain falls on the posterior portion of the capsule of the hip-joint. Under these circumstances, as Hamilton explains, the thigh is converted into a lever of the first kind, the fulcrum of which is formed by the pelvis (against which the bone is thrown by adduction), the shaft of the femur being the long arm, while "the weight" of the body is connected with the short arm, the head of the femur only by the posterior portion of the capsule and the muscles.

Under the sudden wrench and strain the capsule is torn through at its acetabular attachment, or more frequently the rim of the acetabulum is torn off just as the tip of the internal malleolus is sometimes in Pott's fracture. The same course of events would follow the fall of a heavy weight on the pelvis while a person was stooping; or a fall while carrying a heavy weight on the back.

Rarely the head of the femur is driven with great violence upwards and backwards, and the posterior portion of the acetabulum is carried away,[1] or the head itself is fractured.[2] Of four cases of fracture of the posterior portion of the acetabulum given in the table, in three the rim only was torn off.

Passing now to comment on some of the interesting features of the two cases of direct dislocation backwards narrated, it will be observed that in Case 2 the dislocation was "*above the tendon*," in Case 1 beneath (that is, between the tendon and the bone) and "*below the tendon.*"

Other undoubted instances of direct dislocation "below the tendon" are on record. Mr. Wormald's case, given in the table, is an instance, and it is cited by Bigelow as such.

Mr. William Adams[3] has also described the dissection of a "direct dislocation between the obturator internus and externus," in which the capsule was torn through in its posterior half.

A distinction between the dislocations "above" and "below the tendon," appears to be practically important, owing to some differences in the mode of reduction.

As already mentioned, I found by experiment that the head of the femur could, in direct dislocation above the tendon, be returned through the opening in the capsule by which it escaped, but that it was necessary to abduct the limb somewhat widely during circumduction in order to effect reduction; otherwise, the head of the femur passed over (posterior to) the obturator tendon, which thus lay between the head and the socket.

The failure of the ordinary manipulation in Case 2 was

[1] *Vide* Case 4, Table. [2] *Vide* Case 8, Table.

[3] 'Path. Soc. Trans.,' vol. xxi, p. 306.

probably due to this cause, and Mr. MacCormac points out that this would have occurred in his case of direct dislocation.

When the margin of the acetabulum is fractured the dislocation is easily reduced by traction on the flexed and slightly abducted thigh. This manipulation was successful in Case 2, also in a case recently admitted to the hospital, which was believed by the majority of those who saw it, to be a direct dislocation backwards with fracture of the acetabulum.

In direct dislocations "below the tendon" the head of the femur is returned to the socket through the cotyloid notch, the untorn portion of the capsule being, as already shown, no obstruction to reduction.

In Case 1 reduction was readily effected in this manner after dissection.

It is therefore evident that a dislocation cannot be assumed to be indirect because the head of the femur returns to the socket through the cotyloid notch, since a *direct* dislocation "below the tendon," and therefore not produced by abduction, is reduced in the same manner.

The rigidity of the femur in Case 1, due to the head being bound down by the obturator internus, has been already referred to.

It would probably have formerly been ascribed, as Bigelow points out, to locking of the head of the femur in the sciatic notch. The principal conclusions arrived at may be stated as follows:

a. That direct dislocation backwards does occur without fracture of the acetabulum.

b. It is probable that direct dislocation backwards is comparatively common, and frequently associated with fracture of the rim of the acetabulum.

c. That a direct dislocation "above the tendon" can be reduced by flexion and circumduction of the thigh, through the opening in the capsule by which it escaped.

d. That the untorn inferior portion of the capsule is no obstacle to reduction in direct dislocations "below the tendon."

ON A CASE

OF

ANEURISM OF THE SUBCLAVIAN ARTERY

TREATED BY

AMPUTATION AT THE SHOULDER-JOINT AND THE INTRODUCTION OF NEEDLES INTO THE SAC.

BY

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(Received December 3rd, 1879—Read January 27th, 1880.)

The treatment of subclavian aneurism by amputation at the shoulder-joint and ligature of the main artery on the face of the stump was suggested many years ago by Sir William Fergusson, who, however, had no opportunity of putting the suggestion into practice. He says ('Practical Surgery,' p. 542): "It is known that amputation at the shoulder-joint is generally a very successful operation; as far as the wound is concerned, then, there might be little to apprehend, but the effect on the tumour is not so easily foretold. Ligature of the axillary artery on the face of the stump might be reckoned like Brasdor's operation, yet there is a vast difference, for in the latter case the same amount of blood which previously passed towards the upper extremity would still find its way down, and probably part of it would run through the sac, whereas were the member removed, as the same

quantity would no longer be required in this direction, the tumour might be much more under the control of pressure."

Professor Spence carried out this suggestion in 1864 on a naval engineer, in whom, six months before, the right arm began to feel cold and numb, and the ring and little fingers insensible, but who was unaware of any tumour until fourteen days before admission, when he was found to have a pulsating tumour commencing in the interval between the two heads of the sterno-mastoid, extending outwards and occupying the whole subclavian space. Mr. Spence amputated at the shoulder-joint on April 6th, and secured the axillary artery at the lower border of the pectoralis minor. On June 24th, the patient being convalescent, compression of the aneurism was begun by means of plaster, and iodide of potassium was administered, with the result that the aneurism became "very decidedly smaller and harder," but was not cured. The patient lived four years after, but the aneurism did not disappear, and dying at a distance from Mr. Spence no post-mortem examination was obtained. (Spence's 'Lectures on Surgery,' vol. i, p. 610, and 'Medico-Chirurgical Transactions,' vol. lii, p. 306.)

Mr. Luther Holden, in November, 1876, amputated at the right shoulder-joint in the case of a man aged 44, suffering from a subclavian aneurism, after the failure of direct pressure and restricted diet. The aneurism at first diminished in size and became harder. The main ligature came away on the twenty-fourth day, at which time there was considerable swelling, heat, and tenderness over the aneurism. The patient got pleurisy and bronchitis, the sac of the aneurism gave way, and the patient died on the thirty-seventh day. ('St Bartholomew's Hospital Reports,' 1877.)

Mr. Henry Smith has obliged me with the unpublished notes of a case in which, in February 1877, he performed amputation at the right shoulder-joint for an aneurism of the subclavian which had resisted proximal and distal pressure, complete rest, and the use of Esmarch's elastic bandage to the arm. The pulsation greatly diminished

for some days after the operation, and then increased again, and the patient died suddenly three weeks after the amputation from a thoracic portion of the sac giving way into the lung.

These are the only cases, so far as I am aware, prior to my own, and it must be allowed that the results are not encouraging, for, at the best, Mr. Spence's case was only relieved, and in the other cases the improvement was very temporary.

My own case is remarkable, in the first place from being distinctly traumatic, the consequence of a broken clavicle, and, secondly, from the fact that failing to obtain a cure by the amputation, consolidation of the contents of the sac was induced by the introduction of needles.

For the very elaborate report I am indebted to a former distinguished student of University College, Dr. Easmon, now of Sierra Leone.

James S—, æt. 48, was admitted under Mr. Heath on September 12th, 1878, with comminuted fracture of the left clavicle and simple fracture of the six upper ribs on the same side. While under treatment bronchitis supervened, but he was discharged on September 27th and made an out-patient. Previous to November 1st, 1877, patient had never had a fit, but since then he has suffered from epileptic attacks. In May, 1878, he was attended by his medical man for "weakness of the legs" without any actual paralysis, though difficulty in walking was experienced. His arms, however, were "all right." He had never suffered from rheumatic or scarlet fevers, but twenty-seven years ago had an attack of gonorrhœa (?) followed by a rash on the face, accompanied by sore throat. No history of any sore on the penis at this time or of enlarged glands in the groins. At present no scars on the genitals can be seen. It was during one of the above-mentioned fits, in September, 1878, that he fell down and was run over by a van, sustaining the injuries for which he was admitted. On admission the radial pulse on the injured side was normal. There was myosis.

Since September 29th he had attended as out-patient regularly, but it was not till October 31st that any undue pulsation was noticed. The surgical registrar, Mr. S. H. Burton, is quite certain that on September 21st no evidence of an aneurism of the subclavian existed.

The patient was re-admitted on October 31st, 1878.

Present state.—Occupying the lower part of the left posterior triangle of the neck and extending beneath the clavicle for a short distance is a pulsating tumour, the pulsation of which is expansile, and most marked at the upper part; the tumour is about the size of a Tangerine orange, the centre of its upper margin being about two inches above the clavicle. A perpendicular line drawn along the inner margin of the tumour strikes the clavicle an inch and a half from the sternal end. At a point two inches outside this a similar line drawn from its outer margin strikes the clavicle. The lower border is felt half an inch below the clavicle. There is apparently union of the fractured clavicle, the extensive external provisional callus being now to a great extent absorbed. Distinct pulsation is felt over the clavicle, and a double bruit heard, the systolic being the louder.

The left radial pulse is much smaller than the right, and the left temporal also smaller than its fellow of the opposite side. There is some œdema of the left arm. (It has been in a confined position for some time since the accident.) The veins over the left shoulder are more visible than on the opposite side. There is no pain in the tumour, nor shooting sensations down the arm, but the left arm is almost completely paralysed. The muscles look and feel flabby; the degree of wasting is not quite evident on account of the œdema.

Chest.—Lungs present no abnormal or adventitious phenomena. Heart: apex-beat in normal position, but the impulse is more forcible than natural; no murmur. Base: systolic murmur at pulmonary cartilage, none over aortic; second sound, however, accentuated.

Abdomen.—No evidence of disease.

Urine.—No sugar, no trace of albumen, no deposit.

General appearance.—The patient is of short stature and short-necked. His face has a bloated appearance and the eyes are somewhat prominent. He is fairly well-nourished and possesses no deformities. His expression is listless and vacant; muscular power below par. He looks too old for his age. No rigid arteries observed.

The aneurism increased considerably in size after the patient's admission, and it was found to be impossible to apply any pressure on the proximal side. The sac being apparently likely to give way and the arm being completely useless from paralysis, Mr. Heath recommended the patient to lose the limb, and amputated at the shoulder-joint on November 4th, being so well assisted by Messrs. Barker and Godlee that very little blood was lost. The axillary artery was secured with a catgut ligature, but was not dissected out at all, and the whole proceeding was conducted with antiseptic precautions.

November 5th.—The pulsation of the upper part of the tumour is as marked as before, but diminished at the lower part and over the clavicle, due to diminution of arterial tension. The whole tumour seems to have diminished in size, but as the clavicle is raised, the extent of this cannot be appreciated with exactness. The top of the aneurism seems to be not so high above the clavicle as before the operation. Temp. 99°. He is to have extra strong beef-tea, and two-drachm doses of brandy every hour. No respiratory disturbance.

6th.—Passed a quiet night. Pulse improved in character, 100. No sickness. Tongue slightly furred. Temp. 99°.

8th.—Stump dressed at 2 p.m. and looks well. Small drainage tube substituted for the larger. The aneurismal tumour has increased in size both laterally and upwards, and pulsation is more marked. Superficial veins over and about the shoulder more evident. Much hiccough (which last night had to be relieved by hypo-dermic injection of morphia), probably due to carbolic acid poisoning. The

urine is dark coloured, but less smoky, slightly acid, no sugar. Albumen equals one eighth.

12th.—Urine, sp. gr. 1010, acid, slight trace of albumen. No sugar. Hiccough gone. Stump dressed and looks well, flaps united except for drainage tube. Slight discharge. Drainage tube removed altogether. Temp. 98°. Pulse 99.

14th.—Hæmorrhage took place at 6.30 p.m. from the stump, the quantity of blood lost being, according to the house-surgeon, about four ounces. Dressings were removed and ice-bag applied. The general condition does not appear to be affected. Temp. 98°. Pulse 100.

18th.—Temp. 99°. Much discharge from the opening where the drainage tube had been inserted. Stitches giving way and edges of wound swollen and red. Passed a good night. Urine, sp. gr. 1010, acid, with still a trace of albumen. Evening temperature 99·2°.

26th.—The tumour appears somewhat larger. The area of pulsation measures $2\frac{1}{2}$ inches in its transverse diameter, and $1\frac{3}{4}$ antero-posteriorly, occupying the middle $\frac{3}{7}$ths of the clavicle.

29th.—The size of the aneurism still increases gradually, but the patient's general condition improves. The amputation wound is nearly healed, except at the extremities. The sac of the tumour is much attenuated at its most superficial upper and inner border, the skin over which is slightly reddened. The ice-bag is still applied. The temperature for the past week has been about 98·2°, on an average, at 10 p.m. Pulse 100.

December 3rd.—Wound progressing favorably, very little discharge. The size of the tumour is, if anything, slightly increased. Temp. 99°. Pulse 88, of fair volume, but still compressible.

6th.—To-day the patient was fitted with a leaden shield prepared as follows:—A cast of the shoulder was taken in leather, from which a model of plaster of Paris was made, and upon this the lead, lined with wash-leather on its under surface, was moulded. The wound doing well. The size of the tumour does not diminish. Temp. 98°. Pulse 95.

12th.—The tumour having continued to increase in size Mr. Heath injected yesterday at 5 p.m. two drops of a solution of ergotin (= one grain of the alkaloid) subcutaneously at the *outer* border of the tumour, which to-day appears larger and somewhat flatter at its most superficial part, although the pulsation is decidedly diminished. The area of pulsation, which now assumes a more globular form, measures 3 inches transversely, and $3\frac{3}{4}$ inches antero-posteriorly, the flattened superficial part measuring in the same direction $1\frac{1}{4} \times \frac{3}{4}$ inches, and being a little more to the inner and anterior aspect of the whole tumour.

18th.—The size of the aneurism has increased to a very perceptible extent, but the pulsation is not appreciably increased, though it is somewhat more distinct than on the 12th instant. The total area of pulsation measures $3\frac{3}{4}$ inches transversely by $4\frac{1}{2}$ antero-posteriorly. The more prominent superficial part is also increased in area and is of a rectangular form, and measures $1\frac{1}{2} \times 1\frac{1}{4}$ inch. The skin over it is not tender or thinned, but slightly reddened. A vein is noticed crossing from its external border inwards. Urine, sp. gr. 1015, slightly acid, no sugar, faintest trace of albumen.

25th.—Aneurism has increased in size to a slight extent. General condition not affected.

January 1st, 1879.—During the past month the temperature of the patient at 10 a.m. was 98·2°. To-day it is 99°, this being due to inflammatory action in the tumour, the size of which has greatly increased, and its walls are consequently getting much thinned. Mr. Heath introduced into the sac three pairs of fine sewing needles with sealing-wax heads, each pair being made to cross internally. Except for the inflammatory action about the parts, the patient makes no complaint.

5th.—Tumour much harder and flatter, pulsation less distinct. Needles removed to-day. In connection with two only was there bleeding, which was easily arrested, and the pad and shield were continued. Temp. 98·4°. Tongue moderately clean.

7th.—Complains of a little pain in the aneurism, which feels harder. The skin over it is not so red as on the 5th inst., having been relieved by the application of belladonna and glycerine. Patient has slept well. Began the following yesterday :

℞ Potassii Iodidi, gr. xxx ;
Infusi Quassiæ, ℥j.
fiat haustus ter in die sumendus.

9th.—Patient has an acute attack of coryza ; conjunctiva injected and watery ; nose very watery ; face seems bloated. The œdema over the tumour is greatly increased, extending anteriorly and externally toward the stump, which it also invades. Redness, heat, and pain increased. Glycerine of belladonna applied. Temp. 99°. Pulse 115.

Urine.—Sp. gr. 1010, neutral, slightly turbid, albumen = $\frac{1}{10}$th, no sugar, small amount of phosphates.

Heart examined.—The systolic bruit at base already described is rather faint.

11th.—There is much redness about the parts. Temp. 99°. Pulse 135. Discontinue iodide of potassium and take the following :

℞ Pilulæ Scillæ compositæ, gr. v.
nocte maneque.

14th.—Pulse 88, of good volume, and not readily compressed, but slightly irregular. Respirations 32, rather short, and diaphragmatic, but alæ nasi working only slightly. Coryzal symptoms are decreased. Temp. 98·6°. Patient does not complain of pain. His back was examined by the surgical registrar (Mr. Silcock), but no pneumonia detected. Belladonna is still applied to the tumour.

15th.—The redness of the skin over the aneurism is almost gone. The œdema, slightly diminished, is still evident. The whole tumour feels harder and firmer and though no distinct pulsation can be made out, yet the most prominent (apical) part of the tumour (externally) exhibits distinct fluctuation. The wound of arm still

secretes a little pus, and the house-surgeon thinks this is increased upon pressing on the aneurism (?).

Tongue deeply coated with a yellowish-white fur, except at apex and edges (due probably to milk coagulated). He seems altogether weaker. Bronchitis continues, but no evidence of pneumonia.

17th.—During the night the patient suffered from dyspnœa and dysphagia. He is now almost moribund. Diarrhœa is profuse and vomiting present. Temp. 98·8°. Pulse 106, very irregular, small, feeble, and compressible. Patient is now suffering from extreme dyspnœa.

In the afternoon Mr. Heath noted that during expiration the cardiac impulse was transmitted to the tumour, and further, that when the patient coughed the parts about the tumour became more tense. He was still able to feel fluctuation over the tumour at the point before described.

18th.—The dyspnœa increased during the night, and with it profuse diarrhœa. Exhaustion soon became extreme and the patient comatose. At 3.20 a.m. he died.

Autopsy, thirty-one hours after death.—Rigor mortis well marked. Some post-mortem lividity. Subcutaneous fat considerable. The second, third, fourth, fifth, and sixth left ribs strongly bent in at their junction with the cartilages, and fractured three inches posteriorly to those joints.

Lungs.—Collapsed, emphysematous, and crepitant throughout, with much congestion at bases. Large quantity of serosity is poured out, especially on squeezing, particularly from the lower lobes. No part sinks in water. Right weighs 26½ oz., left 18 oz. *Left pleura* contains about 2 oz. of a bloody serous fluid in its cavity, with some old adhesions. *Right pleura.*—A few old adhesions posteriorly at apex, and about 3 oz. of serous fluid.

Heart.—Pericardium contains about ½ oz. of almost clear serous fluid. Its opposed surfaces are adherent by recent bands of lymph, and both its layers injected and covered at most parts with fibrinous exudations, which in some parts appear quite recent, in others old. Heart weighs

15½ oz. The walls of the *left ventricle* are hypertrophied, measuring ¾ in. at thickest part. Its cavity not dilated. Mitral valves healthy. Circumference of orifice = 4½ in. Aortic valves normal, except for a small patch of atheroma on the posterior semilune, which, however, is not incompetent. *Left auricle* normal. *Right auricle* and *ventricle* normal. The consistence of the walls of the heart everywhere is natural. Præcordial fat abundant. *Aorta* exhibits patches of atheroma around the orifices of the coronary artery and opposite the edges of the semilunar valves. The arch of the aorta is also extensively atheromatous, especially about the origin of the great arteries of the head and neck and upper limbs. In some parts this has broken down into ulcers of sizes varying from a pin's head to a bean; in other parts of the transverse parts of the arch calcareous plates are seen; in fine, every stage of the atheromatous process may be observed.

The *abdominal aorta* exhibits patches here and there of atheroma, but less both in extent and stage of development than in the thoracic portion. At the bifurcation, and in the common iliacs, hardly any patches are observed. The *left subclavian* and *carotid* arteries, except close to their origin, exhibit only an occasional patch. The *innominate* artery shows more.

Abdomen contains 3 oz. of clear fluid in its interior.

Spleen rather soft and congested, weighs 2¾ oz.

Liver normal, 57 oz.

Kidneys.—Capsules adherent, and so also the fat around them; each weighs 4¾ oz. On removing capsule a portion of kidney substance is torn with it, opening small cysts thereby. Surface granular, and covered with small cysts of varying sizes, from microscopic to that of pea, the larger ones formed by coalescence of the smaller. Section shows the cysts to be most numerous in the cortex, which varies in thickness from one third to half an inch. The pyramids seem smaller than normal, and are surrounded by a zone of congestion, their outline ill-defined and

pressed upon by the hypertrophied interpyramidal portions. The whole organ is tougher and firmer than normal, and measures about 4 × 2½ in. (at widest part).

Stomach, intestines, bladder, and *urethra* normal, *i.e.* macroscopically.

Brain normal, except for thickening of the membranes and general increased consistency.

The tumour.—On dissecting from above, the skin and subcutaneous tissue are found matted together, and the points of entrance of the needles can be traced deeper than the skin. Some parts of this matted tissue are deeply stained, and notably the posterior and outer part of the sac. Apparently, the sac of the tumour seems to be formed by condensed layers of cervical fascia.

Irregularly lobulated and somewhat solid in feel, and situated at the posterior aspect of the middle third of the clavicle, is the tumour, extending partly above and partly below the clavicle, between it and the first rib, for about equal distances each way. Overlapped anteriorly by the scalenus anticus and parts of sterno-mastoid, trapezius, scalenus medius and posticus muscles, it rests below on the outer half of the supra-spinous fossa, on the supra-spinatus, and upper border of subscapularis muscles; and stretched over the upper surface the omo-hyoid and platysma, scarcely recognisable, are observed. The cords of the brachial plexus at the inner and lower border are pushed aside by, and closely adherent to, the sac, and seem flattened out, as if by pressure.

There had been a comminuted fracture of the clavicle about three inches and a half from its sternal end, *i.e.* a little to the outer side of its centre, the innermost fragment overlapping the external for quite an inch. This fracture is now firmly united, and at the lower margin—close to the communication between the artery and sac—a spiculum of bone projects. The sac of the aneurism is closely adherent to the clavicle at the seat of fracture. The first rib was found to be fractured close to the subclavian groove, the fracture being ununited.

The aperture of communication of the artery with the aneurism is about an inch and a half from the thyroid axis, and measures about, but not more than, one third of an inch, and is situated on the upper and anterior part of the vessel.

The supra-scapular artery could not be found. The external jugular vein was represented by a thick fibrous cord passing over and adherent to the sac. The subclavian vein was also adherent to the sac and impervious beyond the clavicle.

The wound from the amputation had healed except for sinuses leading to the glenoid cavity, wherein the cartilage was still visible, absorption commencing in the centre.

The tumour was removed, with the scapula and clavicle and first rib, and preserved for careful dissection. The following is an account of it after dissection:

There is an irregularly lobulated heart-shaped tumour situated behind the clavicle opposite the middle, and extending above it for an inch, and below for an inch and a half. The widest and thickest part of the tumour is internal *i.e.* towards the spine. It measures in its greatest length $3\frac{1}{8}$ inches. The breadth about the centre is about $3\frac{1}{8}$ inches also, but at the inner part (base) it measures $2\frac{1}{4}$ inches, and at the outer or apex $1\frac{1}{2}$.

There can be little doubt that this patient was syphilitic, and therefore predisposed to arterial disease. It is impossible that the artery can have been punctured by the comminuted clavicle, for no immediate symptoms were observed, but it seems probable that the vessel may have been bruised or stretched by the broken first rib, and that a diseased spot in its coats may then have yielded. It will be noted that the aneurism is very distinctly sacculated, the opening into the artery being very small, whereas spontaneous aneurisms of the subclavian are more generally tubular.

The rapid progress of the aneurism, the impossibility of applying pressure on the proximal side, and the paralysed condition of the arm, seemed to justify a resort

to amputation, and I do not think the patient suffered materially from the proceeding. The effect upon the aneurism must, however, be confessed to have been negative, for any little modification of the pulsation noticed during the first few days rapidly disappeared, and the sac continued to increase in size. It will be seen in the specimen that the main artery is secured at some distance from the sac, and with a large subscapular and other branches intervening; and it may be suggested that a ligature should have been applied higher, as in Mr. Holden's case. I was deterred, however, from attempting this, both by Mr. Spence's experience, and by the difficulty which would have been experienced in controlling any hæmorrhage should it have occurred. The proceeding partakes more of the character of Wardrop's than of Brasdor's operation, and we know that the presence of intervening branches does not militate against the success of the former operation. Seeing, however, the want of success, as regards cure of the aneurism, which has followed the adoption of Sir W. Fergusson's suggestion in four cases, it may, I submit, be doubted whether the proceeding should be any further entertained.

The application of ice and the subcutaneous injection of ergotin having no effect in staying the progress of the disease, I had recourse to the introduction of steel needles into the sac, a plan by which my colleague, Mr. Marshall, had on more than one occasion produced temporary clotting to a considerable extent in an aortic aneurism. Three pairs of needles introduced obliquely so as to cross one another must offer some obstruction to the flow of blood, and favour the commencement of coagulation, and the very well-developed masses of fibrine seen in the preparation are mainly due, I believe, to their presence. The introduction of needles in the manner described was first suggested by Velpeau, and unsuccessfully practised by Dunville and Agnew (*vide* 'Agnew's Surgery,' vol. 1, p. 568, Philadelphia 1878). The method appears to me to have all the advantages claimed by the

late Mr. Charles Moore for iron wire, and by Mr. Bryant for horsehair, introduced into the sac of an aneurism, while it has the great advantage that the needles can be withdrawn at any moment. In withdrawing the needles on the fifth day there was only slight hæmorrhage from two of the openings, which was readily arrested with collodion.

I did not have recourse to galvanism, because I do not think we yet know enough of its action in relation to aneurisms. In the cases in which I have seen it employed there seemed to be considerable risk from the development of gas in the sac and the caustic effect upon the walls of the aneurism.

The administration of iodide of potassium in thirty grain doses, which was begun two days after the withdrawal of the needles, was undertaken partly on account of the reputation of the salt as a coagulator of the blood, but more as an antisyphilitic. The coryza which rapidly supervened necessitated the discontinuance of the medicine, and the general low condition of the patient forbad its resumption. The chest symptoms, which gradually supervened and proved fatal, were dependent partly upon the severity of the weather and partly upon the generally weakened condition of the patient, whose nervous system became latterly so enfeebled that it was difficult to obtain information from him as regards his sensations.

CONTRIBUTIONS

TO THE

EXPERIMENTAL PATHOLOGY OF SPIRILLUM FEVER.

BY

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(Communicated by Dr. JAMES ANDREW.)

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No. 1. The Communicability from Man to a Quadrumanous Animal.

Introduction.

The object of inquiry was the elucidation of the pathology of spirillum fever (relapsing fever) by means of comparative experiments, which might afford fresh facilities, furnish new suggestions, or illustrate by analogy, and this object has been partly accomplished.

Attempts to reproduce the fever in pigeons, rabbits, and dogs failed here in 1877; in 1879 they were successful as regards one of the Quadrumana, and this animal—the *Macacus radiatus* of systematic authors—has been alone made use of, from its being readily procurable in

Bombay, whither it is brought from the adjoining hills and woods. The specimens employed were all young and small (average weight 3 lbs. avoird.), fresh and in good health, and commonly males. They were kept in one large room and regularly fed; though tied apart, they were not so continuously isolated as to preclude the possibility of occasional contact.

The material employed to test the infective nature of the fever was commonly the blood, and occasionally the saliva; no antiseptic precautions were taken in collecting the blood, or during the defibrination usually practised prior to inoculation. Injections were made with the ordinary hypodermic syringe, not deeply, but into the subcutaneous connective tissue, the point of a lancet being also occasionally used; the site was near the groin, and the quantities (approximate) such as are named in the table appended. The method of inoculation did not seem to materially affect the issue.

The subjects furnishing the first and most frequently used material were native patients in the Goculdas Tejpal Hospital, Bombay; cases unselected and of ordinary character.

Attention was directed chiefly to alterations in the bodily temperature of the monkey, as ascertained by clinical thermometers placed in the axilla at two- or three-hour intervals throughout the whole day, and to changes in the blood which were to be found upon microscopic examination of specimens taken simultaneously from the digits. The temperatures were verified by myself, and both fresh and dried specimens of blood repeatedly scrutinised, whenever it appeared desirable. All notes were preserved, and the facts are stated without reserve.

The dates of experiment range from the close of February to the middle of August, 1879; their number and kind are shown in the index attached.

Dr. Henry Cook, Principal of Grant Medical College, Bombay, was good enough to verify the results of

Experiment 10, which is a sample of the rest. The infected condition of the monkey's blood in my first essay was promptly recognised by Prof. Cohn, to whom a specimen was forwarded, and Dr. Koch has successfully repeated similar inoculations in Europe (*vide* the 'Deutsche Medicinische Wochenschrift,' Berlin, No. 16, dated April 19th, 1879, and No. 25, dated June 21st).

A third object, viz. to learn, if possible, the mode and place of origin of the blood spirillum, has as yet been only partially attained, but enough appears, at some inevitable sacrifice of life, to warrant the inference that this parasite grows abundantly in the spleen and liver, and probably in the vascular endothelium and white blood-cells. A large collection of parts preserved in absolute alcohol still awaits attention.

Finally, cultivation-experiments were made with specimens of the blood used in inoculation, and a note on this subject is appended. The parasitic organism may be induced to grow and expand into an open network of exceeding beauty, the spiral contour of the threads being preserved throughout. Further information on this topic may be hereafter offered to the notice of the Royal Medical and Chirurgical Society, the present details being submitted with an acknowledgment of the many imperfections inevitable to a first essay in a new and highly complicated subject.

The experiments will be described first, and then commented on. Temperature charts and specimens of infected blood accompany this paper.

PART I.—DESCRIPTION OF EXPERIMENTS.

A. Series 1.—*Positive results.*

(For appended Charts see p. 120.)

Inoculation from the Human Subject.

Invasion attack.

First Experiment.—N. P., a stout young man, an extra hand on duty for the previous ten days in a medical ward containing several "fever" cases, was suddenly seized with the disease, which in him exhibited its typical features of first and second attack, with an apyretic interval. On the fourth morning of the invasion, temp. 103°, the blood plasma was noted as being rather turbid; it contained some, but not many, spirilla, and otherwise was not abnormal in aspect. (Chart appended.)

Nos. 2 and 3.—Two healthy monkeys brought from the woods only a few hours before, and consequently quite wild, had each about fifteen minims of defibrinated blood injected into the thigh. Temperature at the time as high as 104·4°, on account of their violent struggling on being handled; blood seemingly normal. Next morning the temperature had fallen to 99°—100°. Twenty-six hours after inoculation one of the animals (marked W. B.) was killed to furnish specimens of the viscera at the stage of incubation. The other (marked G. P.) was allowed to live, and forty-eight hours after injection, temp. 102·2°, a few active spirilla were seen in its blood, which otherwise appeared normal. The animal was not at all distressed, and the temperature did not rise for the next twenty-three hours, when at 8 a.m. a smart febrile paroxysm came on; at 1 p.m. the temperature was 105·4°, and at 4 p.m. 103·6°; in the evening at 8 it was 102·6°, and next morning at 8 it was 101°, after which there was a rise again towards night, and two days afterwards another exacerbation, also a third four days subsequently. Thence forward, for the remaining five days of detention, the temperature

was tolerably uniform. (Chart appended.) On the morning of the day when the brief paroxysm of "fever" occurred, the blood plasma was rather clouded, contained much small protoplasm, and a very few delicate spirilla were detected on careful scrutiny; at 2 p.m., or about the acme of fever, not a single parasite was to be seen, and at 4.30 the blood plasma was clear, with very little protoplasm, no specks, and not a trace of movement. On all subsequent examinations of the blood the same negative results were arrived at, even when the temperature had risen or was rising, so that there was no evidence of a "relapse" in this case; the brief paroxysms subsequent to the specific attack were probably mere rebounds of temperature, such as are seen in other animals and in man. The monkey showed no marked signs of illness at any time; the specific pyrexia was very brief, and it will be noticed that the parasite had disappeared at its acme. The chart of W. B. is added in interrupted lines on the first and second days.

Second experiment.—H. S., an aged female mendicant, paraplegic, two months after admission into hospital was attacked with relapsing fever acquired by contagion; the invasion lasting seven days, the apyretic interval ten days, and the relapse four days. (Chart appended.) The attack was pronounced and uncomplicated. On the fourth evening of invasion the blood plasma was clear, there was seen active protoplasm, no specks, and red corpuscles normal; there were several active spirilla of the usual aspect.

2. W. T., a healthy monkey, had ten minims of this blood (defibrinated) injected into the thigh; temp. 102·2°. It was kept apart, and subjected to examination every three hours on each of the following days. The temperature was rather high, but normal in course on the second day, depressed on the third and fourth, and on the fifth there was a brief paroxysm of fever; subsequently, for fifteen days, the temperature was low and level or normal. In the night of the third day, or fifty-four hours after inocu-

lation, the blood first showed the spirillum, which for twenty-seven hours longer was associated with a temperature rather below the mean natural, and the animal was lively. The specific pyrexia was sudden, brief, and moderate (max. temp. 104·6°), and was not attended with much obvious distress. The blood parasite was never abundant and did not seemingly increase with the fever, being sparsely found at its acme, and disappearing within five hours afterwards; it was never again detected. The chart is appended. The sign plus-minus at 11 a.m. of fifth day indicates that in the fresh blood no spirillum was seen, and that after treatment with acetic acid some were detected. This double method was followed throughout, but its results were particularly noticeable on this date.

Third experiment.—I—, a Mussulman sailor, shipwrecked and destitute in Bombay, who had been in contact outside with other sick men (one was in hospital at this time with spirillum fever), showed on admission all the characteristic symptoms of a first attack, which was followed by a marked relapse, and a second briefer recurrence. (Chart appended.) Two days before the first deep fall the blood-plasma was clear, fibrillation distinct and close-set; there was granular protoplasm and very many active spirilla, so that the material for infection seemed well-suited.

2. B. 3, a fresh monkey with normal blood, had five minims of the above (defibrinated) injected into the thigh; temp. 104·4°. A normal rise, slightly exaggerated, occurred next day, then a decline, and on the fourth day a very mild febrile paroxysm (max. temp. 103·6°), which was the sole representative of the specific attack, thence forward the temperature did not vary beyond normal limits, except on the tenth day when it rose somewhat (no blood parasite visible), and the observations were discontinued on the twelfth. The blood was scrutinised regularly four times a day. Fifty-four hours after inoculation some appearances only of the spirillum were seen, which continued for eighteen hours longer, when

during the febrile attack a single organism was detected in the fresh blood, and after treatment with acetic acid a few more; subsequently none were to be found. The chart is appended.

Remarks on the above series. In all three experiments healthy animals were inoculated with blood decidedly contaminated (once even remarkably so), and taken during the first and most pronounced attack of relapsing fever in man; yet the induced pyrexia was singularly mild, especially when least expected, namely, in the third experiment. In every instance there was a slight rise following inoculation, a depression with the advent of preliminary infection, and fever so mild and brief that it might have been overlooked. Rebounds of temperature are indicated; they are more fully shown in the longer original charts, which it did not seem necessary to reproduce throughout. The paucity of the spirillum in conjunction with very mild pyrexia is worthy of special notice.

Inoculation at First Relapse of Man.

Fourth experiment.—H. T., æt. 22, a low caste Hindoo belonging to the scavenging community (frequently affected with severe relapsing fever), showed, after a prompt crisis, a quick rebound, and two days after (eleventh day of illness) high fever set in which never left him till his death on the eighteenth day; there was lobar pneumonia on the right side three or four days at least before the end. Blood was taken by cupping on the fourteenth day (temp. 105°), and displayed a rather clouded plasma, much protoplasm, few white cells or specks, and red corpuscles mostly shrivelled, coagulation partial, and no twitching movements indicative of spirilla; but subsequently a very few organisms were detected in a stained preparation of this blood. The man was very ill of fever, seemingly idiopathic and not specific, as the local complication had not manifested itself, and the

blood showed no parasite until specially treated, upon this presumedly first day of relapse.

2. W. P. 2, a healthy monkey, several days in custody, had five minims of defibrinated blood as above, injected in each thigh. For three entire days the normal course of temperature was maintained only at lower and decreasing level, and the animal seemed to be weakened. On the fifth evening (temp. 101·2°) spirilla were detected in the blood, and the temperature fell in the night to 99·6°; it then rose next night to 102·8°, and on the seventh day fever was decided (temp. 104·4°) and the parasite abounded; a sudden fall and very sharp rebound took place in the after part of the day (spirilla absent). This was the end of the specific attack, and the temperature remained low for three days; on the fourth there was a brief rise (temp. 103·4°, and nothing peculiar seen in the blood just after) with a quasi-normal temperature (no spirilla) for ten days longer. The acute isolated paroxysm at the close of specific pyrexia was unusual in these animals; it occurs sometimes in man. W. P. 2 showed the usual signs of illness during its short attack, and subsequent recovery was prompt; the advent of infection was here unusually deferred, even after allowing for long intervals of observation, and this may be connected with the state of his blood at inoculation from H. T.

Fifth experiment.—B. M., an adult negro of stalwart build, was admitted towards the close of invasion attack, and after an apyretic interval of five days went through a well-defined relapse; there was no subsequent recurrence. The chart is appended. On the second evening of the relapse some blood was drawn, which presented the following characters:—Plasma clouded, fibrillation indistinct, few white cells, a little protoplasm, chiefly small, spirilla several and active.

2. R. T, a fresh, healthy monkey, had three or four minims of partially defibrinated blood injected beneath the skin of the thigh, it was then kept apart with a companion not subjected to operation. No prominent

symptoms were noticed until five day after inoculation, when the animal appeared depressed, refused food, and was hot and shivering; temp. 105·8°. The blood was then examined by gas-light; it set quickly, plasma clouded and so full of active spirilla that the whole field of view appeared in incessant motion. Next morning the fever had remitted (temp. 102·8°), and the monkey though distressed took a little food; at noon temp. 103·4°, with renewed prostration, as indicated by the lowered head and folded limbs, hiccup, rejection of all food and drink, and a sleeplessness in marked contrast with the frequent dozings of its companion; the hair was rough and staring, yet not wet. Upon examination, the blood-plasma appeared tolerably clear, fibrillation coarse and open, white cells and protoplasm scanty, a few specks; the red corpuscles were in constant movement owing to the abundance of active spirilla. At 2 p.m. the temperature had risen to 104·8°, at 4 p.m. it was 103·8°, and at 6 p.m. 102·4°; at 9 p.m. it had fallen to 100·2°, and this probably represented the crisis which thus took place some twenty-seven hours after observed initiation of high fever. At 4 p.m. the blood was found to have regained its normal aspect; plasma clear, fibrillation distinct, white cells not numerous, red corpuscles piled and quiescent; there was some granular protoplasm, and a few minute specks and filaments of indeterminate character. With cessation of fever the monkey rallied, moved languidly, and began to eat; its appetite, indeed, was ravenous; it slept. There was no rebound of temperature, and for ten days no sign of relapse; convalescence was prompt, four ounces in weight being gained in six days; examination of blood on the second, fourth, and fifth days after fever negative. The chart in this case is withheld, being imperfect, as no night observations were made.

The companion monkey did not become infected, although in close alliance throughout; its chain caused some chafing, which doubtless induced the transient feverishness occasionally noticed; blood repeatedly examined with

negative results. In the course of these experiments similar accidents to these little animals were not unusual.

The above was my first essay, date February 26th, 1879, specimens of the infected blood were forwarded to Professor F. Cohn, and their character verified.

Sixth experiment.—I. S. B., a lad of 14, whose father had relapsing fever, was not admitted until the first apyretic interval, but in a specimen of dried blood taken from him at their home I found the parasite in abundance. His relapse was much shorter than his father's, and though the blood spirillum was at first very plentiful, his illness seemed to be of the mildest. On the morning of the second day (temp. 102·6°) the liquor sanguinis was clouded and contained some large protoplasmic bodies, and there were numerous active organisms to be seen. Ten minims of the defibrinated blood were injected hypodermically in each of the following monkeys at 10 a.m. 10th July.

2. Chain, a fresh animal brought to hospital the previous night, small and wild, had normal temperature and blood. The temperature took a normal course and was rather below the mean for the first three days, it then declined and rose into a mild paroxysm prior to the onset of high fever on the fifth day; the blood was examined only twice daily, and forty-eight hours after inoculation a few active spirilla were found in the very clear plasma; next day there were more, and at last the blood was filled with the parasite. The animal was then ill, though still active, and it was killed for pathological inquiry. In this case high fever was preceded by a minor or quasi-abortive rise.

3. Rope, a similar subject and similarly treated, showed healthy blood and a temperature which, pursuing a normal course, was somewhat more pronounced than usual, until the advent of the blood parasite, forty-eight hours after inoculation. Death was then inflicted for the purpose of research at this commencement of specific infection. All charts are appended.

The concordance of date upon which the spirillum was

first seen in both these animals, supplies evidence of uniform conditions and effects to the exclusion of accidents, such as a possible contagion; and since in the other two instances, in which more than one animal was simultaneously submitted to inoculation, a similar concordance was noted, I feel justified in relying upon the majority, at least, of my essays, which were always made in like manner.

Seventh experiment.—B. L., a destitute immigrant labourer, exhibited in hospital, after the close of the invasion, a first and second relapse with subsequent minor perturbations of temperature, all fairly characteristic of famine fever. The chart is appended. On the third day of the first relapse (temp. 103·4°) the state of the blood was as follows:—Plasma clouded, fibrillation indistinct, white cells few, some large protoplasmic masses, no free granules, red corpuscles heaped; there were several active spirilla to be seen. At noon (temp. 104°) two drachms of blood were abstracted and defibrinated.

2. Y. T., monkey, healthy, had twenty minims of the mixed serum and corpuscles injected into the thigh; temp. 101·2°. Next day the temperature rose a little as usual, and on the third day it had subsided. At 10 a.m. it was rising again (102·8°), and the animal's blood was then (forty-six hours after inoculation) found to contain a few active spirilla; plasma rather clouded, but fibrillation seen; white cells few, some large protoplasm, no moving specks, the red corpuscles piled. The monkey looked a little drowsy, though not sick; it was now killed in order to furnish specimens of the viscera at this early stage of infection. (Chart appended.) Y. T. had previously been inoculated with dried spirillar blood, and had remained unaffected.

Eighth experiment.—F. J., æt. 28, ship labourer and immigrant, after a most pronounced first crisis in hospital, and somewhat prolonged first interval, underwent a relapse, striking from its intermittent character. On the evening of the third day, during a mild mid-paroxysm,

the temperature was 100·4° and the blood plasma was clouded, white cells very few, no moving granules, red corpuscles piled, and amongst them indications of two or three spirilla which, with the Albrecht process, were seen to be slender, though not seemingly immature. The man was not suffering much. (Chart appended.)

2. B. 1, a large monkey, rather thin and weak from the effects of a late experiment with sputum, but not now feverish, had ten minims of the above-named blood (defibrinated) injected into the thigh. Its condition as to the temperature and blood was then ascertained at three-hour intervals for the next six days. The spirillum made its appearance fifty hours after inoculation (temp. 100·6°), and was found for forty-eight hours longer, though sparingly, until high fever set in, when the parasite became abundant, and individual examples seemed to be dividing about the middle. The temperature was normal on the second day, higher than usual in the morning of the third, and with advent of the visible infection, declined considerably on the fourth and fifth days (min. 99·4° on last morning), rising at the end to 102·4°, prior to the abrupt onset of pyrexia on the sixth day (temp. 105·4°), when death was inflicted for the purpose of ascertaining the state of the venous blood of different organs. The animal showed hardly any signs of illness till the last day, when it became languid and opened its mouth frequently. (Temperature chart appended.)

Ninth experiment.—S. B., father of the lad in experiment No. 6, after a slowly declining first crisis, and tolerably level first interval, had a well-marked relapse, at 2 p.m. of the third day of which (temp. 102·5°) blood was drawn, and the serum allowed to separate; it contained some red discs and several spirilla, some being extended and quiescent, and others in active movement.

2. W. T. 3, a healthy monkey, had ten minims of this blood-serum injected into the thigh at 5.30 p.m., June 24th; temp. 102·4°. The temperature declined at midnight to 101·4°, and then rose steadily next day until 2

p.m., when it reached the maximum of 106·8°, which was also the acme of a brief paroxysm of specific fever. It declined even more rapidly than it rose, to 100·4° at midnight of this second day. A slight rebound followed, and thenceforward for twelve days a tolerably level, though low and slightly irregular, course was preserved. Here infection was very prompt, the blood spirillum being found sixteen hours after injection, and possibly being present at an earlier period, as the temperature was already 104° on first employment of the microscope after inoculation; it was then abundant. Three hours after the climax of the acute isolated paroxysm (temp. 105·7°) the parasite had quite disappeared from the blood. None but the briefest preliminary infection could have occurred in this remarkable instance, fever setting in so soon as twelve hours after inoculation. The sharpness and brevity of the attack, and its prompt decline, with absence of decided rebound, are other striking features of the case.

The experience was unusual and even unexpected, since the man, S. B., was suffering considerably, and his blood might be supposed to be highly infective. However, I should observe that blood-serum was the vehicle used (whence possibly the rapid infection of the monkey), and that in the two hours and a half after the blood was drawn and had set, some of the spirilla had ceased to be active, whence possibly the milder infection, as manifested by brief duration of pyrexia. This was the only instance known of fever coming on so soon as twelve or fourteen hours after inoculation, and it must be viewed apart from all the rest.

Tenth experiment.—R. Y., a low caste Hindoo, one of others attacked outside, was admitted into hospital just after the invasion, his chart shows only the relapse, and is appended. The symptoms were marked, but the state of the blood was at no time characteristic as usual, owing to opacity of the plasma, and sparseness of the parasite. On the fourth evening of the relapse (temp. 105·4°) the spirilla were so obscurely indicated by a rare twitching of the red

corpuscles, that their existence might easily have been overlooked; but on treatment with acetic acid, their presence in small numbers was fully established. The other characters of the blood were as follows:—Plasma clouded, fibrillation slow and not distinct, much protoplasm, large and small, many small rounded bodies or specks (quiescent), white cells numerous, some large granule cells, red corpuscles in small piles, and often shrivelled. The patient was affected with asthma and bronchitis.

2. W. T., a healthy monkey, had about fifteen minims of defibrinated blood injected subcutaneously in the thigh (temp. 102°); it remained well to all appearances for three and a half days, when fever was noticed at my morning visit, temp. 104·2°; aspect depressed and shrunken. Blood plasma clear, very few specks, white cells not numerous, protoplasmic masses rare, red corpuscles piled and heaped; "there are vast numbers of very active spirilla, which seem to be rather larger than in man." In the afternoon at 3 the animal became worse, its head hung so low that only the radiating scalp was visible, and its general aspect resembled that of R. T. in the fifth experiment (No 2). At this time Dr. Cook saw the blood and became assured of the validity of the observation. At 11 p.m., temp. 103·8°, still crowds of spirilla, and next morning also; no abdominal tenderness evinced, and spleen not felt; at 2 p.m. of the second day of fever the animal was killed with chloroform, in order to furnish specimens of the viscera during pyrexia; blood defibrinated after death showed abundance of active spirilla, so that the vapour of chloroform does not act as an immediate poison on these organisms. The temperature chart of this early experiment is not complete; it shows a maximum of 106·8 in the evening of the first day, a decline to 103·4 next morning, and an interrupted rise on the second day to 106°, when death was inflicted; the temperature was below the normal prior to fever setting in suddenly.

Eleventh experiment.—The first relapse of the man

furnishing the material for experiment 3 was very pronounced, and on the fourth day (5 p.m.) the blood exhibited the following characters:—Plasma tolerably clear, fibrillation distinct, some large granular protoplasmic bodies, no specks, there were swarms of spirilla, fully formed and active, and besides some long wavy beaded filaments were seen, which possibly were immature forms.

2. W. T. 2, a monkey with normal blood, had five minims of the above (defibrinated) injected into the right thigh at 6 p.m., its temperature was 102°, falling in the night to 100° and rising next afternoon to 102·4°; then sinking on the third morning to 99°, it rose abruptly to 105°, again declined, and on the fourth day of experiment ascended at about noon to 105·2°, when the animal was killed. Here was a distinct paroxysm preliminary to what was probably the main attack, and the same event is occasionally seen in man. (The chart is appended.) Blood: at the first examination, fifteen hours after inoculation, a few spirilla were detected in the otherwise unchanged fluid, they appeared to be slender, but were active; thenceforward, during the brief non-febrile period, and as well the isolated paroxysm, to the end, the parasite was invariably present, being at last very abundant. The animal did not show unequivocal signs of distress at any time previous to the last day, and even then its general state was not marked by much apparent malaise; had its life been prolonged, possibly with high fever the usual symptoms would have supervened, yet this circumstance is noteworthy (it has been seen also in children attacked with relapsing fever), as indicating that with even pronounced blood-infection the system is not necessarily overcome. Another striking feature was the rapidity of induced contamination with blood taken during the relapse, as compared with the delayed and mild results of inoculation with that taken during the invasion attack as shown in Experiment 3. I should mention that W. T. 2 had five weeks before undergone an earlier attack of induced spirillum fever (described in

Experiment 2), and upon recovery had been returned to the bazar, whence it was again brought on the day of the present proceeding (April 26th).

Inoculation at Second Relapse.

Twelfth experiment.—R. M., a stout but scorbutic young negro of the same family as B. M., in Experiment No. 5, was admitted after the end of the first attack, and underwent a second and third in hospital. On both these occasions the blood parasite was so sparse that its presence might easily have been overlooked in ordinary examination. The second relapse consisted of an isolated paroxysm of less than one day's duration, but terminating in a fall of 10·6°. At the acme of this brief attack (temp. 104·6°) the blood exhibited the following characters :— Thin, devoid of fibrine, brownish in hue, but brightening on exposure, plasma clouded, little white matter, red corpuscles shrivelled and dispersed, a single spirillum after long search was detected. The patient suffered very little at the time. (His chart is appended.)

2. B. B., a healthy monkey with normal blood, had eight minims of the above (defibrinated) injected hypodermically into the right thigh at noon, 29th March; temp. 101·4°. For three days the blood presented no obvious change and the temperature did not vary beyond normal limits. Early on the 2nd of April, the fifth day of experiment, or under ninety-four hours after inoculation, the animal seemed well, though being kept alone it cried out occasionally; temp. 101·8; blood plasma clear, fibrillation not distinct, very little protoplasm, and no peculiarity except the presence of several active spirilla. The temperature now declined to 99·8 at midnight, and on the next day to 98·6 at 6 a.m.; but at 8 a.m. it began to rise and at 8 p.m. it was 105·6; it remained at 104·5 during the whole of the following day, and the animal was evidently ill. Early on the 5th the temperature declined to 100°, and

subsequently took a quasi-normal course. The spirilla were few at first (1 to 4 in the field), and with the accession of fever their number increased (6 to 12), till at last they swarmed in the plasma ; at 10 p.m. of the 4th April there were many of full size (temp. 104°), but at 2 a.m. of the 5th (temp. 100°) not one was to be found. Unpromising as this experiment seemed, its results were comparable with those of No. 5, and I have no doubt that had the same early scrutiny been practised then, a similar ante-febrile blood infection would have been detected.

B. B. quickly recovered, and it was kept under notice, and daily examined for eleven days in order to ascertain the likelihood of a relapse ; the result was negative. Yet on the twelfth day after fall I found that the spirillum had reappeared (temp. 102·8°), and this was the beginning of a second attack, which had a preliminary infection period of at least thirty-six hours, and culminated in a smart febrile paroxysm (temp. 105·2°), of twenty-seven hours' duration, the parasite, at first sparing, was then abundant ; there was hardly any constitutional disturbance, and return to the normal state was prompt, and maintained for the next thirteen days. I am unable to assign the cause or character of this fresh attack, seemingly it was spontaneous, and the probability of contagion remote, yet with other contrary experience I cannot view the event as a true relapse, unless it be that repetitions of fever by auto-inoculation are, in the monkey, subject to irregularities not perceptible in the human subject. Contagion was not proved amongst these animals when it might have been looked for, but it had possibly occurred in this case. (Two charts are appended.)

Inoculation at Stage of Incubation.

Thirteenth experiment.—S. B. (*vide* Experiment 9) had a somewhat prolonged first interval, and on the ninth day, or two days before the relapse (temp. 98°, and health

fair), his blood was described as clear, white cells few, protoplasmic bodies small, red discs normal; after treatment with acetic acid only a few specks and short rods were noticeable as possibly unusual ingredients.

2. R. T. 2, monkey, had ten minims of the above (defibrinated) hypodermically injected into the thigh; it remained unaffected to all appearance for fully ten days, the temperature being level, except on the seventh day, when a brief rise to 103·4° took place; the blood was unchanged. After so long an interval this experiment was regarded as a failure, and another injection was about to be made, when I found in the blood (temp. 102·6) several active spirilla, and the animal appeared sluggish, though not ill. About twenty hours afterwards a brief febrile paroxysm occurred, which lasted six hours and ended abruptly. The parasite was detected prior to and at the acme (temp. 105·4°), and had disappeared three hours afterwards. The monkey was ailing for the time, but soon and permanently rallied.

Respecting this irregular example, I have to observe that four other monkeys were inoculated on successive days of the same apyretic interval. R. T. being the third in this series, and in all the rest the result was negative; further, it would be quite extraordinary if so long a period as ten days' incubation were conceivable in these animals, and I am therefore inclined to suppose that in the present instance R. T. 2 had acquired the fever it displayed by means of contagion from W. T. 2., (*vide* 11th Experiment), although I had given orders for the latter to be kept apart; were this so the incubation period would be reduced to five days, and the anomaly would be accounted for. Observation has led me to infer that, like men, these quadrumana differ individually in their susceptibility to infection, and though direct attempts were made to test contagion amongst them ineffectually, I think it probably took place in the present instance, and in the 12th and 17th Experiments as the *vera causa* of the second attacks then noticed. This opinion is inferential only, and in the

appended table I have felt bound to give the figures actually found.

Inoculation from the Monkey.

Stage of High Fever.

Fourteenth experiment.—W. T. (*vide* Experiment 10). On the first day of observed pyrexia (temp. 104·2° at 11 a.m.), and general symptoms of illness manifest, the state of the blood was as follows:—Plasma clear, fibrillation slow, white cells and protoplasm scanty, a very few moving specks, and red corpuscles piled and heaped; there are vast numbers of most active spirilla, which appear to be rather larger than those of human blood.

2. R. T., monkey, in fair condition, though rather thin in consequence of an earlier attack (*vide* Experiment No. 5), this day, March 16th, being the twelfth after the fall, and no sign of relapse being manifested during the interval, a minute quantity of blood was taken by puncture from the thigh of W. T., as above, and inoculated with the same lancet in the thigh of R. T., the clot, not larger than a pin's head, being introduced beneath the skin; no bleeding ensued. The animal remained well, and its temperature was tolerably uniform until midday of March 20th, or four complete days after inoculation, when an abrupt rise took place; the attack thus initiated lasted sixty-six hours, and was continuous; the maximum temperature of 106·4° was noted about midway, the decline occurred at 6 a.m. of the 23rd, with a prompt descent to 100·6°. The general symptoms of the fever were the same as seen on other occasions, and the monkey was killed at the fall in pursuit of further inquiry. (Chart appended.)

This early example of communicated fever was unmistakably clear, and it is noteworthy as proving that no protection against reinfection is afforded by even a recently foregoing attack; it also shows that no predisposition to a recurrence, as estimated by comparative

length of incubation period, can be said to result from such previous attack; and, lastly, it demonstrates that a very small quantity of blood, or hardly more than the amount of lymph commonly employed in vaccination, will suffice, when introduced beneath the skin, to convey a pronounced spirillar infection.

Fifteenth experiment.—W. T. (*vide* Experiment 10), on the second day of observed fever, or upwards of thirty-two hours after its initiation, the defibrinated blood, obtained after death by chloroform, was found to contain abundance of active spirilla, which were seemingly unaffected by the inhaled narcotic, and it was therefore employed for further testing the communicability of spirillum fever at first remove from man.

Three monkeys had each a few minims of blood *minus* fibrin subcutaneously injected in the same manner and hour; of these two were fresh acquisitions from the bazar, and the third (C. below) was the companion of R. T. (*vide* Experiment 5), which, although in closest propinquity to that fever-stricken animal, had never shown any sign of contagion. The results are briefly described below, and the fever charts are appended entire, with the exception of the first two days, when night observations were not complete.

2. N. W. T. (also termed "biter" from its viciousness). The temperature rose beyond the mean for a few hours, and then became nearly normal; on the third day it rose again simultaneously with that of the two other animals, and rather higher, being at 11 p.m. 106°, and the monkey still very wild; the blood contained many spirilla. Next day the temperature subsided a little, being 103·8° at 11 a.m.; the animal now seemed ill; its blood was highly contaminated, and death was inflicted when there was every prospect of the attack continuing as in its neighbours.

3. R., the other fresh monkey, showed but little variation of temperature for forty-six hours, when fever came on rather gradually but decidedly (104·2° in the first

evening, and 106·2° on the next); then, after a morning abatement, again rising (third day), and so again (fourth day), when the crisis took place (fifth morning) in a decline to 101·4° at noon. Apparently this ended the specific febrile attack, which had thus lasted for about ninety-four hours or four complete days; and now began a sharp and prolonged rebound, the temperature at 4 p.m. having suddenly risen to 107·2°, and this exacerbation persisted throughout the next day (at 4 p.m. temp. 106·2°); but on the third the body heat declined at first slowly, then rapidly, until the animal died at 4 p.m. The blood was repeatedly examined throughout the entire illness, and found to contain numbers of the parasite on the first day and second, and even at the considerable decline on the morning of the fourth day (temp. 102·2°), when it swarmed with clusters of spirilla. On the true fall occurring next day at noon, not a trace of the growths was to be seen even with the $\frac{1}{24}$ in. immersion lens, or indeed any abnormal appearance. The secondary pyrexia, or rebound, was also marked throughout by an absence of the spirillum, and so its real character was made manifest. At the beginning of its illness the monkey was very quiet, and sweating of the palms was noted; next day, though fever was high, it sat up and took food in the morning, but at noon it joined the other two, and all huddled together, being evidently very sick. On the third day both survivors presented the characteristic aspect, drooping, pallid, and shrunken, refusing rice, nibbling only at fruit, and drinking water, though not with eagerness. At the fall the symptoms were mitigated, but with the renewed pyrexia, shiverings and depression were noted, and finally prostration, semi-consciousness, rapid breathing, and lowering of temperature till death. An autopsy was made and the parts preserved. I may here observe there was found no localised inflammation sufficient to account for the fatal termination, unless some extreme redness of the small intestine be regarded as inflammatory; the

animal seemed to die of fever as human subjects occasionally do.

4. C., a monkey, some time under observation, and affected with an accidental sore, but never the subject of experiment, showed a high temperature shortly after inoculation, which had subsided next day. On the third the temperature again rose, but somewhat more gradually than in the other two animals, declining in the morning, to ascend permanently at night to 106·2°. On the fifth day there was a morning decline and evening rise, and so on the sixth, when the acme of 106·8° was attained a few hours before the critical fall to 101·6° in the morning of the seventh or following day. Except at the beginning, the course of the pyrexia was similar to that in No. 3. The animal was lively on the second morning when the blood was found to be full of spirilla; soon after it became sick, and remained so till the fall. On the last day, at 5 p.m., the blood contained many active parasites; an hour later there were none, and at 8 p.m. the fever reached its turning point. I had intended to watch the case by minutes, but was called away to perform an amputation; sufficient was seen, however, to show how abruptly the blood parasite disappears at this period of febrile crisis, and the observation was even frequently confirmed in the wards of the hospital. A prompt and pronounced reaction of temperature followed the cessation of specific pyrexia, and it was prolonged for four days, slowly subsiding at the last, and the monkey then quickly became convalescent. The blood was free from visible contamination at this time, and no relapse followed during the eighteen days after crisis; the animal was closely watched.

The above experiment is decisive of the inoculability of spirillum fever, of the similarity of symptoms under similar conditions, of the greater severity of the fever induced by infection at first remove, *i.e.* from monkey to monkey after a prior infection from man; and, finally, of the similarity of the comparative phenomena to the human, with the

exception of the relapse, so-called, which was not seen in the lower animal surviving.

Sixteenth experiment.—From monkey No. 2 of the last experiment, the blood taken twenty-six hours after initiation of pyrexia was swarming with spirilla; it was allowed to stand for three hours and a half, and then had separated with considerable distinctness into clot and serum. The serum was found to contain many moving parasitic organisms and a few red discs unchanged, with no other visible particles; the clot was dark and firm.

2. B. T., No. 1 in the chart, had ten minims of the slightly-tinged serum injected beneath the skin of the thigh at 5.30 p.m. of 20th March; temp. 103°. The body heat rose the next day to somewhat above the normal mean, and still more on the third and fourth days, descending to the mean at 8 a.m. of the fifth day; it then rose promptly to 105·4°, and continued high on the following day; early on the seventh the critical fall took place, and very soon after a sudden and sharp rebound (temp. 107°); on the eighth day the temperature declined, at first moderately, and at last very quickly, the animal dying this night (the last entry, temp. 96·4°). The blood was examined at intervals, and the spirilla were found before fever set in, being then few and small; with rise of temperature they became numerous, the animal not seeming to suffer much. On the sixth day, with continuous fever, the parasites were fewer, seeming gradually to decline at first, then towards the end of the specific attack they were abundant and aggregated in clusters; at the fall on the seventh day all traces of them had disappeared; with the rebound of temperature the blood was found to be loaded with plasmic material only. The animal was very ill, as if overpowered by depression, the ears and extremities being cold, and the head and trunk very hot; no spots could be seen on the skin; it remained in this state, the body cooling, until its death. The stomach and small intestines were apparently much inflamed, and there were some hæmorrhagic spots.

3. O. T. Two bits of the above blood clot, not larger than a pin's head, were introduced beneath the skin of the left thigh; they had been slightly washed in a weak solution of sulphate of soda, and were not examined microscopically, it being supposed that a few spirilla would be still entangled in the fibrin meshes. The temperature maintained a quasi-normal course for two days, and on the morning of the fourth a few active spirilla were detected in the blood. Pronounced fever did not come on till the following day (temp. 105·6°), it was less next day, and on the seventh, when an exacerbation took place (spirillum present), which ended in the fall. After a few hours a sharp rebound set in, and at 4 p.m. of the eighth day the temperature was 106·8°. This renewed fever was a veritable relapse, resembling in form and duration, and exceeding in intensity, the preceding attack, but distinguished from it by the entire absence of spirillar blood-infection; at the close the animal was so exhausted that it died. At the autopsy I found some inflammation of the stomach and small intestines; there were some petechiæ on the heart.

Remarks.—The similarity of results in this experiment is manifest, even in particulars, and the series may also be compared with the last, their common feature being increased severity of infection with maintenance of all characteristics. The rebound, or secondary fever, or "relapse," properly so-called, was present in all these instances of inoculation at removes from man, and I infer that the infective property of the blood was increased at each successive remove. As both these last animals died, there seemed no occasion to pursue this subject by means of fractional inoculation, and my experience had not been such as to warrant confidence being placed in dates and changes of temperature as criteria of intensity of blood poisoning in this particular form. I note that although the infection in Experiment 16 was sufficiently intense to lead to death, yet the incubation-period of the fever was not shorter than the mean, nor was the specific tempera-

ture unusually high ; and Experiment 20, and those made with saliva, show that other organic poisons operate differently to the spirillar. Here the varying state of the blood at different stages of the "fever," and probably idiosyncrasy of subject, are elements of calculation as yet not enough known. There could hardly be a more intense degree of blood-infection than was displayed in my experiments, and all the deaths occurred during secondary fever; perhaps some elucidation of the character of this last-named sequela might be elicited by the fractional method.

Seventeenth experiment.—W. B., in Experiment 1 (*vide* chart), had twenty-six hours previously to furnishing the infecting material used below, been injected with spirillar blood from a human subject, which was proved (in its comrade, G. P.) to be capable of conveying disease. In this material no sign of the parasite was detected; plasma rather clouded, fibrillation visible, white cells few, little protoplasm, specks few and quiescent, red discs piled and heaped.

2. E., a healthy animal with normal blood, had a few minims of the above defibrinated blood injected in the usual manner at noon, March 25th; it remained well till the 29th, the temperature being tolerably uniform in its daily oscillations, though rather higher than usual at maximum (103°). The blood was examined twice daily (excepting on the 28th), and at 10 a.m. of the 29th, or ninety-two hours after injection, it was found to contain a few active spirilla without being otherwise peculiar. Next day there occurred a smart febrile paroxysm (temp. 106·2°) of about eight hours' duration, and soon after its acme the blood was found to be free from the parasite, only some moving granules being detected; the temperature then declined to 101°. There seemed no reason to doubt that this brief attack was due to artificial infection, and hence the inference that the germs of specific pyrexia are not necessarily represented by the blood parasite.

Relapse.—E. continued well for six days, or about 150

hours, during which time the temperature oscillated between 100° and 103°, and whenever examined (once or twice daily) the blood was normal in aspect. In the afternoon of 6th April, one or two active spirilla were detected (temp. 103°), and again next day (temp. 102·2°). On the 8th, or thirty-six hours after their first advent, a smart febrile paroxysm took place (temp. 105°) which lasted ten or twelve hours; at its onset the parasites became numerous, and at its decline they had all disappeared. The animal looked pale and thin at this time; it was kept under observation for eleven days longer with negative results. (Charts appended.)

Remarks.—The interval between the first and second attacks of fever was longer than the first incubating period as nine to five, and this points to a character of relapsing fever in man; both attacks were alike in the preliminary non-febrile infection, and in the form and duration (nearly) of pyrexia, and this circumstance also seems in favour of their essential connection; the alternative view is that contagion was the cause of the second attack. Compare with Experiment 12, where the "relapse" came on nearly twice as late, thus showing a suspicious want of conformity which is of the rarest in man.

As regards the invasion-attack, this experiment corresponds to No. 13, in which also incubation-blood seemed to prove infective; both attacks were mild, and so far alike, yet there is a wide discrepancy in their dates of onset, which it is difficult to comprehend, except upon the supposition that the spirillum fever of the monkey is liable to hidden modifications hardly consistent (as it might appear) with the open manifestations in the human subject; here, too, the alternative view was that of incidental contagion.

Series 2. *Experiments furnishing negative results.*

The fact of communicability under certain conditions being established, I proceed to narrate some other experiments made in similar manner, which may assist in elucidating the nature of these conditions.

Failure of Spirillar blood to infect.

1. *Of man, at invasion attack (a).*

Eighteenth experiment.—K. G., a pauper immigrant, æt. 55, had a severe attack of spirillum fever, which was said to be the first and seemed to be much prolonged (fourteen to fifteen days); the end only was seen in hospital, and it was accomplished by slow descent extending over three days, during two of which the blood parasite was abundant. Immediately afterwards a sharp rebound took place, with head symptoms, and the patient died (cerebral hæmorrhage). His daughter was in hospital, and also died after her attack (thrombus in the femoral veins); the wife had had "fever" a short time before, and had recovered. There were no other members of this family. On the first day of decided decline (temp. 100·4°), the blood contained many spirilla, and a minute freshly clotted portion was used for inoculation. (Chart appended).

2. Y. T. 2, a monkey, with normal-looking blood, had the above fragment introduced beneath the skin of the thigh (temp. 102°). The same evening the temperature rose to 102·6°, when the blood was still normal (red discs somewhat irregular); four hours later the temperature rose to 105°, falling promptly to 101·8° next morning (blood healthy), and subsequently for eight days there was only such oscillation of body heat as might be referred to the presence of sores on the body, the blood remaining unchanged in aspect.

Here inoculation failed, though made after the same fashion as in the successful Experiment 16, No. 3; there is no reason to suppose that the brief rise after inoculation was of specific character, and upon consideration, I infer that when the spirilla, as a rare event, persist till near the end of the crisis, their presence does not necessarily imply infective quality of the blood. Under artificial cultivation, I found that at this time the parasites in the blood of K. G. (who was unusually ill) did not grow as other specimens had done, whence it is probable that these organisms do not themselves evolve the germs of future broods, except under certain conditions which do not pertain to the close of a febrile attack. Yet upon the hypothesis that early "broods" or "crops" of the parasite usually leave behind them, in the blood itself, the germs of a succeeding growth to be developed with the "relapse," it might be supposed that blood at or near the "fall" would be both infective and culturable; the contrary being the case, I infer that the "germs" are lodged (if not produced) outside the blood-current. Perhaps the conditions vary, for reproduction is not invariable.

At first Relapse (b).

Nineteenth experiment.—Blood taken on the third day, or the day after the successful attempt described under Experiment 5, from the man B. M., had the following characters:—Plasma not clear, a few white cells, some moving granular protoplasm, many specks, and several spirilla in active movement.

W. and B., two monkeys in good health, had the entire blood injected, namely, a few minims beneath the skin of one, and the same into or around the femoral vein of the other; no local or general ill effects followed, both animals continuing unaffected for eight days. Special notice was taken on the fifth day, or the date of fever in the earlier experiment, and I conclude that the present attempt

was a failure. Why it should have been so is not manifest, for both animals were afterwards successfully inoculated, and the material employed did not appear to differ materially from that used the day before with signal results. I notice, however, that at the time the temperature of the man B. M. was at its highest (105·8°)—and it is probable that at high temperatures, whether mid or final, the parasite loses something of its dynamical activity—the man was sweating when the blood was abstracted; and upon examination of the latter by the acetic acid process, I did not find so many parasitic organisms as was anticipated. The alternative view would be that some fault or accident in either procedure or instrument had intervened to prevent infection; none was perceived.

Twentieth experiment.—The patient named in the successful experiment, No. 4, had, two days at least before his death, pronounced pneumonia (it seemed to be of pyæmic character from the post-mortem examination) in conjunction with spirillum fever; thirty-six hours before decease (temp. 104·6°), nothing definite of peculiar aspect was seen in the blood as ordinarily scrutinised, but I found upon treatment with acetic acid a very few spirilla, besides many small protoplasmic particles—infection, therefore, was possible.

2. R. T., a fresh, healthy monkey, had ten minims of the above, defibrinated, injected into the left thigh. The effect was high and continuous fever ending in death on the fifth day, and the blood never showed any signs of specific contamination; twelve hours after inoculation, the temperature rose and shortly attained 105·8°, it remitted on the third and fourth morning, and the animal died on the fifth, there being some local sero-purulent infiltration above the side of injection, and possibly septicæmia. Here, I suppose, there existed a poison in the patient's blood, which in the monkey over-powered the spirillar infection, checking its development and inducing a fatal toxæmia. A similar instance afterwards occurred (*vide* No. 24), and I do not regard either merely as exceptions

to the rule of spirillar reproduction; both were complicated cases and otherwise interesting as showing the antagonism of blood poisons.

Twenty-first and twenty-second experiments.—S. B. (*vide* No. 9) displayed the relapse ten days after complete fall; as usual, it began abruptly, and blood was taken morning and evening of the day of onset.

1. The morning temperature was 97·4°, pulse 84, the patient convalescent, the fresh blood was noted as containing clusters of white cells, very little protoplasm, but numerous active granules, and by the acetic acid process a few small yet distinct spirilla were also detected.

2. G. T., a healthy and excitable monkey, with normal blood, had ten minims of the above injected after defibrination; for five or six days afterwards the temperature showed marked daily paroxysms with occasional high range (as 104·4° on the sixth afternoon), and yet the blood, examined twice daily, furnished no evidence of spirillar infection. The animal itself continued well. Observations were made until the fifteenth day, with like negative results, and it must be supposed that the parasitic organisms were too few or too immature to induce specific fever, supposing that they represent the infecting agent, the daily perturbations of temperature may be attributed to the excitability of the monkey.

3. The man's relapse set in at 4.30 p.m., and at 5.30 the temperature was 102°, pulse 100, the blood then showed a few spirilla, with small protoplasm and some granules; ten minims, defibrinated, were injected.

4. F., a fairly healthy monkey, but so restless that the chain it was bound with, caused, by chafing, a large ulcer round the waist, and feverishness in consequence. The chart shows this mild pyrexia to be continuous, and as the blood was carefully examined daily with negative results (only protoplasm and white cells abounding) I conclude that there was an entire absence of specific infection in this experiment also. Possibly the local irritation may have rendered the animal's system unfit, or the injected

parasites were too few or immature; two days later the blood of this patient proved to be promptly infective in another monkey (*Vide* No. 9).

2. *Failure with Spirillar Blood of the Monkey.*

During Fever.

The animal, No. 2 in Experiment 6, furnished blood-serum containing many active spirilla at the time of its decease.

Twenty-third experiment.—Four minims were injected beneath the skin of Y. T. 2, a monkey previously the subject of ineffectual trial and troubled with sores. The usual observations were made for eight days continuously, and I became assured that no infection took place. Soon after, Y. T. 2 lost health and died with nervous symptoms. It was probably an unhealthy animal, and seemed to be insusceptible to this particular fever.

During Preliminary Infection.

Twenty-fourth experiment.—Blood taken from B. B. twenty-four hours after the relapse was noted, temp. 102°, and several small but active spirilla being present (*vide* Experiment 12), was introduced on the point of a lancet beneath the skin of B. 1. By oversight, next day B. 1 was injected with human saliva from a fever patient (first interval), and immediately high pyrexia set in which was not of the spirillar character. This experiment, therefore, was a complicated one, and it shows only that one kind of blood contamination is capable of overcoming or preventing another. (*Vide* also No. 20.)

Twenty-fifth experiment.—From R. T. 2 (*vide* No. 13), whilst the temperature, though rising, was still not in excess of the normal, a minute quantity of blood containing several active spirilla was taken, and on the point of a lancet introduced beneath the skin of the thigh of B. B.

This animal had been previously the subject of experiment, but was now in very good health. No result of marked or specific character followed during the next eight days, and why this was so did not appear; perhaps there was some defect in the manipulation, or the blood of R. T. may have been in a peculiar condition, for brief and high fever came on in six hours, and then the spirilla disappeared.

During the Earlier Incubation Period.

Twenty-sixth experiment. — Twenty-four hours after inoculation of the animal mentioned in No. 3, and whilst the temperature and blood were seemingly normal, two half-drops of blood were taken, and on the point of a lancet introduced beneath the skin of both thighs of B. 2, a healthy monkey, though troubled with sores on the body which made it fretful. The temperature rose on the next and succeeding two days, but was within normal limits for the following six; and the blood regularly examined never showed any sign of spirillar infection. Here inoculation with blood at early incubation stage failed to act, whilst in Experiment 17 it seemed to be efficient.

This essay forms a transition to another negative series of attempts I next proceed briefly to narrate.

B. Series 3.—*Experiments with Non-spirillar Blood, with Dried Spirillar Blood, and with Saliva.*

To test further the validity of the instances of successful inoculation, I employed, in the common method, and upon similar subjects, the blood taken at the "fall" or crisis of the spirillar attack, also at dates preceding the expected "relapse," and, finally, during the secondary fever or "rebound" which sometimes follows specific pyrexia.

Seeing that a spirillar organism similar to that detected in the blood is to be found in the saliva or fluids of the mouth, I also made use of this sputum as material for

injection, taken from both febrile and non-febrile subjects.

Lastly, blood that had been dessicated was employed in other experiments, with a view of ascertaining whether or not the blood containing spirilla or their germs (supposed) was infective after being dried up.

Blood at "Fall."—Man.

Twenty-seventh experiment.—S. B. (*vide* No. 9), at the close of first attack, presented characteristic symptoms with a critical fall so prolonged as to resemble decline by "lysis." Near the end the morning blood (temp. 101·6°) contained many active spirilla, whilst that of the evening (temp. 98·8°) was absolutely free from the parasite, and served for the following operation.

2. R. T. 3, a new and healthy monkey with normal blood, had ten minims of the above, defibrinated, hypodermically injected in the thigh; next day the temperature rose to 104·2°, and declined to 99·8° on the following morning; afterwards it was tolerably level, subsiding slightly for the nine days of observation; the animal appeared to be unaffected. The blood was carefully examined, and the result was negative. The rise of temperature on the second day was attributable to irritability of the subject, which for a few hours once escaped custody altogether.

Monkey.

Twenty-eighth experiment.—R. T. had a well-defined attack of spirillum fever (*vide* No. 14) terminating with a prompt fall (temp. 100·6°), three hours after which it was killed and furnished blood at this time with clouded plasma, partial fibrillation, few white cells, little protoplasm, large red discs normal and no movements.

2. Chain 2, a fresh restive animal, had twenty minims of the above, defibrinated, injected into the thigh (temp. 102·6°), blood normal. The temperature rose in the night

but speedily subsided, and thence maintained a uniform course till the seventh day, when there occurred a brief febrile paroxysm; afterwards it was level till the sixteenth day. The blood was repeatedly examined with negative results; the exacerbation noted seemed clearly attributable to irritation of the chain, ending in an ulcer, and it was neither attended nor preceded by specific changes in the blood.

So far as they go, these two experiments are decisive as regards the non-infecting properties of the blood taken at or immediately after the crisis of an attack of spirillum fever.

Blood at "Rebound."—Monkey.

Twenty-ninth experiment.—B. T. 2, of trial No. 16, after a well-defined attack of specific fever, and at the fall a pause of three hours, had a sharp rebound of temperature from 102° to 106·6°; the animal was then very ill, and eventually it died. The state of the blood at the onset of this secondary fever was as follows:—Plasma clouded, fibrillation partial, much protoplasm, some large granule cells, several white cells, red discs heaped, shrunk, and blended, no spirilla.

2. W. T., a healthy animal with normal blood was inoculated in both thighs with the dark, thin blood of B. T. 2, as above; temp. 102·4°, rising towards the close of the second day but subsiding on the third and fourth, and again rising on the fifth and sixth. The blood was repeatedly examined and always with negative results, and the experiment shows the non-infecting character of blood taken after the specific fever, when even more pronounced secondary fever occurs. I consider, also, that the nature of this rebound is indicated by the present trial, in so far as its relationship to septicæmia or other special blood contamination is concerned, for the effect of injection was here nil. W. T. was subsequently inoculated with success (*vide* No. 2).

Blood during the Apyretic interval, prior to expected Relapse.—Man.

Thirtieth and thirty-first experiments.—A., a man, admitted with high fever (specific), which, according to his account, was a first attack, displayed the usual level apyretic stage following the crisis, and on the third day his temperature was 98·4°, and his blood free from any signs of the parasite.

Ten minims after defibrination were injected into the thigh of a monkey (Chain 2, of No. 28), temp. 102°; next day there was a rise, on the third none, and on the fourth a smart paroxysm (temp. 104·8°); afterwards the temperature continued to be level for ten days. The blood was frequently examined and nothing abnormal was detected in it, excepting on the morning of the fourth day, just before the paroxysm above alluded to, when small curved filaments were seen, which at the time I regarded as resembling imperfect spirilla; from subsequent experience, I should not lay any stress upon such appearances, unless in the same specimen undoubted parasitic organisms were also to be found.

On the sixth morning after the fall, the temperature of A. was 97·4°, and blood only rather clouded.

A second fresh monkey (B. 1, of No. 24), with normal blood and temperature 101·4°, was injected similarly to the first; slight exacerbations of temperature followed on the first, second, and fourth days, and on the eighth the observations were closed, all having been negative in results, including the frequent scrutinies of the blood.

The man himself had no relapse, nor did his blood display any marked changes at the dates when the second attack usually supervenes.

Thirty-second, thirty-third, and thirty-fourth experiments.—The patient named in No. 9 had quite rallied after the invasion attack, and showed a level (temp. 97° to 98°), and somewhat prolonged first apyretic interval. In anticipation of the relapse, I abstracted and used for

inoculation blood drawn on the seventh, eighth, ninth, and tenth days after the main fall and disappearance of the parasite. The same process was followed on each occasion, the morning blood being taken, promptly defibrinated, and about ten minims injected subcutaneously into the thigh of a healthy monkey. The four animals were then kept apart, the temperature every three hours taken and recorded, and their blood scrutinised twice daily for ten days.

The result was in every instance negative, the temperature charts showing nothing peculiar and the blood being unchanged. That there was no want of susceptibility in three of the animals is apparent from two of them being afterwards successfully inoculated, and the third is that case described in Experiment 13, where contagion probably occurred; the fourth monkey, however, seemed to be impervious to infection (*vide* Y. T. 2., in No. 23).

This series is continuous with that narrated under Nos. 21 and 22, which was concerned with the onset of the relapse, and furnished similarly negative results, whilst No. 9 shows successful inoculation with blood taken during the recurrent attack. I consider Experiments 30, 31, 32, 33, and 34, prove that the non-spirillar blood of the apyretic interval in man is incapable of conveying infection.

c. Series 4.—*Experiments with Dried Blood.*

At the late period in the epidemic at which my inquiries began, the supply of infecting material from hospital patients was only intermittent, and as the source from animals was necessarily of brief duration, I had early to consider if spirillar blood might not display its characteristic property in the dried as well as in the fresh state. Several specimens of blood proved by experiment to be infective, were accordingly dessicated with care in thin layers, either simply under cover at temperature of the air (about 80° F.), or over sulphuric acid, or over caustic lime, and some of these attempts at preservation were so far successful that the dried blood was found by the

microscope to show, when moistened with distilled water, the parasitic organisms hardly altered in aspect, and but little intermixed with new growths. This material suspended in water was freely used, and I also added in two instances some acetic acid with a view of better liberating the spirilla or their germs from the albuminous substance in which they were embedded. These essays to conserve the blood intact without the aid of antiseptics (the use of which seemed undesirable) have all failed; they form the Series No. 35 to 42 inclusive.

Thirty-fifth experiment.—Blood taken at the crisis of a well-marked first attack in a man, dried in vacuo, and hermetically sealed was, after an interval of eight months, inoculated in B. T. monkey, about a grain's weight being used; the result was negative.

Thirty-sixth experiment.—Blood taken on third day of first relapse, dried under cover, and kept for five days only, was inoculated in C. monkey. The temperature rose for the first three days, and then became normal; there was no visible change in the blood of the animal. The same material produced no effect in two men who had small quantities placed under the skin. In both the above instances the monkeys readily acquired the fever upon inoculation with fresh blood.

Thirty-seventh experiment.—The blood of W. T. monkey, known to be highly infective, was dried over sulphuric acid, and two months afterwards employed on B. B. When moistened with water some bacteria were found, but the spirilla were numerous, and on the addition of acetic acid appeared to be unchanged. The softened fragment and drop of fluid were introduced beneath the skin of the thigh, the temperature was taken, and blood examined with care for twelve days in succession, and there was no sign of illness or spirillar contamination.

Thirty-eighth and thirty-ninth experiments.—The blood of man (first relapse) and of a monkey, known by experiment to be infective, was dried without undergoing any apparent change, and nearly two months afterwards

inoculated in two monkeys, which never showed any evidence of being affected thereby. In the man's blood no trace of the spirillum was detected after desiccation, but that of the monkey showed them distinctly. Bacteria were present in a dried condition, and these produced no effect on inoculation.

Fortieth and forty-first experiments.—The same materials were moistened with acetic acid, and so rendered soft and translucent; they were then used for inoculation in two fresh monkeys, and still without result.

These failures contrast with the success so readily obtained with the preserved blood of animals affected with the so-called splenic fever; and they serve to show that the Spirochæte possesses more delicate physiological properties than the Bacillus.

D. Series 5.—*Inoculations with Saliva.*

The following experiments display a notable contrast with the results of the previous successful inoculations:

Forty-second experiment.—About the same time as the blood was employed of the patient No. 1, I made use of his saliva for injection. High fever being present the fluids of the mouth were scanty, the saliva had a milky aspect and gave a whitish sediment; it contained large epithelial scales, much granular matter, and many active bacterioid forms, including the spirillum. As contrasted with the parasite in the blood, this of the saliva was larger or thicker and more sluggish, never twisting or lashing into knots and rings, and never clustering together; perhaps the more tenacious medium was concerned here. I observe that the Spirochæte proper of Ehrenberg, as I found it in tank water, is much larger than either of the above, and always preserves its spiral contour in movements, however active; whereas the organisms in man straighten out in their contortions and display the screw-like form only when nearly or quite quiescent.

About twenty minims of the above fluid were hypo-

dermically injected in the thighs of two fresh and healthy monkeys.

2. B. (temp. 102·6°). Next day the animal was ailing and the temperature had risen to 104·5°, remitting but slightly; on the third day of the fever it remained high (105·4°) and nearly continuous, and death took place early on the fourth. The blood was quite free from specific contamination throughout the attack. At first, coagulation was slow, and small protoplasmic masses were seen; towards the end there were large granular protoplasmic masses, with plasma clear in the morning and faintly granular in the evening, with little protoplasm and few white cells, red discs shrunk and blending; no moving particles of any kind were detected.

The animal was evidently poisoned by the saliva injected, yet it did not present the same aspect as seen in spirillum fever. There was inability to sit up and the head did not droop low, but was leaned against the wall; attempts to eat were shown, which were ineffectual through debility, as it seemed, and finally, there were signs of paralysis on the left side of the body. After death, meningeal hæmorrhage was found over the right hemiphere of the brain, and there was no inflammation of the small intestines, such as I found in the body of a monkey dying on the same day of spirillum fever.

3. S. Temp. 101·2°, rising next day to 104·6°; the fever being continuous it was 104·8° on the third day, and 105·4° on the fourth. Slightly remitting, it rose to 105·6° on the fifth day, and further remitting, attained the same elevation on the sixth; on the seventh day there was a complete intermission, with an evening paroxysm (105·6°); on the eighth a remission at first, and then an intermission (temp. 101·6°), followed by exacerbation on the ninth (105·8°); again a decline and rise, but now not so high (temp. 104°), and the animal sank early on the eleventh day of experiment. At first the blood resembled that of B., then it became clear and showed small clumps of protoplasm, the red discs being unchanged; on the fourth day there was

nothing peculiar visible, white cells were few, and so next day; on the sixth sloughing of the nates on the right side had began, yet the blood was not altered in aspect; in the intervals of pyretic abatement the monkey rallied a little, but gradually became exhausted by repetition of fever and extension of the local necrosis. At the autopsy no striking lesion was apparent.

This animal also died from the effects of the inoculation, but more indirectly; probably plugging of the vessels had occurred in both animals. Any influence which the spirilla in the saliva may be supposed likely to exert when introduced into the blood was clearly overcome by other toxic agents, of which more than one was doubtless present in the very composite sputum.

Forty-third experiment.—A patient, who had a severe attack of relapsing fever (*vide* No. 3) on the second day of the first interval, was rallying from the "fall" after invasion, and furnished saliva which contained epithelium, bacteria forms, granules, and *débris*, but no spirillum was seen. A small quantity was injected in the usual manner.

2. B. 1, a healthy monkey, temp. 101·8°. After twelve hours, fever set in, which was high (105·6°) and of intermitting character for the first three days, and then became continuous at equal elevation for six days. On the tenth day there was a decline, followed by a sharp rebound (temp. 106°), after which the temperature slowly declined to a sub-normal level in the course of four or five days more. The blood had a dark hue; it was regularly inspected throughout this prolonged attack, and never showed any signs of the spirillum or other forms of bacteria, even after treatment with acetic acid; its characters varied only within quasi-normal limits. The animal suffered considerably, and at first the most. It became prostrate on the second day and lied down, refusing food, the body hot, and the limbs cold, and the left arm seemingly powerless. When the paroxysms ceased and pyrexia became continuous, the monkey seemed to rally, sitting up and looking about; it grew thinner and weaker, how-

ever, and did not attempt to eat. Presently, and before fever had ceased, improvement began, and at last convalescence was prompt; the rebound did not appear to entail additional suffering; the palsy of the left arm was persistent.

By the only mishap which occurred in my experiments this animal had been injected with spirillar blood the day before inoculation with the saliva. I do not think the prior injection had any considerable influence upon the results noted, which were similar in character to those of the preceding experiment, No. 42. B. 1 was shortly afterwards again inoculated with spirillar blood, and the successful result is described under Experiment 8.

Forty-fourth experiment.—The saliva of a perfectly healthy man contained epithelial scales, micrococci, bacteria, vibrios, and granular matter; spirilla were not detected in the fresh state, but after treatment with acetic acid, wavy filaments were seen, which had all the aspects of them. Ten minims of this fluid were injected in the usual manner and place.

2. B. B., a monkey in healthy condition, though a few weeks before the subject of spirillum fever, showed on the day following some inflammation of the thigh, and a small abscess at the site of injection three days later. There was no fever (maximum temperature on fourth day, 103·2°), or only a slight exaggeration of the normal daily cycle, with a tendency to decline below the mean, and the blood was unchanged on the first four days; convalescence was complete.

The above experiments indicate the presence of a toxic agent in saliva which is not the spirillar, and should the latter exist, it seems to be overcome by the former, whatever its nature. Healthy saliva is irritant, and during and after the spirillum fever this quality is greatly intensified, so that even death may ensue from the inoculation of febrile sputum. Germs of the mouth-parasite are not developed within the blood of an animal susceptible of direct contamination from the blood of another infected animal.

CHARTS

Illustrating Dr. V. Carter's Inoculation Experiments.

Series 1.—*Relapsing Fever* (spirillum fever) *in Man.*

(In Diagrams I to IV.)

The temperatures are axillary and shown in the usual manner.

The state of the blood is indicated by the signs + and −, signifying respectively the presence and the absence of the spirillum at the time of examination.

By the sign ± is meant the apparent absence of the parasite in fresh blood, and its subsequent detection after drying of the specimen and treatment with acetic acid.

N.B.—These charts will also serve to show the identity of the fever as seen in Bombay, with its European forms.

Series 2.—*Artificially induced Fever in the Monkey.*

(In Diagrams V to VIII.)

The temperatures are axillary, and are given for every three hours of the day.

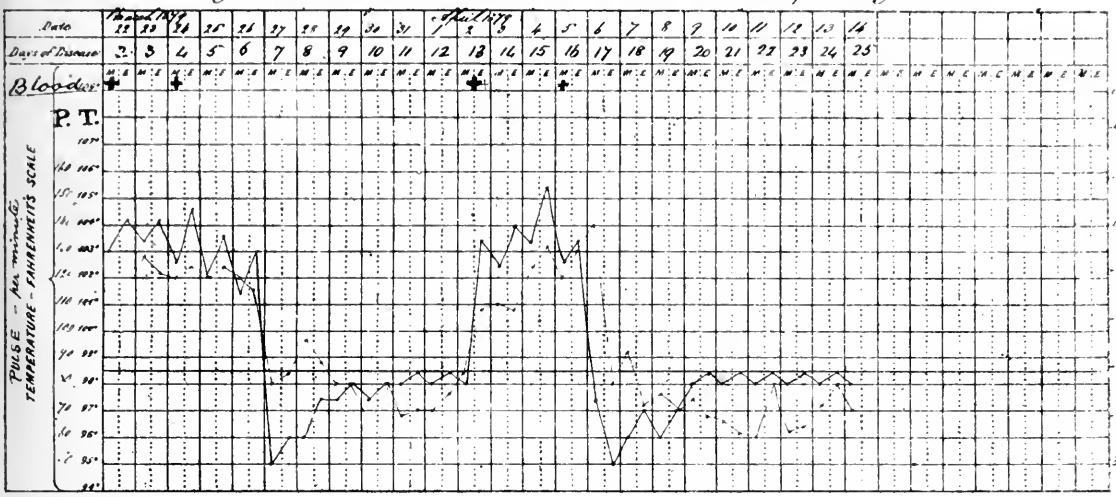
The state of the blood is shown by the signs + and −, which respectively signify the presence and the absence of the spirillum at the time of examination.

The sign ± means spirillum not seen in fresh blood, but seen after drying and the application of strong acetic acid (Albrecht's process).

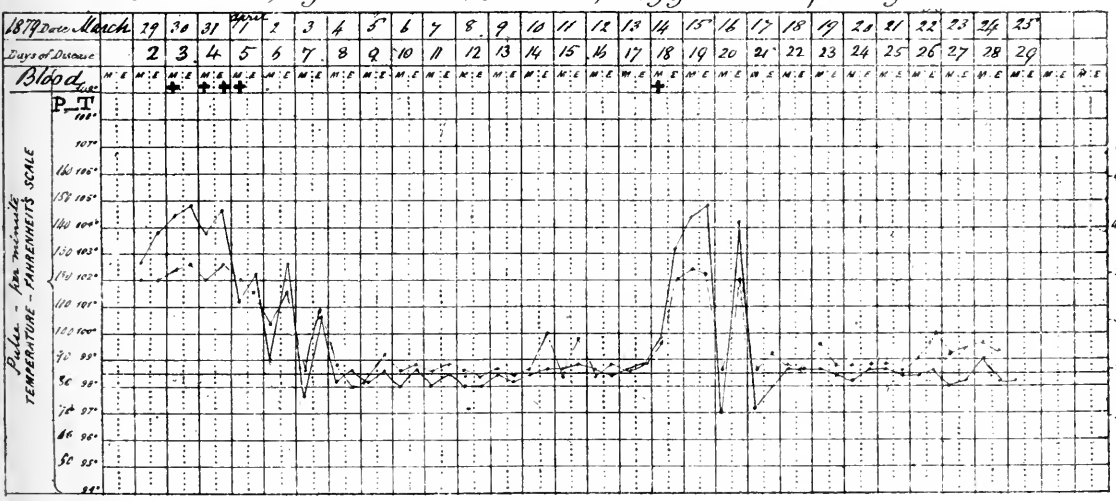
? Indicates appearances not wholly affirmative.

N.B.—The dotted line immediately above that of 101° F., represents the mean normal temperature, viz. 101·3° F.

Diagram I. Series I. Experiment 1.
N.P. age 28, Hindoo, Ward attendant, Relapsing Fever.



Experiment 2.
H.S. Female, age 60 Mussulman, Beggar. Relapsing Fever.



Experiments 3, 11, 43.
E.S. age 25, Mussulman, Coal Trimmer. Relapsing Fever.

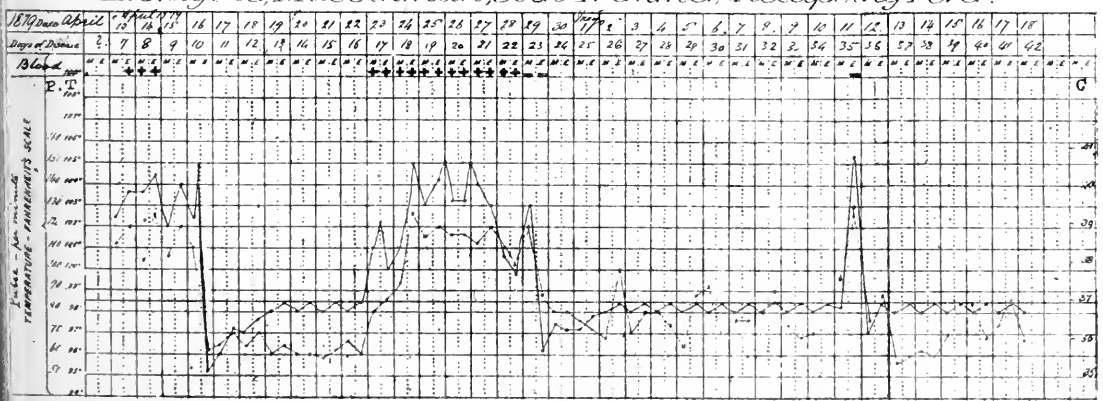
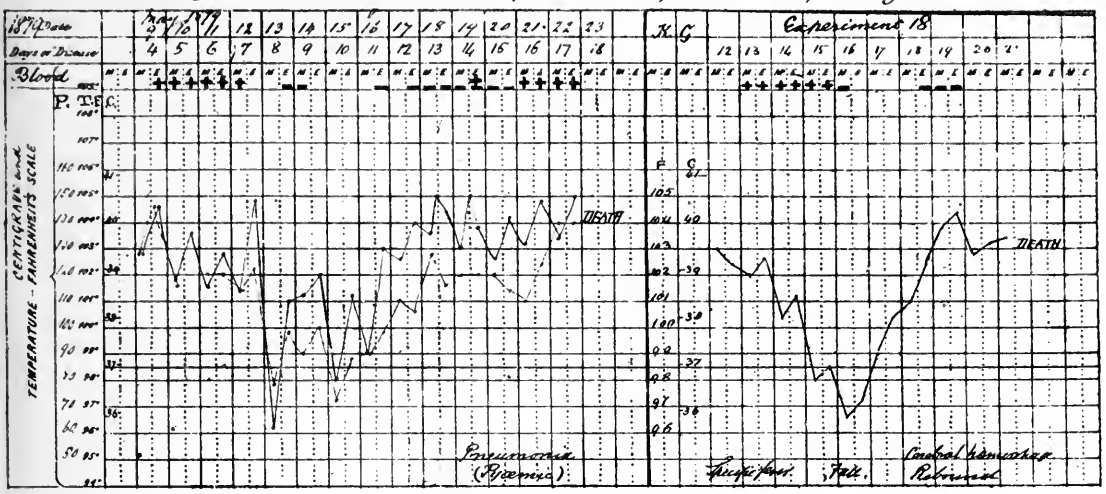
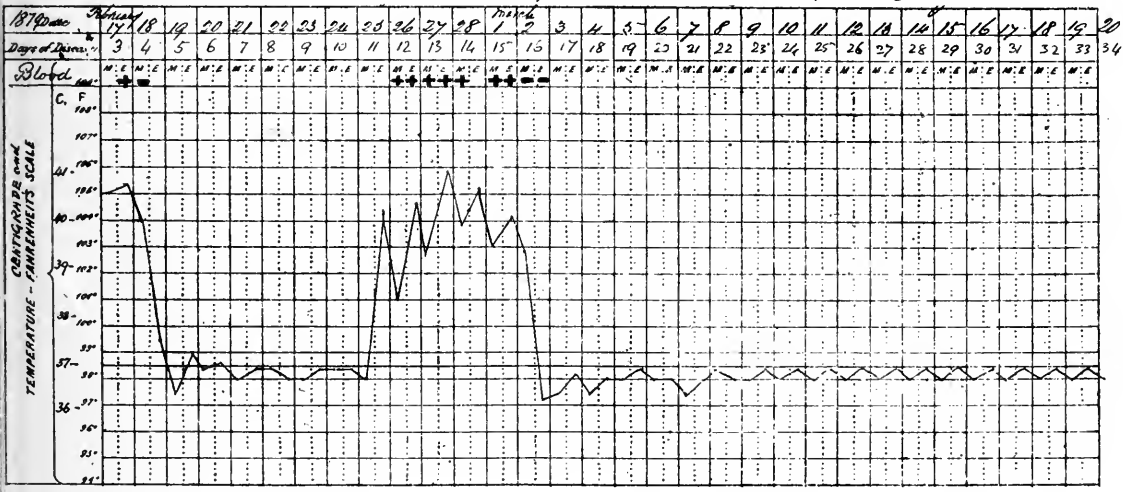


Diagram II. Series 1. Experiments 4, 20.
H.T. age 22 Hindoo, Municipal Sweeper; Relapsing Fever.



Experiments 5, 19.
B.M. age 30, Mussulman, Ship Labourer Relapsing Fever.



Experiment 6.
I.S.B. age 14, Mussulman, Coolie Labourer, Relapsing Fever.

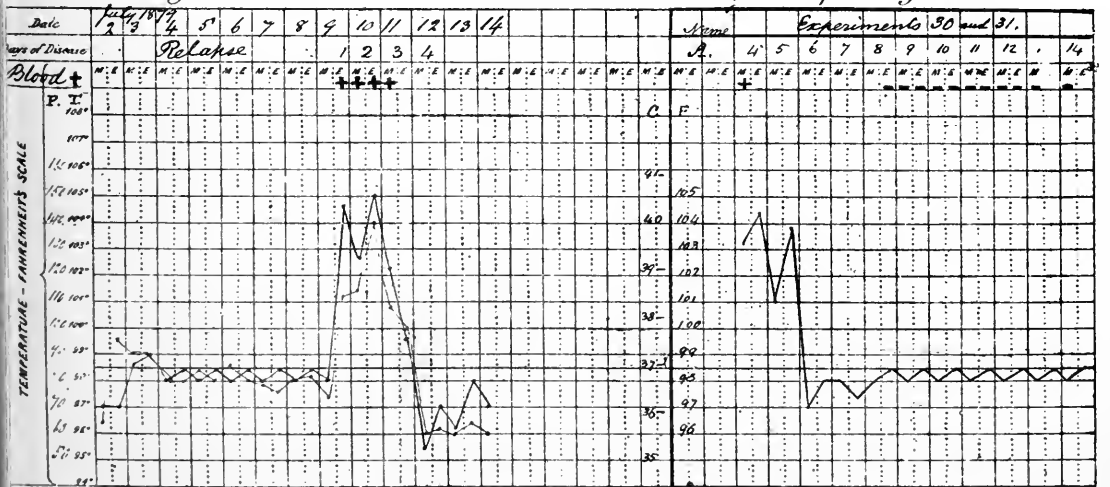
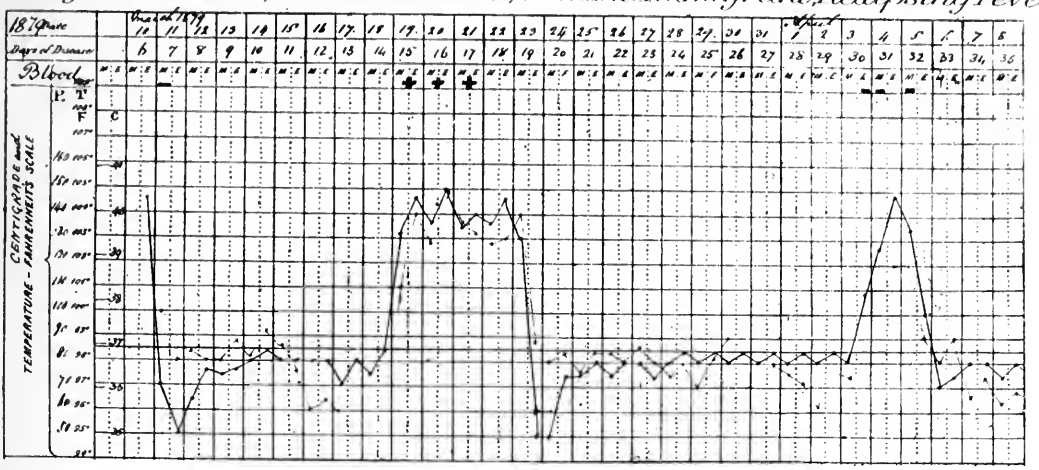
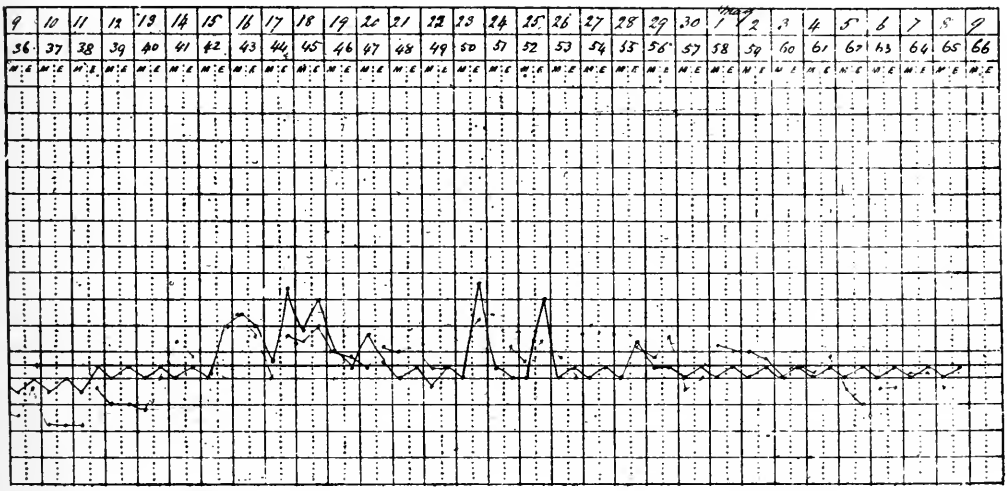


Diagram III. Series 1. Experiment 7.
B.L. age 34. Hindoo, Coolie Labourer; Famine Immigrant, Relapsing Fever.



Experiment 7 continued.



Experiment 8.
F. J. age 28, Mussulman, Ship Labourer; Relapsing Fever.

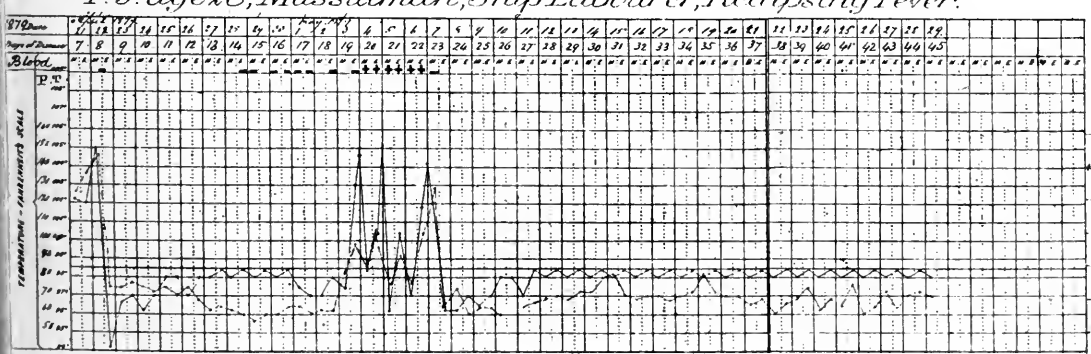
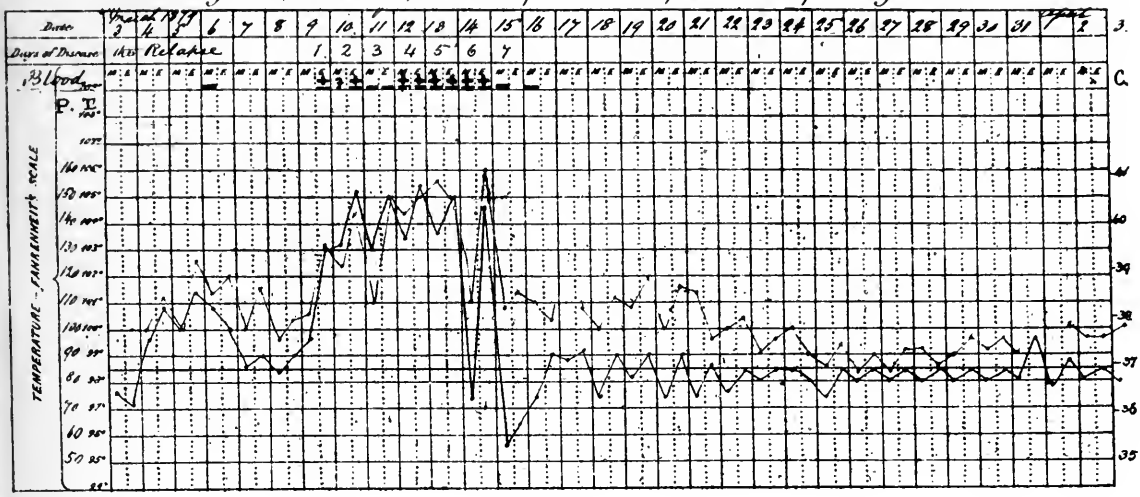
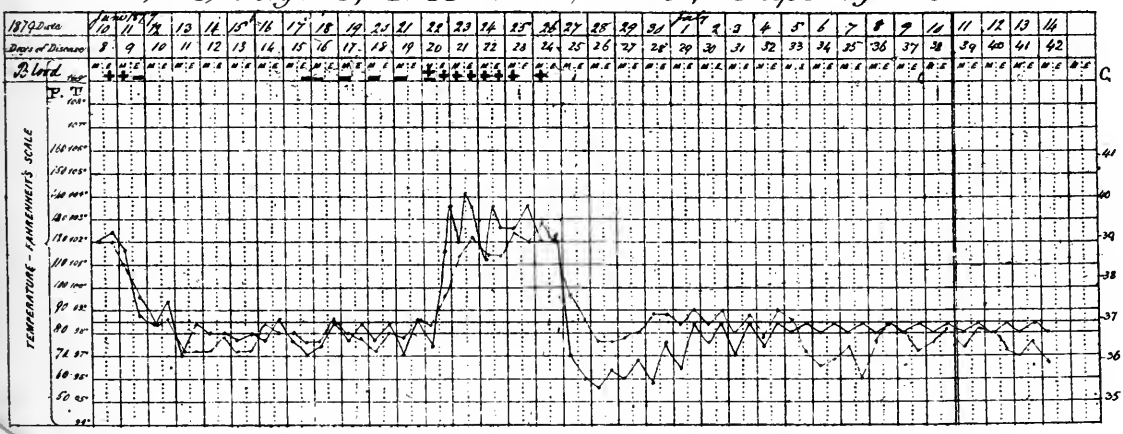


Diagram IV. Series I. Experiment 10.
R.Y. age 24, Hindoo, Municipal Sweeper; Relapsing Fever.



Experiments 9, 13, 21, 22, 27, 32, 33, 34.
S.B. S.R. age 45, Mussulman, Weaver; Relapsing Fever.



Experiment 12.
R.M. age 30, Mussulman, Labourer; Relapsing Fever.

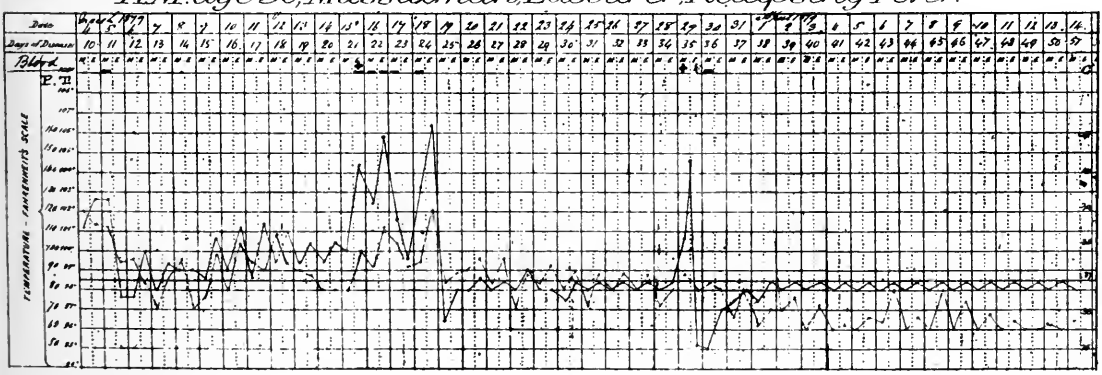


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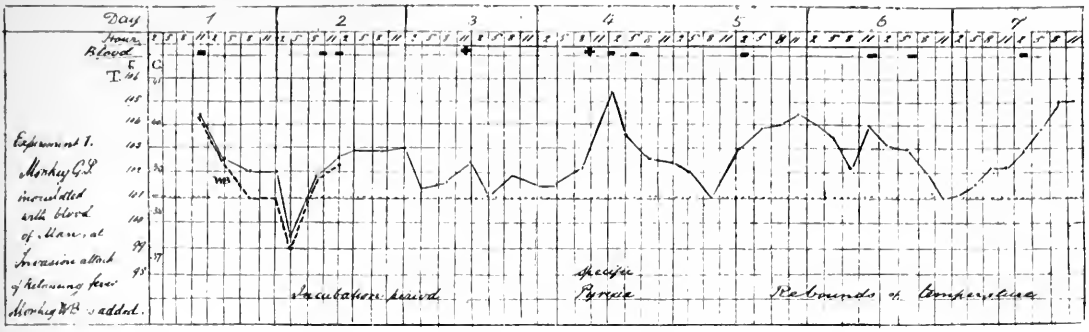
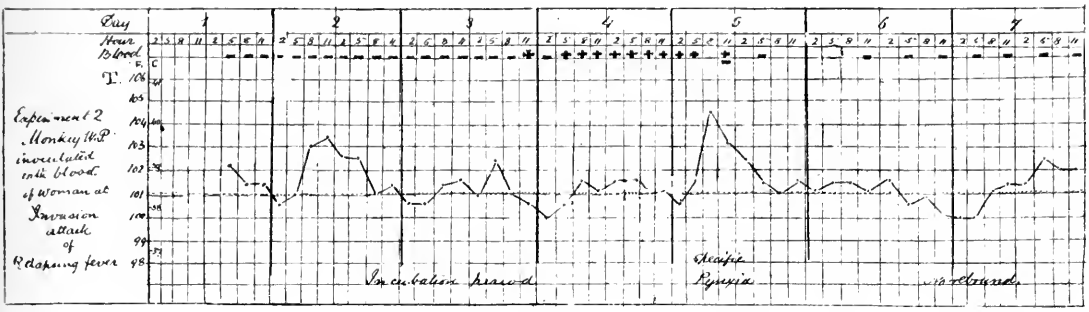
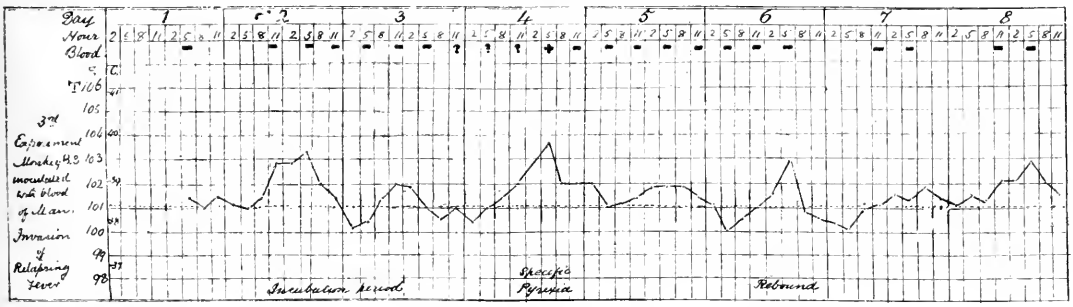
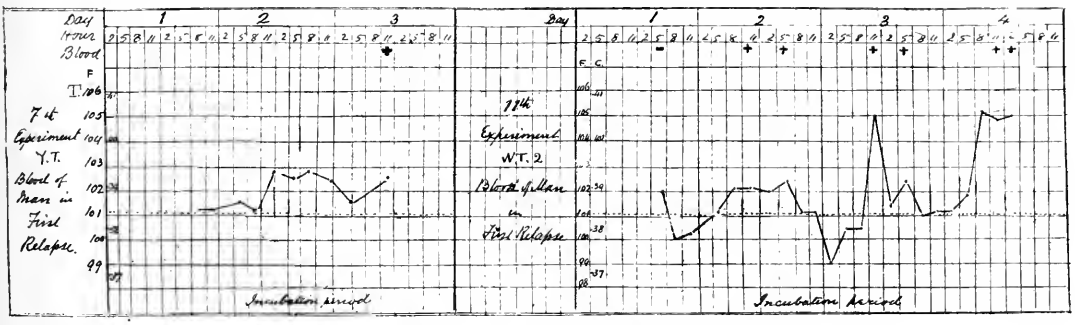
Diagram V: Series 2. Experiment 1, 2. Monkey G P, W B, & W F.





Experiment 3, 7, 11, Monkey, B, 3, Y T, & W T. 2.





Experiment 6, Monkey, 1, Chain.

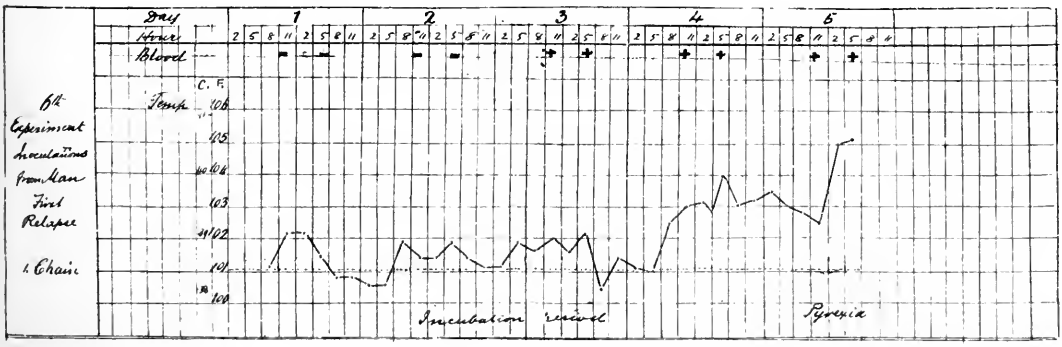
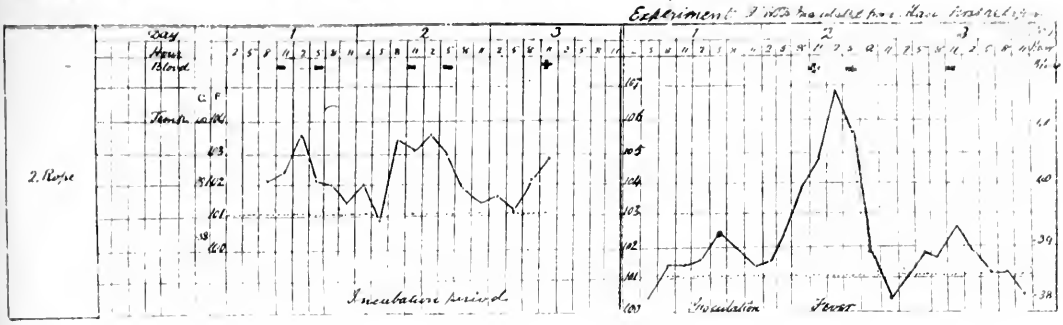
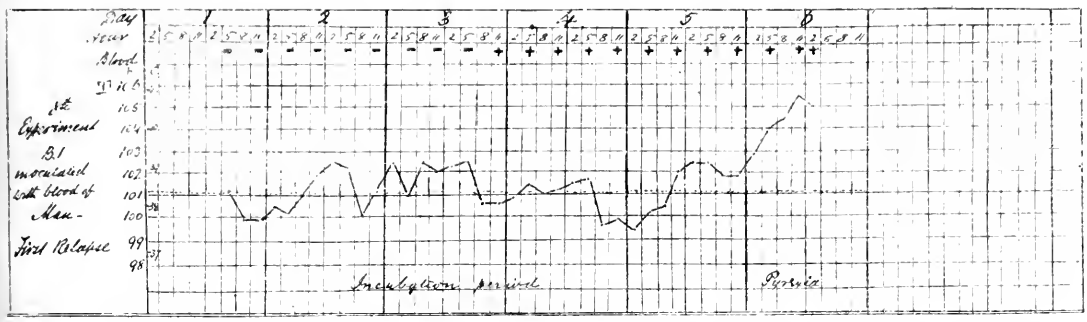
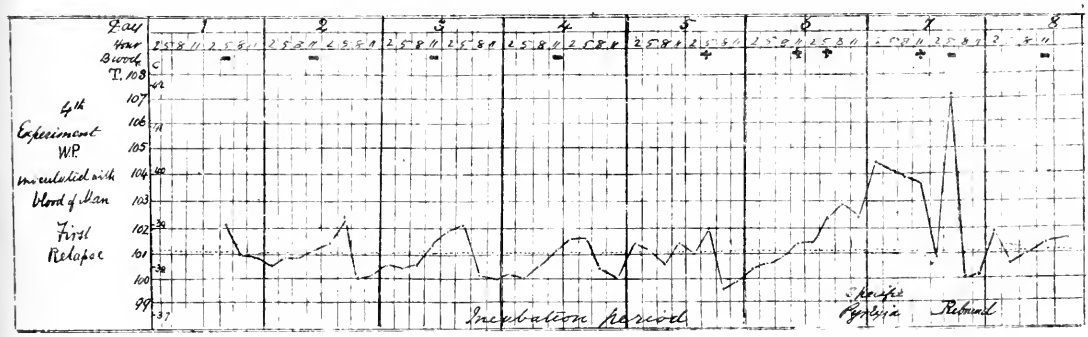


Diagram VI. Series 2. Experiment 2, Rope, 9, WT, 3.



Experiment 8, B4, WP





Experiment 12, BB.

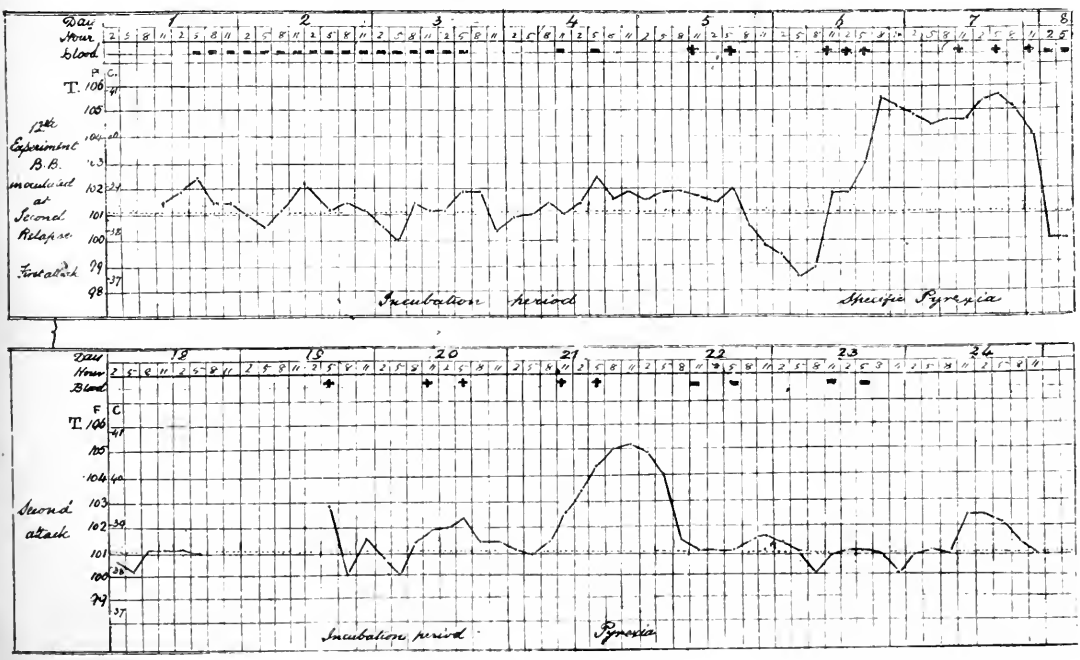
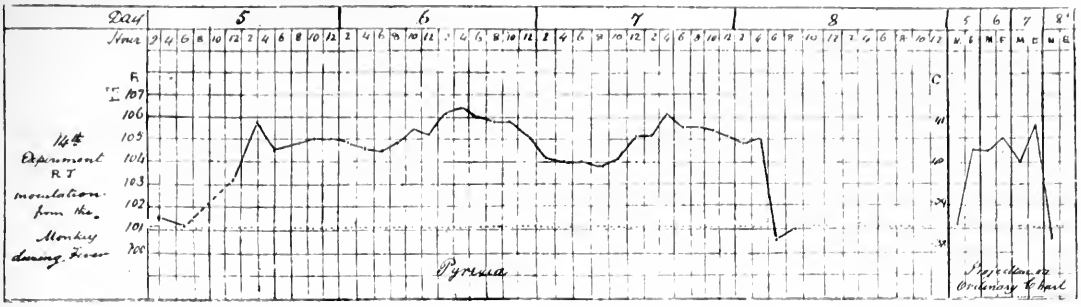
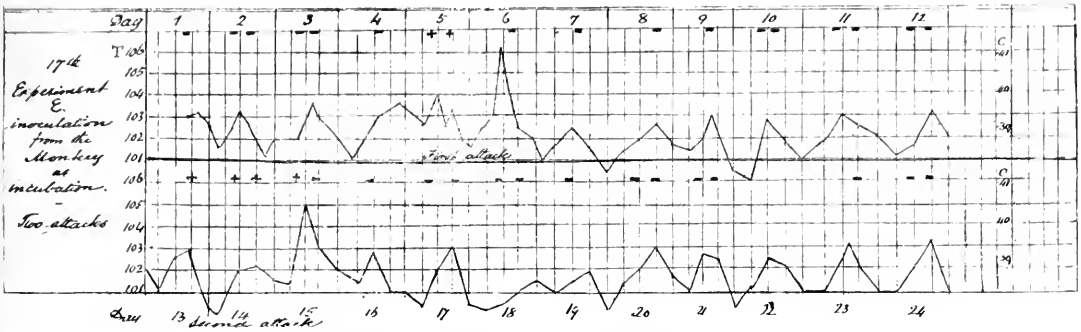
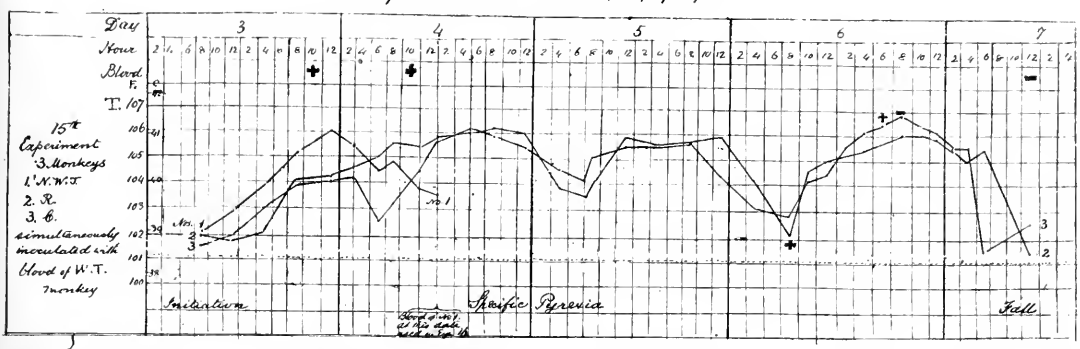


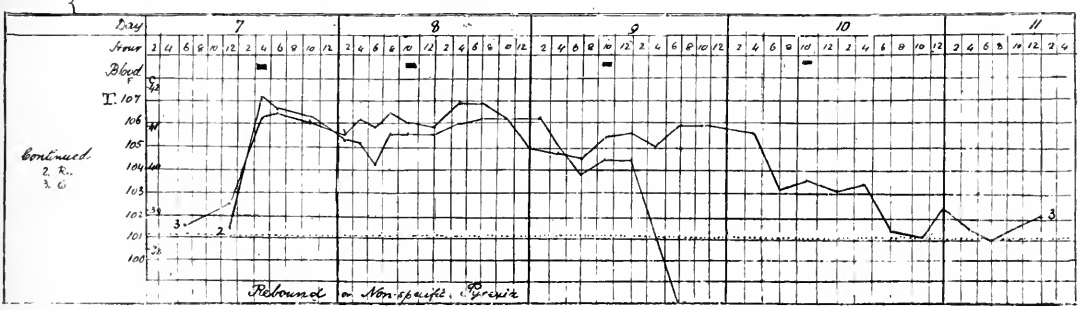
Diagram VII. Series 2. Experiment 14, R.T, 17, E





Experiment 15. N, W, T, R, & C.





Experiment 16. B.T, & O.T.

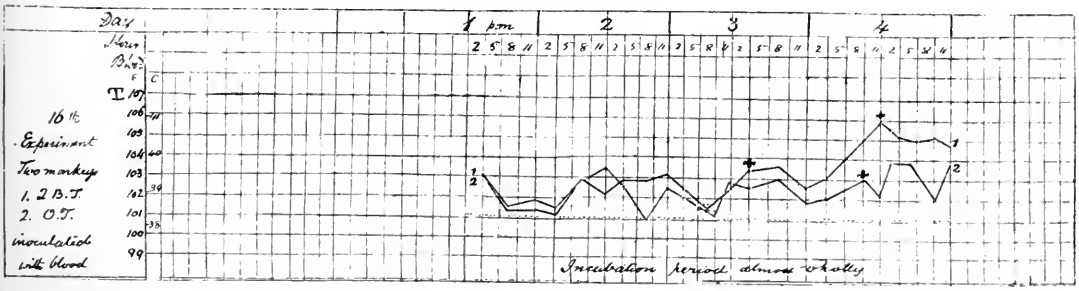
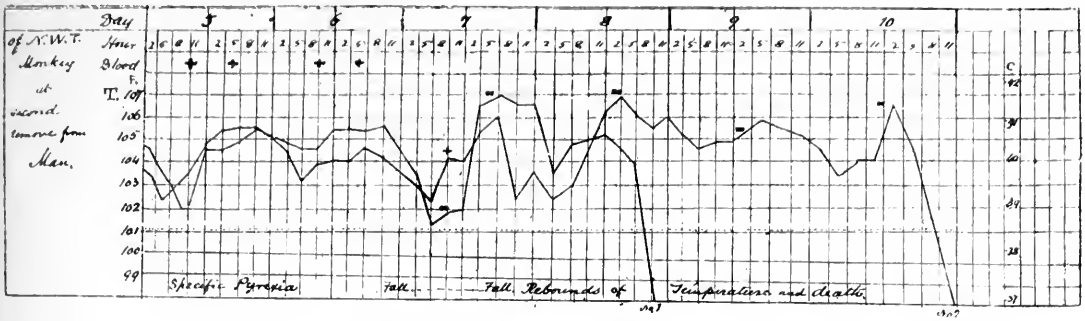
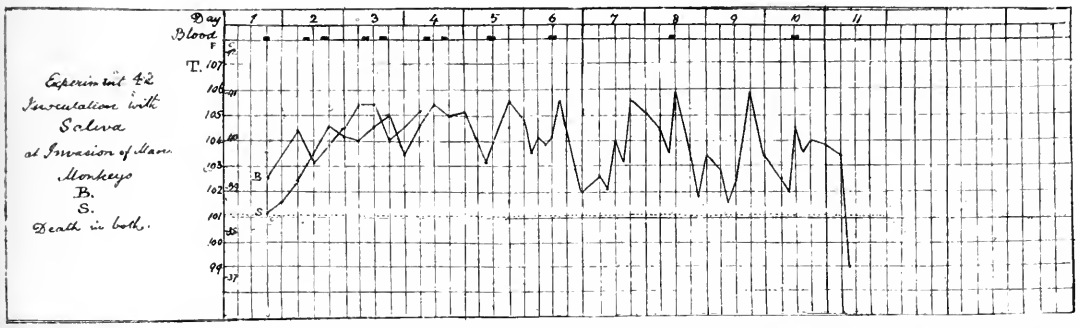
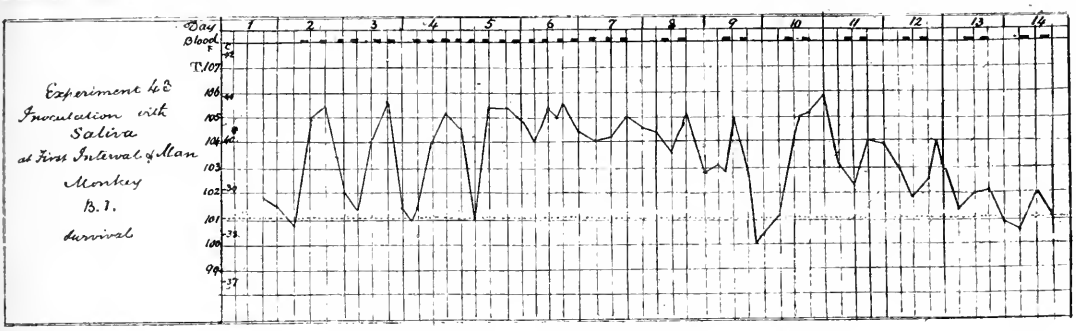


Diagram VIII. Series 2. Experiment 16, continued.

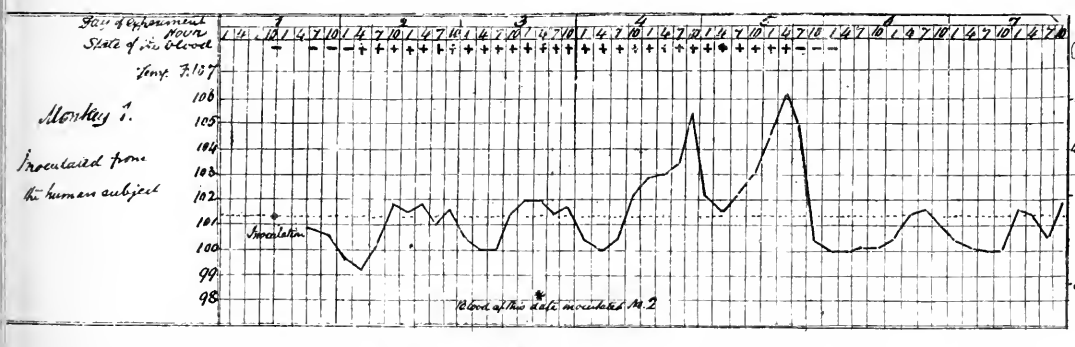


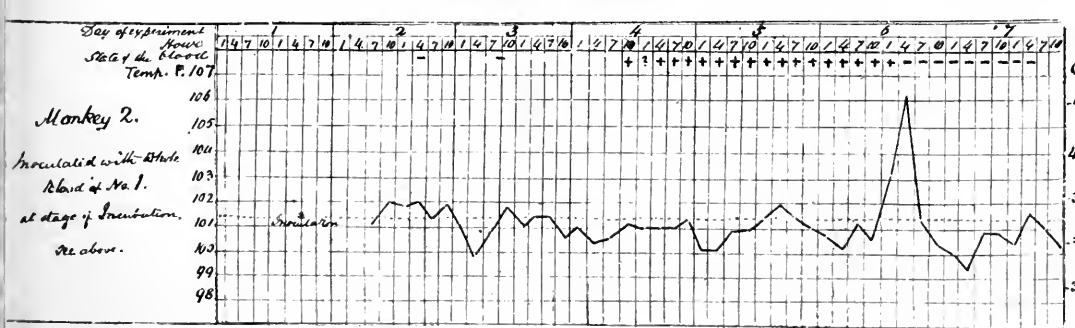
Experiment 42. B,S; 43,B1





Appendix. Monkey 1 & 2.





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INDEX TO EXPERIMENTS.

PART II.—COMMENTARY.

The above forty-four experiments were made with material derived from man (in thirty-one instances) and from the monkey (in thirteen), upon fifty-one of the lower animals. Serious attempts upon the human subject become unnecessary in the light afforded by my comparative data, for I presume there is no doubt that the blood of an individual suffering from relapsing fever is capable of communicating the same disease to an unaffected subject, just as it does to the healthy quadrumana, and also to these animals amongst themselves.

The chief group embraces the essays made with blood containing the spirillum. Of these there were twenty-one inoculations from man with six failures (*i.e.* 1 to 3·5), and ten from the monkey with three failures (*i.e.* 1 to 3·3), or more properly nine with two; the total is thirty-one with nine failures. Even thus crudely stated, the preponderance of successful inoculations is so decided as to render it certain that the spirillum fever is even readily communicated from man to the lower animals, and between the latter themselves; and this being so, the exceptions to the rule claim special notice, not only from their intrinsic interest, but from that bearing upon a series of complementary experiments, which were made with a view of testing the positive results. The significance of this further series of nineteen essays giving negative results, would be much modified were it established that infective material was commonly or even frequently inoperative, and I will, therefore, at once point out the circumstances under which the somewhat unlooked-for failures occurred, with the inferences that may hence be drawn.

A. *Negative Series.*

Remarks on the second series, Nos. 18 to 25 inclusive.—Whilst under most conditions spirillar blood is capable of conveying infection, the following states seem unfavorable to this result :—A period prior to the onset of fever (No. 25), or at the very beginning of high fever (Nos. 21, 22, 23), when I may here observe the parasite will not grow readily under artificial cultivation ; again, at the acme of attack with high temperatures (No. 19), or at the very end (No. 18), when also cultivation fails. The present series shows, too, that when pyæmia is present in conjunction with spirillar infection, the blood conveys not the latter, but a contamination possibly like the former (No. 20), and, finally, if a second poison (*e.g.* of saliva) be injected immediately after the spirillar, it overcomes or annuls the latter (No. 24). Upon review, therefore, these anomalous instances appear not inexplicable, and, further, they become highly suggestive.

Remarks on some other discrepancies.—Experiment 17 showed that blood at early or non-spirillar incubation stage may convey infection, whilst No. 26 displayed only negative results in a similar trial. There is also to note the discordance of Experiment 13 with Nos. 32, 33, and 34, for as they stand these data show that the blood of man and animal, during the ante-febrile stage, may or may not convey infection. When all the evidence is summed up, however, the balance is decidedly against the likelihood of the non-febrile incubative period being an infective one ; and of the two instances to the contrary just named, in one (No. 13) there seemed an inferential probability of contagion, and in the other (No. 17) the monkey appeared unusually susceptible, for it presented a "relapse," and this, at least, may have been incidental. I would further remark that the advent of the spirillum during the pre febrile stage being intermittent, it seems

possible that an incubation experiment may succeed at one hour and not at another during this period.

Remarks on contagion as a disturbing influence in these experiments.—The possibility of an affected animal communicating its disease to another by means of contact was early recognised, and I made some trials to test this with negative results. Contagion was never actually proved, in so far that no animal not operated on ever showed the specific fever; possibly the hairy integument was a protection more or less complete, yet bearing in mind the case in man, I do not deny that amongst the lower animals contagion may have occurred in spite of some measures taken to prevent it, and it is not impossible that the anomalous instances of Nos. 9 (quasi-immediate infection), 13 (infection at first interval), 12, and 17 (relapses), may owe their peculiar features to that agency; for this reason, which is only inferential, they will not be insisted on in the final summary. In the great majority of successful inoculations there was so much concordance in time, that I do not hesitate to exclude the influence of contagion. Whether or not the monkey could acquire the fever by simple contact with human patients is not known; none of the persons handling the sick animals were attacked.

Remarks on the negative Series 3 *and* 4.—In Series 3 are nine experiments with non-spirillar blood, showing no ill effects, namely, one with blood at period of incubation (No. 26), two with blood at or just after the "fall" (Nos. 27 and 28), one with blood of "rebound" (No. 29); also two with blood of man in "first interval" when no relapse occurred (Nos. 30 and 31), and three with human blood in "first interval with relapse" following (Nos. 32 33, and 34). Upon consideration I am disposed to consider these instances as valid evidence, that in the absence of the blood-parasite inoculations fail; and I add that under the test of artificial cultivation the same non-spirillar blood never evolved the specific parasite.

The negative effect of inoculation with dried blood

taken during fever or at the fall, I am disposed to consider as fully settled; and some other means than desiccation is needed to preserve intact the contagious property of infected blood. This bears on the *rôle* of fomites.

As regards Series 5, it seems equally obvious that the spirillum in the sputum does not, upon injection of the saliva entire, induce the specific fever; whether or not it would do so in an isolated state cannot well be known. The different effects of febrile and non-febrile saliva, though structurally alike in abundance of bacteria, &c., is worthy of notice.

In bringing these preliminary remarks to a close, I will only observe that the discordant results elicited in my inquiries produced some surprise until it was considered that the propagation of infective diseases in man is by no means uniform, invariable, or comprehensible as regards exceptions to the rule. These experiments show that (like ordinary typhus) the spirillum fever is not conveyed at all stages or upon all occasions, and they point to some of the exceptional conditions, which I need not recapitulate. Supposing that the contagious element is represented by the spirillum, I find grounds for believing that the parasite is not equally active at all periods of the fever, and not at all so during the ante-febrile state; the discrepancies, indeed, in my experiments are marked enough to render it doubtful if the spirillum itself does represent the contagium proper, and not rather some other agency which at certain periods is associated with it. Such doubt seems warranted so far as regards the earlier scanty proportion of the parasite, its dynamical properties at high temperatures or immediately preceding its total disappearance at the "crisis," and lastly, when associated with some other blood poisons (also possibly parasitic).

Additional inferences might be drawn from the above negative series as regards, for instance, the condition of the animals operated on, which will be suggested by the experiments themselves, and there are, besides, several important points not even alluded to; but I claim for my

work no more completeness than commonly attends a first essay, especially as regards negations which are difficult of proof, and the following series is, perhaps, the more satisfactory.

2. *Positive Series.*

The data I have to offer concern the changes of temperature and state of the blood consequent upon spirillar infection artificially induced; a few remarks upon the post-mortem appearances at different periods of infection are added, and some collateral evidence derived from culture-trials.

Memorandum on the normal temperature of the monkey.— It is necessary to be aware that these small creatures have a greater body-heat than man. The axillary temperature of four healthy animals was taken in the usual manner, and under common conditions, at three-hour intervals during from two to five days; total observations 97; mean temperature of air 80° Fahr. The data obtained were at least as uniform as have been stated for man, and are summed up as follows:

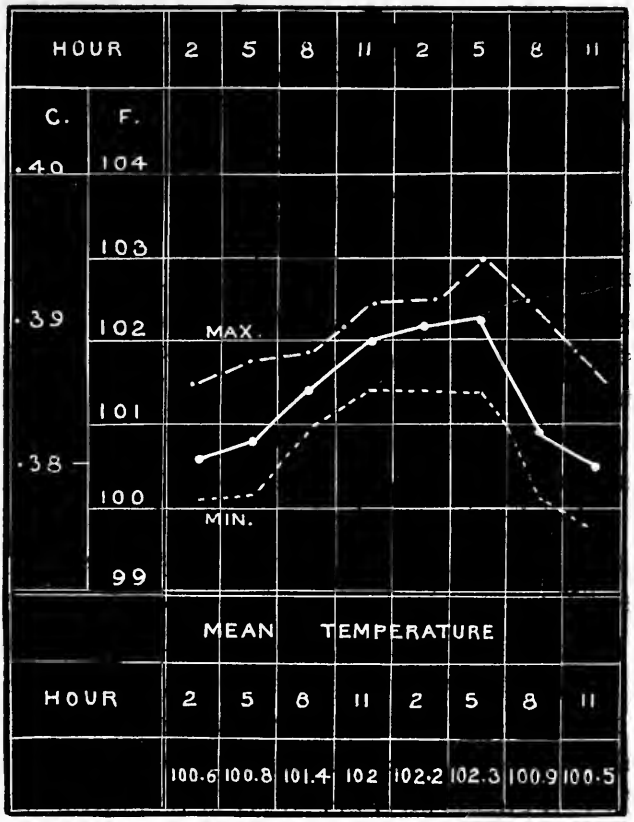
Daily mean temperature . . .	101·35° F.
Mean daily range	100·4° to 102·3°
Maximum	103°
Minimum	100°

The daily mean temperature is nearly the temperature at the hours of 7 a.m. and 7 p.m.; the maximum was found at 5—6 p.m., the minimum at 10—11 p.m.

Course: the lowest temperature occurs about midnight (100·5°); a gradual rise takes place after 2 a.m., continuing till the afternoon, when (5 p.m.) the highest point is reached (102·3°); then follows the evening depression, which proceeds more rapidly than the morning rise.

As compared with man the mean temperature is nearly 3° higher; the range of 1·9° is about the same, and the course followed throughout the day is very similar. The

Normal Temperature of Monkey.



mean daily temperature of healthy natives, taken in the usual manner, I find to vary in range, and oftenest to be rather below that of Englishmen at home.

Normal variations.—The data show these to be not very considerable. They consist in tendencies to a narrower range, to a slight rise soon after midnight, and to an elevation preliminary to the common mean maximum near the hour of sunset.

Incidental variations.—A temporary exacerbation follows restiveness or excessive muscular exertion; and the presence of sores due to biting, with chafing of the band or chain holding the animal, also leads to perturbations of temperature. In cool and damp weather the body heat declines, especially at night.

Some quasi-normal temperatures.—Without entering

into details, I would here allude to my records of abortive experiments. When an experiment made with infecting or non-infecting material fails to produce an effect on the monkey, the temperature retains nearly or mostly its normal characters, and the same is true for the period immediately following an ordinary spirillar attack. This statement is not, however, exhaustive; for although I found that desiccated blood and the blood during the incubation period, at "fall," "rebound" and at date of expected relapse of man, did not induce decided constitutional disturbance, as measured by the bodily heat, yet some perturbations of temperature may ensue, and it has still to be positively determined that these do not represent the mildest or quasi-abortive developments of the blood spirillum. In my observations even at three-hour intervals some minute or very brief blood changes, really specific, may have been overlooked which would correspond to these temperature perturbations when there was no other apparent influence at work; and it would be highly significant, if the spirillar infection, in whatever degree, was established under any of the conditions named above. The chief obstacle to advance in this direction is the want of definite knowledge regarding the development of the spirillum itself, by which to interpret such blood changes as are not seldom seen; and I have many times reluctantly recorded a negative observation owing to this ignorance.

General conditions of experiments.—70 per cent. of my essays, including the first and last that were made, proved successful; and I am of opinion that, with the precautions above intimated as necessary, a much larger proportion of successes might be obtained. In the seventeen trials noted below, twenty-two monkeys were employed, and I find that when two or three were used in a single experiment the result was sufficiently alike, as regards both time and degree, to exclude the idea of contagion as an intervening influence; and as regards the occasions when the same animal was at successive intervals subjected to

repeated inoculations, I had no reason to conclude that the earlier essay materially influenced the later ones. The prime source of infection was the blood of human patients at first, second, or third attack of relapsing fever of ordinary type; subsequently blood inoculations were made from one animal to another.

The induced fever in the monkey can here be compared only in part with its human prototype; yet I am able to say that there was throughout a degree of concordance in general symptoms of oppressive character, which clearly indicated that the "fever" was virtually the same. Post-mortem appearances correspond in the main.

1. *Observations on the Pyrexia.*

Its general features.—The attack in the monkey being initial is to be compared with the first or invasion attack of man, which in him has a strikingly uniform duration, course, and intensity, and in three fourths of cases is followed by a relapse. In the lower animal there is much more variety of form, the date of onset also varies, and a relapse is so rare (viz. at most as one in eight) and so irregular, as to appear altogether incidental or possibly wanting. No such foretelling of events is practicable here as is feasible in the human subject.

These results were not anticipated, and upon reflection I observe, as regards the monkey, that the spirillar infection being new to the species a greater range of susceptibility was likely; besides, the inoculated material was not always the same in character or amount; it may have become modified in transmission, and it was implanted in an artificial manner in healthy subjects.

The human subject, on the other hand, seems to have become uniformly susceptible to attack, is infected in a "natural," possibly uniform, manner, or at certain stages only of the fever; commonly, too, the frame is at the time predisposed by a departure from the normal state.

It is the same thing to say that the contagium itself may have become modified in its passage through the human system; its specific characters may even be due to gradual evolution within the same sphere.

According to my observations the relapse is an acquisition in man, and not an essential part of the fever.

Particular Features of the Pyrexia.

Incubation-period.—This is commonly regarded as a breeding time, ending in maturity of the pyrogenic agency, and reckoned as lasting from infection to fever, general symptoms being seldom well defined until towards its close, when the prodromata appear. My experiments serve to illustrate the varied duration and form, and probable nature of this period, especially as regards the nearer pre-febrile stage corresponding in date to the premonitory symptoms.

I should here remark that, correctly speaking, the relapsing fever of man is a composite affection embracing a series of distinct attacks, and therefore cannot, except in a very restricted sense, be classed as one of the "continued" fevers. The corresponding affection in the monkey, on the other hand, comprises but a single febrile event, which is probably the fundamental, as it is the simpler, manifestation of spirillar pyrexia.

Judging from analogy, the several apyretic intervals of the recurring variety should be regarded as incubation-periods belonging to the attack which they precede, and the accuracy of this view is confirmed by the fact that the specific blood-contamination invariably commences one or two days (at least) before the onset of fever in man and animal, that is to say, towards the close of the apyretic incubation-period; whilst it is never prolonged beyond the pyrexial attack, but promptly and wholly ceases with it. Particular scrutiny is needed to detect the spirillum during the incipient apyretic infection of

man, and in him data are necessarily wanting for the first incubation-period, or that preceding the invasion-attack; but in the lower animal this preliminary blood-infection, besides beginning earlier, is more readily detected during the first and sole incubation-stage here witnessed, and thus, by experiment, the whole series of observations is rendered complete.

Each of the non-febrile periods in question thus becomes divisible into an earlier incubation-stage proper, and a later stage of apyretic infection, during which there is present the same visible blood-contamination as characterises the pyrexial condition, the only difference being that, with the onset of fever, the parasite becomes more abundant. I was not able to correlate in the monkey any premonitory symptoms beyond occasional languor and impaired appetite with the preliminary infection stage, nor have I yet in man observed further concurrence; future inquiry may show such other coincidences as might reasonably be anticipated.

The following details of temperature are available from thirteen selected experiments:

Non-spirillar stage.—After incidental excitement has passed off there occurs but slight perturbation of body heat, the tendency in prolonged instances being to a decline, connected with effacement of normal daily range. The mean duration was 60 hours, which is probably in excess of the actual, but such wide variation is apparent (viz. from 15 to 96 hours), that it becomes evident there were conditions differently influencing each group of experiments; these might be concerned with the quantity of injected material (though hardly likely so), or with the stage of fever, or state of the blood inoculated, and of this the number and activity of the spirillum may not be a complete measure. In general, this stage was twice or thrice as long as the succeeding one.

Stage of spirillar infection.—The ascertained mean temperature at first appearance of the parasite was 102°, or so completely within normal limits as to show conclu-

sively that the mere presence of the spirillum does not entail high fever; subsequently the body heat even declines from ·3° to ·9° below the normal mean of corresponding hours at day-rise especially, and if this stage be prolonged the temperature may sink to 98·6°, or below the normal minimum. I have verified this course in the human subject. The mean temperature immediately prior to the estimated hour of febrile onset was 101·6°, or hardly above the normal mean; once it was 99·8° and once 103°, when fever came on more gradually than usual. The charts show a quasi-normal course, with depressions rather pronounced.

Duration.—This varied from 15 to 50 hours, but the average was near enough the mean of 26 hours to permit this stage being regarded as frequently a well-defined one.

By combining the above two stages the entire incubation-period is obtained; its mean duration was about 90 hours, range from 30 to 126, average rather over the mean, or nearer 4 days than 3. It varies, in general, with the intensity of ensuing fever; thus, five severe cases had a mean total incubation of 64 hours (range 53 to 84), and four mild cases a mean of 86 (range 66 to 118), these differences being determined chiefly by the length of the second or spirillar stage.

The elements of calculation are submitted in the table appended, from which Experiments 9 and 13 may be eliminated, 17 may be exceptional, and 14 is ill-defined. Most of the data being necessarily approximate, the needed corrections would be an abbreviation of the first with corresponding lengthening of the second stage; estimate of fever was arbitrarily fixed at first observation after rise above mean normal. After correction the second, third, and fourth columns of the table would be still unharmonious, and upon the strength of two experiments more precise than usual (viz. Nos. 2 and 8), a natural variation of the incubation-period, as yet obscurely understood, must be admitted, however unlikely

this at first sight would appear; upon arrangement of each series a fair average commonly appears, with rarer extremes in either direction, and more cannot now be be advanced. I would only add that these remarks are in accordance with clinical experience.

REMARKS ON THE ANNEXED TABLE.

(Page 134.)

The figures in the second to sixth columns are hours; the blank spaces in the second to eighth columns are due to observations cut short or incomplete. The first thirteen experiments were made with the blood of man, the last four with blood of the monkey. The figures in the second column are overstated where the blood examinations were made at long intervals, those in the third column being equally understated. In the fifth column fever is reckoned to begin with rise to and decline until normal upper limits (102·3°); comparison with the charts may be made. The fourth column is the sum of the second and third, and the sixth column the sum of the third and fifth.

Incubation-period.—Assuming my data to be free from fallacy, the length of this period is seen to be not only varied in particular instances, but inharmoniously so with regard to current ideas of infection; thus, the three mild attacks after inoculation of human invasion-blood had a mean incubation-period of seventy-five hours, and equally so the two severe and fatal ones of comparative infection at second remove from man (No. 16). This fact seems conclusive of the little significance of incubation-periods as respects the spirillum fever. Nos. 6, 7, 8, and 10 conform in pre-spirillar and apyretic infection, and 11 in the latter only. The longer delay of visible infection in No. 4 (1 Rel.), 12 (2 Rel.), 17 (Incubation), and still more in No. 13, and the two relapses 17 and 12, are not susceptible of collation; if the latter were the result of contagion, the interval in question remains undetermined. No. 9 is wholly exceptional in the other direction.

Fever: Man.—The invasion series Nos. 1, 2, and 3 are concordant, and almost equally brief attacks occurred after inoculation with incubation-blood (Nos. 13 and 17, with relapse). No. 9 had also a brief attack. The sharp rebound of No. 4 is not reckoned above. No. 12 (second relapse) stands alone; the period of spirillar infection (sixth column) was nearly alike in first and second attacks. *Monkey.*—This series is tolerably concordant, and offers a great contrast to the corresponding invasion-series of man, the mean duration of fever being as 72 to 7—a prominent fact.

Table of Conditions and Successful Experiments.

No. of experiment.	Pre-spirillar incubation.	Spirillar incubation.	Total incubation period.	Duration of pyrexia.	Total spirillar infection.	Max. temp. noted.	Character of attack.	Date of inoculation.	Nature, quantity, and quality of material inoculated.
1	48	23	71	8	31	105·4	Mild	4th day of invasion	Defibrinated blood, minims xx. Spirilla several.
2	54	27	81	8	35	104·6	Do.	Do.	Do. do. x. Do.
3	54	18	72	6	24	103·6	Do.	8th day of invasion	Do. do. v. Spirilla numerous.
4	96	24	120	27	51	104·4	Do.	1st day, 1 relapse	Do. do. v. Spirilla very few.
5	—	—	? 120	?30	—	105·8	Marked	2nd ,, do.	Do. do. iv. Spirilla several.
6	48	50	98	—	—	105·2	—	2nd ,, do.	Do. do. x. Spirilla many.
—	48	—	—	—	—	—	—	2nd ,, do.	Do. do. x. Do.
7	46	—	—	—	—	—	—	3rd ,, do.	Do. do. xx. Spirilla several.
8	50	48	98	—	—	105·8	—	3rd ,, do.	Do. do. xv. Spirilla few.
9	?	?	12	15	?	106·8	Mild	3rd ,, do.	Serum of blood do. x. Spirilla many.
10	—	—	85	—	—	106·6	—	4th ,, do.	Defibrinated blood do. xx. Spirilla few.
11	15	48	63	—	—	105·2	—	4th ,, do.	Do. do. v. Spirilla numerous.
12	94	20	114	42	62	105·6	Marked	1st day, 2 relapse	Do. do. viij. Spirilla few.
—	264	36	300	27	61	105·2	Moderate	Relapse	—
13	240	20	300	6	26	105·4	Mild	9th day, 1 interval	Defibrinated blood, minims x. No Spirillum seen.
14	—	—	96	66	—	106·4	Severe	1st day of attack	Whole blood, a quarter of a drop. Spirilla many.
15	—	—	53	—	—	106	Do.	2nd day of attack	Defibrinated blood, a few minims. Many spirilla.
—	—	—	53	90	—	106·2	Do.	Do.	Do. do. do.
—	—	—	57	86	—	106·8	Do.	Do.	Do. do. do.
16	65	24	89	62	86	105·6	Do.	Do.	Blood-clot, the size of a pin's head. Spirilla unknown.
—	48	12	60	67	79	105·8	Do.	Do.	Blood-serum, minims iij. Spirilla many.
17	92	24	116	8	32	106·2	Mild	1st day, incubation	Defibrinated blood, a few minims. No spirillum seen.
—	150	36	186	12	48	105	Do.	Relapse	

Stage of manifested Fever.

Beginning.—Fever sets in from 1½ to 5 days (at most) after inoculation, the mean period being 75 to 80 hours, after excluding an exceptional instance. The hour is commonly near noon or somewhat later, but there is no rule, and it may be at night or early morning; since the majority of inoculations were practised at 5 p.m., no relationship of time here appears. A high temperature is attained on the first day, and generally within a very few hours (3 to 6), it may possibly be earlier, and is sometimes later (8 to 12 hours); this abrupt beginning of high fever is also a character of the attacks in man. The mean temperature at observed initiation was 104·8° for a morning rise, and 105° for an evening rise, both figures being doubtless within the actual, yet sufficient to show how decided is the onset of fever. As the normal temperature of the monkey is higher than that of man by 3°, the absolute initial rise is not so great as in him, yet the upward limit being nearly the same on this first day it is observable that the spirillar pyrexia again displays its special character.

Course.—As to duration, the induced fever is briefer than that of man, never lasting longer than 3½ days (86 hours), in the mean barely 2 days (43 hours), and at the shortest only 6 hours. Such very varied duration has no counterpart in the first attack of man, but it accords with the varying length of "relapses" in the human subject; so that the single comparative attack may be said to represent almost all known degrees of fever. Proportionately to its bulk of body the lower animal suffers more than equally, and so does the human infant, hence the hypothesis of a limited *materies morbi insitæ* has no application here. One half the attacks might be called mild, the fever lasting not more than 24—30 hours; the

rest were pronounced, lasting twice or thrice as long, and being sometimes fatal in the rebound. The form and degree of pyrexia may be stated as follows:—Fever is paroxysmal in the briefer attacks, and remittent, or even continuous, in the severer. Five mild instances had a total mean duration of 16 hours, and the form of an isolated paroxysm, declining somewhat more slowly than it rose, and more deeply than its starting-point; the temperature attained was in the mean 104·7°, at maximum 105·8°, and at minimum no more than 103·7°, or not more than 1½° above the normal level. A rebound was rare, yet prominent once (*vide* Experiment 4). In two pronounced attacks, lasting 24—48 hours, the fever was practically continuous, with a depression at early morning hours more or less marked; the second wave was rather less prominent (105·2°) than the first (105·6°). Five severe attacks lasted from 62 to 86 hours, or in the mean rather over three days; in all, the fever was continuous when viewed at short periods, but when projected from the daily maxima and minima its remittent character was very apparent; high pyrexia was rarely sustained for 24 hours together, and the single instance showing this had a low initial temperature at starting, the animal dying (*vide* Experiment 15); the remissions generally took place in the early morning, and varied from 1° to 3° or 4°. A daily cyclical range was so apparent that it may be confidently looked for in fever lasting two days or longer, and according to the duration will be the number of daily exacerbations and remissions. The mean temperature of these epochs was the following:—First or initial rise 105·5°, first remission 103·1°, second rise 105·8°, decline 103·8°, third and final rise 105·9°, and fall (crisis) 101·7°; there is a visible tendency to gradual augmentation of pyrexia till the acme (maximum temperature 106·6°) immediately prior to crisis of attack. The mid-febrile remissions amounted in the mean to 2·3° (first) and 2·1° (second), or about one half the final fall.

All these severe attacks were succeeded by a sharp

rebound or secondary fever, during which three animals died, and a fourth would probably have succumbed had it been allowed to live as long. Excepting in absolute briefer duration, these comparative phenomena agree with human experiences, and as the contemporary blood-infection is the same, I conclude that the specific pyrexia of man was actually reproduced in the lower animal.

Crisis.—This was always well-defined, yet not so extremely pronounced, or commonly quite so abrupt as in the human prototype. Profuse sweating or incipient collapse were not noticed, and in the milder cases recovery was prompt. The main fall was usually recorded at the 5 a.m. observations; the absolute decline varied from 2·8° to 6·6°, mean about 4° Fahr., the mimimum temperature noted at this time was hardly below that sometimes recorded in healthy animals (100° Fahr.), whereas in man it may be 2° or 3° lower than normal minimum, and this difference may be referable to the greater constitutional robustness of the *feræ*.

Relationship of Pyrexia to Incubation-Period.

No invariable rule was here manifested, only it may be said that whilst for all degrees of fever the earlier non-spirillar stage had a mean duration of near 60 hours, that of apyretic infection had a mean of 26°; divergencies as regards the intensity of ensuing pyrexia were more variable than the conditions of experiment, which was hardly to be anticipated, and I regard the absence of an appreciable regularity of sequence in this respect, an incubation-period of almost identical length preceding, in individual instances, both mild and severe attacks, as indicative of some intrinsic difference of the contagium which is not displayed until fever sets in. The following note bears upon this topic.

Comparative Severity of Fever in the Monkey.

A mild or severe attack in the human subject does not necessarily entail a corresponding one in the lower animal, but inoculation from a monkey infected directly or at one remove, especially from man, always produced a severe attack; and hence it appears that the human virus becomes intensified in passing through the monkey. The following tabular statement of results of seventeen inoculations, more or less completely followed out, illustrates this point; the data are means only, and those of Experiments 9, 13, 14, and 17, have been excluded as being least certain or precise.

Events.	Inoc. from man. Mild attacks.	Inoc. from monkey. Severe attacks.
Spirillar infection appears at latest after .	56·4 hours	56 hours
Spirillar infection lasts, at least . .	28·5 „	18 „
Total incubation period	90·7 „	62·5 „
High fever lasts	22·5 „	74 „
Total spirillar manifestation . . .	42·6 „	82·5 „
Deaths known	0 : 12	3 : 5

N.B.—Though derived from common data, these figures may seem not to tally with others previously given, but there is no real discordance.

Somewhat parallel with this striking augmentation of infective properties by repetition (if it be nothing more), is that indicated by the experiments made with human blood at successive pyrexial stages of relapsing fever; thus, infection at "invasion" (Nos. 1 to 3), resulted in the mildest form of attack. whilst the "first relapse" supplied more active material (No. 5), and the barely visibly contaminated blood at "second relapse" (No. 12), was also highly effective. Similar results might be looked for in human experience, and if the successive attacks of

"recurrent typhus" have anything of a cumulative character, or together be comparable with the longer sole attack of ordinary typhus, it might be remembered that the infective powers of the latter fever are greatest at, if not limited to, its terminal stage; nor is the instance a singular one of a contagium slowly gathering in force.

Rebound of Temperature or Secondary Fever.

This is the commoner sort of "relapse" met with, and the phenomenon is a striking one. After mild specific attacks it assumes the form of a smart but brief reaction, and is promptly recovered from; after the severer attacks it is commonly contemporary with local inflammation (see the note on autopsies), and may lead to death. The blood is always free from spirillar contamination here, as in the human subject. Rebounds set in either immediately after the crisis or in the course of a few hours, the milder lasting six hours, the complicated from one and a half to three and a half days, or as long as the prior specific fever. According to their duration, the pyrexia is wholly paroxysmal, or it is sustained and of remittent type, with a tendency to become continued; the onset is prompt and so is the final decline. The temperature attained equals or more commonly exceeds by about 1° Fahr. that of the specific attack, and the highest recorded in my series, viz. 107·2° occurred at the beginning of secondary fever (see the chart of Experiment No. 15); all the maxima were over 106°; the remissions varied from 1° to 3·4°. At the decline, body-heat did not descend below normal limits, except in the three fatal cases, when it probably sank till death.

Secondary fever followed all the severer specific attacks seen throughout, and of four instances only one survived after serious illness; its occurrence after milder attacks

was irregular. Experiment No. 4 is a good example. I need only add that this event furnishes evidence of a similarity, descending even to incidental characters, between the human and comparative attacks of spirillum fever.

Relapses.—The only two known instances have been described in Experiments 13 and 17; they resembled in all features ordinary mild attacks and occurred six and twelve days after the primary event. The extreme range in man was 5 and 12 days in an infinitely larger series than the present, and the average interval was practically invariable, hence my hesitation to insist upon this limited and irregular comparative experience. The absence of a relapse would be no argument against the identity of the spirillum fever of the monkey with the relapsing fever of man, for in the human subject second attacks are wanting in one fourth of cases, or even oftener, and still the invasion preserves its usual characters. I am not aware that relapses were overlooked in these experiments.

The Post-mortem appearances in Spirillum-Fever.

Twelve autopsies were made of nine animals killed at various stages, and in three dying of fever. During the incubation-period (three cases) commonly no striking change was noticed, but once deep congestion of the mucous membrane of the stomach, with minute petechiæ, about the middle of the viscus; liver and spleen congested. In five deaths during fever, vascularity of the stomach about the middle, with small petechiæ, and vascularity of the duodenum and the lower end of the ileum, with congestion of liver and spleen. In one animal bled to death the liver was almost translucent and the spleen small, flabby, and pale, which shows how little the solid parts were altered. Twice, however, there was pulmonary apoplexy; the brain,

heart, and kidneys were not notably changed. In one death at "full," the viscera showed nothing peculiar. In the three animals dying during secondary fever the liver was congested and once enlarged; the spleen large and congested; kidneys healthy-looking; the mucous membrane of the stomach was once unchanged in aspect, and twice inflamed (being once of a deep maroon colour) about the middle. In two animals dying on the third day of rebound the small intestines were inflamed throughout, beginning abruptly at the pylorus and ending at the ileo-cæcal valve, there being hæmorrhagic spots also. In one animal dying a little later, this mucous membrane was very vascular and the intestinal walls were very thin; the large intestine was comparatively unchanged, the rectum only being rather vascular. Petechiæ were seen twice on the lungs and on the heart in these three cases; the brain was pallid only. These data form part of the evidence showing the identity of the "fever" in the man and animal.

After death from poisoning by saliva (two cases) inflammation of the stomach and extravasation into the arachnoidal sac were once noted.

Portions of the above viscera are preserved, and may show minute textural changes (possibly in the blood-vessels), elucidating the essential seat of morbid lesion better than coarser appearances; at present, I have detected no peculiar changes except within the blood.

Microscopic Observations on the Blood.

From a very large number of data I am able to state that there obtains the closest correspondence in aspect between the blood of the monkey and of man at all stages of the spirillum fever, including that of apyretic infection; nor has any discordance come to light. Respecting the less peculiar constituents, various forms of protoplasm deserve

mention from their possible connection with development of the spirillum; but as such "clumps" and "filaments" may be independent, attention will here be limited to the parasite alone.

I regard it as manifest that this organism is identical in both species, and its comparative prevalence in time is also alike. Thus, on first detection in the early non-febrile stage its numbers are sparse (*e.g.* one or two in field of view), and being only rather commoner in the monkey; it then has a fully-formed aspect and would seem to be introduced into the blood *ab extra;* no change occurs until the onset of fever. The spirillum may, indeed, somewhat diminish in numbers, and even temporarily disappear at this time, but with, or even before the rise of temperature, it rapidly augments (*e.g.* six to twelve in the field), and hence sometimes I have been able to predict the advent of fever. During the course of pronounced pyrexia the parasite positively swarms in the monkey's blood (appearing almost as numerous as the red discs); it is little less abundant with remissions of temperature, but it promptly and wholly disappears at a period, sometimes computable by minutes, immediately prior to initiation of the critical fall of temperature, vanishing at the *perturbatio critica* when this phenomenon occurs. The intermittent appearance of the spirillum during high fever, and its continuance at a complete intermission of temperature or during the fall by "lysis," are exceptional phenomena hitherto seen only in man; and the inevitable inference from my comparative studies is wholly in favour of there being the closest relationship between abounding spirillar infection and pronounced pyrexia. The evidence of interrupted parasitic growth, or accession by "crops" or "broods," has not been so manifest here as in the longer attacks of man.

The great variety of form assumed by the more active spirillum in the freshest possible blood does not seem to be remembered, and the ordinary delineation of a straight spiral filament pertains rather to a more quiescent state

supervening some time after withdrawal from the body; in quickly abstracted blood I found the organism in incessant movement, presenting the shape of curved and twisted filaments, and of loops, knots, and rings, such as were never assumed by the spirilla and spirochæte of tank-water, which I also submitted to scrutiny for comparison. These last preserve their rigid spiral contour in all their movements, and do not unfold, as it were. On the other hand, I have seen the Bacillus (subtilis ?) of similar water part with its straight or wavy outline, and take on a twisted, spiral form, which evidenced nearly as much flexibility and extensibility or contraction as is shown by the blood parasite, and my impression is that the latter organism might be defined anew.

The spirillum of the saliva has been alluded to already in Experiment 42.

For thorough scrutiny of the blood it is necessary to employ the Albrecht process, with or without subsequent staining; and simple dyeing of the blood elements with anilin violet will also be found very useful. By either of these means a multiplication of the spirillum by mid-fission may be seen in most specimens of infected blood, and oftenest, I think, when the parasite is increasing. Often, too, the presence of immature and possibly growing organisms is thus made apparent, the primitive granules and short curved filaments having a diameter somewhat larger than that of the perfect parasite. A dotted or even partially beaded condition of the latter may sometimes be noted, which seems indicative of a mode of growth. The same processes applied to the venous bloods issuing from the spleen, liver, lungs, kidney, &c., during the height of fever, reveal a striking aspect of the plasmic contents, which I can only interpret as significant of active spirillar development; the splenic venous blood is especially rich in endothelial and white-cell forms containing the germs (?) and incipient spiral filaments; this subject is still under inquiry. I should add that there are some fallacious appearances pertaining to blood protoplasm, and (in Bom-

bay, at least) to minute incidental fungus growth which require discrimination.

As to the mode of disappearance of the spirillum at the close of fever, this organism has been found in preserved specimens of blood to quickly melt away leaving no immediate trace behind; sometimes the amœboid masses common in the plasma seemed to involve and remove the quiescent filaments. That the destruction of the parasite, now or previously, is an active process was shown by the fact that in specimens of infected blood kept for several hours the spirillum had been preserved in activity outside the body, whilst it had altogether disappeared from blood drawn afresh within the same period, having evidently been destroyed by some natural process not operating outside the living frame. It was not clear that this process pertained only to a high temperature.

The activity and life of the spirillum are independent of the life of its host; in the living subject this organism is to be seen only in active movement, and thus its detection is rendered comparatively easy. Its movements may persist for many hours after decease of man or monkey (*e.g.* 24 or 36,), and their cessation is not a sign of its own death, for they cease before the filament begins to grow under the influence of artificial culture.

Reserving other details, I will only add here that the parasite of spirillum fever may be readily induced to multiply and grow into a delicate mycelioid network, which in favorable conditions is very luxuriant and striking. Sometimes spores seemed to arise from this fructification, but I was never able to see the production of a second generation of free spiral organisms.

On the connection between the results of Inoculation and of Artificial Cultivation of the Blood.

There exists a certain accord between the experimental series above described and another carried out about the

same time and with the same or similar material, in which attempts were made to induce growth or reproduction of the spirillum outside the body, and the results are worthy of brief mention here, even if it be not admitted that the blood parasite is the "cause" of fever.

Thus, in eight essays at culture I found the spirillum is never developed in blood not containing it at the beginning of experiment, and this is in accordance with the negative results of inoculation practised with similar material. It would, therefore, appear that the germs of the parasite are not habitually present in the blood.

Again, in eighteen cultivation experiments made with visibly infected blood, some (eight) failed whilst others (ten) succeeded, just as is the case with inoculation experiments which are only partially successful. The failures were two of incubation-blood, one of invasion-blood at "acme," one of invasion-blood at "fall," both specimens taken from the same patient as in Experiment 18, which was also unsuccessful at the last of these dates; three failures were of blood at first day of "relapse," which is in accordance with Experiments 21 and 22, and the blood employed was the same as in No. 22; one was of relapse-blood at fall, which also accords; and, in short, these negative data agree throughout, nor are there discrepancies. It therefore appears that growth of the parasite within the blood is, at least, not usual at certain stages of the fever. The successes were six, viz. four of invasion-blood of man from second to seventh day inclusive, and two of monkey's blood at first and second day of high fever, both series agreeing with positive inoculations. Since success in culture implies the growth and increase of spirilla in the medium employed, its relationship with successful inoculation becomes obvious; and the inference favours the view of the blood parasite being essentially connected with the production of pyrexia.

Conclusions.

1. "Relapsing fever" is readily transmissible from man to a quadrumanous animal, and from one of these animals to another, by inoculation of the blood, and it then commonly assumes the form of a single febrile event of highly varied intensity, type, and duration. So far as evidence goes the "relapse" is much more incidental in the monkey than in man. The conditions of infection are multiple.

2. In the comparative attack the incubation-period is characterised towards its close by a non-febrile spirillar infection of the blood, and the same fact has been verified for the first apyretic interval of man. It therefore appears that all incubation-periods are divisible into two stages of a prior non-specific and a later specific character respectively. The duration of these periods, or even of their stages, bears no fixed proportion to the intensity of ensuing febrile phenomena.

3. The relationship of spirillar blood-infection to pyrexia cannot hence be regarded as an immediate one, yet, in the monkey, fever was an invariable sequel to infection, beginning and being conterminous with, and in degree generally corresponding to, augmentation of the parasite in the blood. In milder degrees of infection, however, it is conceivable that the terminal development of pyrexia may be wanting, and hence the spirillum disease would become essentially definable as a *mycosis sanguinis prope cum febre.*

4. Nothing definite has been elicited respecting contemporary pyrogenetic agencies of a specific character, other than the spirilla. Such there may be, yet the tendency of my comparative observations was towards the inference that exceptions to the relationship indicated are explicable upon idiosyncrasy of subject or other complication, the non-specific causes of "fever" being numerous.

APPENDIX.

Successful Inoculation with Blood of Monkey at Incubation-period.

M. No. 1, new animal, had five minims of defibrinated blood taken from a woman, æt. 35, at last day (a.m.) of a characteristic invasion-attack of relapsing fever (temp. 104·8° F.), injected hypodermically in the thigh; material employed contained a great number of active and quiescent spirilla; blood and temperature of the monkey normal.

On the second day at 4 a.m., or 16 hours after injection, the parasite was found in the animal's blood (temp. 99·2°), and for 48 hours longer there was no fever; then a brief intermittent attack ensued, marked, as usual, with increased visible contamination of the blood, and ending with abrupt fall and concurrent disappearance of the spirillum (*vide* Chart, Diagram VIII).

M. No. 2, a female, freshly brought from the bazaar, had hypodermically injected in the thigh ten minims of entire blood drawn from the ear of No. 1 about mid-period of non-febrile, specific incubation-stage (*vide* Chart, Diagram VIII). At this time very few spirilla were to be seen in the blood employed for inoculation, even on testing with the acetic acid process. For two days (possibly less) there was no change in No. 2, but on the fourth day, or 64 hours after experiment (temp. 101·2°), a single organism was detected in its blood, and thenceforward others for 48 hours longer, still without fever, when a brief febrile paroxysm occurred, which terminated the attack.

There were no peculiarities in the state of the blood, temperature, or general symptoms of this case, which seems to show conclusively the possibility of infection during the incubation-stage of fever, due precautions having been taken to avoid error from incidental contagion.

Previously negative results are therefore qualified by this positive issue, and the significance of the spirillar state is, perhaps, increased thereby.

BOMBAY; *December*, 1879.

A CONTRIBUTION

TO THE

NON-GLANDULAR THEORY OF ORIGIN

OF

MOLLUSCUM CONTAGIOSUM.

BY

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Of late years there has been a difference of opinion, chiefly amongst foreign observers, as to the nature of the pathology of molluscum contagiosum. By some the process is looked upon as simply a hyperplasia of the sebaceous glands, together with certain degenerative changes taking place in the cells composing them. This is the old view. It has stood its ground in this country since the days of Bateman, whose name was first associated with the disease, and continues to receive almost universal assent. Some Continental observers have, however, maintained that molluscum contagiosum has nothing to do with the sebaceous glands, being in fact an overgrowth from the Malpighian layer. An attempt will be made to support this latter view; moreover, it will be shown that the degenerative changes peculiar to molluscum contagiosum

are closely connected with the superficial layers of the epidermis.

Perhaps it may be as well in this place to give a brief *résumé* of the writings of some of the observers whose names are associated with the non-glandular theory of the disease. References to their articles will be found appended to this paper.

Virchow, writing in 1865, says:—"If a section of one of these tumours be made (molluscum tumour) an appearance as of a lobulated gland is seen. Two distinct parts may be recognised: one soft, occupying the interior, which may be pressed out, the other firmer, following the walls of the cavity; it is the Malpighian layer greatly developed. The soft substance is composed of epithelial cells, polygonal, and mostly without nuclei, having depressions, in which one or more peculiar bodies (molluscum bodies) are lodged. Nowhere can be seen the fatty granules and oily globules which are seen in sebaceous glands. These (molluscum) bodies have the greatest resemblance to swollen starch granules, but no characteristic reaction can be obtained with iodine and sulphuric acid. They are due to a peculiar degeneration of the epithelial cells." This author considers molluscum contagiosum to begin in the hair-follicles, the folded gland-like appearance being due to a hyperplasia of its epidermal lining. He suggests the name of epithelioma molluscum.

Boeck, writing in 1875, says, concerning the molluscum bodies found in molluscum contagiosum, that "they are epidermal cells which have undergone a peculiar degeneration. This consists in an alteration in the protoplasm of the cell, which appears always to begin round the nucleus. In a vertical section through the lower part of a molluscum at first, several layers of normal rete cells are met with, and these gradually give place to other cells, in which the nucleus has a peculiar shining margin, generally quite distinct from the nucleus itself; this margin increases in width in other cells, until at last the new material fills the whole cell, which becomes larger than before, and of

a more regularly oval shape. These oval cells when aggregated together in vast numbers form the body of a molluscum wart, and have been falsely regarded as of the same nature as those of a sebaceous gland. They contain no trace of fat, nor do they give the reaction of amyloid degeneration with iodine and sulphuric acid."

Lastly, Lukomsky (1875) "was not able to find out the nature of the molluscum bodies by treating them with various reagents. He considers the disease to be one of the Malpighian layer, and not of the sebaceous gland, the Malpighian projections grow rapidly, the peripheral portion being lined with cylindrical epithelium; on these follow two or three layers of the mucous cells. In the centre of the projections appear large granular cells, which as they advance upwards pass into molluscum bodies. The molluscum bodies come from cells which lie in the mucous layer, but they arise from wandering cells, which are always to be found in this situation."

Although some confusion existed formerly between the two varieties of molluscum, namely, "molluscum simplex," or "fibrosum," and "molluscum contagiosum," yet the strongly marked, and now more widely known, characteristics of the latter affection have caused it to be easily recognised, even in countries where it is of comparatively rare occurrence. Error in diagnosis, therefore, can hardly be taken into consideration in accounting for the discrepancy of opinion.

As is well known, the little tumours of molluscum contagiosum vary in size from that of a minute papillary elevation, scarcely recognisable above the surface, to that of a pea or small marble; for the most part they are of the same colour as the surrounding skin, and in delicate anæmic subjects present a sodden, greasy appearance.

Before they attain the size of millet seeds a central depression or "umbilication" is seen; this is, in reality, a widened natural orifice of the skin, probably indicating the original point of departure of the morbid process.

Whilst the tumours are small and only slightly elevated

above the surface, the central depression or umbilication confers on them an appearance which has been likened to that of a small pearl shirt-button. As they become larger, they overhang their narrow bases, and may be said to be pedunculated, although they rarely if ever become pendulous. In this stage the umbilication often presents more the appearance of a comedo orifice. There is a marked tendency for neighbouring lesions to become confluent, and thus some tumours present a lobate appearance, with two or more comedo-like depressions, according to circumstances. On pressure, there exudes from the "umbilication" a thick, pultaceous, or thinner gruel-like, matter; sometimes a milky fluid is discharged in a jet. If the pressure be continued, and especially if the umbilication or comedo-like orifice be enlarged with the point of a lancet, there enucleates a firm, pinkish-white mass, which, to the naked eye even, appears finely lobulated and gland-like. The shrivelled capsule is left behind, oozing some drops of dark venous blood.

Through the kindness of Dr. Stephen Mackenzie, material from a child with molluscum contagiosum, attending the out-patient department at the London Hospital, was procured. Some of the smallest papillary elevations were selected; these were snipped off with curved scissors, together with some of the neighbouring skin; larger tumours were also taken. The material was hardened in methylated alcohol, and stained with logwood in the usual manner.

Fig. 1 represents a vertical section through a molluscum tumour the size of a large pin's head; the new growth is seen partly spread out beneath the epidermis, and continuous with it in places, as at *i*, partly in isolated patches (*d*). Those portions of the growth which have undergone molluscous change (molluscum bodies) are at once recognisable by the deep blue staining. The section has traversed the central depression or umbilication, hence the ease with which the continuity of the new growth with the epidermis is demonstrable. The lobular

appearance, which has gone far to suggest the gland origin of the disease, is especially well seen at *d*, where outlying expansions from the growth have been divided transversely, and appear as it were cut off. Commencing molluscous change (molluscum bodies) are seen here and there (*f*).

It is convenient in this place to allude to a few facts as to the structure of the epidermis. The latter, as is well known, is divided into four layers. The deepest layer is the Malpighian layer proper, one of its chief peculiarities being the elongated vertical cells (palisade cells) which are placed next the papillæ. As the cells of this layer become flattened towards the surface they pass into the "granular layer;" this, in the normal condition, has a thickness of two or three cells, which, on section, appear diamond-shaped or spindle-like, with granular contents and clear nuclei. Above the granular layer is the "stratum lucidum" (not demonstrable in these specimens), and above this the corneous layer. The granular layer can be traced into the mouths of the hair-follicles but no farther; it is not represented in the sebaceous gland or its duct.

Fig. 2 represents a vertical section through a small papillary molluscum, but more highly magnified than that shown at Fig. 1. The section does not involve the central depression or umbilication, consequently no communication of the molluscum growth with the surface is seen, nor is the continuity of the growth with the Malpighian layer directly traceable. At the periphery of the lobular mass are seen the elongated and vertically-placed cells, corresponding to the palisade cells of the normal rete. At *e* the molluscum bodies are seen, round or oval-shaped, vitreous looking, and lying in a granular meshwook, which in places (*f*) becomes merged into groups of diamond-shaped, granular cells, bringing to mind those of the granular layer of the epidermis. The granular reticulum ceases somewhat abruptly, and the more superficial molluscum bodies become more transparent and closely

packed. At *h* is seen a trabecular, apparently fibrous structure, splitting into processes, which become continuous with divisions between the molluscum bodies.

Fig. 3 represents the two ingrowths from the Malpighian layer seen at *g* in Fig. 1, but more highly magnified. The hypertrophied layer of granular cells is at once noticeable at *c*; it is seen to dip down and becomes continuous with the granular reticulum between the molluscum bodies. The hypertrophied corneous layer is more intimately connected with the superficial molluscum bodies.

Fig. 4 shows a vertical section through a minute molluscum growth, together with some of the adjacent skin. Three layers of the epidermis are easily recognised on the right, namely (*c*), the Malpighian layer (*b*) the granular layer, and (*a*) the corneous layer. The Malpighian layer becomes identified with the more peripheral portion of the molluscum growth, the palisade cells being seen in uninterrupted continuity. The granular layer becomes merged into that part which has undergone the peculiar molluscous change (molluscum bodies), and is especially associated with the granular reticulum, while the corneous layer, becoming gradually hypertrophied, expands into the more superficial molluscum bodies, which, as in all specimens, are seen to bear a different character to those lower down. A fibrous structure (*g*), similar to that at *h*, in Fig. 2, is seen; also a definite layer of the same nature (*h*), but continuous with the corneous layer, intervenes between the granular cells and the molluscum bodies.

Fig. 5 represents part of a lobule, highly magnified to show the nature and mode of formation of the molluscum bodies. The peripheral cells are elongated, and placed vertically to the surface; next follow crowded cells with large oval nuclei; then there appear oval or ovoid bodies considerably larger than the cells amongst which they lie; these are the "molluscum bodies." At first they are made up, more or less, of coarse granules and a refractile substance; but soon they appear hyaline or vitreous, as

at *c*. They are seen to lie in a granular matrix (*d*), which, under a low power, puts on the character of the granular reticulum before alluded to.

The appearances as detailed above apply to specimens mounted in glycerine. They differ somewhat in those prepared with Canada balsam; for instance, the nuclei of the cells situated towards the periphery of the lobules become possessed of a bright border, occupying part of the circumference, due to commencing vacuolation. Perfect vacuoles, with the dark and shrunken nucleus pushed aside, are also to be recognised. It is probable that the dark spots seen in the molluscum bodies, as at *c*, in Fig. 5, represent these shrunken nuclei; a similar appearance is figured by Bizzozero and Manfredi. On examining the superficial molluscum bodies with a high power they are seen to be transparent and almost structureless; there are no granules traceable between them, and in fact they are separated merely by the single or double contour of their opposed surfaces.

If the granular cells be examined with a high power in situations such as at *f*, in Fig. 2, where they may be supposed to be undergoing molluscous change, the latter seems to consist, primarily, in an enlargement of the nucleus, the contents of which subsequently become modified to form the molluscum body, while the granular matter occupying the remainder of the cell is pushed aside to form the matrix or reticulum.

It would seem, then, that molluscum contagiosum is a disease of the epidermis. As may be seen in Fig. 4, all three layers take part in the changes, but it is difficult to determine which is primarily at fault. Probably the hyperplasia of the rete is reactive, following the molluscum changes in the more superficial layers; of these, the granular layer is associated with the more deeply placed molluscum bodies, the granular reticulum betraying their genetic connection. (In Fig. 5, accompanying Lukomsky's article, the deeper molluscum bodies are seen lying in a granular matrix.) The more

superficial molluscum bodies, different in character, may be directly descended from those more deeply placed, but it may be conjectured that the corneous layer is concerned in their production. This theory is supported by the appearances seen in Figs. 2 and 4. The trabecular masses have the same fibrous character as the corneous layer of the epidermis, a peculiarity conferred by the sectional view of flattened cells; moreover, the layer (*h*) in Fig. 4 is in direct continuity with the corneous layer. It seems probable, then, that these are masses of unaltered cells, bearing the same relation to the surrounding molluscum bodies as the groups of granular cells (*f*, Fig. 2) bear to those more deeply placed. The hypertrophy of the corneous layer as the molluscum growth is reached is another fact in this direction (Fig. 3).

If it be conceded that the molluscum changes essentially belong to the granular and corneous layers, the sebaceous-gland theory at once falls to the ground, for, as before mentioned, the granular layer does not extend to the sebaceous gland. As a proof that the peripheral cells of the lobular masses are descended from the rete Malpighii there is the fact that with a high magnifying power the prickle cells of Max Schultz may be recognised. Now these have been described as occurring in the external root-sheath of the hair-follicles, as well as in the rete, but not in the sebaceous gland (Fig. 6). Bizzozero and Manfredi figure minute striations between the peripheral cells of the molluscum lobules.

Lastly, may be mentioned a negative fact against the gland-theory of the disease. The normal appearance of a sebaceous-gland cell is very characteristic. The large nucleus occupies the centre of the cell, while the remainder has a peculiar reticular appearance. In the specimens examined no such reticular cells were traceable in any relation with the molluscum bodies, such as would suggest their pathological connection.

What the peculiar degenerative change may be, giving rise to the formation of the molluscum bodies, it is not

the purport of this paper to discuss; it seems, however, that it first affects the nucleus of the cell, causing it to enlarge and push aside the remaining protoplasm of the cell. This description applies especially to the way in which the deep molluscum bodies are formed from the granular cells. Boeck and others have shown the changes not to be of a fatty or amyloid nature.

HENDERSON and PATERSON, Edin. Med. Jour., vol. lvi, 1841, pp. 213, 280.

BARENSPRUNG, Anatomie und Pathologie der menschlichen Haut., Leipzig, 1848.

RICHARD PAYNE COTTON, Edin. Med. and Surg. Journal, 1848.

VIRCHOW, Archiv v. Virchow, vol. xxxiii, 1865.

DUCKWORTH, St. Bartholomew's Hospital Reports, 1868.

RETZIUS, Deutsche Klinik., 1872.

FAGGE, Guy's Hospital Reports, 1870.

BIZZOZERO and MANFREDI, Revista Clinica di Bologna, 1871.

C. BOECK, Vierteljahr. für Dermat. und Syph., 1875.

LUKOMSKY, Archiv v. Virchow, vol. lxv, 1875.

OTTO SIMON, Vierteljahr. für Dermat. und Syph., 1876.

VIDAL and ANDRE, Mémoires de la Société de Biologie, 1877.

KAPOSI, Vierteljahr. für Dermat. und Syph., 1877.

HUTCHINSON, Lectures on Clinical Surgery, vol. i, part i, 1878.

FOX, (Drs. T. & C.), Trans. Path. Soc. Lond., 1879.

DESCRIPTION OF PLATES III AND IV.

Origin of Molluscum Contagiosum (ALFRED SANGSTER, M.B.).

PLATE III.

FIG. 1.—Vertical section through a small molluscum tumour.

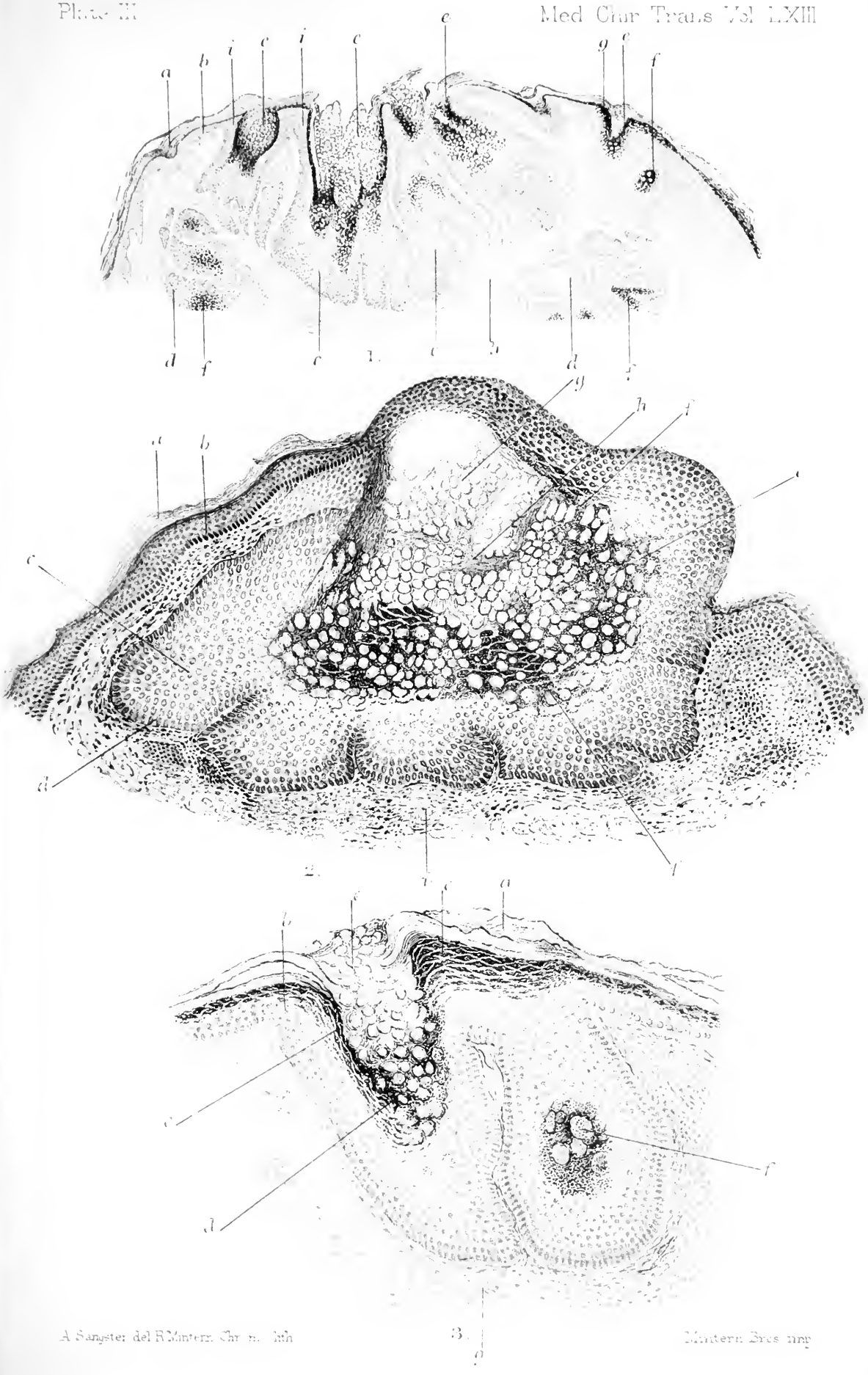
a. Superficial layers of epidermis.
b. Malpighian layer.
c. Lobular new growth from rete cut longitudinally.
d. Lobular new growth cut transversely.
e. Molluscous change (molluscum bodies).
f. Commencing molluscous change.
g. Ingrowths from rete, magnified in Fig. 3.
h. Fibrous tissue of corium.
i. Junction of Malpighian layer with new growth.

FIG. 2.—Vertical section through a minute papillary molluscum. (Hart., oc. 3, obj. 4, tube out.)

a. Superficial layers of epidermis.
b. Malpighian layer.
c. Lobular expansion of molluscum growth.
d. Peripheral cells elongated and vertically placed.
e. Deep molluscum bodies enclosed in granular reticulum.
f. Groups of unchanged granular cells.
g. Superficial molluscum bodies.
h. Trabecular masses (?), unchanged corneous cells.
i. Fibrous tissue.

FIG. 3.—The ingrowths from rete, Fig. 1, *g*. (Hart., oc. 3, obj. 4, tube out.)

a. Hypertrophied and irregular corneous layer.
b. Malpighian layer continuous with peripheral portion of ingrowth.
c. Granular layer dipping down to become continuous with *d*.
d. Granular matrix or reticulum.
e. Superficial molluscum bodies associated with corneous layer.
f. Commencing molluscum change.
g. Fibrous tissue of corium.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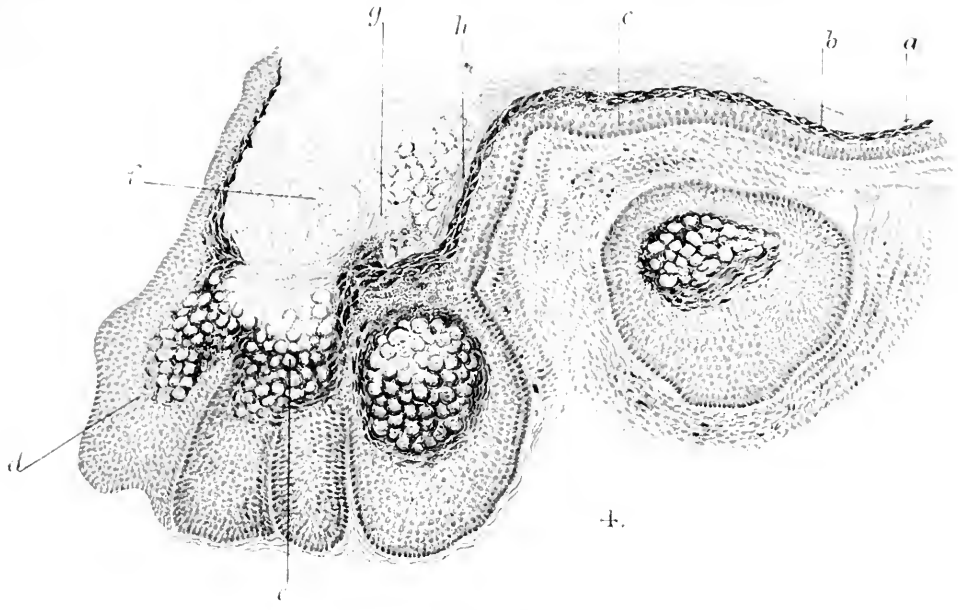

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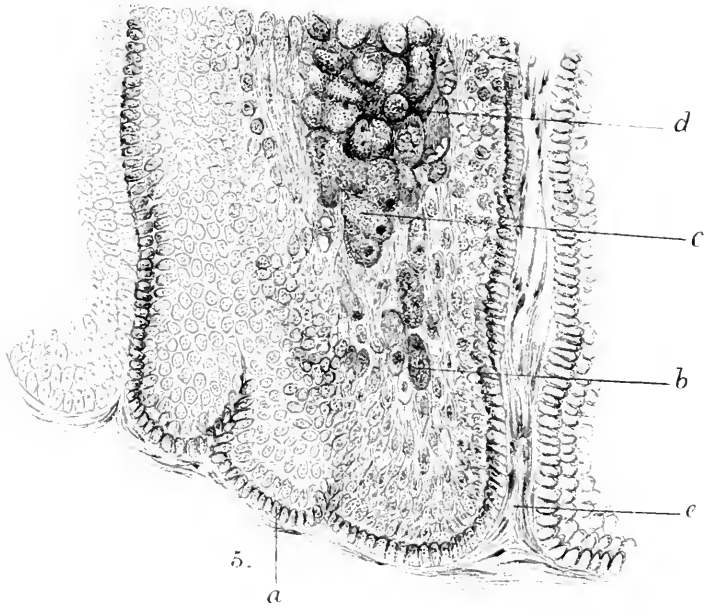

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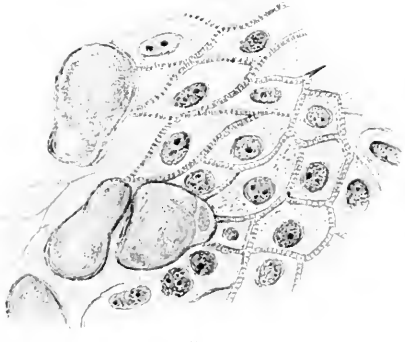

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Plate IV.

Fig. 4.—Vertical section through a minute molluscum-growth, probably a widened and altered hair-follicle. (Hart., oc. 2, obj. 4.)

a. Corneous layer of epidermis.
b. Granular layer.
c. Malpighian layer.
d. Peripheral cells of molluscum growth continuous with rete.
e. Molluscum bodies, with granular reticulum continuous with granular layer.
f. Superficial molluscum bodies.
g. Trabecular masses (?), unchanged corneous cells.
h. Fibrous layer, continuous with corneous layer, intervening between granular cells and molluscum bodies.

Fig. 5.—Part of a molluscum lobule. (Hart., oc. 3, obj. 8.)

a. Vertical peripheral cells.
b. Molluscum bodies in early stages of formation.
c. Fully formed molluscum bodies.
d. Granular matrix (reticulum).
e. Fibrous septa between lobules.

Fig. 6.—Part of molluscum lobule, highly magnified, showing molluscum bodies lying amongst prickle cells; somewhat diagrammatic. (Hart., oc. 3, obj. F. Zeiss.)

THE ANTISEPTIC THEORY

TESTED BY THE STATISTICS OF

ONE HUNDRED CASES OF SUCCESSFUL OVARIOTOMY.

BY

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THERE is hardly any department of medical practice in which the reasoning used to support any particular proposition is raised above the ordinary level of the statement of convictions or impressions of a purely empirical character, based solely upon the personal observation of the arguer. This is certainly the case in surgical practice, though here, perhaps, if anywhere, we might expect it to be possible to argue upon a purely physical basis. As soon as this does become possible we shall have made a gigantic stride in establishing rules of surgical proceeding, and shall thereby obviate much of the useless discussion with which we now occupy our time.

With these views, for which I hope a charitable consideration will be granted, I venture to indicate a line of research in which results may be obtained of a more definite and conclusive character than any yet laid before us, and if I have ventured to apply it to the discussion of

the antiseptic system it is because I think that this particular instance affords the best ground for arguments of a purely physical kind, based as it is upon a theory now so well supported that it may practically be taken to be one of the great laws of biology.

To those who have followed closely the elaborate researches upon the phenomena of putrefaction, which have occupied some of the greatest minds of our time during the last fifteen years, it must, I think, be admitted as an established fact that these phenomena arise from the presence of minute living organisms in the air which can be removed from it by a variety of physical means, by which the air is rendered absolutely harmless to the substances which are experimented upon. Further, that so far as we know, no phenomena of putrefaction do occur without the admission of these so-called germs to the substance putrefying, that the putrefactive processes depend entirely upon them and the organisms to which they give rise, and that the origin of such organisms within the putrefying fluid, independently of a sowing of seed in the fluid, though by no means to be regarded as an impossibility, is not yet proved as an actual occurrence. To any who take a general view of the biological scheme from the aspect of the evolutionary philosophy I think it must be clear that the so-called "spontaneous" generation of the early forms of life is a necessary corollary, but it is by no means certain that it is a part of the present process, nor is it likely, in my opinion, that we now have upon the earth such conditions as would render biogenesis possible.

For my present purpose, therefore, it is enough for me to assume, as I do most fully, that the germ theory has been completely substantiated, and that no known process of putrefaction does occur save by the admission of resting spores or swarm spores of some of the many minute living organisms which are invariably associated with putrefactive changes. But concerning this there is another constant position associated with these phenomena. The

materials upon which the experiments have been made, of infinite variety of kind and constitution, have all been dead, and no one has yet pretended that, by the admission of germs to living matter, he has produced the phenomena of the putrefactive changes which constantly result in matter which is dead. To quote the apt illustration given by Dr. Wm. Roberts in his masterly exposition of this most difficult subject, the ordinary hypodermic morphia syringe will inoculate inevitably a sterilised solution of dead organic matter, but amongst the hundreds and thousands of hypodermic injections which are made daily, no one has yet declared a single instance of putrefactive changes resulting from it in the healthy, or even in the diseased, human body.

It will, therefore, be seen that the application of the facts of the germ theory of putrefaction to the phenomena of diseases of living tissue is met at once by an overwhelming difficulty, to the removal of which none of the adapters, so far as I have seen, have as yet applied themselves. Granting that the same germs which would inevitably produce putrefaction in a dead infusion of beef are constantly admitted to wounds, there is not the slightest particle of evidence that they do produce any change whatever upon living tissue, still less is there any evidence that the changes which occur in the numerous varieties of what we call blood poisonings, even when they are of an undoubtedly local origin, have the slightest analogy to those seen in a putrefying dead infusion. The mere presence of bacteria in the fluids of wounds, or in fluids enclosed in cavities, whilst offering many difficulties to the adapters of the germ theory, prove nothing for their position until they have shown that these organisms ever do occur in fluids or tissues which are truly living.

The difficulty, therefore, is this, that what we call vital action, for want of a name based upon a better understanding of what it is, places living tissue in an altogether different category from tissue in which the phenomena of life are no longer present.

Now, this is consonant with every-day experience. If a decaying hyacinth bulb or a rotting apple be examined, the presence of the minute forms of life is found to be absolutely confined to those parts where the changes have been effected, whilst those parts to which the rot has not extended are found absolutely free from them, and the difficulty of the adaptation of the germ theory is simply this, that its advocates have assumed that the invasion of the germs is the cause of the decadence of the vital phenomena and the ultimate death, whilst there is the alternative still undiscussed and certainly undismissed—that the decadence of the vital powers, due to some cause possibly yet unknown, is that which gives the germs their protential ascendancy, and enables them to do what, during full vital action, they were wholly unable to effect.

If the views of the germ theorists were correct we ought to expect that no operation could be done successfully without rigid antiseptic precautions. The slightest cut of the skin ought to be followed by septic poisoning. There ought to be no difference in the mortality of operations in small and in large hospitals, in town and in country. In fact, if germs could have had the unbounded influence which is claimed for them by many antisepticists surgery must long ago have been an extinct art, if, indeed, it ever could have struggled into existence.

The uniform experience of operating surgeons has taught them that the success of their work will depend upon three factors—the condition of the patient, the condition of his surroundings, and the nature and extent of the operation performed.

Of these three, most undoubtedly the most uncertain factor is the first. What condition of the system it is which is favorable to operations is almost unknown. I must base my conclusions chiefly upon my own work, and in my special operation of ovariotomy I am perfectly certain that apparent perfect health is by no means a certain indication of a power of resistance to those conditions, whatever they be, which result in so-called septic poisoning.

The second of the factors, the condition of the surroundings of the patient, contains elements of far greater certainty. It has approached the position of a statistical law that the death rate is in constant harmony with the density of the population, and that when the density exceeds a certain minimum of safety there are introduced specific septic diseases, as typhus fever, which are wholly unknown under other conditions, and which, even after the danger density has been reached, attack certain individuals only, and not all, for reasons which can be expressed only by saying, as I have already said, that the living tissues of those affected could not, and did not, resist the septic influence.

Every advance we make in sanitation shows that this factor, the condition of the surroundings of the patient, is of extreme importance.

The third factor which influences surgical success is the extent and importance of the operation performed. Everybody knows that while amputation of a finger is probably fatal in not more than one in ten thousand cases, nearly one half of all amputations of the thigh die. Now, if the adaptation of the germ theory to surgical practice were as promising and as legitimate as some of its supporters allege, we should have had the remarkable result, previous to its application, that amputations of the fingers and of the thigh ought to have approached one another in mortality to an infinitely larger extent than they have done.

If the contact of a bacterium germ upon a wound could be the source of blood-poisoning then the size of the wound and the nature of the operation could make but small difference in the result, and a wound into the theca of a finger tendon, and one of similar size into the peritoneum of another patient in the same ward, ought to have had very similar risks. But, as a matter of fact, they did not, and we are forced to the conclusion that, even if bacterium germs lighting on wounds were the cause of much surgical mortality, that the power of vital

resistance by the tissues or the condition of the patient, and the extent and nature of the operation, are of infinitely greater importance as factors in the general result. This logical difficulty has evidently occurred to many of those who carry out Mr. Lister's adaptation of the germ theory to surgical practice. I have seen a rigid antiseptic occupy an hour and twenty minutes in making incisions a fraction of an inch in measurement, and barely skin deep, for the purpose of laying bare the tympanic membranes of an infant in whom they had been congenitally covered, the protraction of the operation being due solely to the antiseptic precautions. Such a proceeding produced in my mind a variety of emotions, chief of which were admiration for the enthusiastic consistency of the operator and sympathy for his evidently wearied audience. The just criticism of such a proceeding is, Has any one ever seen such a trivial operation result fatally from septic poisoning, unless in some such hospital, as is described by John Howard in 1780, as the Hotel Dieu, with three patients in each bed? I certainly never have, and I have performed some thousands of them; and if it were necessary to take one hundred minutes to do what I could do in three I, for one, should seek my livelihood in some employment other than that of an operating surgeon.

The logical conclusion to be made from the facts is, therefore, that in minor operations germs have never, or at least hardly ever, any influence at all, and that in major operations the condition of the patient is of immense importance in enabling him to resist the influences, whatever they may be, which result in what we call the septic condition.

In any examination of the question there will of course be the primary difficulty, that it is by no means agreed as to what constitutes a major operation, and that between different operations which are admitted as major, there are known to be very different rates of mortality. Thus, amputation of the leg is proved, in my book on 'Hospital

Mortality,' to be more than twice as fatal when performed for accident as when performed for disease. It must be perfectly evident, therefore, that any examination of this question must be conducted upon the usual rules of statistical investigation, the chief of which is that similar and not dissimilar accidents should be grouped together. Any mere statement, therefore, of the general percentage of deaths from septic diseases on the general hospital or other population are absolutely worthless unless they be most carefully analysed, and they are, of course, open to the still further objection that what constitutes a death from septic disease is by no means a perfectly accepted definition.

There is a popular belief that statistics can be made to prove anything, than which there is no popular belief more erroneous. Statistics alone seldom prove anything, certainly they never explain anything. Thus, the Registrar-General's tables tell us that there are certain death quantities which are perfectly constant, and they establish the fact that half of all our human mortality occurs before the fifth year of life. But this neither proves nor explains the cause of this mortality, nor does it even explain its factors, until a more careful analysis of individual cases is made. Therefore, nothing whatever can be proved for or against the adaptation of the germ theory to surgical practice by mere statistical statements. But in spite of this, statistics may be made to show exactly in what direction analysis of individual instances should be made, and, therefore, they alone are capable of forming the first step of accurate inquiry. First, let us ascertain as fully as possible what the facts are, and then analytical arrangements of them will certainly afford a more or less complete explanation of their method of production.

Some of the warmest supporters of the antiseptic system uphold it, on the ground that under its protecting influence operations can now be undertaken successfully which formerly were impossible, such as laying open joints, &c. Before I proceed to the immediate subject of

my paper, I desire to point out that this is an argument which cuts both ways, and which seems to me to form one of the great dangers of antiseptic surgery. The immense favour with which the antiseptic system has been so widely received, is most undoubtedly due to the fulness of its promise as a royal road to surgical success, as a something which puts the skilled and the competent upon a level with the inexperienced and incompetent; and I know that there have been abundant instances of bitter lessons already, that even an antiseptic spray will not condone the want of manipulative dexterity or the absence of readiness in emergency.

There is, further, an inevitable result in the full acceptance of this germ-theory adaption, that the other factors —the condition of the patient and his surroundings—will be relegated to unimportant positions, and we shall have a great risk of inducing an inattention to general hygiene and the incursion of rash experiment, which will do more harm than antisepticism will do good, even if everything claimed for it is true. That this is no fancy sketch, is proved by what Mr. Spencer Wells narrated in his lectures at the College of Surgeons on abdominal surgery.

Even if it were true that joints can be opened now as they could not be before, the question must first be answered before the fact can be credited to the antiseptic system—Is this more than was to be expected from general surgical advance, seeing that in 1866 ovariotomy was opposed as an operation of an altogether unjustifiable character, and yet *before* the antiseptic system was applied to it, it had become more justifiable than lithotomy or amputation below the knee?

It is, therefore, evident that for the proper estimation of all results a most careful analysis must be made of all known or possible factors contributing to them. I have already said that no rough aggregate of operative results would be of the slightest use for any purpose whatever. The estimation of the results in one particular operation, such as ovariotomy are, on the contrary, of immense import-

ance, not as proving or explaining anything, but as pointing to the directions in which research may be made with profitable result. Thus, it must be evident to every one that a large group of one hundred ovariotomies must present features more similar to those of another set of a hundred than can probably be got in any other surgical comparison which is possible; and it is a probably correct assumption that if the same surgical skill and patience, the same attention to minute details, and the same state of the surroundings were common to the two groups, their resulting mortality would be identical or nearly so. But if there is one thing we value more than another, as being likely to contribute to success in surgical operations, it is personal experience; and we, therefore, may fairly expect that with each succeeding hundred ovariotomies the mortality will diminish, owing to the increasing skill of the operator. And this is the case notably in the practice of Dr. Keith who, beginning with 11 per cent., went successively down to 8 and 6, before he began to use antiseptics; and of my own experience I can only say that whilst I had nineteen deaths in my first fifty operations, I had only three in my second fifty, and I fully expect that in my third group these good results will be at least maintained.

Now, can such a method of examination, that is, by mortality results, indicate anything more than the mere increase of general skill? Can it be made to show for some individual proceeding an advantage over some rival practice? Most certainly not. A recovery after an ovariotomy is the sum of a number of details, all of which were efficient. A death, on the contrary, may be the failure of one only, and that may be or may not be under the control of the surgeon. Thus, of my three fatal cases in my second fifty, two were deaths due to details wholly beyond my control, and having no relation whatever to either the antiseptic system or any other of the operative details. The third death was due, as far as I could determine, to the irritative effects of thymol used with full antiseptic details. Two of these deaths were antiseptic out

of twenty-nine cases treated antiseptically, whilst of twenty-one cases treated without antiseptic precautions, I had only one death, and as she died within three hours after the operation, the want of antiseptic precautions could have had nothing to do with her death. From this group of cases, therefore, the argument would be wholly against the antiseptic system, and though my impression is that the conclusion would be a just one, yet the argument is absolutely fallacious, as all such are.

In the discussion of this question, which occurred a few months ago, the only statistical argument of the slightest importance was given by Mr. Spencer Wells, who said that a very marked improvement had occurred in his results since he had used antiseptic precautions. But nearly concurrently with his adoption of germicides, he adopted the intra-peritoneal method of dealing with the pedicle, a method which has been superlatively successful in the hands of Dr. Keith, and to which chiefly I attribute my own rapidly increasing success. Thus, Mr. Wells' mortality improvement argues nothing in favour of antiseptics, but probably far more, in my opinion, for the short ligature.

Here, for instance, are my own results, which point conclusively in this direction.

		Per cent. mortality.
(17 cases).	Ligature, non-antiseptic .	5·9.
(29 cases).	Ligature, antiseptic . .	3·45.
(36 cases).	Clamp, non-antiseptic . .	25·
(26 cases).	Clamp, antiseptic . . .	27·

Dr. Keith, in the record of his cases, does not give completely such details as afford a perfect statement of his results based upon the various methods of treating the pedicle, but he tells us that in his first 50 cases he used the clamp 48 times with 9 deaths. In his second 50 he merely indicates that his confidence in the cautery is returning. In his third 50, the clamp was used 34 times, with 7 deaths, and the cautery and short ligature 15 times without a death; and now I understand from himself he

has entirely abandoned the extra-peritoneal method of dealing with the pedicle, as indeed has everybody else, by reason of Dr. Keith's unprecedented success with the intra-peritoneal method, even before he adopted antiseptics.

It is made very probable by this, as far as my practice is concerned at least, that the improvement is due chiefly to the introduction of the intra-peritoneal treatment of the pedicle, and as far as can be seen there is nothing to be credited to antiseptic precautions, for the difference in result between the ligature used under antiseptic precautions and without them is no more than can be safely referred to increased personal experience, as the non-antiseptic cases were of course of earlier date. However, I am not prepared to say the antiseptic system is absolutely without result, for it certainly must be admitted that occasionally an operation will be done under some unfortunate circumstances, so that immediate and direct poisoning of the wound may take place; and that any substance having the properties of carbolic acid, and used according to Mr. Lister's plan, may have the effect of preventing disaster. My own belief is that if this were effectual only once in a hundred operations we should adopt these precautions in the whole hundred, unless it could be shown that they were productive of greater harm in other directions, and I am bound to say I do not think they are.

In a previous communication to the Society I have already said that some of the details of the antiseptic method prevent the healing of wounds, and this is an absolutely uniform experience in my practice. I have never yet got an ovariotomy incision to heal with Lister's dressings, as I nearly always did with Mr. Wells' dressing of plain dry lint, and to the dry lint I intend to go back. Some of the antiseptic disciples have told me that my failure in this direction is due to the inefficacy of my methods, and it is said of others who fail equally that it is due to their want of belief. Now, such arguments are really childish. In my own case the want of belief is not

a difficulty, for I have the most profound belief in the germ theory of putrefaction, but I utterly fail to appreciate its application by Mr. Lister. The statement that my methods are faulty is, of course, a charge against either my intelligence or my honesty, and I reply that both are quite equal to the average, that I as earnestly desire to cure my patients as can any of my confrères, and that, in spite of the failure of the method, my patients get well in a proportion which is greater than has yet occurred in the parallel experience of any other ovariotomist, that is to say, in Dr. Keith's second 50 he lost 8, and in Mr. Wells' he lost 17, whilst I have lost 3.

But this statistical research points out that there is a method which, so far as I know, is yet a wholly untried one, and which may give very important indications of the value of any individual detail, such as the antiseptic system, especially one like it, based on a theory of a purely physical kind, and having a claim for preventing febrile complications.

This method is based on an examination of the method of recovery of those in whom the sum of the details is such as to lead to that satisfactory result.

The basis of the antiseptic claim is that the system prevents septic poisoning, that is, septic or surgical fever. Every one who has watched a number of ovariotomies knows that by far the larger number of deaths occur from the incidence of fever, and that the pulse and temperature rise progressively, though perhaps with intermissions, till they reach the fatal vanishing points. With a single exception this is true of all the deaths I have had. If, therefore, the antiseptic system favours a larger number of recoveries by preventing septic fever, it is an absolute certainty that the recoveries will be uniformly and correspondingly facilitated, inasmuch as in non-antiseptic cases the germs will enter every peritoneum and will theoretically produce fever in every case, and only in those cases where there is a sufficiency of an unknown something which counteracts the septic poison will recovery be obtained.

Equally according to the theory will the germs destroyed by the antiseptic precautions enter the peritoneal cavity harmlessly, dead and unfit to produce septic fever.

Another step in the syllogism is that as the temperature and pulse curve are uniformly admitted to represent the course of any case involving febrile action, if the antiseptic system makes its claims justly, ovariotomies performed under its precautions ought to indicate a more even and less febrile course of recovery than the non-antiseptic cases, and this should occur independently of all other details of the operation.

I would put the possible conclusion briefly thus :— If we find a marked difference between the curves of cases treated antiseptically and those not so in favour of the former, then I think I may say that more has been done to establish Mr. Lister's view than anything I have yet seen. If there be no difference, then the question is just where it was; but if there be a difference on the other side, then I think the application of the germ theory to surgical practice will be certain to fade away from professional and popular acceptance just as many as fair-looking visions have done before.

Further, a just grouping of cases may display wherein consists the real road to surgical success.

The line of research which I have taken is already sufficiently indicated, but I may further say that, like Mr. Spencer Wells, I have a separate note-book for every case of abdominal section, in which every particular of the slightest importance is entered. The pulse and temperature observations are taken by trained nurses, and they are constantly subject to my personal testing, so that I think it hardly possible that error of any appreciable extent can have been introduced. If there has been, it is of course quite as likely to have occurred on one side as on the other ; and if on any certainly on both. At any rate, the observations were made without the slightest reference to any purpose other than as a periodical record of the progress of the cases, and therefore any conclusions

they may point to can have no prejudice about them. I may say further, that the remarkable regularity of the curves is extremely suggestive of their accuracy, and I ought to add that in three instances I was able to detect errors by a re-examination of the data constituting points wherein a remarkable deviation from uniformity occurred. My experience in this is so very striking that I feel persuaded that if I could have dealt with hundreds of cases, instead of scores, I should have been able to present curves of absolute standard uniformity. As they are, I believe their value to be great, but not absolute, merely by reason of the smallness of the figures from which they are constructed, and it is almost unnecessary to point out that, under such circumstances, any error or accidental variation has a decreasing importance just in proportion to the amount of material employed.

The plan used is of course very simple. Taking the morning and evening observations of the temperature and pulse for each case during a period of ten days, I constructed for the morning and evening of each day an average of the total observations under discussion, and marked this upon the graphic paper. I took ten days as the limit, because I believed that this exceeded by at least three days the average period of stable recovery in cases of ovariotomy, and because it was the limit to which the observations could be extended with full material. My general impression was that a successful ovariotomy was practically well on the sixth day, but it will be seen from the charts that, like other general impressions, this is quite a mistake, for convalescence is not fully established till the eighth day, and is certainly not complete on the tenth. Therefore, probably my conclusions would have been better with more extended observations. I also see now that my statements would have been more perfect if I had carried out my figures to two, or even three places, but this would have involved an amount of labour for which I may frankly say I was not prepared.

Concerning the mere duration of recovery, some inter-

esting remarks may be made. Taking the curves of Fig. 11, which are constructed from the whole of the 100 cases, it is quite evident from the pulse curve, still more from the temperature curve, that recovery takes a sudden progress forwards on the eighth day, but that it is not then complete. On the sixth and seventh days the temperature gives distinct indications of exaltation, especially nocturnal, and this is clearly seen, on examination of the constituent curves, to be due to the suppuration consequent on the separation of the clamp, and probably, also, on the formation of stitch-hole abscesses.

The consideration of this curve (Fig. 11) leads me to say that I attach less value to the temperature curves than to the pulse curves, for the reason that the temperature during the course of recovery from ovariotomy is liable to extraordinary explosions. I have repeatedly seen a patient's temperature rise three or four degrees, and in one recent case six degrees centigrade, without the slightest apparent reason, the exaltation lasting from half an hour to three or four hours, and then the temperature would fall quite as rapidly, leaving the patient without any appearance of effect, or any record of it, save on the chart. This is not the case with the pulse curve, for if that rises the general appearance of the patient, and other signs and symptoms, amply prove that something is wrong, and the changes of the curve do not occur or give way with rapidity, but always gradually. Therefore, temperature readings require to be far more numerous than pulse readings to give the same uniformity of result. Pulse readings are also not subject to such influence by limited suppuration as temperature readings, and this is shown by the marked difference in the temperature and pulse curves (Fig. 11) on the sixth and seventh days. Further, the temperature rises almost uniformly at night during the progress of recovery, whilst the pulse does not do so after the fourth night, and this confirms my general impression that the fourth night is the critical night of the course of an ovariotomy. My conclusion is

finally confirmed by the fact that, whilst I have seen a case end badly without the temperature rising to any remarkable height, I have invariably found the pulse rise continuously till it disappeared.

The constituents of the curves on Fig. 11 are broken up into minor curves in the preceding ten figures, each such curve consisting of an average constructed from a varying number of cases having some pronounced feature of their treatment in common. Fig. 1 includes 27 cases in which the pedicle was dealt with by the clamp, without any antiseptic precautions having been employed. Fig. 2 includes 19 cases where the clamp was used with full and rigid antiseptic precautions throughout the case. Between these two groups there is really very little difference. The non-antiseptic cases had slightly higher temperatures during the second, third, and fourth days, but during the same days the antiseptic cases had a more pronounced difference between their morning and evening temperatures, and my impression is that this is a more serious condition than a more uniform range, even if fractionally higher. The non-antiseptic cases have a decidedly better pulse curve than the antiseptic cases, and, as far as this indication goes, their recovery was one day shorter than the antiseptic cases.

The curves on Fig. 6 are constructed from 26 cases in which the pedicle was dealt with by three intra-peritoneal methods without antiseptic precautions, these three methods being given in sub-groups in Figs. 3 and 4, and one case in which I used the cautery, but of which I have not thought it necessary to give a special figure. Fig. 6 makes it perfectly certain that in cases where the intra-peritoneal method is employed, recovery is more even and speedy and much less anxious than when the clamp is used, either with or without antiseptic precautions, and this is absolutely in harmony with the corresponding death rates. Judging from both temperature and pulse curves, the non-antiseptic and short ligature method gives a better and shorter recovery than any other; and if

we compare Figs. 4 and 5, where the short ligature intra-peritoneal method is contrasted in 15 cases where it was employed without antiseptic precautions, with 28 cases where they were rigidly carried out, the non-antiseptic cases, we find, make somewhat, though not very much, better recoveries than the antiseptic cases, the difference consisting chiefly in the nocturnal acceleration of temperature on and after the fifth day. This I believe to be due to the uniform suppuration of the wounds which has occurred in the antiseptic cases, and which I believe to be entirely due to the irritating effects of the gauze dressings. This irritation was more perceptible when protective was used than when it was omitted, and I think the whole effect was due to the gauze preventing the drying of the wounds.

I have gone back to the dry lint dressings in my most recent practice with infinitely more satisfactory results in the wounds, and I have just read a paper of Prof. Hegar, in which he says that after having performed oophorectomy a large number of times with antiseptic precautions, he has given them up, as he got better results without them. Here it occurs to me to mention a very singular fact, that after the sixth or seventh day, or at most the eighth, I always gave up the antiseptic dressings and used the familiar red lotion, or oxide of zinc ointments, to facilitate the healing of the wounds. Under these old-fashioned circumstances, germs, of course, were allowed to do as they liked upon the wound, but not a single case died. Why was this? I cannot answer the question, further than by repeating the fact in another form, and supposing that in six or seven days after an ovariotomy the patient gets into some condition which enables her tissues to resist septic or germ influence.

It is impossible not to be struck with the remarkable regularity in the recovery progress of the 15 cases where the short ligature was employed without any antiseptic precautions, forming, as this group does, the most uniform of all the curves. The highest temperature is the re-

actionary rise within twelve hours after the shock of the operation, and that rise is limited to half a degree. After that the temperature is practically normal. The highest pulse rise is at the same time, and amounts only to 110; then from the morning of the third day there is such a rapid and uniform fall of the pulse curve that it becomes practically normal on the fifth day. Contrasting these curves with those of the clamp cases, whether used with antiseptic precautions or without, it is absolutely impossible to avoid the conclusion that the difference in recovery is due to the extra-peritoneal method of dealing with the pedicle, and there is an entire absence of any evidence of influence on the part of Listerian antisepticism.

Again, I have constructed curves in Figs. 7 and 8 for the purpose of contrasting the intra- and extra-peritoneal method of dealing with the pedicle, without reference to antiseptic precautions, and the result is certainly very remarkable. In the intra-peritoneal cases the important temperature exaltations are over on the third day, whilst in the extra-peritoneal cases the high temperature is continued till the seventh day, the evening temperature of that day being exactly the same (37·6°) as the highest temperature of the intra-peritoneal cases, this latter being practically the reactionary rise. The pulse curves of the two groups show still more startling contrast. The highest pulse of the intra-peritoneal cases is 104 on the evening of the second day, the reactionary rise; and from the evening of the third day till the morning of the fifth, it falls uniformly from 102 to 90. On the second day of the extra-peritoneal cases the pulse is 107, and it does not fall below 100 till the morning of the eighth day.

Finally, I have constructed Figs. 9 and 10 with regard purely to the employment of the Listerian method of performing operations, regardless of the methods of dealing with the pedicle, and I must point out that if the claims for this system which are made can really be substantiated in such an operation as this, its influence should be paramount, and should over rule all other details in its effect

upon recovery. But it is perfectly clear that it does not do so. The high temperature on the sixth and seventh evenings, visible in both figures, is derived clearly from the group of clamp cases. The general curve of temperature is decidedly higher for the antiseptic cases than for the non-antiseptic cases, and in the pulse curve the recovery of the non-antiseptic cases is pronouncedly more even and shorter than in the antiseptic cases.

It is not necessary to attribute this delayed and more difficult recovery to the use of Mr. Lister's various processes. It is quite enough to show, as these curves clearly do, that the so-called antiseptic processes of Prof. Lister have not facilitated the recovery of my patients, as is asserted to be the case in the practice of others. These assertions so far have been confined to the statement of general impressions, whilst I have given a physical basis for my impressions, which is capable of accurate estimation, incomplete it may be, but more complete than any attempt yet made, and for any suggestions of improvement in the method I shall be very grateful. In fairness to myself, I must conclude by saying that I have followed closely upon all the published details of those who advocate the antiseptic system, that at first I was strongly impressed by its influence, but that it was only after a large experience of it that I began to suspect that I was attributing to it effects due to other causes. My suspicions are fully confirmed by the facts I have given above, and though they will cause me to modify my conduct of the details, especially in the dressings, yet I shall continue to employ all the antiseptic precautions during operations, as I have done hitherto, until a wider experience decides whether they may not all be given up. I must once again admit the great lesson that the antiseptic system has taught me, that there is no detail in the performance of an ovariotomy, or in the preparation for it, so insignificant that it may be trusted to a deputy; and there is no circumstance so unimportant that a neglect of it may not lead to an important result.

DESCRIPTION OF CHART

OF

TEMPERATURE AND PULSE CURVES

IN

MR. LAWSON TAIT'S ANTISEPTIC AND NON-ANTISEPTIC CASES OF RECOVERY AFTER OVARIOTOMY.

Diagrams I, II.—46 clamp cases.
I.—27 non-antiseptic.
II.—19 antiseptic.

„ III—V.—54 cases (including 1 cautery case), in which the pedicle was treated by the intra-peritoneal method.
III.—10 écraseur cases.
IV.—15 ligature cases, non-antiseptic.
V.—28 ligature cases, antiseptic.

„ VI.—26 intra-peritoneal cases, non-antiseptic (1 cautery, 10 écraseur, 15 ligature).

„ VII.—54 intra-peritoneal cases, antiseptic and non-antiseptic (1 cautery, 10 écraseur, 43 ligature).

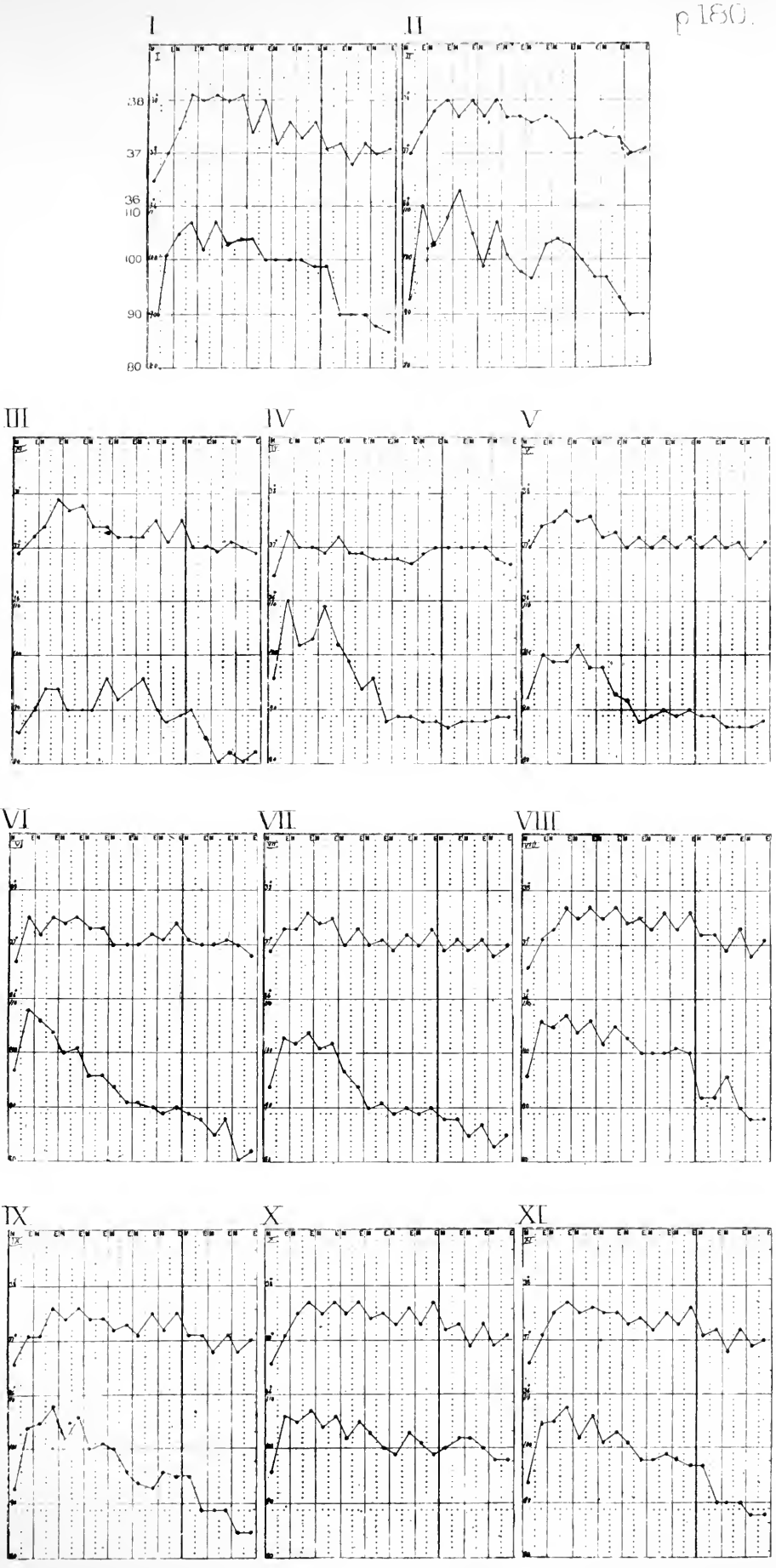
„ VIII.—46 extra-peritoneal cases, antiseptic and non-antiseptic (all clamp).

„ IX.—53 non-antiseptic cases.

„ X.—47 antiseptic cases.

„ XI.—Average of 100 cases.

Med. Chir. Trans. Vol. LXIII
p. 180.



Mintern Bros. lith

NEPHRECTOMY[1] BY ABDOMINAL SECTION.

BY

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As this is an unusual operation I may perhaps claim some indulgence if I explain generally the grounds upon which it was undertaken. This appears necessary from the fact (of which I am assured on high authority) that the whole subject of extirpation of the kidney is not yet familiar to English surgeons. It will, I hope, appear at the same time that my facts and data were in a great measure already gathered and fully weighed before I had any definite prospect of peforming the operation; moreover, that the convictions as to its propriety were of no hasty origin, but the plain outgrowth from the consideration of a larger number of clinical facts than it had hitherto been possible for an operator to collect, owing to the recent and rapid growth of the subject. Abroad, only a few imperfect collections of cases have been made; at home, apparently nothing in this direction has been done adequately.

[1] The term "nephrectomy" is employed throughout this paper as shorter than that of "extirpation of the kidney," and as distinct from "nephrotomy," or simple incision into the kidney from the loin. Observation seems to show that such a term will be found convenient in the future.

Since the perusal of Prof. Simon's (of Heidelberg) memoir a couple of years ago, based upon his first successful extirpation of the kidney, the whole question appeared so attractive for many reasons that I had read all other cases which came under notice with special care, and also a good deal of collateral matter.

It appeared that if anything was needed to prove the correctness of Simon's deductions that one kidney can be safely removed from the human body it was furnished by the two cases in which, in 1878, Martin, of Berlin, extirpated healthy movable kidneys successfully, and with manifest benefit to the patients. The next case met with was that of Smith, of New Orleans, also quite successful. Simon's deductions and the consideration of these four cases seemed to lead to but one conclusion, namely, that the operation in its proper place is not only justifiable, but called for.

On seeing the present case, therefore, I felt clear as to the line of treatment to be advised. This opinion was only further strengthened by the evidence of all the cases published at home and abroad which I then collected and tabulated as soon as possible as below. These furnished additional proof of the correctness of Simon's deductions and supplied many data of great value unavailable to him when he wrote, or, indeed, up to the time of his death.

But as this author's original address must always be regarded as the starting-point in any consideration of the whole subject of "nephrectomy," his being the first case in which the operation was performed designedly and on definite principles, it may be well to review briefly the facts and considerations which led him to calmly weigh its propriety and eventually to carry it out with success.

Simon had been asked to treat a case of fistula of the ureter resulting from ovariotomy one and a half years before. Numerous expedients having failed, and the woman's state being truly wretched, the question of removing the offending kidney occurred to him. But

although the annals of pathology afforded him many instances in which one or other kidney had been *gradually* rendered useless by slow disease without any notable effect on the economy, he was not quite satisfied that the *sudden* withdrawal of the functions of one organ would be physiologically tolerated. His first care, then, was to study this point by experiment on healthy animals. But in doing so it would be necessary to distinguish between the mere primary surgical risks of the operation and the physiological, or those relating to the urinary functions.

The careful observation of some fifteen hysterectomies on bitches contrasted with the same number of nephrectomies on dogs supplied data on this point, and satisfied him that there was no more danger in one operation than the other. Both kidney and uterus, it should be remembered, are in the dog almost completely enveloped in peritoneum. Moreover, he found that after some practice he only lost about one out of five cases of nephrectomy. Also that primary and secondary hæmorrhage were far less prominent dangers than had been anticipated. The greatest risk run was from peritonitis, but many dogs recovered without it. Emboli from the renal vein and pyæmia were never met with in his cases, the clot in the vessel being short and limited to the immediate neighbourhood of the ligature. Nor were the physiological effects of the operation to be dreaded. If morphia had been given and the animals survived, they remained stupefied for about twenty-four hours and vomited freely; they ate nothing, lay upon the belly, and did not rise to call. After twenty-four hours these symptoms gradually disappeared, and on the third day they ran about and took their food. If operated on without morphia they presented no appearance of stupefaction, vomited but little, or not at all and very soon regained spirits and appetite.

Simon points out that in estimating the value of these experiments we must not forget that the kidney in the dog is much larger in proportion to the weight of the body than that of man, being as 1 to 170 against 1 to 230

in the latter. Also that the dog has to secrete proportionately more than four times the amount of urea than the human being. If, then, removal of the kidney produce no uræmic symptoms in the dog, the latter ought far less to be looked for in man.

Some other points also come out in Simon's experiments in relation to questions which have since been settled in other ways, but were then matters of debate. First, the general condition of the animals after nephrectomy was excellent in most cases. Again, no albuminuria was produced by the increased intra-vascular pressure presumably caused by the sudden closure of so large a vessel as the renal. Nor did hypertrophy of the heart ensue. The remaining kidney, on the other hand, was found to increase rapidly in size, and to be quite adequate from the first to the double duty imposed upon it.

That the deductions drawn from these observations hold equally well for the human being was soon after shown by Simon's first nephrectomy on the latter as far as one single case could prove anything. Several others from the collection of cases which I subjoin will, I hope, demonstrate the same. Two of these, unknown to Simon, were prior to his operation, and between twenty and thirty have occurred since, and were unavailable to him.

But there is one other question not specially considered in Simon's address which I venture to think deserves particular attention. It is this: *Are we to look for any very striking effect on the vaso-motor system from ligature so close to the solar plexus of the large sympathetic branches and ganglia which surround the vessels of the kidney? Will this, in short, produce much shock?* This could only be determined by direct observation on the human subject, and under very exceptional circumstances. We should be able, namely, to eliminate the effects of that part of the operation which consists in cutting down on the kidney from those due purely to the ligature of the pedicle and its division. There should, in

fact, be an interval between the two operations sufficiently long to admit of observation of the separate and peculiar effects of each.

Now, at least one, if not two, of the cases in my list appears directly to settle this question in the negative. In both, a gash in the loin exposed the kidney, and led to its prolapse through the resulting wound. After an interval (as long as four days in one case) the second operation of ligature of the pedicle and removal of the organ was performed. It is a most striking fact that neither one nor other of these operations gave rise to any real constitutional disturbance.

Case 1 (No. 11).—An Austrian peasant received a slash with a knife in the left flank on January 3rd, 1873. He lost two or three ounces of blood at the time. A couple of hours later a fit of coughing forced out of the wound a soft, pulpy, vascular tumour. This was replaced by a friend, but prolapsed again on coughing. On examination, twenty-four hours later, by Prof. Brandt, of Klausenburg, this was found to be the kidney, slit in its long axis nearly in halves, and pouring out clear urine. It was not at all painful itself on handling, but when drawn upon much pain was felt in the pedicle, but nowhere else; it could be rotated on the latter without distress. Two days later the patient walked to a studio, remained there for two hours while being photographed in various positions and walked back again In fact, his condition was almost that of perfect health. On the fourth day after injury, the kidney was removed by Professor Brandt, apparently *without an anæsthetic*. The pedicle was transfixed and tied in two portions, and the organ cut away with a knife. Considerable pain was felt during transfixion, much less during removal. He had no bad symptoms after this and but slight pain, and left hospital sixteen days later quite well. This appears to me one of the most important cases of the whole series.

Case 2 (No. 13), recorded by M. Marvaud, surgeon-

major to an Algerine regiment, is almost identical, though not quite so valuable for want of equally full details.

A young Arab woman was stabbed in the right flank with a yataghan, the latter dragging the kidney out of the wound. Here it bled for a short time and then became strangulated. The pedicle was ligatured and the organ allowed to slough off, which it did at the end of some weeks. The patient continued in good health the whole time, the secretion of urine being normal. She was discharged perfectly well two months after admission.

Two other cases, though somewhat less simple, may with advantage be mentioned here.

No. 23. Mrs. H—, æt. 55, came under the care of Dr. Smith, of New Orleans, in April, 1879. Eight years before she began to notice pain in the right side, and a little later a tumour. Not long after "another surgeon performed upon her the usual operation for ovarian tumour, without removing the cause of her trouble." The year following another medical man passed a tape seton through the abdominal wall and the lump, for the purpose of causing adhesions around the latter the movements of which were so painful. This operation was followed by hæmaturia. The setons remained *in sitû* for three months and gave some relief, but then broke and came away. Two other attempts six months later to pass in a seton for a similar object failed, the broken ends of two needles besides remaining in the abdomen. Two months later a tape was introduced as before, but with less relief. Her sufferings now rendered her insane, and she spent two years in a lunatic asylum. During this period the second seton came away.

In November, 1878, she had sufficiently recovered to return home, when the tumour was diagnosed to be a movable kidney. As she pressed for its removal, Dr. Smith operated on June 23rd, 1879. He made the usual lumbar incision, forced the kidney into it by pressure on the abdomen, ligatured the pedicle, and cut the organ

away. Nothing of special note, much less anything untoward, occurred during or after the operation. The wound was dressed with weak carbolic acid solution, it suppurated freely until the ligature came away on the tenth day. Little or no pain was complained of and the patient walked about on the eleventh day without trouble.

No. 5 is equally remarkable. A negro woman, five months pregnant, entreated operation for a most painful condition of the kidney. The latter was removed by Dr. Gilmore through the loin, and the patient recovered well and without any ill effect on her pregnancy, which terminated at the usual time.

All these cases appear to show how little impression on the system a simple uncomplicated nephrectomy may produce.

Turning now to the question of uræmia, the evidence of all the cases in the appended table is convincing that it is not to be feared where the remaining kidney is healthy. This has been the great anxiety hitherto, and has been carefully looked for in most cases. But glancing down the column of "symptoms" we are struck by the fact that only in one instance (No. 18) was anything present fairly and exclusively attributable to disturbance of the urinary functions.

The remote effects of the operation have now to be considered.

In the case of dogs, we have seen that months after they were in a thriving condition, and when killed the remaining kidney was found largely hypertrophied. Of the cases recorded in the subjoined table I am able to furnish evidence on this point in six.

Simon, writing of his first patient seven years after operation, says that "she enjoys the best of health." In a courteous letter received from Dr. Martin, of Berlin, a few weeks ago, he says that he still sees the three patients operated on by him in 1878, and that they are in perfect

health. In a later letter he tells me that there is probable recurrence in one of these whom he has since seen, though there was none a year after the operation. Mr. Jessop, too, of Leeds, kindly informs me that the child operated on by him for renal encephaloid (No. 16) was running about for a time after, but that he had reason to believe that the cause of death eight months later was recurrence. In the reports of the other successful cases there is no note as yet on this point. It is sufficiently shown by these, however, that the general health does not suffer by removal of one kidney.

The next point for inquiry is—For what objects has the operation been performed up to the present? The subjoined table shows 28 cases (excluding my own), which may be utilised as bearing generally on this question. Of these the operation was abandoned uncompleted in 2 (for new growths), on account of difficulties, real or apprehended. There remain, therefore, 26 of actual extirpations of the kidney for one cause or other. Of these the diagnosis was ovarian disease in 4, cyst of the liver in 1, and in 1 the organ was removed unwittingly during the excision of a fibro-cystic tumour of the uterus. Thus, there are 6 in which the original design was not removal of the kidney. Of these, 5 died, 1 recovered. There remain, then, 20 *completed* operations, commenced apparently with the distinct aim of excising the organ. The earliest of these was Simon's first, for fistula of the ureter. He has recently been followed by Zweifel, of Erlangen, with the same object, in a similar case, both successful. We then have 2 for the removal of kidneys acutely painful from some unknown cause, 1 death, 1 recovery. Next, 2 for calculous pyelitis, both fatal, but 1 of these only thirty-one days after from pyæmia. Then 3 for injury of the organ, 2 (mentioned above) complete recoveries, 1 fatal; the latter was operated on during great exhaustion, consequent on profuse suppuration following gunshot injury many months before. Then there have recently been 6 for intolerable pain and constitutional disturbance

due to movable kidney; 2 deaths from peritonitis, 4 complete recoveries. Next come 4 for neoplasms of the kidney (one by Langenbuck, a little obscure, however); recovery in 3, death from septic peritonitis in 1 on the third day. To this set for neoplasms, might now be added those 2 fatal cases in which the operation was abandoned. Finally, we have one operation for pyonephrosis, quite successful.

Although any minute comparison of the number of deaths to successes would, taken alone, be quite worthless at present as bearing on the propriety or the reverse of the operation, a glance at this point is not without interest.

Out of this whole 28 cases, 14 recovered and 14 died. Excluding those 6, where the original aim was not nephrectomy, of the 22 remaining 13 recovered and 9 died. Two of the latter were uncompleted operations, and desperate from the first.

But the consideration of the causes of death in these 9 cases will be more instructive. Of 1 (No. 9) it is reported that the patient died within a week, but that no cause was found post mortem. Four died of peritonitis after abdominal section, 2 of shock ten hours after operation (the aorta had been ligatured in one to arrest furious hæmorrhage), 1 of pyæmia. Of the ninth case the cause of death sixty-five hours after operation is not noted.

The successful cases now offer several points for consideration. First of all, look at the matter as we will, 14 patients, one of whose kidneys has been extirpated, have recovered completely. Let us take, however, only those 13 in which the nephrectomy was designed *ab initio*. Firstly, the immediate object of the operation was attained, and the patients were restored to health in all (possible recurrence in 2 excepted). It is a striking fact, again, that only in 1 (No. 18) were any symptoms exclusively referable to the intrinsic nature of the operation observed, such as drowsiness, rigors, &c., setting in at once. In most of the others it is distinctly noticed that there were no prominently bad symptoms as regards the urinary func-

tions. Again, we have seen that there was no shock after simple cases, nor is any particularly noticed in the others.

As to the mode of procedure, we must be on our guard against generalising too freely upon so few data, but the following points may be noted.

Of our 13 successful cases 9 were lumbar sections, 4 ventral. To the latter might be added that other successful case (No 12), where nephrectomy was not designed from the first.

Of the 9 fatal cases the operation was lumbar in 5, and ventral in 4. To these latter might be added, as in the last instance, 5 others not originally intended to be nephrectomies.

On the surface these facts would appear significantly in favour of the lumbar operation, but they lose much of their force when we study the nature of each case for itself.

If I may be allowed to express a general impression, the result of a careful study of these cases, together with experiments on the dead body, it would be this. The lumbar operation appears best suited for removal of healthy or comparatively healthy kidneys, if this be ever justifiable; also in those cases where there has been much perinephritic inflammation; also for pyo- and hydro-nephrosis, and, perhaps, also for the smaller new growths of the organ, especially if fixed. The ventral incision, on the other hand, seems best suited for taking away movable kidneys, whether healthy or containing neoplasms, especially if the latter be large; also for the ordinary cysts of the organ not fixed by inflammation. The difficulty in the case of neoplasmata of removing the lymphatic glands in and about the pedicle of the kidney (which are known to be one of the earliest seats of recurrence) by the lumbar incision, would influence me much in favour of abdominal section, by which the root can be better reached.

Two more points may here be noticed as bearing more particularly on the case now to be described.

First, I found recorded 6 cases of nephrectomy for

movable kidneys, which were causing intolerable pain and constitutional disturbance; 2 were fatal, 4 quite successful.

Secondly, I had before me 6 cases where the operation was for new growth of the organ. Two of these were desperate, and abandoned before completed. Of the remaining 4, 3 recovered, and 1 died of septic peritonitis.

It was with almost all these facts before me that the following case had to be considered:

S. W—, æt. 21, servant; admitted into University College Hospital, November 3rd, 1879. Eight months previously noticed an aching pain in right loin. This was not severe enough to oblige her to give up her employment. She had had very hard lifting and carrying work for some months before admission. Menstruation irregular for last few years. Three months before admission had a severe attack of pain in region indicated, succeeded by vomiting and the passage of very dark bloody urine. A medical man then detected a small tumour between the crest of the ilium and last rib at the painful spot. When this was handled she felt pain, nausea, and occasionally vomited. Since then she has only been able to get about her work at intervals, having soon to return to bed on account of recurrence of the above symptoms, accompanied by hæmaturia. No cause was assigned for the tumour. The family history is good.

Present state.—A remarkably movable oblong tumour is felt on the right side, its long axis running upwards, backwards, and outwards. The abdominal wall is distinctly bulged out over it, most so at a spot about three inches directly above the anterior superior spine of the ilium. Palpation shows its inner border to reach the umbilicus, the outer a line drawn upwards from the junction of the middle and anterior third of the iliac crest. Above it reaches the last rib, below to within a finger's breadth of the iliac crest. The abdominal wall moves

freely over the mass. The surface of the latter is smooth but nodulated, one lump forming the prominence of the skin already noticed. Its edges are rounded and well defined, its shape irregularly oval; it is tender to the touch and pressure, the latter producing nausea, and sometimes vomiting, especially when exerted in an upward direction. The mass is freely movable from side to side, less so from above downwards. It rises and falls with the respiration; it is unconnected with the liver. A tympanitic note is obtained all round and over it except at the prominence alluded to. There is no hydatid fremitus or pulsation direct or communicated. The right flank does not appear particularly void. The tumour varies with position.

The general health is fairly good, but there is slight anæmia and occasional hæmaturia: constipation marked before admission.

From the study of these facts and the patient's condition, I felt justified from the first in urging the removal of the mass by operation. The argument was this. There appeared to be little doubt that the tumour was renal. I held it to be a movable kidney, with, in addition possibly, a new growth seated in it. But supposing it to be simply a floating kidney, it still ought to be excised. For the patient's life was rendered comparatively useless as well as wretched by the recurrent attacks of pain and other symptoms. That the suffering was very severe we had opportunities of observing, besides her own statements. I had read Keppler's very interesting analysis, in 'Langenbeck's Archiv' of 11 cases of movable kidney producing intolerable suffering and much constitutional disturbance, and rendering the patients' lives a burthen to themselves, and in some cases to others. So much so was this the case in one instance that the sufferer became melancholic and committed suicide, the diagnosis of floating kidney being confirmed by the autopsy. Out of these 11 Keppler had prevailed on Martin to perform

nephrectomy in 2 (both successful). The same operator soon after removed a kidney similarly affected, also with success. In Smith's case (No. 23) the patient was eventually driven out of her mind by the same condition, but on leaving the asylum was relieved of all her troubles by the removal of the floating organ. Gilmore's case of the pregnant negress seemed significant too. I felt satisfied that the present patient's case was as bad as these, and called for operation as urgently. But if the copious hæmaturia and other symptoms, together with the rapid increase in the size of the tumour observed by the patient, were to be interpreted as pointing to a malignant growth of the kidney, there was all the more pressing need for its removal. The operation was of course a grave one. But it appeared clear from careful study of this series of cases that there was nothing intrinsically dangerous in the removal of the kidney apart from the ordinary risks of ventral and lumbar section. I was satisfied, and still am, that there is no more risk than in ovariotomies. But this was more urgent than many ovarian cases, if malignant much more so. It was not more contra-indicated than many well-recognised operations of expediency.

These, then, were the chief grounds for removal of the tumour. And when some weeks later the case was placed in my hands by the kindness of Mr. Heath, I was quite ready to operate as soon as possible. This feeling was further confirmed—1st, by the changes found in the tumour when I could now examine it; 2nd, by the several recent attacks of intense pain and profuse hæmaturia; 3rd, by the conviction that we had before us a malignant growth of the kidney; and, finally, by the now unanimous opinion of my colleagues, Dr. Graily Hewitt, Dr. Wilson Fox, Dr. Williams, and Mr. Heath, that an operation was justifiable.

The points noticed after admission were briefly as follows:—First, that for the fifty days the patient was in the house hæmaturia was almost constant. The urine is only once noticed as free of blood. Hæmaturia was

profuse about every six or eight days, gradually decreasing to rise suddenly again. This bleeding almost inevitably made its appearance after manipulation of the tumour, and was preceded and accompanied by intense pain in the latter. The pain also was most constant in the same way, and was accompanied with nausea or vomiting during the paroxysms. For two and a half days before operation these prevented anything lying on the stomach. The growth of the tumour was very remarkable. Just before operation it appeared quite double the size observed on admission. It now measured in the transverse axis of the body $7\frac{1}{2}$, in the vertical $4\frac{1}{2}$ inches. It was distinctly more fixed, and the right flank had now lost its tympanitic note. I therefore regretted much the delay in operating in view of the possible generalisation of the neoplasm. But this regret was redoubled by observing the attack on the Saturday preceding operation. The patient had been particularly well all day and for some days previously, but on that night the worst access of pain she had yet experienced in the tumour set in. This was accompanied by hurried breathing and rise of temperature to 100°, and later by constant nausea and vomiting, Her sufferings were intense all through Sunday, and on that night her pulse, taken three different times, was respectively 170, 180, and 150. She was somewhat relieved by morphia, and next morning her pulse was only 130, but the temperature had risen to 101·5° during the night: her breathing was not so hurried. She was besides pale and anxious-looking, and quite unable to sit up in bed as before. The operation was performed on the next morning (Tuesday, December 23rd, 1879). At this time the pulse was from 125 to 130, the respirations quieter than before, but the vomiting had not yet ceased. She was altogether much exhausted, though, as she told me, she thought she would have been able to walk over from the hospital to the private room prepared for her across the street, she had felt so well on Saturday. As it was she was so prostrate that her removal had had to be deferred. On the same

morning she had passed a large quantity of almost pure blood, and it was noticed that the tumour had shifted *against gravity* two and a half or three inches towards the left side.

As to the operation itself, it was not really difficult, and I was not obliged to depart from the plan I had sketched beforehand in detail. The first incision from the umbilicus downwards was limited to three inches at first, in order that, while the left kidney was being first explored, and then the tumour on the right, the intestines should not protrude. An early escape of the latter and their exposure was much feared, the abdomen not being distended, as in ovarian cases. As a matter of fact, no part of them ever rose above the level of the wound during the whole operation; they were hardly ever seen. For this I have in great measure to thank Mr. Meredith's perfect administration of the anæsthetic.

The end of the tumour lay under the wound, with the ascending colon over its left border. The left kidney was in its normal position as found by the finger. The incision was now enlarged to about three inches above the umbilicus. On passing my hand into the abdomen the tumour in the first place was felt to be distinctly renal; it was freely movable laterally but fixed somewhat towards the aorta. It was unevenly tuberculated, softer, and more elastic in spots, and the end which lay under the liver was pointed. The peritoneum was stretched tightly over it but was smooth and unimplicated. Several large veins were also seen running transversely over the dark purple surface of the growth and under the serous covering.

I now cautiously tore through the external layer of the ascending mesocolon with finger-nail and forceps transversely to the axis of the body, then, inserting the finger under it, slowly and gently swept round the growth. After the anterior portion had been freed this was not difficult, but so far the utmost care had to be observed lest some of the soft parts of the tumour should be broken into, and I should lose myself in its pulpy vascular sub-

stance. Soon the lower end of the mass could be protruded through the peritoneal rent, and the rest, easily but slowly separated from the perinephritic tissue, presently followed. The ureter was now found at its lower and left border, and was divided between two ligatures. The tumour could then be partly extruded through the abdominal incision, but not fully until another rather firm band, lying a little higher up between the mass and the aorta, had been divided. This was found subsequently to contain an aberrant artery and vein. Then the whole mass came out, though still attached by the renal vessels and a fibrous band posteriorly. The vessels were not visible, however. The pedicle was now transfixed with a blunt aneurism needle bearing strong (No. 30) twisted silk, and was tied in two portions; then in the same way, a little nearer to the aorta, the last ligature being finally brought round the whole *en masse*. The pedicle was then divided with a scissors, and the renal artery found dry and gaping; its mouth was ligatured.

The edges of the peritoneal rent falling nicely together I simply sponged the areolar tissue dry, and then the abdomen, replaced the colon, and stitched up the abdominal wall in the usual way. An antiseptic dressing completed the operation. The latter was almost bloodless from beginning to end, no vessels requiring ligature except the renal. The patient bore it very well. Mr. Meredith has kindly supplied me with the following note on this point: —"Pulse about 124 after the operation (it had been 125—130 before), small, quite regular, and of fair strength. During the time she was on the table it had remained steady throughout, and never gave the slightest cause for anxiety."

Looking at the operation now I believe some time might have been saved in its performance. It lasted over an hour, the patient being dressed and in bed in an hour and twenty minutes. But for any slowness of execution I am prepared to accept the responsibility; it was to some extent premeditated. First, this was a

pioneering operation, not having been done before in this way in England. Then I feared from the first that on opening the abdomen the soft vascular mass might be found nearly or actually protruding through its serous covering. This was almost the case. I realised the risk in stripping off the peritoneum of thrusting the finger into this brain-like substance, and so losing my way in it, as had occurred in two foreign cases (Nos. 15 and 22), leading in them to what I think may be called catastrophes. The first of these is thus described by the operator, Professor Czerny:

"The external layer of the mesocolon was divided over the tumour, and the separation of the mass encapsuled under this begun with blunt instruments. Further advance showed that the soft growth had not only broken through the capsule internally, but even the inner layer of the mesocolon. In this soft mass one had nothing to hold on by. A profuse hæmorrhage could only be arrested by temporary compression of the aorta, returning as soon as this was taken off. All the signs of a speedy death from hæmorrhage manifesting themselves, nothing remained to me but to throw a ligature round the aorta. This stopped the bleeding. The abdominal wound having been closed the patient was put to bed. He came to himself thoroughly, but after two hours complete anæsthesia of the lower limbs set in, which persisted until his death, ten hours after the operation."

These are the operator's own frank words, and require no comment. The other case (No. 15) was almost similar.

Again, there was great danger of wounding some of the large veins ramifying over the tumour, and so flooding the field of operation, as had also occurred in one case. Great difficulty, too, had been experienced by another operator abroad in avoiding the distended renal vein. In another case the ligature had slipped off the vessels after division of the pedicle. I thought, therefore, no care too great to avoid these risks. They were all real, and it

appeared better to sacrifice a little time (especially as the intestines were not exposed) to deliberate work, and to avoid all chance of having to adopt any desperate expedient or abandon the operation, which in that case I should have regarded as a complete surgical failure. An examination of the tumour afterwards justified the caution observed. There were spots on its surface almost diffluent, and which an unwary touch would have easily broken into, letting out either its soft material or the blood effused into it.

The patient's condition after operation was briefly as follows:—She recovered from the anæsthetic in the usual way. There was hardly any shock, the temperature only falling to 97·6°. In the evening she was still vomiting; pulse 108. Next morning at 6 a.m. the temperature had risen to 99·2°; pulse 130; respirations 22. At 10 a.m. the temperature 99·4°. Patient now looked quite as well as on the morningof operation. At 2 p.m. still vomiting; temperature 99°. She seemed to be doing so well that it was decided in consultation not to trouble her with nourishment or stimulants as she was still vomiting. At 6 p.m. as before. At 10 p.m. she was plainly not so well; respirations 36; pulse 140; temperature 99·3°. One could not fail to be struck with the similarity between these symptoms now suddenly setting in and those manifested on the preceding Saturday night. Two hours later Dr. Williams kindly saw the patient with me. Her symptoms were as before. On the one hand, the absence of abdominal rigidity or distension, or the characteristic pulse excluded peritonitis, besides the temperature was only 100·2°. Moreover, she had not the look of that disease. On the other hand, the pulse was too good for rapid *general failure*, and, further, the temperature ought to have fallen for this condition. Besides this, there was something very peculiar in the character of the hurried breathing. On the whole, we inclined to the belief that a clot was forming in the vena cava.

She remained in this state for some hours longer, and

then began really to fail. The pulse became feebler, the respirations shallower, the vomiting continued, the right lower limb became numbed, and she died forty-five hours and a half after operation. She was perfectly clear-minded to the last, and had no head symptoms.

Autopsy twenty-seven hours after death.—Cause of death pulmonary thrombosis. Body fairly well nourished, somewhat anæmic. Slight discoloration over the course of the veins of the right thigh and leg, as noticed some hours before death.

Operation wound accurately and soundly united, without a trace of inflammatory action or puffing. It was seven inches long, three above and four below the umbilicus. Abdominal cavity opened an inch to the left of it to show its serous surface. Union had also taken place here in the most satisfactory manner, the edges being glued firmly together without any *visible* lymph. A small shred of omentum was loosely included in the uppermost stitch. The intestines were normal in appearance and position. There was a trace of stained serum in Douglas' pouch, but no visible plastic exudation anywhere, or any blood-clots. The colon had returned to its normal position, and overlapped the rent in the mesocolon, being firmly adherent to it by invisible cementing material which required considerable force to make it yield. The rent itself had contracted considerably. It led into a space in the perinephritic areolar tissue, which was ecchymosed with the blood of the operation. There was only one small blood-clot here, about the size of a date seed; no trace of pus or visible lymph, though the tissues had already begun to consolidate. In this areolar space lay the vessels of the kidney tied as described, the ligatured end of the ureter a little ecchymosed, and the aberrant renal artery mentioned above. The renal artery was tied at about one inch and three quarters from its origin, and filled with firm clot. The vein had been ligatured exactly at its termination in the cava. Its shortness seems to have been due to the fact that the

cava was drawn away from the aorta, but whether by the weight of the tumour or traction during operation it is hard to say. At all events, the same ligature which closed the artery at about one inch and three quarters from its origin included the vein at its termination. The vena cava and iliac veins were partly filled with fluid blood without clots, either opposite the ligatured renal vein or elsewhere.

The liver was normal, but pale and slightly yellow-tinted. There were one or two minute whitish specks on its surface, suspiciously like secondary deposits. The gall-bladder was engorged.

The left or remaining kidney weighed five ounces. It was slightly hyperæmic, but quite healthy, and without secondary deposits. Its pelvis contained some thickish turbid urine.

The bladder contained a little turbid bloody urine, with a small washed-out clot.

The stomach was pale, but spotted with small ecchymoses, and covered with thick mucus.

The pericardium contained a little straw-coloured serum.

The heart substance was normal. Right auricle full of soft dark coagula. Right ventricle almost filled with a firm, toughish, pale, yellow ante-mortem thrombus, entangled in the columnæ carneæ, but not very firmly adherent to the walls. This was traceable far into the pulmonary arteries, where it ended in ordinary post-mortem coagula. The left ventricle contained only post-mortem coagula.

Both lungs were anæmic anteriorly, with slight turgescence posteriorly; otherwise quite healthy, except for a few small secondary deposits of the neoplasm, varying from the size of a small nut down to that of a millet seed, scattered here and there on the surface. These had the same texture as the tumour.

Some small glands at the root of the kidney were infiltrated with the new growth, but no further traces of the latter were found in the lymphatic system.

The supra-renal capsule was found to have been left behind by the operation, and to be quite normal.

Now, while looking upon the operation as the cause of death, it may not be uninteresting to glance a little more closely at the various factors in the process of dying. First, there can, I think, be but little doubt that thrombosis of the pulmonary artery was the immediate cause of the fatal issue. That it was not peritonitis is quite clear. Nothing could have been more satisfactory than the condition of the whole abdominal cavity and intestines. There was exactly that process of repair going on which we desire in such cases, a glueing together of the wounded surfaces (and of them alone), and yet no trace of lymph visible to the naked eye. Again, it was just as clearly not septicæmia. The temperature never rose above 100·2°, and we had none of those appearances, before or after death, usually associated with that condition. Nor was it the immediate effect of shock. The fact that the temperature only fell six tenths after operation, and had then slowly risen to 100·2°, together with the good character of the pulse for the first twenty-four hours, settle this. It was, also, not hæmorrhage. The patient did not lose a couple of ounces of blood during the operation, and lost none after. Again, there were no uræmic symptoms, the mind being perfectly clear to the last.

I was at first inclined to attribute the fatal issue to those secondary neuro-paralytic effects sometimes met with after severe operations, where (to put it briefly) the system is so depressed by the latter that it is unable to respond to the call made upon it when the time for repair of the wounds comes round, and thus heart and lungs fail, and with them all the other powers. But this cannot be so here. There was no hæmorrhage, no exposure of the intestines, and no shock of any note.

But in saying that the patient died of pulmonary thrombosis, we do not advance much further in explanation. Why did the clot form in the right heart? It is

as difficult to account for this phenomenon here as it is in other cases where it is found. Here there was no evidence of embolus, no trace of clot at the mouth of the ligatured renal vein or elsewhere. The great primary thrombus in the heart also gave no indication in its texture of such an origin. It was limited to the right ventricle, and only extended secondarily into the pulmonary arteries. It was plainly of considerable standing. Whether it had some little shred of the soft encephaloid growth as its starting-point is, I think, a fair subject for speculation, although sections do not show this. If such were so, the thrombus might well have commenced to form a couple of days before the operation, in view of the sudden symptoms of dyspnœa, &c., which had set in and persisted to a greater or less extent until the latter. This thrombus, very small at first, might have been added to when the weakening effects of the operation and the commencement of inflammatory repair had altered the force of the blood-stream and its plasticity. The latter may also have been considerably influenced by the almost constant bleeding for at least seven weeks before from the kidney. That small portions of the growth did pass through the heart at one time or another is plain, for there were secondary deposits in the lungs. Buds of the soft neoplasm, too, were found projecting into the lumen of some of the larger renal veins (fig. 1). Whether the handling of the tumour during operation may have dislodged one of these, which only reached the heart on the second day, is also a fair question. Such a shred at all events had been swept down the ureter a couple of days before, where I found it, and recognised its texture microscopically on the preceding Sunday.

How long the patient would have lived had she survived the operation it is hard to say, probably not very long. The deposits in the lungs, however, were still small, the largest about the size of a nut. But I cannot help thinking that, had we operated before the last attack of dyspnœa, pain, &c., had set in, she would have survived

the immediate effects of the operation; and if when she first came in, that we should probably have forestalled recurrence, which was manifestly recent.

Examination of the tumour; naked-eye appearances.— The kidney on removal weighed 30½ ounces; its length was 7, breadth 4½, and depth 4⅔ inches. It was larger below than above, where its only healthy portion lay. This had the usual appearance of smooth, pale renal tissue. The rest of the organ was uneven and lumpy, with dark purple patches over the irregularities. The largest prominence was below and anteriorly, and equalled the area of about a crown-piece. Here the surface was soft, and even fluctuating under the finger. In no spot did the growth actually pierce the capsule of the kidney, which was smooth everywhere, but in several places it very nearly did so.

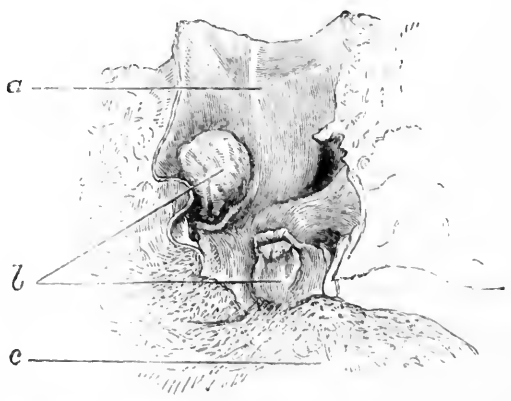
The ureter, cut about three inches long, looked normal; its infundibulum was dilated. The renal vein was severed close to the kidney, as also the arteries, which appeared of normal size. The perinephritic areolar tissue presented nothing abnormal.

On section of the organ in the usual way its upper portion is seen to be quite healthy for about as much as the upper quarter of a normal kidney. Below this the organ is almost completely converted into a mass of encephaloid new growth from the apices of the pyramids to the great convexity. Here, somewhat towards the front of the mass, is a soft collection, about the size of a hen's egg, consisting chiefly of blood held together by very loose trabeculæ of the growth, and encapsuled in a very tough fibrous covering about $\frac{1}{12}$ inch thick. The latter is lined by decolorised fibrin deposited in layers of different density and hue. Outside of this encapsuled mass the growth extends in all directions as a more or less spongy tissue, made up of loose fibrous trabeculæ enclosing much softer material, and here and there extravasated blood. This softer substance is almost diffluent in parts, out of which can be picked up shreds

of very delicate texture. It appears manifest that the youngest parts of the neoplasm are those towards the cortex of the kidney, the oldest at about the position occupied by the apices of the pyramids, although most of the latter are obliterated by the new growth. The expansion of the latter has elongated the pelvis to a large extent, and drawn out the calyces into tubular structures. Seven of these can be made out. One of them laid open shows the growth commencing to fungate through its fibrous wall. Over the area of a sixpenny-piece an irregular flocculent bleeding mass projects into its lumen. A second calyx is nearly filled with another such projection. The large encapsuled mass mentioned above is also nothing but one of the calyces distended by one of these portions of soft growth, and the blood extravasated into it at different times. The greater part of the rest are healthy as to colour and texture of their lining membrane.

The renal arteries can only be traced a short distance into the fungous mass of the tumour and are soon lost.

FIG. 1.



a. Vein laid open; normal.
b. Two buds of new growth piercing through the intima of the vein.
c. New growth outside the vein.

The vein too is soon lost. But before it has passed far inwards its walls are found to be pierced by small pulpy projections of the new growth. These can be studied

in every stage, from that of mere raising of the inner coat of the vessel to actual projection of spongy tufts into the vessel. (See Woodcut, fig. 1).

On the whole, a careful dissection of the organ appears to show the following to be the plan of growth of the neoplasm.

1. That it commenced in one or other of the pyramids in the lower part of the kidney, and spread from this centrifugally, the cortex having been but recently invaded.

2. That the calyces and pelvis were much stretched before their walls were actually pierced.

3. That one calyx was much distended by such a mass and by the blood poured out by it. And also that this blood was poured out at different periods, and with sufficient intervals to admit of thick separate layers of fibrin being deposited from it on the walls of the calyx.

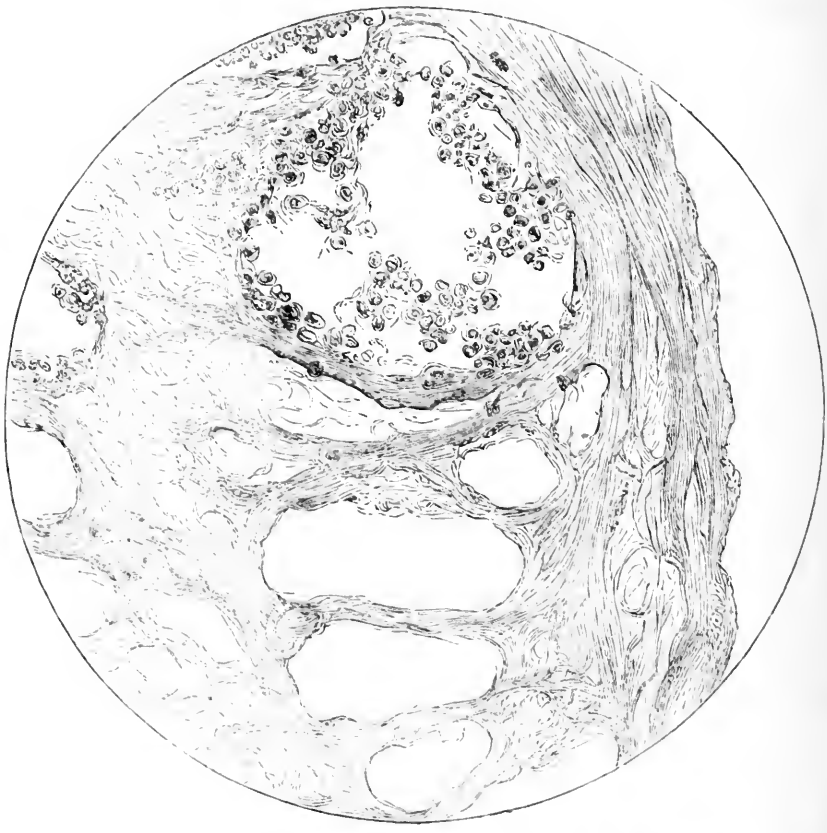
4. That the other calyces pierced by the growth presented the process in a more recent stage, with no coagulation of blood *in sitû*, but simply projecting tufts.

5. That the renal vein had comparatively recently been pierced by the neoplasm, explaining probably the very small extent of its generalisation in the lung.

6. That the neoplasm was probably a small round-celled sarcoma.

Microscopic structure.—The bulk of the growth consists essentially of small round indifferent cells crowded together without any particular arrangement in large masses. (See Woodcut, fig. 2). These are bounded by bands of fibrous tissue of more or less firm texture. In the younger parts of the growth the small aggregations of cells occupy alveoli, of oval form and tolerably even contour, but varying much in size and arrangement. This alveolar structure is very striking in many places. Whether the individual loculi were derived from the distended tubules of the organ or not could not be determined from the sections examined. In none of them, however, were there any traces of epithelial elements, nor were there any other traces of renal structure discovered here. The cells of the neoplasm were in all points identical with those of the small round-

FIG. 2.



Section near the periphery of the growth, showing a somewhat alveolar structure. Some alveoli contain the small round cells, others have been emptied in preparation.

celled sarcomata, to which group the growth is plainly to be referred.

The following table gives the proportion of urea passed before and after the operation :

The amount of urine passed in 24 hours was estimated on five occasions.	The amount of urea passed in 24 hours was estimated on six occasions.
ozs.	grs.
50	342
69	390·5
46	499
38	369
68	185
	400
5) 271·0	6) 2185·5
Average for 24 hours...54·2	Average for 24 hours...364·25

The amount of urine passed in the twenty-four hours following the operation was twenty-six ounces, or a little less than half the average quantity.

The amount of urea passed in the twenty-four hours following the operation was 200 grains, or 164·25 less than the average quantity, or nearly two thirds of the average whole.

Finally, if this case has done nothing more it has demonstrated that the diagnosis of cancer of the kidney is, in certain instances, arrived at with sufficient certainty to justify operation, should this not be contra-indicated by other considerations.

I cannot conclude now without acknowledging Mr. Heath's kindness in placing this case, in which I was so much interested, in my hands for operation. I have also to thank Mr. Erichsen for kindly being present to aid me with counsel, and Dr. John Williams and Mr. Beck for their able assistance.

The appended table is compiled from a careful study of the original reports of the several cases in every instance except three, viz. Nos. 1, 13, and 23, where the originals were not accessible, but only abstracts.

Table of all cases of Nephrectomy (two

No.	Operator and original record.	Sex and Age.	Diagnosis or aim of operation.	Evil dates back.	Date of operation	Seat of incision.	Form of dressing.	Treatment of pedicle.	Symptoms after operation.
1	Wolcott (America), 'Med. and Surg. Rep.,' Phil., 1861, vol. vii	M. 58	Cyst of liver	6 yrs.	1861	—	—	—	—
2	Peaslee (America), 'Ovarian Tumours,' p. 158	F.	Ovarian cyst	—	1868 Apr.	Ventral	—	—	No coma; free sec tion of urine
3	Simon (Heidelberg), 'Chirurg. d. Nieren,' Erlangen, 1871, Th. i, p. 33	F. 46	Fistula of ureter after ovario-hysterotomy	18 mos.	1869 2nd Aug.	Lumbar, extra peritoneal	Not antiseptic	3 silk ligatures, thick No. 2	"Neither headac coma, delirium, convulsions;" tient vomited fr the chlorofor urine reduced first, but "no all men found"
4	Esmarch (Kiel), 'Archiv f. Gynæk.,' Bd. i, Hft. 3, p. 415	F. 19	Ovarian cyst	12 yrs.	1869 15th Nov.	—	—	Ligatures, 16 in all	Only noticed "thirst pain in back co tinuous
5	Gilmore (America), 'Am. Jour. of Obstet.,' May, 1871	F. 33	Painful kidney: patient 5 months pregnant	4 yrs.	1870 Dec.	Lumbar	—	1 ligature of one vessel after removal	—
6	Bruns (Germany), 'Würtemb. M. Corresp. Bl.,' 1871, No. 14	M.	Gunshot wound on Dec. 2, 1870	4 mos.	1871 23rd Mar.	Lumbar 9 inches long	Not antiseptic	Ligature very difficult	Great pain and sire to urina Spoke quite clear
7	Meadows (England), 'Brit. Med. Jour.,' 1871	F.	Ovarian cyst	—	1871 24th June	Ventral	—	Ligature	"No inflammat anywhere"

abandoned) hitherto recorded or authenticated.

Result, immediate and eventual.	Hæmorrhage at or after operation.	Condition of organ removed.	Remarks.
ath 15 days after from haustion of suppura-on	—	Encepha-loid of right kidney; weight 2½ lbs.	—
ath 50 hours after of eritonitis	—	—	The original record is exceedingly scanty, giving little more than these notes.
t of bed in 6 weeks: ne three ligatures re-ained fixed until the th month, at which time atient was apparently uite well: after they ame away the sinus ealed in a few days	During, but easily arrested	"Quite healthy in all parts"	The original record is most valuable. N.B.—Analysis of the matters vomited showed no trace of urea.
ath 36 hours after; dema of lungs and dys-nœa marked	During, smart from adhesions, none after	Large cyst of left kidney	This operation is incorrectly attributed to Schetelig by one or two writers; Schetelig only recorded it by Esmarch's permission.
covered well; did not bort	—	Shrunken, fibrous, marked by ribs	The patient was a negress, and suffered intensely before operation; the nature of the body removed was not quite clear until after removal.
ath 10 hours after rom shock	Inconsider-able	Converted into a large abscess	The patient was very weak before the operation, and bore the chloroform badly. The operation lasted nearly 2 hours. The right kidney was also dotted with small abscesses.
ath 6th day after of æmorrhage from pedi-le	—	Large cyst	The original record is very scanty.

No.	Operator and original record.	Sex and age.	Diagnosis or aim of operation.	Evil dates back.	Date of operation	Seat of incision.	Form of dressing.	Treatment of pedicle.	Symptoms after operation.
8	Simon (Heidelberg), 'Chr. d. Nieren,' Th. ii, p. 148	F. 30	Calculous pyelitis	12 yrs.	1871 8th Aug.	Lumbar, rapid	Charpie packed lightly into wound	Ligatures 2, not catgut	Vomited, but n uræmic symptom Pain in abdomen i direction of ureter
9	Durham (London), 'Brit. Med. Jour.,' May, 18, 1872	F. 43	Painful kidney	2 yrs.	1872 14th May	Lumbar	—	—	None bad
10	Peters (N. York), 'N. Y. Med. Jour.,' Nov., 1872	M. 36	Calculous pyelitis	19 mos.	1872 16th May	Lumbar difficult and slow	Lint	Ligature 1, strong silk	Vomiting, but n uræmic symptoms
11	Brandt (Austria), 'Wien Med. Wochensch,' Nov., 1873, p. 1079	M. 25	Injury by a knife slitting kidney in loin	4 dys	1873 7th June	Lumbar original wound	Not antiseptic	Ligature double silk	Nothing uræmic b fore or after, b sweated a good de
12	Cambell (Dundee), 'Edin. Med. Jour.,'1874, vol. xx, p. 36.	F. 49	Ovarian disease	18 mos.	1873 2nd Dec.	Ventral	Modified antiseptic	Ligature hempen	No uræmic; fr flow of urine fro first
13	Marvaud (Algiers), 'Revue de Méd.Milit.,' Oct., 1875	F., 'young arab'	Wound by yataghan	Few days	1875	Lumbar, original wound	—	Ligature silk	No uræmic; go flow of urine fro beginning
14	Langenbuch (Germany), 'Berl. Klin. Wochensc.,' No. 24, 1877	F. 32	Sarcoma of lumbar muscles or of kidney	18 mos.	1875 7th Dec.	Lumbar	Antiseptic	Ligature difficult, slipped off	None bad; no feve
15	Kocher (Bern), 'Deutsch. Zeitsch. f. Chir.,' Bd. ix, 1878	F. 35	Sarcoma of a freely movable kidney	18 mos.	1876 20th Apr.	Ventral high	Antiseptic failed	Not reached	Only thing remar able was cysti second day

Result, immediate and eventual.	Hæmorrhage at or after operation.	Condition of organ removed.	Remarks.
eath 31st day after from yæmia, setting in on 1st day	None from incision; severe from pedicle at operation	Small, fibrous, calculous with blood clots	"Very favorable conditions until the 21st day," when pyæmia manifested itself. Original report is most carefully made, and very valuable. Operation lasted ½ hour.
eath within a week	—	—	Original report most scanty.
eath 65 hours after	Very little during or after	Enlarged and granular	The operation lasted 2½ hours.
covery; left hospital n the 16th day; ligaures came away on 6th ay	None before or after	Healthy; weight 58 grammes	Original report most instructive. After the first knife-wound patient was able to walk about without trouble in spite of the protrusion of the kidney from the opening into which it had been forced by coughing.
covery, but tedious; ound quite healed 84th ay; left hospital on 13th day	Considerable oozing nothing more	Cyst of Lower ¼th of left kidney	—
covery; left hospital two months quite well	Smart before	—	The ligature was allowed to cut its way through the pedicle, and the kidney to slough off, which it did in "some weeks."
covery; left hospital an., 1876	Slight when ligature slipped	Doubtful, a hollow organ	There is some doubt as to the nature of the diseased mass removed, the specimen having been thrown away by the inadvertence of a servant.
ath 3rd day, from peritonitis	None noted	Sarcoma extensive	Operation abandoned; the growth spreading too far into transverse mesocolon to be separable; abdominal incision stitched up.

No.	Operator and original record.	Sex and age.	Diagnosis or aim of operation.	Evil dates back.	Date of operation	Seat of incision.	Form of dressing.	Treatment of pedicle.	Symptoms after operation.
16	Jessop (Leeds), 'Lancet,' 1877, vol. i	M. 2½	Renal encephaloid	1 year	1877 7th Jan.	Lumbar	Not antiseptic	Ligature whipcord	None bad; no vomiting; good flow of urine
17	Kocher (Bern), 'Deutsch. Zeitsch. f. Chir.,' Bd. ix, 1878	M. 2½	Renal new growth	2½ yrs.	1877 27th Sep.	Ventral	Antiseptic	Ligature double catgut	No uræmic; no vomiting; free flow of urine soon after
18	Martin (Berlin), 'Langenb. Archiv,' Bd. xxiii, 1879, p. 540	F. 49	Painful floating kidney	8 yrs.	1878 15th Mar.	Ventral	Antiseptic strictly	Ligature, 1 *en masse*, 2 transfixion	Rigors, pain, nausea; drowsiness at first
19	Martin (Berlin), loc. cit.	F. 30	Painful floating kidney	5 mos.	1878 25th Aug.	Ventral	Antiseptic strictly	Ligature, 4 transfixion, 1 *en masse*	Some vomiting and pains
20	Martin (Berlin), loc. cit., and private letter	F. 25	Painful floating kidney	—	1878	Ventral	Antiseptic strictly	—	—
21	Martin (Berlin), 'Berl. Klin. Wochense.,' 1879, No. 22.	F. 53	Sarcoma of kidney	2 mos.	1878 9th Dec.	Ventral	Antiseptic strictly	Ligatures 9, silk	—
22	Czerny (Heidelberg), 'Cent. Blt. f. Chirurg.,' No. 45, 1879.	M. 50	Malignant tumour of kidney	2 yrs.	1879 19th Jan.	Ventral	—	Not reached	—
23	Smith (N. Orleans) 'N. Orl. Med. & Sur. Jour.,' Aug., 1879.	F. 35	Painful floating kidney	8 yrs.	1879 3rd June	Lumbar not difficult	Not antiseptic	Ligature "strong"	No uræmic; slight fever; free suppuration

Result, immediate and eventual.	Hæmorrhage at or after operation.	Condition of organ removed.	Remarks.
Recovery; child up and running about soon after	—	—	Mr. Jessop kindly informs me that he satisfied himself by examination 8 months later that recurrence was taking place, probably in the lumbar glands; the child died a few weeks later at its home; no autopsy.
Death 3rd day of septic peritonitis	Considerable during; none from pedicle	Adeno-sarcoma 15 × 16 × 10 cm.	—
Recovery; out of bed on 17th day; wound healed 26th; strong, working 3 months	None from pedicle; some from peritoneum	Perfectly healthy	Next to Simon's monograph this is the best article on the subject yet written. The great distress of patient was quite relieved by operation; latter lasted 36 minutes.
Recovery; out of bed on 13th day walking about; wound healed 17th; discharged	—	Perfectly healthy	A more striking case than last; symptoms very slight, recovery very rapid; operation lasted 50 minutes.
Recovery, complete	—	—	—
Recovery, complete; out of bed 8th day, went home 17th day	None	Malignant new growth weighed 28 ozs.	Operation rendered particularly difficult by presence of large engorged veins running over the tumour; operation lasted 45 minutes.
Death 10 hours after of shock	Profuse at operation from renal artery	Soft spongy mass left *in sitú*	Having broken into the soft mass of the tumour during operation profuse bleeding set in from torn renal artery; the aorta was then ligatured.
Recovery uninterrupted; walked about on 11th day	—	Healthy; showed scar of seton	Ovariotomy attempted in 1873, no relief; a seton was passed through abdominal walls and tumour in 1874 to cause adhesion; temporary relief.

No.	Operator and original record.	Sex and age.	Diagnosis or aim of operation.	Evil dates back.	Date of operation	Seat of incision.	Form of dressing.	Treatment of pedicle.	Symptoms after operation.
24	Zweifel (Erlangen), 'Archiv. f. Gynækol.,' B. xv, Heft. i, p. 1.	F. 29	Fistula of ureter into uterus after labour	2½ yrs.	1879 8th Jan.	Lumbar	Antiseptic strictly	Ligature silk, left long	Vomiting, pain, and restlessness on 1st day, nearly absent 2nd day
25	Czerny (Heidelberg), loc. cit.	F. 32	Pyonephrosis	4 yrs.	1879 22nd May	Lumbar	Chl. Zinc. Thymol. gauze	Ligature silk, and elastic	None noted
26	Quoted by Mr. Wells, 'Med. Tim. & Gaz.,' 1870, Jan. 8th.	—	Fibro-cystic tumour of uterus	—	—	Ventral	—	—	None uræmic or referable to loss of kidney
27	Martin (Berlin), Private letter.	F. 48	Painful floating kidney	—	—	Ventral	Antiseptic strictly	—	—
28	Martin (Berlin), Private letter.	F. 24	Painful floating kidney	—	—	Ventral	Antiseptic failed	—	—

Result, immediate and eventual.	Hæmorrhage at or after operation.	Condition of organ removed.	Remarks.
ecovery; stay in hos-pital prolonged for sepa-ration of ligatures	Moderate from torn kidney	Small, atrophied; ureter large	The removal of the organ produced no ill effects.
ecovery; hardly a trace of fever; up on 25th day, discharged on 42nd	Pretty free during operation	Large abscess; burst at operation	A portion of the 11th rib was removed during the operation for free access to pedicle. There was a perinephritic abscess and fistula beforehand.
eath on 3rd day	—	Healthy	The original notice is very brief, but the kidney appears to have been removed inadvertently being adherent to the tumour.
eath from peritonitis	—	—	—
eath from septic peri-tonitis	—	—	—

ON

THE ESTIMATION OF URIC ACID

BY ITS REDUCING ACTION UPON THE

AMMONIATED CUPRIC TEST.

BY

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Through uric acid more or less serious trouble is not unfrequently produced. A natural constituent of the urine to a limited extent, it possesses more important pathological than physiological bearings. It is its appearance as a deposit which gives rise to harm, and to obviate this is a task often placed before us as medical practitioners. The task may be readily enough accomplished by the administration of certain agents which lead to the formation of a soluble product, but at present our knowledge does not extend much beyond this, and when the agent is discontinued, it but too often happens that the deposit again shows itself.

As a means of studying the various conditions which tell favorably and unfavorably upon the uric acid diathesis, and likewise as a guide during the management of a case

of the occurrence of the deposit, the information afforded by the quantitative determination of the uric acid voided forms an item of the first importance. It is to the consideration of the manner in which this determination may be satisfactorily effected, that this communication is devoted.

The methods hitherto employed for the estimation of uric acid cannot be spoken of as suitably meeting the requirements. Both as regards precision and convenience or facility of application, they leave much to be desired. The simple collection of the crystals which settle from a specimen of urine after the addition of an acid constitutes a very unsatisfactory process, as, on account of the partial solubility which uric acid possesses, only a portion of it present becomes thereby estimated. A more accurate plan is that of evaporating a given amount of urine to dryness, washing the residue with alcohol, and then treating it with dilute hydrochloric acid. In this way the uric acid is separated from the other constituents of the urine, and the quantity determined by weighing. This process, from the time it occupies, and from the manipulative conveniences required, is not adapted for general clinical use, nor is it altogether free from affording a lower estimate than should be yielded.

The process which it is the object of this communication to bring under the notice of the Society is one by which the estimation of uric acid may be effected not only speedily and accurately, but in such a manner as to be susceptible of being carried out by the medical practitioner.

It is well known that uric acid possesses, like glucose, the property of exerting a reducing action upon the oxide of copper. With the ordinary copper test this property does not admit of being turned to account for the estimation of uric acid in urine, as it does in the case of sugar. In the ammoniated copper test, however, which I have recently introduced and am employing with such a satisfactory result for the quantitative determination of sugar,

not only have we an agent which is available for the estimation of sugar, but also for that of uric acid.

The test in question was described by me in communications to the Royal Society published in the 'Proceedings' (vol. xxviii, p. 260, and vol. xxix, p. 272). It is prepared by adding ammonia to the ordinary cupric test solution, the effect of which is to exert a solvent action upon the reduced oxide formed when the reagent is employed. Reduction occurs under the influence of the reducing agent, whether sugar or uric acid, but instead of the reduced oxide falling as an orange-coloured precipitate, it is held in solution by the ammonia in a colourless state, the change noticeable being the gradual removal of the blue colour belonging to the original liquid as reduction proceeds.

The instructions for preparing the liquid are as follows:

Let in the first place the ordinary cupric test solution be made according to the subjoined formula.

Cupric sulphate	34·65 grammes.
Potassio-sodic tartrate (Rochelle salt) .	170 „
Potash	170 „
Water to a litre.	

For the ammoniated liquid, 120 c.c. of this test solution are mixed with 300 c.c. of strong ammonia (sp. gr. 0·880), and water added so as to bring the volume to a litre.

The liquid thus produced is prepared for sugar estimation, and is adapted in strength to give an even numbered expression of value. The reducing power of uric acid bearing no correspondence with that of sugar, the value in uric acid presents itself as a fractional number. The strength might, of course, if desired, be so arranged as to correspond with any given amount of uric acid, but it seems scarcely worth while for the sake of a few figures in calculation, to provide an additional liquid for use.

The test is employed in the same way as for the estimation of sugar. Twenty c.c. of the ammoniated liquid are placed in a flask, which is affixed to a cork suspended by a piece of vulcanised tubing from a burette containing

the product to be examined. The flame either of a spirit lamp or gas having been applied underneath the flask, its contents are brought to a boiling point, and the liquid in the burette is then dropped in under the control of a clip placed on the vulcanised tubing, until all trace of blue colour is just made to disappear. To obviate the inconvenience arising from the dispersion of the evolved ammonia into the air, it may be conducted by a piece of vulcanised tubing into a vessel of water. The end of the tubing which dips into the water being plugged at its extremity, and having a transverse slit through three fourths of its extent just above, supplies a valvular arrangement which permits of the escape of air and ammonia vapour from the flask, but prevents the sucking back of water with the sudden condensation that is liable to occur should it happen that the liquid is run in a little quickly from the burette. Messrs. Griffin, of Garrick Street, Covent Garden, supply the required apparatus.

Having at the outset learnt that in the application of the liquid for the estimation of uric acid the point of complete decoloration was easily susceptible of discrimination, and that uniformity was recognisable in the result, it next became necessary (in order that quantitative expressions might be given) to ascertain the precise reducing capacity appertaining to the principle.

Solutions of various strengths that had been prepared from carefully purified uric acid were submitted to trial, and the results obtained were such as to very closely agree with the formula representing three atoms of oxide of copper as reduced by one atom of uric acid. Actual observation, for instance, gave 0·03747 gramme of uric acid as the quantity taken to reduce 40 c.c. of the ammoniated cupric liquid. Theoretically, upon the basis of one atom of uric acid reducing three atoms of cupric oxide, the quantity would stand at 0·03732 gramme. From the close accord thus existing, I think the theoretical relationship mentioned may be justifiably accepted as the expression to be adopted.

A. Burette containing the diluted urine.

B. Flask containing the ammoniated cupric test.

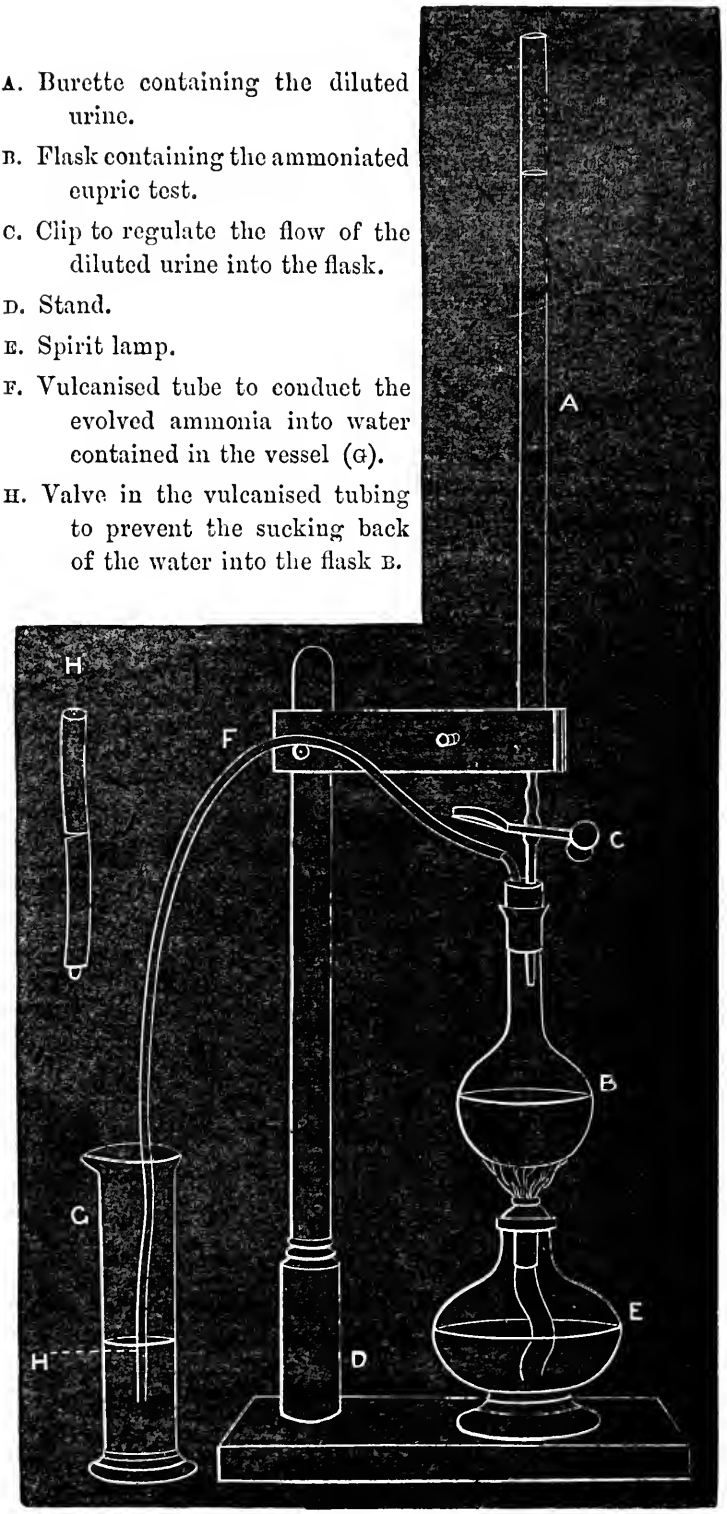
C. Clip to regulate the flow of the diluted urine into the flask.

D. Stand.

E. Spirit lamp.

F. Vulcanised tube to conduct the evolved ammonia into water contained in the vessel (G).

H. Valve in the vulcanised tubing to prevent the sucking back of the water into the flask B.



Reckoning, then, that one atom of uric acid reduces three atoms of oxide of copper, 20 c.c. of the ammoniated test will stand equivalent to 0·01866 gramme of uric acid.

In the application of the test to the determination of uric acid in urine, 20 c.c. of it constitutes a convenient quantity to be taken, and experience shows that it is desirable to add after its introduction into the flask an equal bulk of water. This dilution diminishes the interference produced by the colour of the urine, and with a specimen of urine of average concentration it is even advisable to dilute this with a corresponding bulk of water before placing it in the burette.

In this state it is found that somewhere about 8 or 10 c.c. of liquid are required to decolorise the 20 c.c. of the cupric test.

With a little experience there is no difficulty in determining when the required removal of blue colour has been effected, although the point of completion is not so sharply defined as in the estimation of sugar, on account of a slightly yellowish tint becoming developed during the operation performed. The contents of the burette must be dropped in till no sign of blue or green is left.

The actual determination of the uric acid in a specimen of urine is effected by taking the reducing power before and after precipitation with acetate of lead. The reducing action before the addition of acetate of lead is due partly to uric acid and partly to the small amount of sugar naturally present in urine. It is doubtful if there is any other body worthy of consideration to exert any sensible reducing effect.

The reducing power, expressed as uric acid, of one portion of the urine, is first determined in the ordinary way. Fifty c.c. of another portion of the urine are then taken and treated with 5 c.c. of a cold saturated solution of acetate of lead. It is necessary that enough acetate of lead should be added to secure complete precipitation, but it is desirable that there should not be a large excess left in solution. Unless the urine is very concentrated, the

quantity of acetate of lead recommended will be found to be sufficient. The precipitate having been separated by filtration, the filtrate is treated with a small fragment of caustic potash, which first throws down the oxide of lead from the surplus acetate, and then redissolves it. The reducing power, again expressed as uric acid, of the urine thus operated upon is now taken, and to the figures yielded an addition of one tenth is made in order to supply the correction required for the dilution with the lead solution.

As an illustration, let it be supposed that 20 c.c. of the ammoniated cupric test, which are equivalent to 0·1866 grammes of uric acid, are placed in the flask and, after being brought to a state of ebullition, that the urine diluted with an equal volume of water is allowed to run into it from the burette. If we assume that 10 c.c. (equivalent to 5 c.c. of urine) are required to effect decoloration, the amount of reducing power, represented as uric acid, is $\frac{\cdot 01866 \times 1000}{5 \cdot 0}$, or $\frac{18 \cdot 66}{5}$, or 3·732 per 1000.

This completes one step of the operation, and it gives the total reducing power (only a portion of which, however, is due to uric acid) of the urine. Let it be next supposed that 50 c.c. of the urine have been taken and mixed with 5 c.c. of the solution of acetate of lead, and that 7·5 c.c. of the filtrate are required to decolorise the 20 c.c. of the cupric test. We have here the reducing power of the urine minus that of its uric acid, which has been precipitated by the lead solution; and after making the necessary correction for dilution with the lead solution and working out the second calculation, the required data are furnished for showing the amount of uric acid that is present. The second calculation stands as follows:— $\frac{\cdot 01866 \times 1000}{7 \cdot 5}$, or $\frac{18 \cdot 66}{7 \cdot 5}$, or 2·488 per 1000, and correcting for the dilution $2 \cdot 488 + 0 \cdot 248 = 2 \cdot 736$ per 1000.

Having learnt that the total reducing power was 3·732 per 1000, and the reducing power after the precipitation of

the uric acid 2·736 : 0·996 per 1000 is given as the amount of uric acid present.

To ascertain if reliance could be placed upon the uric acid present being revealed by the method of procedure described, weighed quantities of it were added to specimens of urine and the estimation undertaken. The following results show that a close accord between the amount of uric acid added and that indicated was obtained.

	Uric acid found before the addition of uric acid, per 1000.	Uric acid found after the addition of uric acid, per 1000.	Uric acid indicated as having been added, per 1000.	Uric acid actually added, per 1000.
Obs. I ...	0·597	1·194	0·597	0·600
„ II ...	0·168	1·474	1·206	1·200
„ III ...	0·653	2·649	1·996	2·000
„ IV ...	0·653	1·613	0·960	1·000

A satisfactory illustration was also afforded by these observations of the complete precipitation of the uric acid that is effected by the acetate of lead. The filtrate from the lead precipitate belonging to the specimen in which uric acid had been added, presented no essential difference in reducing power from the corresponding specimens to which no uric acid had been added.

These observations suffice to show that whatever uric acid is present is revealed through the difference existing between the reducing action before and after precipitations with acetate of lead, but they do not prove that the difference is wholly attributable to uric acid. There might be something present besides uric acid precipitable by acetate of lead, and if so, it would be reckoned as uric acid. To ascertain whether the existence of such a source of fallacy is to be apprehended, observations were conducted, in which the results yielded by the process described were checked by the actual collection and subsequent determination of the uric acid.

For these check observations to be of any value, the method of procedure adopted must be secure against any loss of uric acid. After considerable attention, and trials made with added known quantities of uric acid, the fol-

lowing is the plan I have been led to have recourse to. Fifty c.c. of urine are treated with acetic acid and evaporated to dryness. The employment of hydrochloric acid is avoided on account of the coloured product which is developed by it. The dried residue containing the uric acid in a liberated state is extracted and well washed with spirit. On account of its insolubility in spirit the uric acid is left in conjunction with some saline matter, which does not interfere with the subsequent part of the analysis. Instead of employing a filter paper, the insoluble residue is best collected upon an asbestos plug introduced into the neck of a funnel, and the plug and residue, when the washing is completed, are transferred to a beaker and treated with a weak solution of potash. This dissolves the uric acid, and the solution is separated by filtration, and brought to the volume of the urine started with. The estimation of the uric acid thus isolated is effected volumetrically with the cupric solution, and on account of this plan of estimation being adopted, the complete purification from saline matter (necesssary where weighing which has hitherto constituted the means of determination had recourse to) is not called for. In this method of procedure the employment of an aqueous liquid in washing is dispensed with, which is a material item, in consequence of the slight solvent action upon the uric acid it is capable of exerting.

Looking at the nature of this process, there does not appear to be any room for questioning that the results furnished by it may be taken as representative of uric acid; and, that it is susceptible of being worked so as to give a close approach to absolute accuracy is proved by the results derived from the observations which have been conducted upon specimens of urine to which known quantities of uric acid were added. In these observations the amount of uric acid was first determined in the urine itself by the process in question. Uric acid was then added and the estimation again undertaken. Subjoined are the figures yielded in three sets of observations.

Estimation effected by the separation of the uric acid and its subsequent determination where known quantities had been added.

	Uric acid per 1000 in the urine.		Uric acid per 1000 indicated as having been added.	Uric acid per 1000 actually added.
	Before the addition of uric acid.	After the addition of uric acid.		
Obs. I .	1·00	3·06	2·06	2·00
	—	3·02	2·02	2·00
Obs. II .	0·67	2·67	2·00	2·00
	—	2·56	1·89	2·00
	—	1·67	1·00	1·00
	—	1·62	0·95	1·00
Obs. III .	0·71	2·62	1·91	2·00
	—	2·69	1·98	2·00
	—	2·62	1·91	2·00

Having learnt that reliance may be placed upon the process of separation and determination which has been described, the means are supplied for testing the accuracy of the process which forms the special subject of this communication. The two processes have been applied to various specimens of urine, and, as the following tabulated representation shows, a close accord is noticeable in the respective results obtained. With this evidence before us, sufficient grounds are afforded to justify confidence being placed in the estimation founded upon the reducing power displayed before and after precipitation with acetate of lead.

	Uric acid per 1000.	
	Estimated from the reducing power of the urine before and after precipitation with acetate of lead.	Determined from the uric acid separated from the urine.
Obs. I	1·45	1·49
„ II	1·21	1·24
„ III	0·71	0·84
„ IV	0·72	0·70
„ V	0·70	0 77
„ VI	0·90	0·84
„ VII	1·05	1·00
„ VIII	1·47	1·42
IX	1·24	1·26
X	0·67	0·62

This completes what I have to say about the process I have described for the estimation of uric acid. The next matter to deal with is the results obtained by its application to the urine under various conditions of health and disease. This is a matter of some magnitude, which I must reserve for the subject of a future communication, but I may here state that, according to the observations I have conducted, the figures that have been hitherto given as representing the amount of uric acid present will have to undergo considerable alteration.

A CASE

OF

EXTREME PHARYNGEAL STENOSIS

THE

RESULT OF SYPHILIS.

WITH REMARKS.

BY

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(Received February 10th.—Read April 13th, 1880.)

R. E—, æt. 47, a lodging-house keeper, was admitted into the Royal Hospital for Diseases of the Chest, on the 29th of October, 1878, under the care of Dr. Gilbart Smith.

As regards her family history, she stated that her parents were healthy almost up to their death from old age, but that her two brothers had died from the effects of violent cold at the ages respectively of 58 and 47. She had no sister. Of her personal history and illness, she gave the following account, viz. That during girlhood and until her marriage, twenty-six years ago, she enjoyed good health, but that a year after marriage she contracted syphilis, for which, after three months, she was

treated by Professor Millar, of Edinburgh, for a period of nine months, when she considered herself cured. However, a vaginal discharge, lasting many months, troubled her while she was pregnant with her first child, which was born three years subsequent to marriage. Although this child presented a healthy appearance, it succumbed to an attack of inflammation of the lungs when eight months old. Besides this child she has had four others, of whom two are living and healthy. She has had no stillborn child and but one miscarriage.

About seven years after marriage she was afflicted with an eruption over all her body, which, however, yielded in six or seven months to treatment; at this period also she suffered from what in all probability was an attack of iritis of the left eye.

Some ten years ago she was for the first time attacked with a soreness of the sides of the tongue, which slowly ulcerated. The ulceration extended to the back of the tongue and throat, rendering deglutition painful. This condition, sometimes better, sometimes worse, continued for four years, when it disappeared, leaving a tendency to sore throat on the slightest exposure to cold. However, four or five years ago it returned, and has since then remained, gradually increasing, until eight months ago respiration became involved, and her voice was for the first time affected, while articulation was difficult, in consequence of the impeded motion of the tongue.

For two months previous to her admission she has been subject to sudden and alarming seizures of dyspnœa, coming on, as she describes them, "like a fit," many times a day.

Her difficulty in swallowing solid food first appeared last May, since which time she has only been able to take food in a liquid form, but this dysphagia has the last week or so somewhat improved. Besides the above she has had no other ailment, and has never suffered from rheumatic fever, hæmoptysis, or any lung symptoms.

Her condition on admission is thus described:—"The

patient is emaciated and weighs six stone eleven and three quarter pounds. She is of dark complexion and has a sallow, anæmic appearance; her cheeks are deeply dimpled as a result of the contracting ulceration. The left pupil is small and adherent, and the sight of that eye somewhat impaired. Her voice is muffled, the pronunciation indistinct and difficult to understand. She complains mostly of the dyspnœa and prostration, also of dysphagia, emaciation, loss of voice, and slight occasional night perspirations. Appetite fair could she but gratify it. Tongue moist and red, protruded with difficulty. Temperature normal; pulse 76, thready and feeble; respirations 24, with stridor accompanying both inspiration and expiration. Urine acid, sp. gr. 1026, containing neither albumen nor sugar. Body emaciated; lungs normal; heart's impulse feeble, sounds weak but natural; abdomen healthy; no anasarca of legs or feet."

On examining her mouth it was observed that, owing to contractions, chiefly involving its right base, the tongue could not be protruded beyond the teeth, which were much decayed; the right posterior pillar of the fauces was drawn backwards and adhered to the pharyngeal wall, whilst the uvula and a considerable portion of the soft palate had disappeared, leaving a clean semicircular border to the part that remained.

The laryngoscope revealed the following changes. The epiglottis, rima glottidis, and vocal cords were invisible; but a small aperture was detected, one eighth of an inch in diameter, situated at the bottom of a funnel-shaped depression to the left of the middle line, and apparently on a level with the epiglottis. It was separated by a thick cicatricial band from another and deeper depression to the right, which terminated in a *cul-de-sac* containing some greenish-yellow pus. Several bands were observed to cross from the root of the tongue towards the posterior wall of the pharynx, effectually glueing the parts together, so that it was doubtful whether there was any communication with the œsophagus save through the above-mentioned aperture.

Fig. 1.

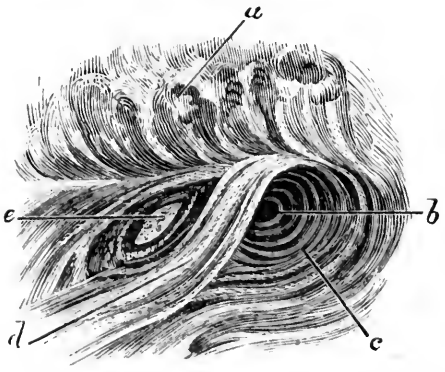

Fig. 2.

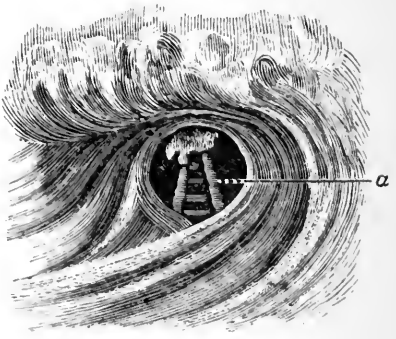

Fig. 1—Showing stenosis in lower pharynx. *a*. Base of tongue. *b*. Aperture for food and air. *c*. Funnel-shaped depression. *d*. Cicatricial band. *e*. Ulcerous *cul-de-sac*.

Fig. 2—Showing condition of the pharynx on the patient leaving the hospital, Dec., 1878. *a*. Enlarged orifice, through which the irregular-shaped epiglottis and vocal cords are seen.

On November the 1st tracheotomy was performed by Mr. Walsham without chloroform. It was borne well; there was no hæmorrhage, and the insertion of a metallic tube gave immediate relief. On the next day one of Mr. Morrant Baker's india-rubber tubes was substituted for the metallic one. The operation (tracheotomy) was, however, followed by an attack of subacute bronchitis, with rise of temperature and considerable expectoration, which gradually yielded to treatment, and on November 19th the following note is recorded :—"Patient feels better; tongue still a little sore; deglutition a little easier; pulse feeble—60. Heart sounds weak. Chest—resonance good, expansion good, breathing rather rough posteriorly and over the apex on the right side."

The pharynx was then again examined, and it was observed that the ulceration looked much less active. In order to satisfy ourselves as to the orifice being common to both larynx and œsophagus, a catheter was passed downwards and found to enter the œsophagus. The tracheotomy tube was then removed, and the catheter, inserted upwards through the trachea, was observed, with the laryngoscope, protruding through the pharyngeal opening.

On the subsidence of the bronchitis the patient was put on a course of mercury and iodide of potassium with tonics.

On November 24th, all signs of active ulceration having disappeared, Mr. Walsham made an attempt to dilate the opening common to the œsophagus and larynx. Various forms of urethral dilators, œsophagus bougies, and laminaria tents were employed, but the greatest success was obtained by introducing into the hole the blades of a small pair of curved dressing forceps, and then forcibly opening them. The tissues, however, were so rigid and unyielding that, although very considerable force was used, the aperture was not materially dilated. The attempts at dilatation, moreover, could only be made for a few seconds at a time, as the presence of the various instruments caused severe spasmodic cough and dyspnœa. Gradual dilatation was also found to be impracticable, as the laminaria or sponge tents when retained in the aperture for more than a few seconds affected the patient in a similar manner.

After the dilatation she was able to swallow some solid food, the first taken for five months.

On the 26th, the second day after the attempts at dilating, the opening was seen to be somewhat larger and to have assumed a triangular shape. Through it was perceived a small movable ridge which was thought to be the epiglottis.

Other attempts at dilatation were made, and the opening again slightly enlarged so that one of the vocal cords could be seen. From this date, November 26th, until December 5th, the attempt at dilatation was continually repeated, but although considerable force was used the tissues surrounding the aperture would not further yield.

It was resolved, therefore, to aid the dilatation by an incision through these tissues.

Mr. Walsham accordingly partially divided the cicatricial band which, as before described, separated the aperture from the ulcerous depression on its right. The incision was made by means of M. Ricord's urethrotome,

but several instruments had been used before one was found adequate to the purpose. The band proved exceedingly tough and creaked under the knife while being cut. The division was attended with but very slight hæmorrhage. On the following day, December 6th, on examining the throat with the laryngoscope the incision was seen gaping, the opening being considerably larger and the parts very tender. On December 8th Mr. Walsham further divided the cicatricial band by means of curved scissors, one blade of which was placed in the aperture the other in the ulcerated depression on the right. The incision was made little by little from the surface downwards so that should any vessel be divided its bleeding orifice might be well in view. After the incision the parts were forcibly dilated by the finger and œsophagus bougie. On the following day the throat was sore and deglutition difficult, a good deal of the food passing into the larynx and trachea, and returning by the tracheotomy tube. However, on the succeeding day there was much less tenderness, and the food no longer entered the larynx.

On December 12th another incision was made in the band at a different spot; the old incision appeared gaping and sloughing. The epiglottis, vocal cords, and arytenoid cartilages could now be distinctly seen on looking through the aperture with the laryngoscope. The epiglottis was red and distorted, the vocal cords white, coming well together on phonation.

On December 14th, the skin about the tracheotomy tube becoming swelled and œdematous, Dr. Hamilton Bland, the house physician (to whom we are much indebted for these notes), removed the tube, when the swelling and œdema immediately began to subside. The opening rapidly contracted, so that by the evening it was completely occluded.

When the patient left the hospital on December the 18th, which she was compelled to do on account of urgent family reasons, she could swallow solid food with comfort; she had no difficulty in breathing, and had gained a stone in weight since her admission. The last examination of the throat

showed the opening to be about ¾ of an inch in diameter and capable of admitting a full-sized œsophagus bougie (see woodcut, fig. 2, p. 232).

On September the 23rd, 1879,[1] she was seen by Dr. Gilbart Smith who once more examined her throat. She stated she was enjoying perfect health, and had no trouble whatever in swallowing solid food or in breathing. The opening in the throat was wider than when she left the hospital, ten months previously, and was from ¾ of an inch to an inch in diameter, allowing the vocal cords to be distinctly seen below an altered epiglottis.

Remarks.—We venture to think that this case presents features which may not prove without interest, and that it possesses valuable indications for the treatment of similar conditions.

As regards the nature of the affection, there can be no doubt that syphilis was the "fons et origo mali,"—that the extreme stenosis was the result of contractions following cicatrisations of repeated ulcerative processes involving the pharynx. From the history of the case it would appear that the constrictions and narrowing of the lower portion of the pharynx had been in progress for many years, slowly advancing step by step from above downwards. The ulceration seems first to have attacked the back of the tongue and tonsils, then to have spread to the soft palate and uvula, destroying the latter, and glueing, as it were, the right side of the soft palate and the corresponding posterior pillar of the fauces to the posterior wall of the pharynx. Subsequently and more recently proceeding downwards it appears to have invaded the walls of the pharynx as low as the level of the epiglottis where the contraction of the cicatrices resulted in the condition described. We have endeavoured to illustrate what we believe to have been the condition of the parts in a rough outline (see woodcut, fig. 3).

Constriction of the pharynx in this situation appears to

[1] This patient was exhibited on the evening of the reading of this paper, April 13th, 1880, when she gave a good report of herself, and the laryngoscope revealed no recurrence of the stenosis.

Fig. 3.

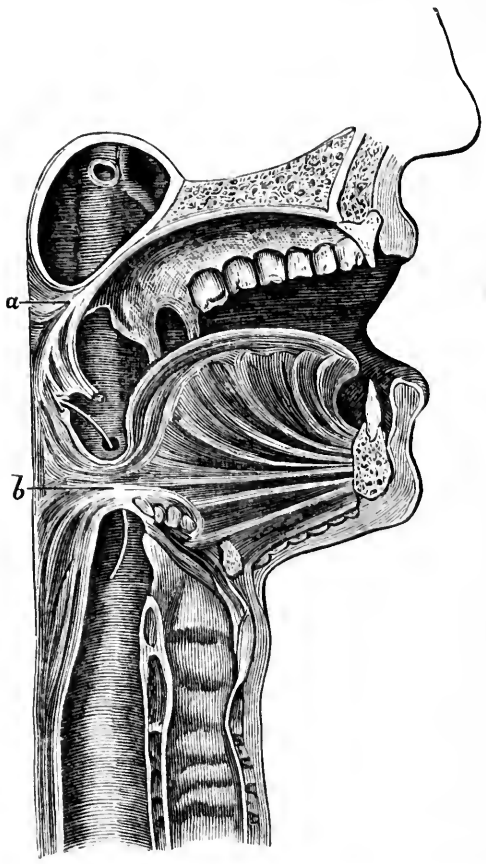

Fig. 3—Showing rough sectional outline of (what we believed to be) the condition of the parts. *a*. Adhesion of the right side of soft palate. *b*. Stenosis of lower pharynx and orifice, with arrow showing position of orifice.

be exceedingly rare. We do not remember having seen any exactly similar case ourselves, and very few, as far as we have been able to ascertain, have been reported. In the upper portion of the pharynx constrictions, although still rare, have been more frequently met with and seem to occur chiefly in two forms. In one the naso-pharynx is partially or completely cut off from the mid-pharynx by adhesions (backwards) of the soft palate and uvula to the hinder wall of the pharynx; in the other the communication between the mouth and pharynx is narrowed by the adhesion of the soft palate and its arches (downwards) to the back of the

tongue. Thus there appears to be three distinct varieties of pharyngeal stenosis, one between the naso- and mid-pharynx, one between the mouth and pharynx, and one, as in this case, involving the lower pharynx. We are well aware that other conditions, such as caries of the cervical vertebræ, post-pharyngeal abscess, perichondritis of the cricoid cartilage, &c., may lead to narrowing of the pharynx; but with such we are not concerned in the present communication. The three forms above mentioned have almost always been due to the contraction of cicatrices resulting from syphilitic ulceration, although from the cases reported by Travers [1] and Wernher [2] there appear to be exceptions to this rule. A few remarks on each form may not be devoid of interest, especially as stenosis of the pharynx has excited but little attention in this country.

Cases illustrating the first form have been reported by Mr. Messenger Bradley,[3] Drs. Cheevers,[4] Ried,[5] Coulson,[6] Scheck,[7] and others.

Partial adhesions in this situation are by no means uncommon, but total adhesion, or entire cutting off of the naso-pharyngeal cavity from the pharynx, is exceedingly rare. The most perfect example we have seen is that exhibited by Mr. Bradley at the Pathological Society, in which "the curtain of the soft palate was continued straight back beyond the posterior pillars of the fauces and became incorporated with the back wall of the pharynx, thus shutting off the communication of the posterior nares with the mouth and throat, with the exception of a small orifice of the size of a pea situated in the position which the uvula had once occupied." Czermak [8] reports a similar case in which the interesting phenomenon was noticed that in spite

[1] Türck, 'Klinik der Krankheiten des Kehlkopfs,' S. 315.

[2] 'Centralblatt für Chirurgie,' 1875, No. 30.

[3] 'Trans. Pathological Society,' vol. xxiii.

[4] 'Boston Medical and Surgical Journal,' vol. xcix, No. 21.

[5] 'Sen. Zeitschrift für Med. und Naturwiss.,' i, 4, 1864.
'Lancet,' ii, 20, 1862.

[7] 'Deutsches Archiv für Klinische Medicin,' B. xvii.
'Sitzungsberichte der Wien. Akad. der Wissensch.,' 1858, No. 8.

of the adhesions contraction of the soft palate (*i.e* rising and flattening) still took place. This is remarkable, for more commonly the muscles of the soft palate undergo atrophy, as in Mr. Bradley's case, in which they were much wasted and replaced by glistening bands of fibrous tissue. This adhesion, although generally due to deep and extensive ulceration, may be the result, as pointed out by Scheck, of a superficial inflammatory process and casting off of epithelium; he also shows that for such adhesions to take place it is not necessary that the ulceration or loss of epithelium should occur on both the palate and the back of the pharynx. This form of stenosis, as we have already mentioned, was present to some extent in our case, and we have also now under observation several patients in whom it exists in a still slighter degree.

The manner in which complete stenosis in this situation takes place we think can be deduced from the study of the various degrees of constriction furnished by these cases. At first catarrhal inflammation leads to various degrees of thickening of the mucous membrane and its epithelium, giving rise to increase of tension of the posterior pillars and hypertrophy of their attachments to the pharyngeal wall. As a result of this there is a greater liability to infiltration and subsequent ulceration from over-stretching of the narrowed arch, and when once this takes place adhesion (following advancing ulceration) from below upwards, accompanied by the formation of cicatricial contractions, is the most likely occurrence unless arrested by treatment. This adhering process is materially assisted by the chronic naso-pharyngeal catarrh usually present; for owing to blocking of the nares by the swelling and secretion of the mucous membrane the respiratory current is absent from the upper pharynx and therefore fails to keep the ulcerated surfaces apart.

According to Scheck, adhesion of the palate to the pharynx is more likely to occur when there is a loss of the uvula or perforation of the palate. For the current of air that is expelled during violent expiratory efforts such as coughing or sneezing passes through the aperture left by the

perforation or absence of the uvula; whereas when these conditions are not present the stream of air falls with its full force upon the adhering surfaces, and so tends to tear them asunder before they have become firmly united.

The second form of stenosis, namely, that in which the contractions interfere mainly with the communication between the mouth and the pharynx, is more rare.

A case of the kind is reported by Mr. West in the second volume of the 'Lancet' for 1872, and we are informed that another was some years ago under treatment at the London Hospital. In the former patient, to use Mr. West's own words, "the soft palate was seen to be ulcerated and cicatrised to the base of the tougue, having on one side an opening only the size of a small pea; through this opening the food had to pass, and the function of respiration had to be performed." He does not state if the communication between the nose and pharynx was cut off. The explanation of this form of stenosis is similar to that which we have already given for the first mentioned variety. Whereas the one is produced by ulceration and contraction of the *posterior* pillars of the fauces, and the drawing backwards and subsequent adhesion of the soft palate to the pharyngeal wall, the other is produced by ulceration and contraction of the *anterior* pillars and the drawing downwards and subsequent adhesion of the palate to the dorsum of the tongue.

Of the third form in which the lower pharynx is affected, cases are reported by Gerhardt,[1] Scheck,[2] Trendelenburg,[3] Schroetter,[4] West,[5] and Cheevers.[6] In Scheck's and Gerhard's cases a membrane extended across the pharynx from the base of the tongue to the posterior pharyngeal wall. In the former case the membrane presented an oval shaped hole in its centre through which the epiglottis protruded;

[1] "Ueber Syphil. Krankheiten des Kehlkopfs," 'Virchow's Archiv,' xxi.

[2] 'Deutches Arch. Klin. Med.,' B. xvii.

[3] 'Archiv für Klin. Chir.,' xiii, 2, p. 335.

[4] 'Jahresbericht,' &c., 1871 und 1874.

[5] 'Lancet,' ii, 1872.

[6] 'Boston Med. and Surg. Journal,' vol. xcix, No. 21.

in the latter case the orifice was eccentrically placed, and altered its shape in speaking; the epiglottis and vocal chords could not be seen, and the membrane felt soft, and seemed to be composed of muscular as well as of mucous tissue, as the movements observed were not communicated to it by the tongue. Trendelenburg's case, which is alluded to by Wendt in 'Ziemssen's Cyclopædia,' resembles the above; but the aperture in the membrane was only the size of a pea and its closure caused dyspnœa. These cases appear to differ from ours, inasmuch as the stenosis depended upon the presence of a soft membrane rather than upon tough cicatricial bands. Schroetter has observed conditions resembling those found in our patient, in which scar-like tendinous bridges or trellis-like bands traversed the cavity of the pharynx, and like side scenes closed in upon its lumen.

Mr. West also alludes to a case in the article in the 'Lancet,' above mentioned, in which M. Ricord "performed tracheotomy on a patient who was almost moribund with advancing asphyxia," and in which after death the pharynx, soft palate and epiglottis appeared to have been affected in a manner similar to that observed in our case. Dr. Cheevers also relates an instance of tertiary syphilis where the pillars of the fauces were ulcerated away, and the passage downwards was gradually closing—the patient becoming slowly unable to breathe.

With reference to the treatment, the first indication was clearly to relieve the patient of the distressing and alarming attacks of dyspnœa which threatened at any moment to terminate life.

This it appeared to us would be best accomplished by the immediate performance of tracheotomy. Considering that the respiratory function was carried on through an aperture so minute, and at any moment so liable to be further diminished, or perhaps even occluded by sudden œdema, any delay would, in our opinion, have been dangerous in the extreme. Moreover, when we reflect that, as was afterwards demonstrated, this small orifice served for the passage of food as well as air, we cannot but wonder that it had not

long since become blocked with a fatal result. The subsequent history of the case has strengthened our belief that opening the trachea was the best and only safe course to pursue, and it is the one we should most strongly recommend in any similar case. It may be objected that tracheotomy was uncalled for, at least until dilatation of the constricted part had been attempted; but we submit that such an attempt would in all probability have added to the dyspnœa, already urgent, and necessitated a hasty performance of tracheotomy, as even after the trachea had been opened, any attempt to dilate caused intense suffocative fits of spasmodic cough and dyspnœa. However, we advocate the performance of tracheotomy, not only as a temporary expedient to obviate death from suffocation whilst some more effectual measure for permanently relieving the constriction is being planned, but also as essential to the satisfactory and safe performance of any such subsequent operation. In the first place, it allows difficult manipulation to be borne with diminished spasm; secondly, it relieves the surgeon of a considerable source of anxiety; and thirdly, it reduces the risks should hæmorrhage take place, for, having secured a free passage for air, blood could be prevented from entering the trachea by plugging the upper portion of this tube. Of this danger from hæmorrhage we were not ignorant, knowing that in a similar case, where tracheotomy had not been performed, death from suffocation had resulted, owing to the formation of a blood-clot in the trachea.

With regard to the measures resorted to for the permanent relief of the stenosis, it is noteworthy that all attempts at dilatation signally failed to produce any real benefit; and gradual stretching by means of laminaria and sponge tents, notwithstanding the opening into the trachea, could not be endured.

Caustics and the use of the galvano-cautery did not commend themselves to us, seeing that in other forms of

pharyngeal stenosis relief afforded by such means had been only of a temporary character.

It was not, however, until we had thoroughly convinced ourselves of the inutility of further efforts at stretching that we resolved to resort to cutting. We felt that not only should we experience the usual difficulties of all operations where the structures needing division can only be seen as reflected and inverted images, which, at the moment of making an incision requiring precise care as to force and direction, frequently disappear, but also we should be in no little danger of wounding important structures in consequence of the altered anatomical relation due to contracting lesions. The marked drawing inwards of the cheeks and the tissues behind the angle of the jaw, together with the impaired movement of the tongue, left us in doubt as to how far the internal carotid artery had been involved in the cicatrices.

We have delayed laying this case before the Society in order to be in a better position to report upon the result of the treatment. As it is now twelve months since the last operation was performed, and as at present no recurrence of the contraction, but, on the contrary, much improvement, has taken place, it appears probable that this improvement will continue and that stenosis will not recur.

Although fully aware of the questionable value of deductions drawn from the experience of a single case, we venture to formulate the following as the conclusions which we think may have a practical bearing on similar lesions :

That tracheotomy is called for both as a temporary expedient to obviate sudden death from asphyxia, and as an essential factor for safe and satisfactory treatment.

That division with a guarded knife presents advantages over other methods of operation.

That several small notches are preferable to a single deep incision, and that, when possible, the parts should be gradually divided from above downwards, so as to allow of a full view of the cut surface of the tissues divided.

That the aperture should be enlarged in a direction so as to permit the passage of liquid food clear of the entrance to the larynx.

In conclusion, we would add that the rarity of the lesion, the difficulty in recognising its exact nature, and the lines for treatment of similar cases which the operative measures here undertaken suggest, are our apology for bringing the case before the Society.

ON THE

PARTIAL METABOLISM, BY THE LIVER,

OF

LEUCIN AND TYROSIN INTO UREA.

BY

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HAVING been taught to regard the presence of leucin and tyrosin in the urine as diagnostic of that exceedingly rare disease of the liver known as acute yellow atrophy, or when occurring in typhus, smallpox, and some of the other exanthemata, as only indicating concurrent acute atrophy of the liver, I, on finding that the urine of a patient under my charge (No. 1 in the list quoted in the following pages) evidenced the presence of leucin and tyrosin, became alarmed for his safety, and it caused me, on the first occasion presenting, to seek the advice and assistance of a Newcastle physician.

The fact that not only did my patient recover, and his urine cease, for the time being, to produce, on analysis, leucin and tyrosin, but that they returned in greater or less

amount with each return of his former symptoms, caused me to look for leucin and tyrosin in the urine of other patients; first, in those similarly situated with respect to their ailments, and, finally, under every form or modification of disease likely to result in interruption of the hepatic functions. The result of my investigations is, that leucin and tyrosin are found in the urine under numerous different pathological conditions, whether affecting the liver intrinsically or from without; and that as often and as soon as the patient recovers, these substitution-products for urea, as they are termed, first diminish in amount, and finally, in most instances, disappear. The numbers, quoted and recorded in the following pages (33), represent individuals, not cases, for some of the patients returned under treatment after a longer or shorter interval on several occasions; so that, counted as cases, they would amount to more than sixty.

Method of preparation of leucin and tyrosin.—The method adopted for preparing leucin and tyrosin has usually been the following:—To four fluid ounces of urine add of a standard solution of lead acetate an excess, so that, on filtering a small quantity of the mixture, and adding thereto a drop of the lead acetate solution, no precipitate occurs. Filter. H_2S is then passed through to throw down the excess of lead. Again filter, and gently evaporate the filtrate in a water-bath to the consistency of syrup. Set aside for a day or two, for the tyrosin, if present, to crystallize out. Pour off and evaporate to dryness. Extract the residue with boiling alcohol. Filter. Evaporate the filtrate to a syrup. If required to purify (the leucin) still further, Hoppe-Seyler's method is applied, viz.:—Dissolve in ammonia, add lead acetate till no further precipitate is produced. Filter. Wash the precipitate with a little water. Suspend it in water, and pass H_2S through it. Filter. Evaporate the filtrate in the water-bath.

Test for leucin.—The smell of amylamine.

Tests for leucin and tyrosin:

Scherer's test for leucin and tyrosin.

Hoffmann's test for tyrosin.
Piria's test for tyrosin.
Microscopic test for both.

Over 500 analyses for leucin and tyrosin have been made by me within the past two years.

Many authorities might be quoted in support of the statement that the almost universal consensus of opinion is to the effect that "the presence in the urine of leucin and tyrosin exists most especially in that very rare disease known as acute yellow atrophy, that they are only occasionally present in some few other diseases, viz. in chronic yellow atrophy and leucocythemia (Beale); in typhus, smallpox, and other exanthemata (Frerichs and Städeler); attributed, however, to the probable concurrence of yellow atrophy (Murchison); and only one to 'remora' or hindrance to the hepatic circulation, due to valvular disease of the heart (Balfour)."

I proceed to give a list of patients suffering from a variety of complaints, in the course of which either leucin alone, or it in company with tyrosin and uric acid, was present in the urine.

No. 1. W. R—, first seen on November 11th, 1877. Jaundice.

Nov. 25th. *Urinary examination.*—Leucin and tyrosin present in large amount. Met Dr. G. H. Philipson, of Newcastle, in consultation, to whom I showed the mounted microscopic slides of leucin and tyrosin.

Result.—Recovery. All but traces of leucin and tyrosin in time disappeared. Occasionally, since then, leucin has reappeared, to again disappear under administration of hepatic stimulants, and notably of euonymin.

No. 2. Ann D—, æt. about 56; a stout, dark-complexioned woman. Heart: area of dulness increased, with mitral systolic and aortic systolic bruits.

Jan. 12th, 1878. *Urinary examination.*—Leucin present. Under treatment she improved, although leucin is, more or less, constantly present.

No. 3. D. McVai, æt. 35; first seen on January 19th, 1878. For years had been a hard beer and spirit drinker. Spleen: area of dulness increased. Liver small, contracted. Skin dry, anasarcous. Abdomen tympanitic, ascitic.

Urinary examination.—Acid, no albumin, bile, sugar; leucin and tyrosin in very large amount. He entered Durham Infirmary as an in-patient on the same day, and was discharged at the end of a few weeks. He died, I am informed, in September, 1878. I saw him once in the interim after his return from Durham, and examined again his urine, which still contained leucin and tyrosin in considerable amount. Cause of death stated to be English cholera: most probably from the form of diarrhœa consequent upon the advanced stage of his disease—cirrhosis.

No. 4. R—, an overman in a coal mine; February, 1878. Complained of giddiness, loss of appetite, &c. Liver: area of dulness increased, sluggish.

Urinary examination.—Bile, sugar, leucin.

Result.—Recovery; leucin disappeared.

No. 5. R. D—, a coalheaver, æt. 23; February, 1878. Suffering from rheumatic fever and hypostatic pneumonia. Pericardium so full of fluid as to almost completely mask the sounds of the heart. Liver: area of dulness increased and very inactive.

Urinary examination. March 17th, 1878.—Leucin in large quantity.

March 25th.—Mere trace of leucin.

Result.—Recovery with, on absorption of pericardial fluid, evidence of mitral insufficiency. Occasionally leucin reappeared.

No. 6. J. G—, æt. 32; February, 1878. Suffering from pneumonia and acute rheumatism.

Urinary examination.—Leucin.

Result.—Recovery, with disappearance of leucin.

No. 7. W. P—, an overman, æt. 44. Suffering from

dyspepsia, with alternations of the uric acid and oxalic acid diatheses. Occasionally suffered from acute nephritis, and some months ago passed a calculus per urethram.

Urinary examination.—Albumin, pus, mucus, uric acid, and leucin.

Result.—After passage of calculus, recovery.

No. 8. J. A—, coke drawer, æt. 19½. January, 1878. Complained of headache, giddiness, anorexia, and loss of strength. He was excessively anæmic. Liver: area increased, tender, sluggish. Spleen area increased.

Urinary examination.—Leucin.

Treatment.—Ferruginous tonics, hepatic stimulants, gentle alvine evacuants, and generous, easily digestible, unstimulating diet. To cease smoking.

Result.—Returned to work in about six weeks. Leucin disappeared.

No. 9. J. W—, a putter, æt. 14. First seen August 19th, 1878. The subject of phthisis, and of intestinal obstruction, caused, most probably, by one or more bands of organisable lymph.

Urinary examination.—Leucin and tyrosin in very large amount.

Result.—Death.

No. 10. J. H—, a publican. First seen July 29th, 1878. Suffering from colic. (The subject, in September, 1877, of fæcal obstruction accompanied with jaundice, and of the presence in urine of bile and albumen.)

Urinary examination.—Leucin and tyrosin in very large amount.

Result.—Recovery, with complete temporary disappearance, first of tyrosin, and then of leucin. Occasionally leucin has reappeared.

No. 11. W—, a coal hewer. August 6th, 1878. Suffering from right pleuro-pneumonia.

Urinary examination.—Leucin and tyrosin in very large amount.

Result.—Recovery.

No. 12. Miss G—, æt. 40. The subject of bronchitis, with great exacerbation, especially if liver sluggish.

Sept. 5th, 1878. *Urinary examination.*—Leucin and tyrosin in large amount. Temperature 103·5° F. Salicylic acid administered; within less that twenty-four hours temperature reduced to 98·6° F., with relief of all symptoms, and increased quantity of urine, which, upon analysis, showed great diminution of leucin and tyrosin, and increased quantity of urea.

This patient takes now, regularly from one and a half to three grains of euonymin, and keeps by her a mixture containing salicylic acid; and although, for years past, a great sufferer from bronchitis, she has for months been nearly free.

No. 13. Mrs. H—, a short stout woman. Suffering from frequent hepatic derangement.

Oct. 13th, 1878. *Urinary examination.*—Leucin in large quantity, to disappear on recovery, and reappear in each successive attack.

No. 14. Mrs. H—, October 22nd, 1878. Suffering from varicose ulcer of the leg, and much inaction of the liver.

Urinary examination.—Leucin in large amount.

No. 15. Mrs. H. R—. Has occasional severe attacks of hepatic derangement.

Oct. 22nd, 1878. *Urinary examination.*—Leucin in large amount, tyrosin in smaller quantity.

Result.—Recovery; both disappeared.

No. 16. T. J—, æt. 66; an active free-living man, with heart displacement to right, intermittent pulse, and emphysema of left lung.

Urinary examination.—Leucin.

No. 17. A female patient in Stockton-on-Tees Hospital, with Bright's disease, and presystolic and mitral systolic bruits.

Urinary examination. Nov. 12th, 1878.—Inter alia, leucin and tyrosin in large amount. She became better, and worked in the hospital for some months, but finally succumbed to Bright's disease, &c.

No. 18. Mrs. T—, senr., æt. 56. Bronchitis, with occasionally spasmodic asthma.

Urinary examination.—Leucin.

No. 19. John S—, a blacksmith. Rheumatism.

Urinary examination.—Tyrosin in large amount. Leucin in smaller. (He drank freely). Urine several times afterwards examined, with result of disappearance first of tyrosin, and then of leucin.

No. 20. H. D—, senr., æt. 65. Pleuro-pneumonia of right side and base of right lung behind.

Nov. 30th, 1878, *Urinary examination.*—Leucin and tyrosin in very large amount. During returning convalescence tyrosin first disappeared, and lastly leucin.

No. 21. R. D—, an overman. Sudden attack of giddiness. Liver very inactive.

Dec. 12th, 1878. *Urinary examination.*—Leucin.

No. 22. Margaret D—, æt. 20. Puerperal eclampsia on January 31st, 1879.

Urinary examination.—Of about five fluid ounces, the total amount passed in the three first days. Albumin copious; uric acid in excessive quantity; urea very deficient; very large amount of leucin and tyrosin (particularly of the latter, 25, 11, 79).

Result.—Recovery, with total disappearance of leucin and tyrosin, diminution of uric acid, disappearance of albumin, and increase of urea.

No. 23. Miss McC—, Salop. Chronic and spasmodic asthma.

Aug. 14th 1879. *Urinary examination.*—Leucin.

Treatment.—Inter alia, euonymin nightly, and gentle evacuant each succeeding morning.

28th.—Considerably improved in health. Leucin reduced in quantity.

No. 24. Thomas A—, Salop. Incipient delirium tremens after much drinking.

Aug. 16th, 1879. *Urinary examination.*—Leucin and tyrosin and large amount of albumin.

25th.—No albumin, but leucin still present. Tyrosin disappeared. Himself well and about.

No. 25. Miss M—, Salop. Phthisis.

Aug. 19th, 1879. *Urinary examination.*—Leucin in large amount.

No. 26. Miss K—, Salop; æt. 24. Chronic asthma with spasmodic attacks.

Aug. 23rd, 1879. *Urinary examination.*—Leucin in very large amount.

No. 27. John M—, Salop. Labourer; a somewhat free drinker. Left lung somewhat emphysematous; heart displaced to right; area of liver increased.

Aug. 25th, 1879. *Urinary examination.*—Leucin.

No. 28. S. T. B. B—, Salop (æt. about 70). Hemiplegia of about four years' standing; about three times epileptic. Liver sluggish. In other respects healthy with vigorous intellect.

Urinary examination. From August 6th to 29th.—Albumin in small quantity. No urinary casts. Leucin.

Result.—Albumin reduced to mere trace; leucin lessened in amount. More urea and more water passed in urine. General health considerably improved.

Treatment.—Euonymin gr. ij or gr. iss nightly, with from fluid ʒiv to fluid ʒviij of Friedrichshall every succeeding morning. Most other medicine discontinued.

No. 29. K. W—, æt. 32. Chronic asthma.

Aug. 30th, 1879. *Urinary examination.*—Leucin and tyrosin.

No. 30. M. W—, æt. 5. Infantile jaundice. A strong, active, healthy child, apparently.

Aug. 30th, 1879. *Urinary examination.*—Leucin.

No. 31. Florence W—, Salop, æt. 7. Pertussis.

Sept. 2nd, 1879. *Urinary examination.*—Leucin; tyrosin in minute amount (25, 11, 79).

No. 32. Old female patient in Stockton-on-Tees Hospital. Jaundice, arising probably from cancer of liver.

Nov. 5th, 1879. *Urinary examination.*—Much tyrosin and leucin.

No. 33. Mrs. R—, æt 44. Puerperal septicæmia with embolism into base of right lung.

Feb. 1879. *Urinary examination.*—Leucin and tyrosin. *Result.*—Death.

I have thus shown the presence of leucin and tyrosin in the course of eighteen or nineteen different pathological conditions: different in two respects; viz. as, first, differing among themselves; and, secondly, differing, inasmuch as they do not include either acute yellow atrophy, typhus, smallpox, or the other exanthemata. If, therefore, these last-named diseases be added to the foregoing, the number of distinct manifestations of disease in which leucin and tyrosin present themselves, is necessarily still further increased.

Physiologically, the sources of leucin and tyrosin are two-fold; viz. as a result of digestive proteolysis, and, secondly, of the metabolism of the tissues of the organism. Whether they can be re-elaborated into more complex bodies, and thus regenerated into albumin is unknown, although it is more likely to be the case with tyrosin. Respecting the further decomposition of leucin, it is certain that it can be split up into urea by a decomposition into ammonia, and into a fat, or be oxidised into carbonic acid.

Besides the leucin and tyrosin arising as a result of digestive proteolysis, most of which passes directly to the liver through the portal vein, the spleen may be regarded as a

great source of the leucin and tyrosin (at least of leucin, if not of both), arising from the destructive metamorphosis of the tissues of the body. The spleen is by some regarded as a possible source of urea, from its metabolism of leucin and tyrosin; but when it is considered that the only organ in which urea is found normally, in any amount, is the liver, and that the whole of the splenic blood passes into the vena portæ, and thence to the liver, it may reasonably be inferred that it is the office and function of the liver to convert these substances into urea, or to otherwise dispose of them, and that the spleen is, therefore, only indirectly a source of urea.

I have frequently remarked that tyrosin only occurs in the urine in the severer forms of hepatic interruption, and that it is the first to disappear during convalescence. The greater difficulty with which it is, if at all, converted into urea, and the greater complexity of its molecule, would seem to render it quite conceivable that it is more easily so conjugated as to subserve a higher use in the organism than leucin; while its appearance in the urine, on the other hand, in only the severer forms of disease, evidences considerably greater diminution of hepatic functional activity, so that it, as well as leucin, is excreted.

The experiments of Rutherford, Vignal, and Dodd, reported in the 'British Medical Journal,' for 1878, abundantly prove that with regard to the preparations of salicylic and benzoic acid, irrespective of their passage through the kidneys as salicyluric and hippuric acid, they greatly increase the secretion of bile. So likewise with euonymin. I had long known the effect of salicylic acid in increasing very greatly the urates of the urine, but until then I had not used euonymin. From and since this time, however, I have employed it somewhat extensively, and occasionally in conjunction with the salicylates of potash and of soda. As an instance of the beneficial influence of salicylic acid, not only in reducing temperature and increasing the amount of urea, but also in diminishing the quantity of leucin and tyrosin, Case No. 12 is instructive. Since this patient has regularly taken euonymin, followed each succeeding morning by a

gentle evacuant, she has enjoyed a remarkable improvement in health. As to the effect of euonymin alone in reducing the quantity of albumen present in the urine, increasing the urea and quantity of urine passed, and lessening the amount of leucin, No. 28 may be cited. I have used euonymin, notably in cases of asthma complicated with hepatic derangement and the consequent presence in the urine of leucin and tyrosin, and always with marked benefit, both in relieving the more distressing effects of this complaint, in lessening the quantity of leucin and tyrosin, and in warding off or at least lessening the severity of succeeding attacks. I must not be understood as inferring that euonymin combines with glycin in its tendency to increase the formation of urea, but I presume that by its very decided action in augmenting the secretion and flow of bile, it in this manner greatly conduces to the re-establishment of the normal hepatic functions, by enabling the liver to set free and utilise its glycin.

I have thus shown that leucin and tyrosin are present in the urine of man in the course of numerous diseases, and that their presence is not, as hitherto universally held, confined to a few. While the researches and experiments of physiologists prove conclusively that leucin and tyrosin can be and are (especially leucin) converted into urea, and afford the strongest presumption that it is a function of the liver so to convert them, the numerous records of the appearance and disappearance of these substitution-products, leucin and tyrosin, in the course of the diseases, and of their treatment, strengthen and support, in the fullest possible manner, the doctrine of the urea-forming function of the liver.

AN ACCOUNT

OF THE

DISSECTION OF THE PARTS REMOVED AFTER DEATH FROM THE BODY OF A WOMAN THE SUBJECT OF

CONGENITAL MALFORMATION

OF THE

SPINAL COLUMN, BONY THORAX, AND LEFT SCAPULAR ARCH;

WITH REMARKS ON THE PROBABLE NATURE OF THE DEFECTS IN DEVELOPMENT PRODUCING THE DEFORMITIES.

BY

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(Received March 11th—Read April 27th, 1880.)

Previous history of the case.—Of the woman J. H—, from whom this specimen of deformed thorax was taken, the following particulars are known:

1. The curvature had existed since birth.

2. She was of average height, and, until uncovered, did not present any noticeable deformity.

3. She had given birth to a living child.

4. Her mother had a curvature of the spine.

5. When undergoing physical examination of the chest, an immobility of the scapula had attracted attention, and on this account she was referred to one of us (Mr. Willett).

6. The immobility of the scapula was found to be due to its union through the medium of a broad bridge of bone with the lower cervical vertebræ.

7. Extremely wide intercostal spaces were noticed, the impulse of the heart's apex being widely diffused and abnormally distinct, as if seen through attenuated structures.

8. She was the subject of mitral disease, and died of cardiac dropsy in her thirty-first year.

Casts taken during life are preserved in the Museum of St. Bartholomew's Hospital.

At the post-mortem examination, the whole thorax, the left scapula, the left clavicle, the upper part of the left humerus, and part of the right clavicle, together with the soft parts connected with them, were removed (see Plate VI and Plate V, fig. 2).

The viscera presented no point of particular interest.

An account of the muscles and other soft parts will be deferred until after the description of the bones, as their attachments and relations will then be better followed.

Description of the bones.—To facilitate comprehension we have thought it advisable to offer a few remarks upon the general appearance of the specimen before proceeding to give a more detailed account of the peculiar abnormalities of the different bones.

General appearance of the specimen.—The portion of the spinal column preserved deviates from the natural condition—1st. In that not only has it lost the posterior convexity, but it has, if anything, a slight anterior curve. 2ndly. There is a sharp lateral curve, with the convexity to the left, in the upper dorsal region, corresponding to the absence of the right half of the third dorsal vertebra. 3rdly. There is a longer and slighter compensatory curve in the lower dorsal region. 4thly. Four and a half of the dorsal vertebra are missing.

The distortion of the thorax corresponds generally to the

altered line of the spine. There are five ribs missing on the right and four on the left side of the chest; the intercostal spaces are abnormally wide. The sternum lies obliquely across the middle line, pointing in its long axis downwards and from right to left. Its upper end is only three quarters of an inch from the spine, its lower end three inches instead of two and nine inches respectively. The cavity of the thorax is diminished in all its diameters, especially in the antero-posterior, in the middle line, whilst this measurement is markedly less on the right side than on the left. The left clavicle deviates notably from the normal. Its general direction from the sternum is upwards, forwards, and outwards, instead of nearly horizontally outwards. It has also only a single posterior curve throughout its entire length.

The left scapula presents a most remarkable deformity. There is a broad bridge of bone extending from the middle third of the posterior border of the scapula backwards and upwards to the lamina and spinous process of the sixth cervical vertebra. The transverse axis of the scapula is elongated, whilst the vertical is shortened and the bone appears rotated forwards, so that the glenoid cavity looks nearly directly forwards, whilst the plane of it is nearly an inch in front of the left sterno-clavicular articulation. The upper border and spine, instead of being directed nearly horizontally outwards from the base, slope downwards and forwards as well as outwards.

Having thus given a general outline of the specimen, we shall now proceed to describe its several parts.

Description of the vertebræ.—Counting the bodies, twelve and a half vertebræ are apparently present in the specimen. Of these the two upper are presumably cervical, the three lower lumbar, and the remaining seven and a half dorsal. We have had considerably difficulty in referring each of these dorsal vertebræ to its number in the normal series. We believe that they correspond respectively to the first, second, half the third, fourth, seventh, tenth, eleventh, and twelfth. The uppermost vertebra in the specimen is apparently the sixth cervical; its body and transverse processes

are fairly normal; its spine is cleft, the left half of the spine and left lamina being fused with the irregularly triangular portion of bone already mentioned, and by it connected to the posterior border of the scapula. The right half of the spinous process is free and projects backwards and to the left, slightly overwrapping the triangular portion of bone, to which it is anchylosed at its base. The left half of the medullary canal, viewed from above in transverse section, is round instead of triangular, as it is on the opposite side. The shape of the vertebra and canal are best explained by the accompanying photograph taken from above (see Plate V, fig. 1).

The second vertebra is apparently the seventh cervical. On the right side it has the characters of the normal seventh, but on the left side the body and transverse process are fused with a process of bone, which appears at first sight to be the first rib. This process of bone we regard, however, as a cervical rib, that is, as the anterior part of the transverse process of the seventh cervical vertebra, a process which, as is well known, is developed from a separate centre of ossification, and frequently grows beyond its normal dimensions, constituting a supernumerary rib, which may either remain free, resembling the asternal ribs of birds, or be joined, as in the present instance, by its distal end to the first thoracic rib. The spinous process of this vertebra is fairly normal, and is fused at its apex with the left half of the spinous process of the first dorsal vertebra. The left lower articular process and the lower border of the left lamina are anchylosed with the superior articular process, and the upper border of the left lamina of the first dorsal vertebra. In the recent state, very free movement existed between the the sixth and seventh cervical vertebræ. The scapula, the irregular portion of bone already mentioned, and the sixth cervical vertebra, moved as one piece at this articulation. The first dorsal presents on each side of its body a facet and a half, as is normally the case, a whole facet above for the first rib and a demi-facet below for half of the head of the second rib; the upper facet on the right side, however, is

higher than that on the left, the portion of bone forming this facet encroaching above upon the intervertebral substance between the seventh cervical and first dorsal vertebræ. The bodies of the seventh cervical and first dorsal are anchylosed by bone stretching between them on the left side. The spinous process is cleft, and the two halves separated except at their base, the left half and corresponding lamina being rotated vertically upwards and anchylosed with the spinous process and lamina of the seventh cervical, whilst the right half and corresponding lamina are rotated downwards and anchylosed with the left half of the spinous process of the second dorsal vertebra, which process is likewise cleft and its halves similarly separated.

This displacement of the two halves of the spinous process must have occurred before each lateral arch of the vertebra had united posteriorly; and when viewed in connection with the coincident displacement of the superior facet for the first rib on each side of the body, suggests the idea that the whole of that part of each lateral half of the vertebra which is developed from the lateral centre of ossification was involved in the displacement.

Looking to the way in which the vertebræ are ossified this would appear not improbable. In the dorsal vertebræ, as is well known, the centre for the body does not extend into the lateral masses, but is limited to that portion of the body which is situated between the lateral facets for the ribs; the lateral portions of the body, and consequently the facets for the ribs, are ossified by the extension into them of the centre for the lateral masses, a narrow cartilaginous interval (the neural central suture) existing on each side of the body for some time between the centre for the body and each lateral mass. As the right facet for the rib and right pedicle are higher than the left, whilst the right lamina and spine are depressed below the corresponding parts on the left side, it would appear that each lateral half of the vertebra, already ossified and moving as a whole, had been rotated on an imaginary axis drawn through the transverse process, the anterior half on the right side being raised

and the posterior part depressed, and *vice versâ* on the left side. The appearance of the parts, we think, justifies such a supposition, and how this displacement might have taken place we shall endeavour to show later on. The second dorsal vertebra presents as usual two demi-facets on the left side of its body for the second and third ribs, but no distinct facets on the right side. The left half of the body is anchylosed to what appears to be the left half of the body of the third dorsal. At first sight it seems as if the right lamina and right half of the spinous process and transverse process were deficient, but on closer examination it is evident that these parts are not absent, but are fused with the corresponding parts of the fourth dorsal vertebra, these parts of the third being wanting. The left half of the spinous process, the left lamina, and transverse process, are distinct. The left half of the spinous process is anchylosed, as before stated, to the right half of the first dorsal. The left lamina is anchylosed to the laminæ above and below, and the transverse process articulates with the tubercle of the second rib, and by its apex is anchylosed to the transverse process of the third dorsal. It would, therefore, appear that the spinous process had remained cleft like that of the vertebra above, and that the right half and right lamina had been rotated downwards in a similar manner to that of the right half of the first dorsal, and in this position had become fused with the fourth vertebra.

The right half of the third vertebra is wanting. Viewed from the front the left half of its body appears as a triangular piece of bone, with its base to the left, wedged in and anchylosed between the bodies of the second and fourth. It presents two demi-facets for the heads of the third and fourth ribs respectively. Posteriorly, the left transverse process, the left lamina, and the left half of the spinous process, are fused below with the fourth vertebra, and above with the second ; the left half of the spinous process, as before stated, is also fused in the middle line with the right half of the spinous process of the second dorsal vertebra. As in front, so posteriorly there are no signs of the existence of a right half, although at first

sight what we have described as the right transverse process, right lamina, and right half of the spinous process of the second dorsal might be taken for it.

The transverse process on the left side articulates with the third rib.

The deficiency of the right half of this third vertebra is the cause of the lateral curvature of the spine, the four vertebræ above all leaning to the right. It may also account for the peculiar displacement of the cleft spinous processes. The next vertebra might be considered as the fourth or sixth; we believe it to be the fourth. Its body presents two demi-facets on each side as usual, which articulate with the fourth and fifth rib in the specimen on the left side, and with the third and fourth rib in the *specimen* on the right side. Above, it is anchylosed on the left side with the half body of the third; on the right side it is in contact with the second. Posteriorly, its left transverse process, left lamina, and left half of the spinous process are fused with the similar parts of the third dorsal, as already noticed. Its right transverse process, right lamina, and right half of the spinous process are fused with the similar parts of the second dorsal. Its transverse process articulates with the fourth rib on the left side, and on the right side with the third rib in the specimen, which has characters intermediate with those of the third and fourth.

The next vertebra is apparently the seventh. It presents two demi-facets on each side of the body, as usual, for articulating with the fifth and sixth rib in the specimen on the left side, and with the fourth and fifth on the right side. Posteriorly, its laminæ and spine are increased in depth, having somewhat the appearance as if they had been fused with similar parts of another vertebra.

The next vertebra presents a single demi-facet on each side above for articulation with the sixth rib on the left side and the fifth rib on the right. Posteriorly, its spinous process appears to have been cleft, the left half being bent towards the right, overhanging the right half, which appears to have been imperfectly developed.

The next vertebra presents one whole facet above on each side of the body for articulation with the seventh rib on the left side and the sixth on the right. Posteriorly, it does not present anything remarkable.

The next vertebra is apparently the twelfth. It presents two whole facets on each side of the body above for articulating with the eighth rib in the specimen on the left side and the seventh on the right.

The next three vertebræ are apparently lumbar, and do not call for any special remarks.

It will thus be seen that four and a half of the dorsal vertebræ are wanting; and those present we are inclined to regard as the first, second, half the third, the fourth, seventh, ninth, eleventh, and twelfth.

Description of the ribs.—In dealing with the ribs we have also had much difficulty in referring each to its number in the normal series. On the right side there are apparently seven; of these four are true, one is false, and two are floating. On the left side there are apparently eight or nine, if we regard the portion of bone articulating with the second vertebra in the specimen as a rib. Of these, excluding the above-mentioned rib, there are five true, two false, and one floating. On the right side the first has the character of the first rib; it articulates with the upper part of the right half of what we believe to be the first dorsal vertebra and with the facet on the transverse process of the same, and by its anterior end with the sternum through the intermediation of a costal cartilage, about half an inch long.

The second and third ribs in the specimen are fused posteriorly in about a fifth of their length. The second, we believe, represents the second in the normal series, although it has some of the characters of the normal third. The third in the specimen, although it too resembles to a slight extent the normal third, resembles to a much greater extent the normal fourth, to which rib in the normal series we believe it corresponds. If this is the right interpretation of these ribs, the rib that should correspond to the normal third is wanting on this side. The reason for this we hope to show

further on. From the upper and inner part of the mass of bone formed by the fusion of these ribs, there projects a tongue-like process continuous with the second rib, and articulating with the sides of the bodies of the first and second vertebræ. It is evidently the head of the second rib. From the lower and posterior part of the fused rib-mass there projects a somewhat similar process, which is continous with the third rib in the specimen, which, as just stated, we regard as the fourth in the normal series. It articulates with the bodies of the second and fourth vertebræ (the third being wanting), and by a facet on its inferior border with the next rib in the specimen. It evidently represents the head of the fourth rib. Near the inner end of the fused rib-mass, and midway between its upper and lower border, is a small foramen, through which the intercostal nerve passed. From this foramen a slight groove extends over the rib-mass to the angle of junction of the two ribs, and clearly indicates the line of their fusion. The free portions of these ribs are flattened laterally in their anterior halves, and their upper and lower borders present a sharp edge, of which the upper is the sharper. The intercostal spaces are very wide, the first space measuring vertically, from costal cartilage to costal cartilage, where they join the ribs, one inch and one eighth; the second space one inch and a half. Posteriorly they are not wider apart than natural. It is difficult to assign the fourth rib in the specimen to its right place, as the vertebra with which it articulates is also doubtful. We are inclined to regard it as the seventh. Unlike the rib immediately above, it is exceedingly slender in its middle. Measured at the junction of its costal cartilage, the third intercostal space is one inch and a half in a vertical direction.

The fifth in the specimen is a false rib. Its costal cartilage articulates with the costal cartilage of the rib above.

The next two ribs are floating; they bear respectively the characters of the eleventh and twelfth, and each articulates by a single facet with what we regard as the eleventh and twelfth vertebræ.

The ribs on the left side.—The first rib in the specimen on the left side we have already stated we believe to be a cervical rib, as the right side of the vertebra with which it articulates bears clearly the stamp of the seventh cervical. It extends for an inch outwards and slightly forwards, and becomes fused, just external to the posterior tubercle of the transverse process of the seventh cervical vertebra, with the rib below. A foramen exists between its neck and the posterior part of the transverse process. The next rib in the specimen, which we regard as the first thoracic, articulates with the upper part of the left side of the body of what we regard as the first dorsal vertebra, and markedly resembles the head of the first rib on the opposite side. After a course of three quarters of an inch it becomes fused with the cervical rib above and with the next rib below, but becomes separated again three quarters of an inch more anteriorly, and is continued to the sternum, with which it articulates by a short costal cartilage. It articulates by its tubercle with the transverse process of the first dorsal vertebra.

The next rib in the specimen, which we regard as the second, articulates with the first and second vertebræ; and after a course of an inch upwards, outwards, and backwards, becomes fused with the rib above, but afterwards separates and continues forwards singly to the sternum. It is anchylosed by its tubercle with the transverse process of the second dorsal vertebra, and also with the first.

The next two ribs in the specimen are fused posteriorly from their angles to the bodies of the vertebræ in a single mass, which mass, half an inch from the body, is perforated by a foramen leading into the spinal canal.

Through this foramen the intercostal nerve emerged. Internally the fused rib-mass presents a slight notch; the portion above the notch evidently represents the head of the third rib. On its upper surface are two facets; the upper one articulating with the facet on the lower part of the left side of the body of the second, and the lower one with a facet on the upper part of the left side of the wedge-like portion of bone, representing the left half of the

third dorsal vertebra. The portion of bone below the notch before referred to clearly represents the head of the fourth rib. It likewise presents two facets on its internal surface, the upper of which articulates with the facet on the lower part of the third vertebra, and the lower with the upper part of the left side of the body of the fourth vertebra. The fused rib-mass also articulates by two processes representing the tubercles with the fused transverse processes of the third and fourth vertebræ. Beyond the angles these ribs are continued forwards separately, and articulate through the intermediation of the two fibro-cartilages with the third bone of the sternum. They are markedly flattened laterally in their anterior thirds, and present a sharp edge above. The next rib (the fifth in the specimen) articulates above with the facet on the lower part of the left side of the body of the fourth, and with what we regard as the seventh vertebra below, By its tubercle it articulates with the transverse process of the seventh vertebra.

The next rib in the specimen is a false one. It articulates in the usual manner with the vertebra above and the vertebra below, *i.e.* with the seventh above, and with what we regard as the ninth below, from its having a demi-facet above on each side of the body. It articulates in front with the costal cartilage of the seventh rib. The next rib articulates with what we regard as the eleventh dorsal vertebra by a single articular facet above. It has more the characters of the ninth or tenth rib than of the eleventh. It articulates in front by a costal cartilage with the costal cartilages of the ribs immediately above. It articulates with the transverse process of the vertebra, as well as with its body. The last rib in the specimen bears the characters of the twelfth, and articulates with what appears to be the twelfth dorsal vertebra. It does not articulate with the transverse process of the vertebra. It resembles in general characters the last rib on the opposite side. It is free in front, that is to say, it is a floating rib.

Description of the sternum.—The sternum is placed somewhat obliquely, the sternal notch looking upwards and to

the right. It measures from the sternal notch to the ensiform cartilage five and a half inches. Like the normal sternum, it apparently consists of three parts (not including the ensiform cartilage), an upper, middle, and lower part. On its right side it articulates with four ribs, on its left with five. The second, third, and fourth ribs articulate with the sternum on the left side at a slightly higher level than on the right. The manubrium appears considerably elongated from above downwards. It measures on the left side, from its upper border immediately to the left of the sternal notch vertically downwards, two inches and an eighth; and on the left side, from the same point of bone downwards, two inches and a quarter. The manubrium is intimately fused with the gladiolus, the angulus Ludovici being scarcely, if at all, marked. The ensiform cartilage is bifid.

Description of the scapula.—The scapula is striking in the resemblance it bears to a scapula at birth. It would seem as if it had retained many of the characters which it possessed at that early period. Thus, it is remarkably deficient in that vertical length so characteristic of the human adult scapula, whilst its spine is imperfectly developed behind, and its supra-spinous fossa extends but a short distance above the spine. The infra-spinous fossa has almost the shape of an equilateral triangle instead of a scalene. The axillary border, which is shorter than natural, slopes nearly horizontally backwards, instead of downwards and slightly backwards. Its superior border is remarkably straight, and slopes upwards, backwards, and inwards. The posterior border first slopes nearly horizontally backwards as far as the spine, then almost vertically downwards, and then downwards, forwards, and outwards, so that it appears as if roughly curved, with the convexity backwards and inwards. To the middle third (the vertical part) is anchylosed a triangular portion of bone, which connects it, bridge-like, with the vertebræ. The spine is slender; the acromion, coracoid process, and glenoid cavity are small, but in other respects fairly normal.

Description of the bridge-like portion of bone.—The bridge-

like portion of bone (Plate V, figs. 1 and 2) connecting the scapula to the spine is of an irregularly triangular shape; its base, which looks outwards, being anchylosed to the posterior border of the scapula, its apex, which looks inwards, to the spinous process of the sixth cervical vertebra. Its inferior border slopes from a tubercle, which remarkably resembles the tip of the left half of the spinous process of the sixth cervical vertebra, downwards, outwards, and a little backwards, and is prolonged in a gentle curve into the lower third of the posterior border of the scapula. Its superior border appears continuous externally with both the superior border of the scapula and with the lower border of the spine of the scapula and runs horizontally and with a gentle curve forwards and inwards towards the spine. The inner third of the superior border is continued upwards and forwards as a tongue-like portion of bone into the lamina and spine of the sixth cervical vertebra. Its base is fused posteriorly with the scapula, except in its upper third, where it is only connected to it by soft tissues, and at a spot in its lower third, where a foramen is left, sufficiently large to admit a pea. Anteriorly a sulcus, filled in the recent state by cartilage, exists between the bone and the scapula. Its anterior surface is concave from side to side and slightly convex from above downwards. Near its lower border and internal angle is a well-marked cup-shaped depression, which is prolonged into a slight groove, running outwards and downwards towards the foramen already described at the junction of the posterior border with the scapula, with which another slightly shelving groove is prolonged upwards and inwards, the two grooves nearly meeting in the centre of the process.

The clavicle.—The photograph of the specimen (see Plates V, fig. 1, and VI) will convey a better idea of the deformity of the clavicle than any verbal description. We may briefly remark, however, that, in place of the two normal curves, the sternal with its convexity forwards, and the acromial with its convexity backwards, it presents a single curve with its convexity backwards. The sternal half appears flattened

out from above downwards, whilst the anterior border is sharp and has a remarkable crenated appearance. The abnormal direction of the bone has already been pointed out. The acromial end is one and a half inch in front of, and one inch higher than, the level of its sternal end.

Description of the muscles.—The pectoralis major on the left side was very thin. It arose in the normal manner from the sternum and clavicle, and from the cartilages of the five true ribs. Its insertion was normal. On the right side it arose from the sternum, clavicle, and the cartilages of the four true ribs. Its insertion was not removed at the post-mortem examination.

The pectoralis minor on the left side arose from the second, third, and fourth ribs; it was well developed and raised the ribs freely. Its insertion into the coracoid process was normal. On the right it arose from the second and third ribs only. Its insertion was not removed.

The deltoid on the left side had a normal origin from the clavicle, acromion, and spine of the scapula; it also arose by an aponeurosis, one inch and a half in length, from that part of the posterior border of the scapula which corresponds with the outer margin of the triangular bridge of bone already described.

The latissimi dorsi were not entirely removed. The left muscle arose from the spines of the three lumbar vertebræ present in the specimen; from the spines of the four lower dorsal, which we regard as the seventh, ninth, eleventh, and twelfth, in the normal series, and from the three lower ribs. It was particularly well developed at its insertion, which was normal. The right muscle had a similar origin. Its insertion was not removed.

The teres major, teres minor, infra spinatus, and subscapularis were well developed, and had normal attachments. The supra spinatus was small; its attachments were normal. These muscles were not removed on the right side. The serratus magnus on the left side arose from the five upper ribs in the specimen; and was inserted, as far as we could determine, into the posterior border of the scapula as usual.

Its upper and lower portions were very oblique and well developed. Its middle portion was exceedingly thin. On the right side it arose from the four upper ribs in the specimen. Its insertions on that side were not removed.

Only the lower portions of the trapezii muscles were obtained with the specimen. They arose, as far as we could make out from all the dorsal spines, which, at the time of their removal, appeared to be five, but which, as we have already described, are made up of eight, variously fused. The left muscle was inserted by thin scattered fibres into the clavicle, acromion and spine of the scapula. The insertion of the right muscle was not removed.

The levator anguli scapulæ on the left side had a normal insertion into the posterior border of the scapula from the superior angle to the base of the spine. It was well developed. Its origin was not obtained. The right muscle was not removed.

The rhomboideus major on the left side was very thin. Its upper part was injured in obtaining the specimen. It appeared to arise from the three upper dorsal spines, which after maceration were found to be four, variously fused. It was chiefly aponeurotic at its insertion. It partly covered the triangular bridge of bone connecting the scapula with the spine. Its exact insertion into the scapula could not be defined. The right muscle had a similar origin.

The rhomboideus minor was injured in removing the specimen. Its exact attachments could not be determined.

The omohyoid had a large posterior belly. Its attachments to the scapula was normal.

The scalene muscles were well developed on both sides. They had normal attachments into the first and second ribs.

The serratus posticus superior on the left side was so irregular that its accurate insertions could not be made out. On the right side the part which was removed with the specimen was attached to the spines of the seventh cervical and first dorsal vertebræ, and was inserted into the second and third ribs.

The serratus posticus inferior on the left side was inserted into the three lower ribs. On the right side it was inserted in a similar manner.

The deep vertebral muscles were very irregular in their origins and insertions. We have not thought it necessary to give their accurate attachments, as they did not, in our opinion, throw any light on the nature of the deformity. We may, however, remark that on the left side both the longissimus dorsi and the accessorius were attached by superficial tendinous slips to the lower border of the irregularly triangular bridge of bone. Their deeper parts, as well as the cervicalis ascendens, transversalis colli, and semispinales and spinales, passed under the bridge of bone to their insertion in the neck.

The intercostal muscles were remarkably thin. They had normal attachments and did not present any tendinous intersections.

Description of the vessels and nerves—The subclavian artery passed over the first rib between the scalenus anticus and medius in the normal manner on both sides.

The subclavian veins were also normal.

The brachial plexuses, as much as was left of them, were normal.

There were eight dorsal nerves on the left side and seven on the right; the third on the left and second on the right emerged from the foramen in the fused rib masses made up of the third and fourth ribs on the left side, and of the second and third on the right side.

Remarks.—Having given a somewhat detailed description of the specimen, we will now offer some remarks on what appears to be the nature of these several deformities. That they were the result of some congenital defect in the development of the embryo, and not of disease, we think so apparent that we will not stay to discuss the point. We may, however, again mention that the deformity was said to have existed from birth, and that the mother herself was the subject of lateral curvature of the spine.

We will first discuss the abnormalities of the dorsal

vertebræ and ribs, and first, with regard to the absence of the right half of the third dorsal vertebra.

Specimens in which a lateral half of a vertebra is absent are described by Drs. Rokitansky, Otto, Goodhart, and others; and there are similar specimens in the Museums of the Royal College of Surgeons, and of St. Bartholomew's and Guy's Hospitals. The malformations of the vertebræ in the specimens in St. Bartholomew's Museum are not referred to in the description of these specimens given in the Catalogue; and as far as we know have never been described elsewhere. As they seem to throw some light on the deformity in our specimen, we will give a brief description of them.

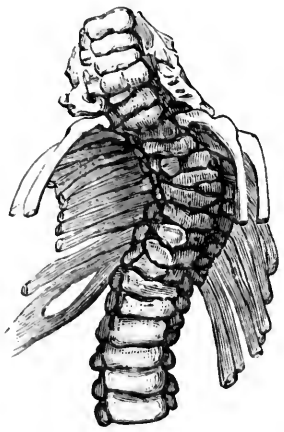
No. 1. (Series A, 133), described in the Catalogue as "dorsal and lumbar portions of a fœtal spine. The spinal canal is wide open behind; the laminæ of nearly all the vertebræ being disparted and turned outwards. They are not deficient in size; rather they are overgrown and expanded at their distal ends." The right half of the ninth dorsal vertebra is wanting, but no mention of this fact is made in the Catalogue.

No. 2. (Series A, 134), described in the Catalogue as "part of a fœtal skeleton from a case of hydrocephalus with spina bifida." Except those of three dorsal and two cervical vertebræ, all the laminæ are deformed and disparted like those in the preceding specimen. The left half of the body, the left lamina, and left processes of the body of the ninth dorsal vertebra are wanting. The eighth and the tenth vertebræ are approximated to each other on the left side, and the left laminæ of these vertebræ, which are also in contact, appear overgrown as if to make up for the deficiency of the absent lamina. The ninth rib is also absent on the left side; consequently there are twelve ribs on the right side, eleven on the left. The eighth rib on the left side is slightly wider than the eighth on the right (as if it had been reinforced by some of the tissues which should have constituted the lost rib). The fifth lamina on the left side is in contact in the middle line with both the fourth

and fifth on the right side; and the fourth on the left side is in contact with, although on a little lower plane than, the third on the right. The bodies of many of the vertebræ appear bilobed.

No. 3 (Series A, 137), described in the Catalogue as "an anencephalous female fœtus. The arches of the vertebræ are separated and everted in nearly the whole length of the vertebral column." (See Woodcut, fig. 1.) The dorsal

Fig. 1.

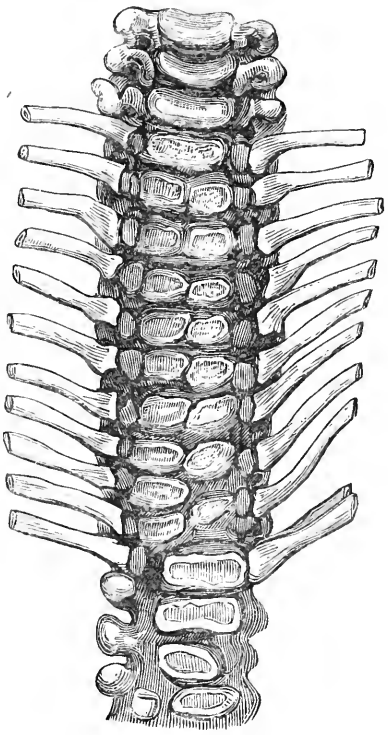


Front View.

spine is very irregularly malformed. The first dorsal vertebra is fairly normal. The second and third are represented by two distinct halves on the left side, but by a single half on the right side; the single half on the right side is opposite the line of division between the second and third half vertebræ on the left side, so that it is difficult to say whether it is the right half of the second or the right half of the third, and consequently, whether the right half of the second or of the third is wanting. The fourth vertebra consists of two distinct halves. The fifth vertebra is fairly normal, but obliquely placed with its left end considerably higher than the right. The sixth is represented by a left half only, the right half being absent. The seventh, eighth, ninth, and tenth, appear bilobed, but otherwise

normal; the eleventh and twelfth are normal. There are twelve ribs on each side. The upper nine on the left side are indescribably fused together from their tubercles to their angles, and the second, third, and fourth on the right side are likewise partially fused posteriorly.

Fig. 2.



Front view.

No. 4 (Series A, 164, see Woodcut fig. 2), described as "the skeleton of a fœtus in which the left lower extremity and left half of the pelvis are deficient." The spinal canal is open in the lumbar and sacral regions, through defect of the vertebral arches; indeed, those of the left side of the sacrum and of the last three lumbar vertebræ appear to have been never formed. No mention is made of the malformation of the dorsal spine.

One dorsal vertebra (probably the ninth), together with the corresponding rib on each side, *i.e.* a whole of one of

the vertebral segments of the body, is wanting. Of the other dorsal vertebræ the upper eight are normal, except that some of them are bilobed. The next vertebræ (the tenth and eleventh, if the ninth is the one absent) are represented on the right side by two halves, but on the left side by a single half, to which the two right halves are united, so that it is difficult to say whether this left half belongs to the tenth or to the eleventh vertebra. It may also be noticed that the eighth vertebra, which is bilobed, has its left half on a lower plane than the right. The twelfth vertebra is normal. Posteriorly there are ten laminæ on the left side, and eleven on the right; the tenth or eleventh, probably the eleventh, being the one that is on this side wanting.

There are eleven ribs on the right side, seven true, two false, and two floating. On the left side there are also eleven, but the eleventh and twelfth are intimately fused, so as at first sight to appear as one.

No. 5 (Series A, 165), described as the "skeleton of a fœtus in which the arches of the lower lumbar vertebræ are deformed, and in the place of the sacrum there is a single distorted bone. The ischia are absent or rudimental, and a single bone representing the heads of both femora articulates with both the imperfect acetabula. The two femora are fused in their upper halves. Their diverging lower halves articulate with the tibiæ fused into a single short conical bone. Behind the tibiæ a series of small bones represent the fibulæ and feet, but cannot be severally recognised." No mention is made of the malformation of the dorsal spine. The cervical vertebræ are natural, with the exception of the left half of the body of the fourth being absent. The body of the first dorsal vertebra is obliquely placed, the left side being much lower than the right. The bodies of the second and third vertebræ appear naturally formed on the right side, but on the left side a single half vertebra, to which they are both anchylosed, takes their place, so that it is impossible to say whether this half vertebra is the left half of the second vertebra or left half of the third; and consequently, whether the left half of the

second or left half of the third is absent. The laminæ corresponding to the second and third on the left side, though somewhat malformed, are present.

The bodies of the fourth and fifth vertebræ appear natural, except that they are bilobed and placed obliquely with their left sides higher than their right.

The body of the sixth is absent, the sixth rib on the right side articulating with a little nodule, which represents that part of the body of the absent vertebra which is external to the neuro-central suture. This nodule is situated between similar nodules belonging to the fifth and seventh vertebræ. On the left side the nodule corresponding to the sixth right half is fused with a similar nodule belonging to the seventh; and the heads of the sixth and seventh ribs on the left side are fused. The laminæ of the sixth vertebra are present on both sides.

The body of the seventh dorsal vertebra is bilobed. It articulates above with the body of the fifth.

The eighth and ninth are fused on the right side, the division between them, however, being distinct. On the left side they are not fused.

The tenth is bilobed, its right side being higher than its left.

The bodies of the eleventh and twelfth dorsal vertebræ are absent on the left side; but the corresponding laminæ are present, though fused into a single mass. The body of the right half of the twelfth is fused to the body of the first lumbar.

There are twelve ribs on the right side, thirteen on the left. The last rib on the left, which is fused with the twelfth, is probably a lumbar rib. The ribs are naturally formed on the right side. On the left side the first and second are fused. The sixth, seventh, eighth, ninth, and tenth, are fused with one another external to their angles, but are separated again further on. The sixth and seventh are fused, moreover, at their heads; the seventh, eighth, and ninth at their tubercles; the ninth and tenth at their heads.

The lumbar vertebræ are so fused one with another that it is next to impossible to determine in what the deformity consists. The laminæ are not united behind.

The malformations of the vertebræ in the specimens in the Hunterian Museum are likewise not described in the Catalogue. A very full account of them by Dr. Goodhart will be found in the 9th vol. of the 'Journal of Anatomy and Physiology.' We have examined these specimens carefully; and although we do not quite agree with the account given of them by Dr. Goodhart, we have to acknowledge the aid we have received from his paper in forming an opinion as to the nature of the malformations.

No. 6 (2879 c a, Museum of College of Surgeons). "The skeleton of an adult male sixty-four years of age. In addition to other deformation there is bony anchylosis along the front of the bodies from the seventh cervical to the fourth dorsal inclusive."

The left half of the first dorsal vertebra is apparently wanting. In consequence of this deficiency the left halves of the bodies of the seventh cervical and second dorsal are in contact. A slender process of bone representing the first rib projects from their line of union. It is about an inch in length, and is anchylosed internally to the two vertebræ and externally to the articular process of the seventh cervical. On the right side the first rib articulates in the normal manner with the right half of the first dorsal vertebra.

The laminæ of the seventh cervical do not meet in the middle line; the right lamina ends singly in the spinous process, while the left is rotated downwards and anchylosed with the right lamina of the first dorsal, thus taking the place of the left lamina of the first dorsal, which is wanting.

On the left side the second and third ribs are fused and anchylosed with the similarly fused transverse processes of the second and third dorsal vertebræ. On the right side the second and third ribs arise in the normal manner from the fused second and third vertebral bodies, and articulate with the articular processes of the same, which are not anchylosed.

The laminæ and spines of the second and third dorsal vertebræ are completely anchylosed, though a ridge still marks their proper separation.

In the ligament running from the spine formed by the fusion of the right lamina of the seventh cervical and left lamina of the first dorsal to the spine of the third is a small bony nodule, which seems to represent the spinous process of the second dorsal vertebra, the laminæ of which are anchylosed with those of the third.

No. 7 (278, College of Surgeons Museum), described in the Catalogue as "the skull and vertebral column with a portion of the ribs and pelvis of a hydrocephalic male fœtus, with distortion of the vertebral column, defective development of the occipital bone, and defective closure of the vertebral canal behind."

In addition to the other malformations the dorsal spine presents the following:

The first dorsal vertebra is normal, the left half of the second dorsal is wanting, the third is normal, but placed obliquely, the right half of the fourth is absent. The remaining ten are normal, except that the upper are bilobed. There are eleven ribs on each side, the left second and right fourth being absent. The lumbar vertebræ are five in number and are normal.

No 8 (1004^{92}, Museum of Guy's Hospital). This specimen is described in the Catalogue as follows:

"A congenital malformation of the spine. This is caused by the fusion together of the bodies of the third, fourth, and fifth dorsal vertebræ and the curvature of the new formed mass. A slight oblique fissure passes across the anterior part, whereby it is seen that the body of the fourth vertebra is wholly destroyed, and the third and fourth perhaps so; the one more on its left side, the other more on its right.

Upon the posterior view three transverse processes are seen to be perfect, those of the lower vertebræ retaining their natural position, and respecting those of the other two vertebræ, on the right side, the upper process is seen entering its proper body, and the middle one attached to a very small

portion of the body of the fourth, which remains; on the left side the two upper transverse processes are fused together and join the remains of the body of the third vertebra. The three spinous processes are correct in number; and the two lower ones come off from the remains of their respective vertebræ in the usual way; but the upper spine is continuous wholly with the right arch of the third vertebra, the left arch of the bone not meeting the fellow at all (the two being developed separately), but is welded into the spinous process and arches of the vertebræ."

Dr. Goodhart, in commenting upon this specimen, says, "The bodies of the third, fourth and fifth vertebra are fused together, but of these the fifth is obviously perfect, and merely altered in shape somewhat by reason of the anchylosis and curvature. The fourth is described as wanting in its body, but from the setting of the transverse processes it is, I think, well represented on either side, whether by original body centres, or merely by a growth forward of that part which helps to make up the body, but comes from the lateral masses, is very difficult to say. However, on the left side it seems that the body of the third is rather wanting; on the right side that of the fourth. This description also tallies with what is observed behind; the main right half of the third vertebra is merely an appendage to the upper border of the fourth, and does not meet its fellow in the median line."

Dr. Goodhart, in the paper above referred to, adopting Professor Humphry's conclusion, "that there is sufficient evidence of the occasional development of the vertebral bodies from two nuclei laterally disposed," seeks to explain the deficiency of half a vertebra on the assumption that in these cases only one such lateral nucleus was developed. As to the occasional occurrence of two nuclei in the body of a vertebra, much difference of opinion has been expressed. For while Professor Humphry thinks that in the complete or typical vertebræ two nuclei should be allowed to this part (the vertebral body), one on each side of the middle line, Professor Flower states that "he has found no

evidence in the study of normal anatomy, from which alone he thinks an opinion ought to be formed, of the occurrence of a double nucleus" (Goodhart); and Professor Humphry himself admits that he has examined many fœtal spines with great care without ever finding two nuclei, and he states moreover that Meckel, in spite of diligent search, could never find more than one.

We have ourselves examined a number of *normal* fœtal spines with a like result.

In the malformed spines already referred to in the specimens of St. Bartholomew's Hospital, and of the College of Surgeons, however, many of the vertebral bodies present a bilobed appearance, as if they had been developed from two laterally placed centres of ossification. We were at first inclined to attribute this bilobed appearance to the bodies having been developed from a bilobed centre, as Müller states that such is the usual form of the centre for the body; and as we were unable for some time to discover any vertebra in which, if two centres had ever existed separately, they were not now united in the middle line. But afterwards by careful probing we found that in a few of the vertebræ in the specimens in St. Bartholomew's Hospital (for example, the third dorsal in No. 2 A, 134) there was no trace of bone stretching across the middle line between the lobes, which clearly indicated that here two nuclei were present.

In that rather rare malformation of the spine, moreover, in which, as the consequence of the non-union of the mesoblastic tissues around the notochord, the bodies of the vertebræ are vertically cleft in the middle line, it is evident that each lateral half must have been developed from two centres of ossification.

The presence of two centres in the specimens under consideration, we believe, can be shown to depend upon a similar, though much less evident, cause.

It would appear, then, that, under normal development, there is no evidence of two centres ever occurring in the vertebral bodies; but that under certain abnormal condi-

tions such may not infrequently be the case. We are not inclined, however, to admit that the suppression of one of these nuclei is usually the cause of the absence of one half of a vertebral body, though we are not prepared to say it may never be so. For we do not see how the suppression of one such centre in a vertebral body could account for the other malformations which so frequently attend upon the absence of half a body, viz. the absence of the corresponding arch, processes, and rib.

As is well known, the arch and processes on each side are developed at or about the same time as the body; the mesoblastic elements on each side of the notochord growing upwards and inwards (the prone position of the embryo here, as in the rest of this paper, being understood) around the medullary canal to form the arch, and inwards and forwards around the notochord to form the body; whilst ossification, which does not begin till some weeks afterwards, starts from three distinct centres, one for each side of the arch and one for the body. The deficiency of the arch on one side, therefore, as well as the deficiency of one side of the body, could only be explained by supposing that the centre for the arch, in addition to one of the lateral centres for the body, was not developed. It could not therefore be said that the deficiency of the lateral centre of the body was the cause of the deficiency of the centre of the arch.

The absence of the corresponding rib, moreover, is not accounted for by this theory, as the cartilaginous rudiments of the ribs, which grow out from the sides of the bodies, are already developed before the centres of ossification for the vertebræ are laid down, and have themselves distinct centres quite independent of those of the vertebral bodies.

Still holding the view that the deformity was dependent upon some defect in the method of ossification, we conceived that it might be explained on the supposition that the centres for one half of the arch and process and the centre for the body had remained undeveloped, whilst the

centre for the arch on the opposite side had extended forwards beyond its normal limit into the side of the body, and so simulated one half of the body. For, as we have already had occasion to remark, the bodies of the vertebræ in the dorsal region are ossified laterally by the centres for each lateral half of the arch extending inwards into the cartilaginous matrix of the body as far as the facet for the rib.

This theory, however, like the former, is open to the objection that it does not explain the simultaneous absence on one side of body, arch, and corresponding rib.

Further, we cannot conceive how any theory of defective ossification can satisfactorily explain these cases. Is it not more probable that these and similar abnormalities are really dependent upon some earlier departure from the normal process of development? Should they not be referred to some fusion, vacuolation, destruction, or non-differentiation of the formative cells or material out of which the parts are developed rather than merely to defects in the methods by which these parts are ossified? Is it not conceivable that they are due to some defect in the development of the mesoblastic elements constituting the vertebral and lateral plates of the embryo at or about the time of the primary segmentation into the protovertebræ or somatomes?

There are many points in these specimens which, we think, favour this view.

First. The condition of the laminæ and spinous processes.

Secondly. Deficiencies of whole vertebral segments.

Thirdly. The absence or defective development of the corresponding rib.

Fourthly. The frequent malformations of other portions of the skeleton.

1st. The condition of the laminæ and spinous processes. These in all the specimens of unilateral suppression of the vertebral bodies we have as yet met with, have exhibited some developmental defect. In the majority of cases the lamina and half of the spine corresponding to the absent half of the vertebra are wanting. This is so in our speci-

men, in 2879 c a and 278, Hunterian Museum, and in Nos. 1, 2, 3, and 4 in the St. Bartholomew's Hospital series. In other cases the laminæ, though present, are in a rudimentary condition, and have not united posteriorly with the corresponding and opposite laminæ in the median line.

In by far the greater number of these specimens, moreover, the laminæ of the vertebræ above and below the one whose body is deficient have also failed to unite. In several specimens this non-union of the laminæ extends throughout the whole spinal column. Now, this non-union of the laminæ is evidently the result of the failure of the coalescence in the middle line of the mesoblastic elements, which normally grow upwards and inwards around the medullary canal; and not of a defect in ossification. For as this coalescence of the mesoblast, as will be remembered, occurs at a very early period (first and second day in the chick) in the development of the embryo, whereas ossification does not commence until a comparatively much later period, any theory founded upon the failure of union of the osseous centres which form in each lateral arch after the mesoblastic elements have normally coalesced above, would only account for the cleft condition of the spine by supposing that subsequently to the union of the mesoblastic tissues and their conversion into cartilage they had again, in some unaccountable manner, become cleft about the period of ossification—an assumption which is surely gratuitous. A rudimentary condition of the laminæ would imply a still further defect in the upward growth of these tissues, and the absence of the laminæ their total suppression. If, then, the non-union, rudimentary condition, or suppression of the laminæ is the result of defective development during the first few days of fœtal life, is it not more probable that the abnormal condition of the body of the vertebra originated at the same early period, that is to say, would not the same changes which led to the absence of the arch and processes also account for the absence of the body? Should not any theory that pretends to explain the absence of one part of the vertebra also explain the absence of the other parts?

2ndly. Another fact in favour of these deformities having occurred at a very early period of development is the deficiency in several of these specimens (for example, in our own and in No. 4) of whole vertebral segments of the body —deficiencies which could hardly be explained on the supposition of abnormal ossification.

3rdly. The absence or defective development of the rib of that side on which the half vertebra is wanting is another point which favours this view. Thus, in No. 2 (A 134, St. Bartholomew's Museum), in 278, Teratological Series, College of Surgeons Museum, and in our specimen, the rib corresponding to the absent half vertebra is wanting. In Nos. 5, 3, and 4 (A 165, A 137, A 164, St. Bartholomew's Museum), and in 2879 C A, Hunterian Museum, the rib corresponding to the absent half vertebra is defective, or defective and fused with the next rib in the series.

4thly. Another fact which supports our view is the coincident malformation of other portions of the body, such malformations, that is to say, which could only have been produced at a very early period of development. Thus in No. 2 (A 134, St. Bartholomew's Museum), and in 278, Hunterian Museum, the deformity is combined with hydrocephalus. In No. 3 (A 137, St. Bartholomew's Museum) the fœtus is anencephalic. In No. 4 (A 164, St. Bartholomew's Museum) the left half of the pelvis and the left lower extremity is non-developed. In No. 5 (A 165, St. Bartholomew's Museum) the sacrum is represented by a single distorted bone, the ischia are absent, the femora are fused in their upper halves, and the tibiæ represented by a single short conical bone.

Believing, then, these abnormalities to be the result of some defect in the development of the mesoblastic elements constituting the vertebral plates, we venture to suggest the following as an explanation of the deformity.

To render this explanation more intelligible we must briefly refer to the development of the vertebræ and ribs.

During the first few days of fœtal life the plate of meso-

blast on each side of the notochord splits, as is well known, horizontally into two layers, one of which attaches itself to the epiblast to form the somato-pleure whilst the other attaches itself to the hypoblast to form the splanchno-pleure. This cleavage, however, does not extend quite up to the walls of the medullary canal; "hence there is left on either side of the canal, between it and the line along which the cleavage begins, a tract or plate of uncleft mesoblast, which receives the name of vertebral plate, the more external mesoblast being called the lateral plate" (Foster and Balfour).

Very soon, however, the vertebral plate becomes segmented by a number of transverse clefts, stretching inwards from the lateral plate to the notochord, and a little later it becomes cut off from the lateral plate by a vertical cleft parallel to the notochord, so that each vertebral plate is cut up into a number of cubical masses, each cubical mass constituting with the corresponding mass on the opposite side of the notochord a pair of protovertebræ, or as they are now more commonly called somatomes.

Such a protovertebra, after giving off on its outer part the muscle plate, grows upwards around the medullary canal and becomes fused in the middle line with the opposite protovertebra which grows upwards in a similar manner. At the same time the lower portion of the protovertebra extends inwards above and below the notochord, and also becomes fused with a similar process extending inwards from the protovertebra on the other side, so that the notochord and medullary canal are surrounded by an investment of mesoblast. The investment of mesoblast is an extension chiefly from the posterior part of the protovertebra and afterwards constitutes the arch of the permanent vertebra. The clefts dividing the protovertebræ from each other now disappear, and the protovertebræ become fused. Soon again, however, fresh segmentation occurs, and the resulting segments constitute the permanent vertebræ, the new line of segmentation occurring through the middle of what was originally each protovertebra. So that it comes to pass that each

permanent vertebra is formed out of parts of two consecutive protovertebræ, for example, the third vertebra is formed of the posterior part of the third protovertebra and the anterior part of the fourth, whilst the arch apparently no longer springs from the part of the protovertebra from which it is an outgrowth, but from the front part of the permanent vertebra to which it henceforth belongs. While the chief mass of the protovertebra is converted into muscle plate and arch and body of the vertebra, a small portion of the exterior grows downwards in the dorsal region as the rudiment of the rib. These rudiments soon, however, become separate from the bodies of the vertebræ—with whose arches they henceforth alternate.

At first the muscle-plates end opposite the point where the mesoblast becomes split into somato-pleure and splanchno-pleure. Soon, however, they extend to a certain distance into the side walls of the body beyond the point of the division into somato-pleure and splanchno-pleure. In the first instance, as is clear from their origin, the muscle plates correspond in number with the protovertebræ, a condition which is permanent in the lower vertebrata.

Thus much of the development of the embryo we have thought it necessary to refer to in order to make clear the explanation we are about to offer of these deformities.

We believe that at or about the time of this primary segmentation of the mesoblast into protovertebræ or somatomes, the tissues which should constitute one of the lateral sections of the body, become completely, or in part, destroyed or non-differentiated, whilst the tissues constituting the segments above and below come into contact with, and become fused to, one another, so that when the secondary segmentation occurs one lateral half of the permanent vertebra, together with its arches, processes, and corresponding rib and muscles, will be wanting.

In our specimen, to briefly recapitulate, the bodies of the first and second dorsal vertebræ are normal, and articulate in a fairly normal manner with the first and second ribs. The right half of the third dorsal vertebra is absent.

The left half articulates with the fused third and fourth ribs, that is to say, with the third above and with the fourth below. On the right side, the third rib in the specimen, which we are inclined to regard as the fourth in the normal series, believing the third to be wanting, articulates with the bodies of the second and fourth dorsal vertebræ, and with the transverse process of the fourth. It is fused in its posterior part with the second rib, and has the characters partly of the third and partly of the fourth rib in the normal series, appearing somewhat as if the two had been fused together. The next vertebra (the fourth) articulates on the left side above with the fourth rib and below with the fifth, which we regard as the seventh in the normal series. On the right it articulates above with the third rib, which we consider the fourth in the normal series, and below with the fourth, which we look upon as the seventh. The next vertebra we regard as the seventh, believing the fifth and sixth to be wanting. It articulates on each side, above with what we have called the seventh rib and below with what we have called the ninth.

Here, then, we suppose that the mesoblastic tissues which should have constituted the third somatome or protovertebra on the right side were affected (*i.e.* destroyed, fused, or non-differentiated), and that consequently the tissues constituting the second and fourth somatomes on that side came into contact, whilst the tissues constituting the third right somatome would be slightly compressed in the form of a wedge, with its apex to the right, the embryo being slightly bent to the side where the destruction of the formative material had taken place. Hence, when the primary segmentation occurred, three protovertebræ on the left side, the second, third, and fourth, would correspond to two on the left, the second and fourth; and therefore, when the second and fourth on the right side, which were in contact with one another, grew up around the medullary canal, and downwards and inwards about the notochord, they would come into contact, not only with the similar processes from the second and fourth on the left side, but also

with that of the third on the left. Hence, as we should expect, the third arch would be deficient on the right side, and the second and fourth arches be more or less approximated, taking up the space left vacant by the absence of the third. But on the left side the third arch would have formed as usual, and would necessarily be fused in the middle line with the second and fourth arches on the right side, which together occupy the place of the absent third, such as is actually the case in the present specimen.

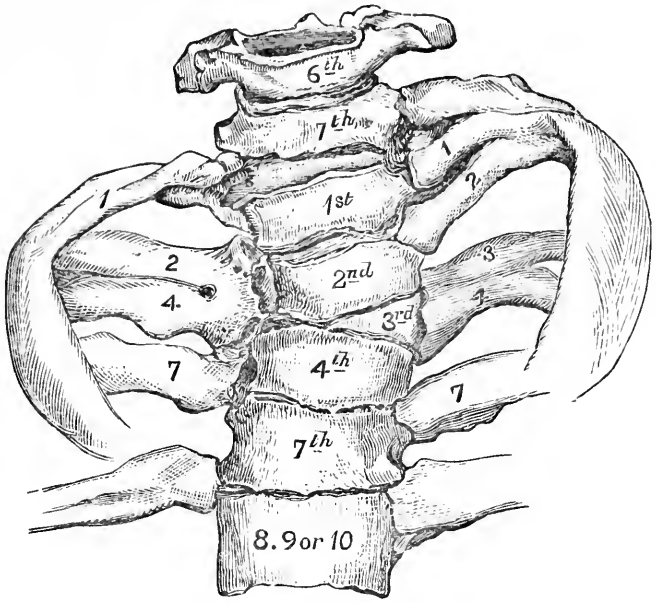
On secondary segmentation occurring, the first dorsal vertebra would be formed from the posterior half of the last cervical protovertebra and the anterior half of the first dorsal protovertebra as usual, whilst the second dorsal vertebra would be formed from the posterior half of the second dorsal protovertebra and the anterior half of the third on the left side, but from the posterior half of the second and the anterior half of the fourth on the right side (see Woodcuts, figs. 3 and 4). The arch of the second, which is formed from the posterior part of the second, would spring from the usual position, *i. e.* from the front of the permanent second vertebra, as it actually appears to do in this case, and would be normal except by being cleft and its right half bent backwards (the embryo being prone) to fill the space left by the deficiency of the third arch. The second rib, which is formed by the growing out of the second protovertebra, should be normal on each side. The third dorsal vertebra, which is usually formed from the posterior half of the third pair and anterior half of the fourth pair, would be formed on the left side in the normal way. But since on the right side the protovertebra was absent, and the anterior part of the fourth, as we have seen, had already united with the hinder part of the second permanent vertebra, it is clear that there would be no material on this side to form the right half of the third vertebra. This vertebra would therefore only exist on the left side. On the left side the third rib would be formed, as usual, by the growing out of the third protovertebra, while on the right side it would be deficient, as it appears to be in the specimen, the fourth being

approximated to the second and taking its place. The fourth vertebra on both sides will be formed from the posterior part of the fourth protovertebra and the anterior part of the next vertebra in the series, as is natural; the anterior part of the fourth, as we have seen on the left side, having formed the posterior part of the third, and on the right the posterior part of the second. It would hence follow that the fourth vertebra would be in contact on the left side with the third, and on the right side with the second; and that the fourth rib, which is formed from the fourth protovertebra on the left side, would articulate with the third and fourth vertebræ, while on the right side it would articulate with the second and fourth.

This theory commends itself to us in that it explains not only the absence of half the body of the third dorsal vertebra, but also the absence of the corresponding arch and rib. Moreover, if it be correct, we should not be surprised to find the second and fourth ribs on the right side (second and third in specimen) fused as they actually are; for it can readily be conceived that the rib processes growing out from the fused protovertebral elements would also be more or less fused. We should also not be surprised to find each of these ribs partaking (as they actually do) somewhat of the character of the lost rib, for it is quite possible that the tissues, which should have formed the third protovertebra, would not have been entirely destroyed, but would in part have remained and become blended with the protovertebræ above and below; so that the tissue elements which afterwards grew out from these protovertebræ to constitute the second and fourth ribs would be reinforced, as it were, by the elements which remained of the partly destroyed protovertebra, and so both the second and fourth ribs would assume, to some extent, the characters of the lost rib.

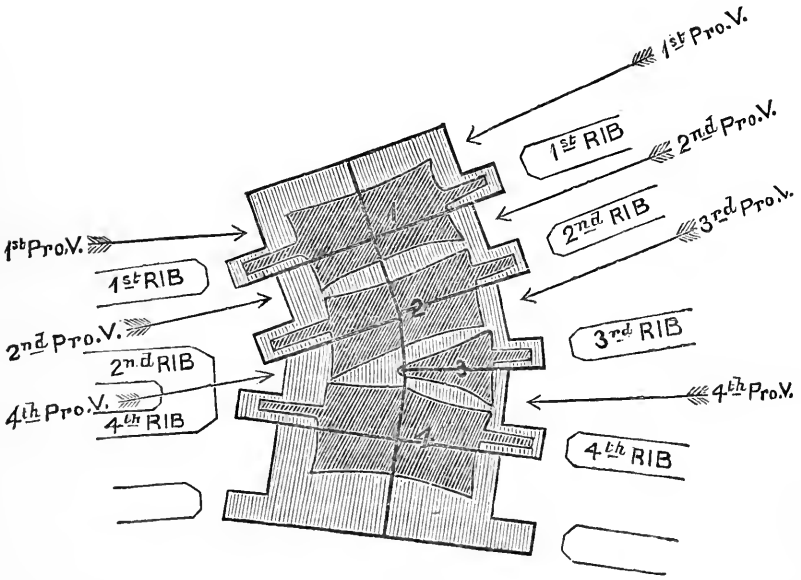
Granting that the fusion of the second and fourth ribs took place in the way we have suggested, it can easily be imagined how, as development advanced, and the fused ribs grew downwards into the lateral plates of the embryo, that the normal formative force would ultimately reassert itself,

FIG. 3.



PATHOLOGICAL FACT.

FIG. 4.



ANATOMICAL EXPLANATION.

The light parts represent the protovertebræ; the dark shaded parts, the permanent vertebræ.

and that the ribs would therefore become separated and continue their growth in a more or less natural direction; although each would still be affected by the reinforcement of the formative material which it had received at starting from the partly destroyed protovertebra, and would permanently retain some of the characters of the lost rib. The fusion of the third and fourth ribs on the left side, on this supposition, is also not difficult to account for, as it would naturally be expected that, although the tissues constituting the protovertebra on one side were chiefly affected, those constituting the corresponding parts on the other side would not entirely escape, but would become to some extent blended, &c., with the tissues above and below, so that the parts on the left side would not undergo complete differentiation, but remain more or less fused or ill developed, as is exactly the case.

The fusion of the cervical with the first and second ribs just external to the tubercle, we think, is due to an entirely different cause. Here it will be seen that the three ribs are not fused where they articulate with the vertebræ, but only just external to the tubercles. This fusion we believe to be the result of the second and third ribs taking an abnormal direction in consequence of the curvature of the spine which was produced by the loss of the left half of the third dorsal vertebra, so that in their growth they would be thrown together at or near the tubercle and become fused.

Below the fourth vertebra the complete destruction of the next two pair of protovertebræ has apparently occurred, as the fifth and sixth vertebræ, with the fifth and sixth ribs, seem to have totally disappeared.

At first sight it may seem improbable that the fourth vertebra should be formed and the fifth and sixth be wanting. We can offer no solution of this difficulty. Still, the specimen presents the fact very clearly. The fourth vertebra, therefore, will have been formed out of the posterior part of the fourth, and the anterior part of the seventh, dorsal pair of protovertebræ. This would account for it articulating above with the fourth rib and below with the

seventh. The next vertebra might be either the eighth, ninth, or tenth, it is difficult to say which, but from its general characters we are in favour of its being the ninth. If this be so, the eighth and tenth are then wanting. The spine of the ninth appears to have been cleft at its base on the right side. The next two vertebræ are apparently the eleventh and twelfth.

The explanation we have offered of the malformation of the vertebræ in our specimen holds good for the similar malformation of these structures in the other specimens to which we have referred. The slight variations observed in many of the latter as regards the condition of the laminæ and ribs, we believe are differences of degree, not of kind—*i.e.* that they depend upon the destruction of the tissues constituting the protovertebræ having occurred to a greater degree in some instances than in others. In No. 4 there appears to have been complete destruction of the corresponding protovertebræ on both sides, *i.e.* of the whole of one of the primary vertebral segments of the body, since the ninth dorsal vertebra, together with the corresponding rib, on both sides are wanting. In Nos. 2 and 278, Hunterian Museum, an entire protovertebral segment on one side appears to be affected, as in No. 2, the left half of the ninth dorsal vertebra (body, lamina, and processes) and the ninth rib are suppressed, and in 278, the right half of the second dorsal vertebra and second rib on the right side and the left half of the fourth dorsal vertebra and fourth rib on the left side are absent. The former of these two specimens is perhaps the most important of the series, as here it is quite evident that the entire half of one of the permanent vertebral segments is suppressed; whereas in the other specimens, including our own, and in 278, Hunterian Museum, such an entire suppression, although we are ourselves convinced of it, cannot be so clearly demonstrated.

In Nos. 3, 4 and 5, St. Bartholomew's series, and in 2879 C a, Hunterian Museum, the destruction of the protovertebræ appears to have been limited. Thus, in No. 4 the left half of the body of the eleventh dorsal vertebra, together with the

left lamina and processes, are wanting, but the corresponding rib on the left side, although fused with the rib below and otherwise defectively developed, is present. Here, then, we may suppose that the lower portion of the protovertebra which grows downwards to form the rib escaped the destructive process which the rest of the eleventh protovertebra underwent, and became partly fused or blended with the like process growing out from the protovertebra below.

In 2879 C a, College of Surgeons, the destructive process, whilst affecting the body and lamina, as in the former specimen, would have appeared to have involved more of the process which forms the rib, as this structure is here merely represented by a very slender process of bone, which has become anchylosed to the transverse process of the vertebra above. In this specimen, moreover, the destructive process was evidently not confined to half of one vertebral segment, but involved many more, as is seen from the fusion of the vertebral bodies from the seventh cervical to the fifth dorsal, and the attending partial fusion of the upper ribs. In No. 3 the destructive process appears to be chiefly limited to the half of a body, as the lamina and the processes and rib on that side were present; the rib, however, presents a somewhat rudimentary condition, and is fused to the rib above.

In No. 4, in which the left half of the eleventh dorsal vertebra is wanting, the left half of the vertebra above, *i.e.* the tenth, is on a lower level than the right half, appearing as if it had fallen down to supply the place left vacant by the absence of the half body, hence the two half vertebræ on the right side correspond to one half only on the left, so that it is, at first sight, difficult to say to which vertebra this half belongs, and conversely, whether it is the left half of the tenth, or left half of the eleventh, which is absent. On closer examination, however, it is clearly seen to be the left half of the tenth. The left half of the vertebra next above is also on a slightly lower plane than the right half. This appearance is readily explainable on our theory; indeed, we think it lends support to it. Now on the

destruction of the tissues constituting the eleventh proto-vertebra on the left side, and the approximation of the tissues of the protovertebræ above and below, it would come to pass that the tenth, eleventh, and twelfth protovertebræ would correspond to the tenth and twelfth on the opposite side, and hence that the anterior part of the twelfth left protovertebra would necessarily be on a lower level than the anterior part of the eleventh on the right side. Now, while the anterior part of the permanent tenth vertebra will be formed from the posterior part of the tenth pair of protovertebræ, its posterior part will be formed on the right side from the anterior part of the eleventh proto-vertebra, and on the left side from the anterior part of the twelfth, so that it follows that its left half would be on a lower level than its right half. The lower level of the left half of the next vertebra above the tenth is also what we should expect, as it is conceivable that by the approximation of the tenth and twelfth the next protovertebra would also be on a slightly lower level than its opposite half. In the vertebræ below the right halves, for the same cause, are on a slightly higher level than the left halves. In our specimen there is an appearance as if a similar state of the parts might have existed at an early stage of development.

Remarks on the triangular portion of bone.—Although we have discussed the probable explanation of the malformation of the dorsal vertebræ first, the triangular portion of bone stretching from the scapula to the spine is perhaps the chief point of interest in this very remarkable specimen. We have already given a detailed description of the bone; we will now endeavour to offer what appears to us some explanations of its origin and significance. We have looked through the Hunterian and several other museums, but have failed to discover any like deformity; neither have we come across any specimen resembling it in published records. We know of no exactly homologous condition of the shoulder-girdle in the animal kingdom, although it bears a resemblance to the supra-scapular bone of some of the lower vertebrates; for example, the frog and

the skate. Development does not materially help us. "The pectoral arch," as remarked by Huxley, "may be connected with the skull or with the vertebral column by muscles, ligaments, or dermal ossification, though primitively it is perfectly free from and independent of both," this arch, as is well known, being developed from a "rod of cartilage" unconnected with the ribs or the vertebræ.

There are several theories, more or less probable, which we beg to submit to the consideration of the Society. These, for convenience, may be classed under three heads, according as they are based upon one or other of the following hypotheses:

1st. That the process of bone was formed in connection with the vertebræ, and afterwards became anchylosed to the scapula.

2nd. That it was originally a scapular element, and secondarily became fused to the spine.

3rd. That it was formed independently both of the spine and of the scapula.

In favour of the first theory is the intimate fusion which exists between the internal end of the bone and the left half of the spinous process and left lamina of the sixth cervical vertebra. We first thought that it might be an outgrowth of the spinous process—an over-development of the epiphysis that normally exists at the apex of this process, and that it might be homologous to the exceedingly high and compressed spinous processes of the ungulata, processes which, as is well known, are developed from a very early autogenous ossification, and soon become united to the upper part of the arch. Such a theory, however, offers no explanation why the process should only have united to the left side of the arch, and why it should have become bent outward and fused to the scapula. The excessive growth of the epiphysis in the ungulata, moreover, only occurs in the anterior dorsal region, not in the cervical. Or, again, the bone is perhaps homologous to one of the laterally projecting processes (called hyper-apophyses by Mr.

Mivart) developed near the extremity of the spinous processes of the third, fourth, fifth, and sixth cervical vertebræ of the Mycetes. In favour of this is the apparent connection of the bone to the side of the spinous process near its apex, the small tubercle marked A in the specimen evidently being the apex of the left half of the spinous process. We are not aware, however, that these hyper-apophyses have ever been observed to grow out to anything like the dimensions of the process of bone in the present specimen, and this theory, like the former, offers no explanation of the anchylosis of the bone and the scapula.

2ndly. On the hypothesis that the bone is a scapular element, it may be regarded as probably connected with an overgrowth of the epiphysis which normally exists along the posterior margin of the scapula, and which, from some unexplained cause, has become anchylosed to the spine of the sixth cervical vertebra. This epiphysis, as is well known, extends normally the whole length of the posterior margin of the scapula. But as the bridge-like portion of bone is only connected to about the middle third of this margin, the overgrowth of the epiphysis, if the portion of bone is due to such, could only have been partial. The partly cartilaginous suture already described, which connects the portion of bone with the scapula, would, according to this view, represent the line of union of the epiphysis with the scapula. To this it has been objected that an epiphysial line apparently exists anterior to the suture before mentioned, that therefore the suture is not the epiphysial line, and consequently the triangular portion of bone is not an epiphysis. There can be no doubt that the epiphysis is united in the lower third, as in this situation the line of union is well indicated by a distinct ridge. On tracing the line upwards, however, it appears to us to be continued into the suture between the bridge-like portion and the scapula, not to pass anteriorly to it, *i.e.* the bridge-like portion appears continuous with the epiphysis, which has united with the scapula along the lower part of its posterior border.

That the upper part of the epiphysis of the posterior

border does sometimes take on an overgrowth, extending backwards and inwards beyond the rest, receives support from some specimens in the teratological series contained in the College of Surgeons Museum.

In 322 and 323 such an outgrowth has apparently occurred. In the Museum of St. Bartholomew's Hospital there is a specimen of a fœtus in which both scapulæ present a backwardly projecting process from the lower third of their posterior border.

If, then, an overgrowth of the epiphysis, the bone may, perhaps, be homologous with the supra-scapula of some of the lower vertebrata. It certainly bears a superficial resemblance to that of the frog and toad, in which creatures, however, it is not united to the spine. As far as we know, the only vertebrata in which the supra-scapula is so united are the skate tribe. "In the thornback skate" to quote Professor Parker, "the cervical spine is closely wedged between the supra-scapulæ, just as in the birds the sacral vertebral spine is wedged between the iliac crests. The supra-scapula of the thornback skate is separated from the scapula by a complete transverse cleft, which results in a perfect joint cavity. Each supra-scapula is irregularly four-sided, its upper edge straight, its anterior concave, its posterior margin sigmoid, whilst its scapular margin has an oblique convex outline, the scapula mounting upwards behind it. The supra-scapula is thick below and thin above, and by its thin margin it is attached by strong fibrous tissue (there is no joint cavity here) to the jutting edge of the vertebral spine. It is gently concave above and gently convex below."

If this bone is really a supra-scapula we may regard its occurrence as an instance of reversion to an earlier type, *i.e.* of a reversion of the human shoulder-girdle to the very rudimentary girdle of the placoid fishes. We merely suggest the idea of reversion as not altogether an improbable explanation of the deformity. We do not attempt to discuss the argument for and against it, as to do so we should have to enter upon the much wider question of

reversions in general, a discussion which would here be obviously out of place.

3rdly. On the hypothesis that the bone was developed independently of both the spine and the scapula, we may ascribe its formation to the ossification of one of the intermuscular planes which stretch between these parts. The suggestion of its being an ossified "rhomboideus minor" can be disposed of at once, as that muscle was found in a nearly normal condition on the dissection of the specimen.

It is further possible that the bone is composed of some of the lost ribs which had been displaced and fused with the spine and scapula in their abnormal situation.

We have already drawn attention to the resemblance which the process of bone bears to a scapula. It may possibly be a rudimentary scapula, and its presence as such may be accounted for by supposing that the upper part of the embryo from which our specimen was developed was originally cleft into a right and a left half, and that the left half, with the exception of the left scapula, a few of the ribs on the left side, and the right scapula (represented by the process of bone), were suppressed or fused to the right half.

This theory, fanciful as it may seem at first sight, is not perhaps without some claim to our consideration. Nearly all pathological museums contain specimens of double monsters, all grades of which are known to exist; but it is that form in which the upper half alone is cleft to which we have to refer as bearing upon the present specimen. The accompanying drawings,[1] taken from Förster, represent some of these malformations. In A the doubling is nearly complete, the individuals being merely attached by the sternum. In B the doubling is much less complete, the individuals being attached, not only by their sternum, but by the pelvic bones and by one of their upper and one of their lower extremities. In C and D it affects all the parts above the sacrum. In E, it is limited to the upper part of the dorsal spine and head and neck. In both D and E one

[1] Shown at the time the paper was read.

half of the double monster is smaller than the other. This irregularity of size must be due either to a disproportion in the rate of growth of the two halves, or to the atrophy of one of them. Now, we believe we are right in saying that it is generally believed by teratologists that the latter is the more common cause. Indeed, it is said by some that atrophy of one half, accompanied by fusion with the other half, may proceed to such an extent as to leave but little trace of the original double condition; and that in extreme cases a third arm or leg, or the rudiments of the same, may alone remain as an evidence of it.

Granting that those cases in which such rudiments are found are due to the atrophy and fusion of one half of the double embryo with the other half, it is going but a step further to suppose that in the present specimen a still greater atrophy, accompanied by fusion, has occurred, and that here the right scapula alone remains as evidence of the original doubling.

If we adopt this view the specimen would consist in greater part of the right half, and to a less extent of the left half of the originally double embryo. Thus the head, neck, upper part of the dorsal spine, with the ribs, scapula, and arm on the right side, would be developed from the right half of the embryo, while the left scapula, the left arm, and a few of the ribs of the left side, and the process of bone would be formed from the left half. The parts of the right embryo which would have been suppressed are the left scapula and left arm; and of the left embryo, the head, neck, upper part of the dorsal spine, and the right arm, the scapula remaining as the triangular piece of bone.

The strong resemblance of the process of bone to the scapula, and the jumbling together of the vertebræ, have been urged upon us as reasons for accepting this theory. It has been pointed out to us, though we fail to see it, that the process of bone presents evidences of a spine and glenoid cavity, and that the inclination of the upper portion of the dorsal spine to the right, and the inter-collation of the half vertebra, are indications of a bifurcation of the spine at this

spot. In the College of Surgeons there is a specimen of a bifurcated spine of a calf, in which the vertebræ at the place of bifurcation are jumbled togther in a somewhat similar manner to those in the present specimen. We believe, however, that this state of the vertebræ has been better accounted for by the explanation we have already given in the former part of this paper. The resemblance of the process of bone to the scapula is not to our mind sufficiently marked to warrant us to regard it as such; and although we admit the possibility of its origin from the doubling of the embryo and suppression of the greater part of one half, we confess that such an explanation appears to us highly improbable.

DESCRIPTION OF PLATES V AND VI.

Dissection of the Parts in a Congenital Malformation of the Spinal Column, &c. (Mr. A. Willett and Mr. W. J. Walsham.)

PLATE V, Fig. 1.—View from above.
Fig. 2.—Back view.

PLATE VI.—Front view.

Fig. 1.

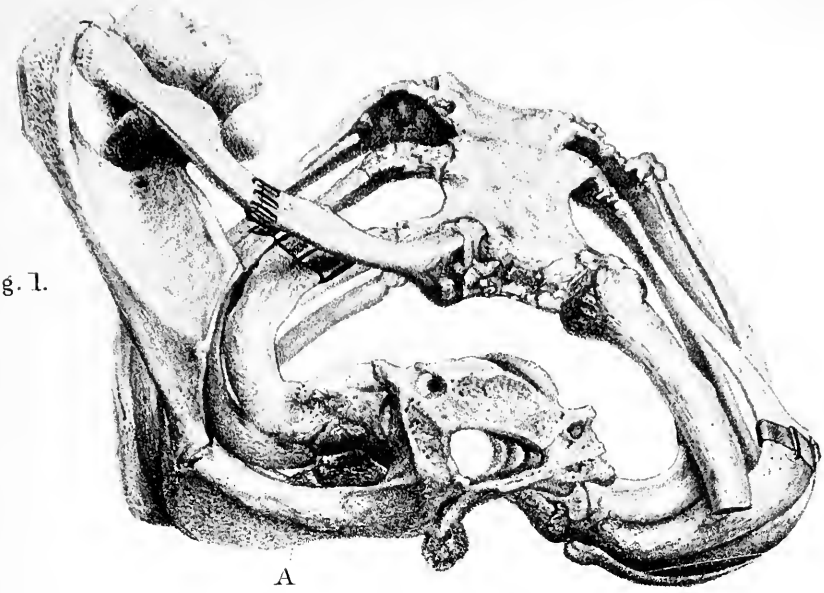
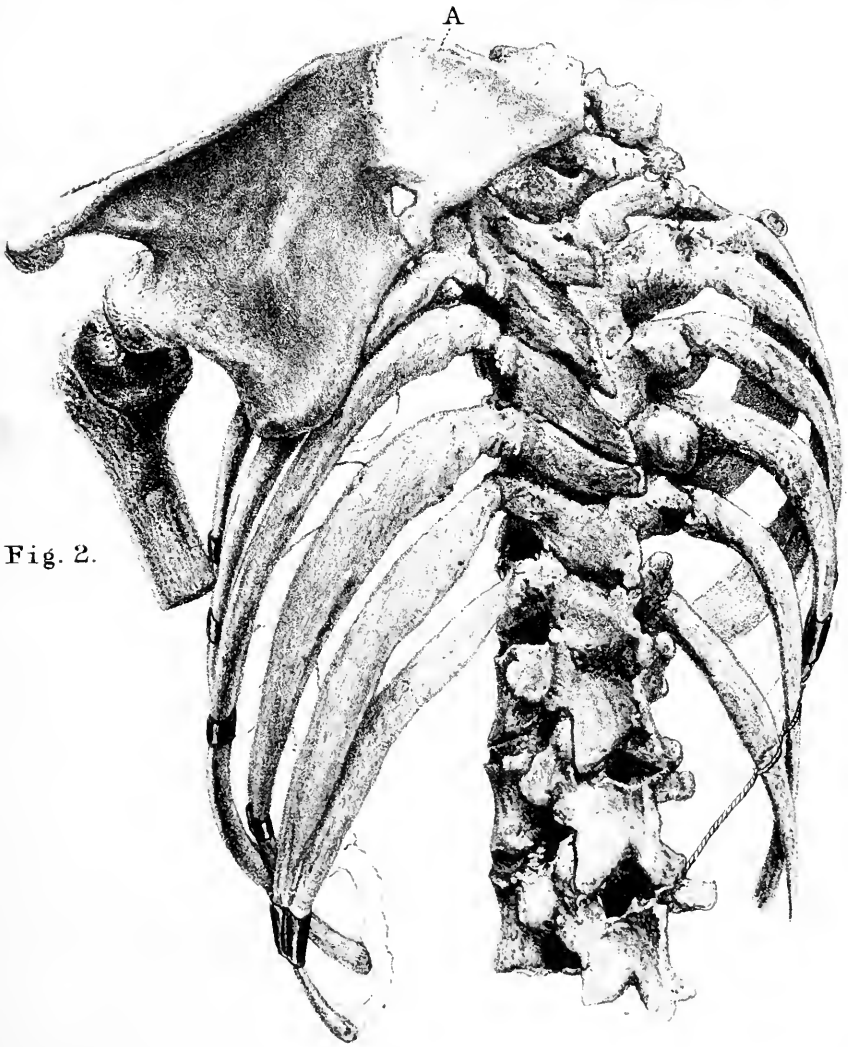
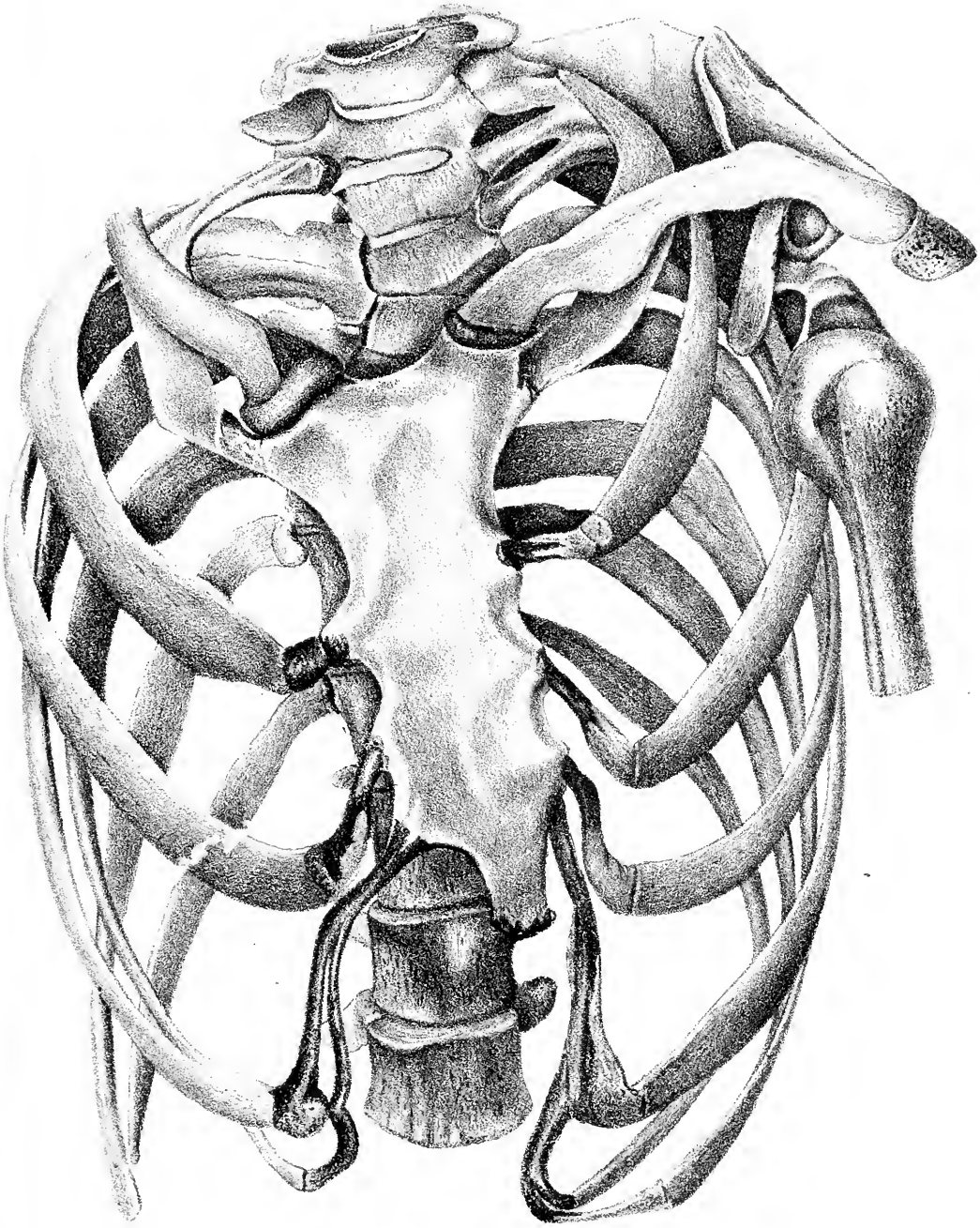


Fig. 2.



Mintern lith. Mintern Bros imp.



COMPOUND FRACTURE OF THE FEMUR

TWENTY YEARS AFTER EXCISION OF THE KNEE-JOINT FOR DISEASE,

THE LINE OF FRACTURE BEING TRANSVERSELY ONE INCH ABOVE THE BONY UNION.

BY

FREDERICK JAMES GANT, F.R.C.S.,
SENIOR SURGEON TO THE ROYAL FREE HOSPITAL.

(Received February 12th—Read April 27th, 1880.)

The previous history of this case is included in a series of joint-excisions which I brought before the Society in the year 1870, and which is published in the 'Transactions,' vol. liii; but it may not be uninteresting to briefly relate the chief particulars of the case in connection with its sequel.

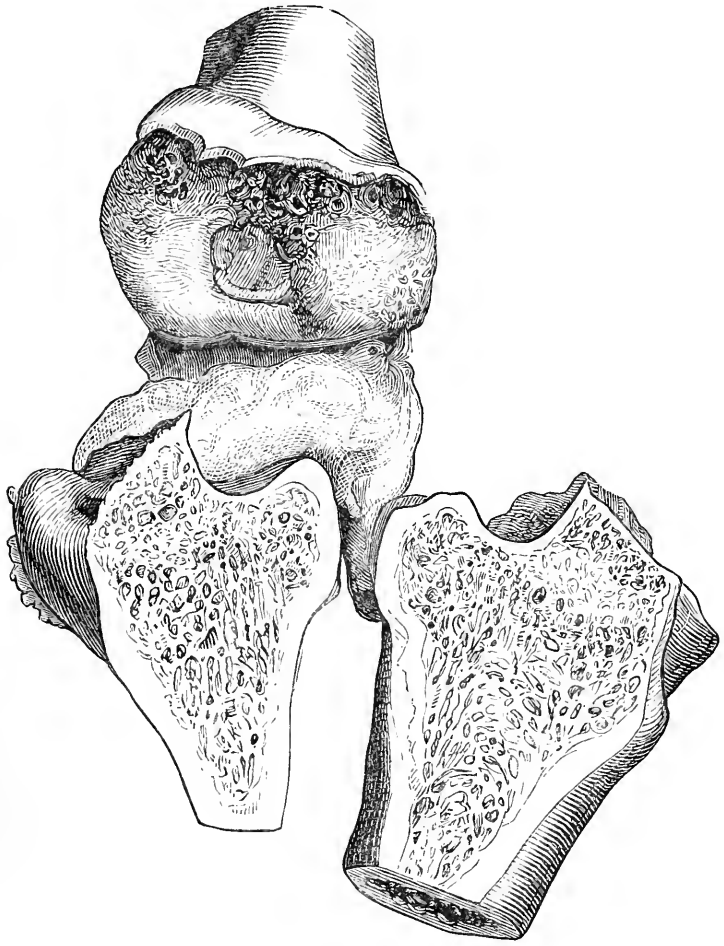
Elizabeth D—, æt. 33, married, without children, had always enjoyed good health. In the year 1853 she injured her right knee, by a wrench inwards, from a fall, which was followed by the swelling and pain of acute synovitis. Treatment seems to have had little permanent effect, for the disease continued in a chronic form. Five years afterwards a rheumatic attack, apparently, affected the same joint. September, 1859, she was admitted into the Royal Free Hospital. The joint was then considerably swollen, and discharging through three sinuses a thin

purulent matter. Grating crepitus and mobility plainly declared that destruction of the articular cartilages had occurred, with—as the operation showed—deeper caries of the adjoining portions of the femur and tibia than is usual in consequence of synovitis, especially of traumatic origin, and the leg being drawn backwards toward the buttock the limb was rendered further useless. The constitutional condition was that of nervous exhaustion from six years' uninterrupted suffering, the patient having obtained snatches of sleep at night by pouring a pitcherful of cold water over the knee, to kill the pain, as she said, in the hope of dosing off before it returned. But there was no hectic nor marked emaciation.

Under these circumstances I excised the joint, and then fixed the limb in a McIntyre splint, an apparatus which I have long since discarded for the back-splint, and interrupted external splint with foot-piece, as constructed for subsequent cases. After operation, the patient's health rapidly improved, and the wound, with simply lint-dressing, healed by primary union, except at the external angle, where healthy granulations sprang up. At the end of a month, the splint was removed for the purpose of cleanliness. In two months, a shorter period than usual by at least a month, after excision of the knee-joint, the splint was finally removed. So firm had osseous union become that the patient was then allowed to move about on crutches, with the leg bandaged only to prevent swelling. The limb remained perfectly straight, and a high-heeled shoe enabled the woman to walk with a carriage quite easy and symmetrical. She often walked distances of four, six, and eight miles a day, without any inconvenience. This result I had the opportunity of ascertaining during the first ten years before the case appeared, as figured Plate I, fig. 1, in the volume of the 'Transactions' referred to.

Ten years subsequently, E. D— slipped downstairs, and doubled the right leg under her. The integument was torn across, above the situation of the knee, to the

extent of six inches, and the femur snapped. Considerable hæmorrhage occurred, but the popliteal artery escaped injury. When brought to the hospital the patient lay in a wandering state of collapse and shock, from which she never rallied. Post-mortem examination revealed extensive changes in various organs, but the



surgical interest in this case centres in the coexisting synostosis and adjoining fracture to which I have drawn attention. The specimen is here represented. Section shows the complete character of the synostosis, the osseous texture of the femur and tibia having become so fused together that the junction is imperceptible, being indicated

only by a transverse line in the cancellated structure, between the tibia and the femoral fragment, as represented in the figure. The strength of this union is attested by its having withstood the extreme violence, coupled with the long leverage of the leg, that produced a compound fracture through the bulky end of the femur. It is very doubtful whether an ossific anchylosis of the knee-joint would have borne an equal force without breaking. Thus, the cure resulting from surgical art may be more effectual than that of nature.

ON THE

TREATMENT OF PELVIC SUPPURATION

BY

ABDOMINAL SECTION AND DRAINAGE.

BY

LAWSON TAIT, F.R.C.S.,

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(Received April 13th—Read May 11th, 1880.)

I HAVE purposely used the words "pelvic suppuration" in the heading of this paper, in order to advocate a principle which I believe to be capable of much wider application than it has already had at my hands. The cases, six in number, in which I have pursued this new method of treatment have all been, so far as I could discover, cases of suppuration occurring in pelvic hæmatoceles; but the difficulties in these cases have been no greater than I think would occur in pelvic suppuration of almost any kind, and the success has been most exceptionally encouraging. My experience is, of course, limited to suppurations of the female pelvis, but I see no good reason why the same proceedings should not meet with equal success in some cases, at least, occurring in the male.

Like others who follow the surgical speciality in which my practice lies, I have had a wide field for the observation of the various conditions classed under the head of

pelvic abscess, and, like others, I have until recently confined my treatment of it to openings made from the vagina or in the neighbourhood of Poupart's ligament. Experience, however, has driven me to the conclusion of Dr. Emmet that, "I cannot regard the introduction of the trocar into the inflamed tissues of the pelvis as a procedure free from danger under all circumstances." It is perfectly true that in very many cases where an abscess undoubtedly exists in the cellular tissue of the female pelvis, the fluid can be reached and removed by the needle of the aspirator. But, according to my experience, the relief obtained in this way is, in a large number of cases, neither complete nor permanent, and, in nearly all, the convalescence has occupied a time not at all commensurate with the extent of the lesion. This is quite as true of abscesses which have been allowed to open themselves or have been assisted to open in the groin. They continue as fistulous openings for years.

In many cases, even when the abscess can be reached by vaginal puncture, the nature of its contents is such as to make its evacuation an impossibility; and I have seen several where a puncture made at random through an indurated pelvic roof has missed the disease. In these cases the symptoms of the presence of pus were conclusive, but no indication of its seat could be obtained. Dr. Emmet speaks of such in these words:—"I can recall a number of cases, which have been under my observation with thickened tissues, where no treatment had the slightest effect, and finally, they have passed into other hands."

The course of such abscesses is so thoroughly described by Dr. West that his words cannot be improved upon, and therefore I give them at length:

"When suppuration takes place the matter makes its way outwardly through the vagina, or through the intestinal canal, in almost all cases in which the inflammation is limited to the parts contained within the broad ligament. In those cases, however, in which the pelvic cellular tissue is im-

plicated, the matter not unfrequently makes its way round between the muscles and the external surface of the peritoneum, and the abscess points and discharges itself through the abdominal walls somewhere in the course of Poupart's ligament, or a little below that situation.

"Though the size of the abscess is not in general very great, it not unfrequently passes into a chronic state, and emptying itself, for the most part, through some narrow passage of communication into the bowel, the patient continues for months or years liable to occasional discharges of pus per anum, the commencement of which dates back to some attack of inflammation of the cellular tissue years before."

In an instance quoted by Dr. West, "occasional discharges of matter took place from the bowel, and pus was often intermixed with the fæces, five years after the first symptoms of inflammation of the cellular tissue about the uterus, the chronic results of which were still evident in a tumour which was closely connected with the rectum and the womb. These chronic abscesses generally contract, and the fistulous passages that lead to them become by degrees obliterated, but exceptions to this now and then occur, two of which have come under my own notice; and Sir J. Simpson has reported some very interesting cases where permanent fistulous communications have formed between the abscess succeeding to inflammation of the pelvic cellular tissue and the bladder, uterus, or intestinal canal."

In my own practice, such disappointing cases have occurred with but too great frequency, and though I have had some successes by the employment of such means as the elastic ligature ('Lancet,' June 27th, 1874) and counter-opening in the vagina ('Lancet,' April 3rd, 1875), yet the progress towards recovery has been so protracted as to contrast favorably only with those cases in which there was no recovery at all.

I have been, therefore, continually on the outlook for some means of dealing with such cases which would bring them as satisfactorily within our means of treatment as

are collections of matter in most other parts of the body. This has been furnished by the wide, free, and successful application of abdominal section for the treatment of pelvic and abdominal tumours, and I have now to lay before the Society six cases, which include the whole of my experience in this novel proceeding, and in which success has been obtained far surpassing anything I have yet seen or heard of. In this comparison I am of course excluding those cases where pointing of the abscess in the vagina is evident at an early stage of the case, but even in these the recovery has always been, in my experience, more protracted than in the six now to be narrated.

A patient was sent to me in February, 1879, by Mr. Gwinnett Sharp, of Walsall, suffering from a pelvic tumour associated with very severe symptoms. She was twenty-two years of age, and had been married nine months. Her menstruation had always been too frequent and too profuse, and six weeks before I saw her it had stopped suddenly during its course, and this was associated with the onset of violent pelvic pain—the leading features of extra-peritoneal hæmatocele. A few days afterwards she shivered, and became very ill and feverish, and these symptoms had become intensified when I saw her ten days after their occurrence. She was then emaciated and hectic looking, with a high night temperature, intense pain and tenderness over the lower abdomen, and when examined a large fluctuating tumour, adherent to and behind the uterus, and going on either side of it, was found to occupy the pelvis and rise about half way up to the umbilicus. The roof of the pelvis was fixed and hard, and no fluctuation could be felt there.

The nature of the tumour could be open to only two suggestions—that it was a suppurating parovarian cyst with peritonitis, or a suppurating hæmatocele. I leant to the latter view, as it was in consonance with the history, and I have never known a parovarian cyst suppurate, whilst hæmatoceles frequently do.

In any case I determined to open it from above, and

this I did. I found a large cavity containing about two pints of fœtid pus with decomposing blood-clots. This I carefully cleansed out, and after having united the edges of the opening into the cyst carefully to the abdominal wound, I fixed in one of Koeberle's glass drainage-tubes, five inches long. Seven days after the operation I placed a three-inch glass drainage-tube, and in another week this was replaced by a soft rubber tube. The patient got up on the twentieth day after the operation, and in ten days more went home perfectly well, with the abscess healed, and she remains now in perfect health (March, 1880).

The second case was sent to me by Dr. Flynn, of Birchills. She was forty-five years of age, and had never been pregnant, save one doubtful miscarriage soon after marriage, nineteen years ago. Symptoms resembling those of hæmatocele had occurred eight months before I saw her, and since that time she had been losing flesh, had lost her appetite, was troubled by constant thirst, and night sweats, and she had a rising night temperature. The uterus was fixed in a mass of effusion occupying the left broad ligament, and partly the right one also, and the mass on the left side encircled the rectum, forming a pronounced stricture, as hæmatoceles of the left broad ligament frequently do. No point of fluctuation could be felt in the pelvis, but the symptoms pointed clearly to the presence of pus. I therefore determined to open the abdomen, and readily obtained the consent of my colleague to this proceeding.

On reaching the peritoneum the two layers were found to be adherent, so that the cavity was not opened. A large abscess was opened just behind the base of the bladder, between which and the uterus it principally lay, but stretching round behind the rectum. The floor and posterior wall of the abscess were found to consist of organised blood-clot, so that its origin was in a blood effusion into the broad ligament. A glass drainage-tube was inserted, and this was changed for one of Chassaignac's wire tubes on the eleventh day after the operation. She

sat up on the twenty-first day after the operation, and the tube was finally removed on the twenty-sixth. She went home on the thirtieth day perfectly well, and has remained so ever since, now nearly ten months.

The third case was a patient of Mr. Hall Wright's, in whom he had diagnosed hæmatocele some four weeks before I saw her. Symptoms of suppuration set in, and I performed exactly the same operation as in the first case, that is, the peritoneum was opened, and the abscess emptied and cleansed, and then the edges of its opening fastened to the edges of the parietal wound, and a glass drainage-tube fastened in. This case also was an undoubted hæmatocele of the broad ligament. Eight days after the operation the glass tube was changed for a wire one, and this was removed in twelve days more.

She left the hospital perfectly well only thirty-three days after her admission, and has since remained in good health.

Mary Ann B—, æt. 30, has been married eight years, and has had four children, youngest fifteen months old. Seen first by Mr. Hall Wright on December 12th, when she stated that she had become unexpectedly unwell about five weeks ago, that this was accompanied by violent pain which has never since been absent. She was seen by Mr. Hall Wright, who diagnosed the occurrence of hæmatocele from the presence of a large hard tumour behind the uterus. About a fortnight previous to my seeing her the nocturnal exaltation of temperature, night sweats, thirst, and increased pain, led Mr. Hall Wright to suspect that the effusion was suppurating, and when I saw her I had no difficulty in confirming his opinion. I therefore admitted her to hospital, and on December 22nd I performed abdominal section, as I found the intensity of the symptoms increasing, and on examination under ether I found that the mass of the effusion seemed too high up to be opened safely from the vagina. I found the tumour to be a large effusion of blood in process of disintegration, contained in a cavity formed by the lifting up of the

posterior layer of the broad ligament, the rectum being carried up in front of it, together with the large vessels of both sides, as high as the bifurcation of the aorta, whilst anteriorly the peritoneum dipped to a most unusual depth, so that had I tapped the tumour from the vagina I must have gone through the peritoneal cavity. The cyst was opened and emptied, and a drainage-tube fastened in, and the peritoneum closed in the usual way. Her recovery was neither so easy nor so rapid as any one of the others, probably because the cavity was the largest of all, and her condition before the operation was very bad, though another case to be related was in the latter respect much worse. The temperature in the present case was 38·4° before the operation, and it rose to 40° on the second day. It did not fall to 37° till the tenth day, December 31st. The drainage-tube was removed on January 10th, and on the 17th the wound had perfectly healed, and she left the hospital on the 26th. I saw her on February 26th, when she was hardly to be recognised, so astonishing was her restoration to health. From a thin, emaciated, and apparently dying woman, she had been transformed into a perfect picture of health, and she stated that she was able to do her work and to get about as well as ever she did in her life.

In this case I am quite satisfied that the delay of the operation for a few days would have been fatal, and no vaginal tapping, even if it had missed the peritoneal layers, would have emptied the cyst of its clotted contents.

Ann S—, æt. 28, placed under my care in January of the present year by Dr. Gordon, of Walsall. She is the mother of three children, the youngest being three years of age. About four months ago she had symptoms resembling those of sudden effusion of blood into the broad ligament. For a month she was able to get about, but during the last three months she has been entirely confined to bed, the subject of symptoms clearly pointing to the occurrence of suppuration. Dr. Gordon had discovered the presence of a pelvic mass behind the uterus,

in which no fluctuation could be discovered, and which was fixed.

I admitted her into hospital, and suspecting the case to be one of suppurating hæmatocele, I opened the abdomen on January 5th, and found the case to resemble the preceding one, save that the disintegrating effusion was not so large. It was dealt with in the same way, and the patient made a much more easy and rapid recovery, leaving the hospital on January 17th, and being restored to perfect health before the end of February.

Mrs H—, æt. 29, was married at 18, had a child within the year, and has never been pregnant since. I saw her, at the request of Dr. Millington, of Wolverhampton, under whose care she had been, in conjunction with Dr. Blackford, of Cannock, whom I met in consultation over the case on the 15th of last January. The history given to me was that about nine weeks previously, when driving in an open carriage with her husband on a very cold day and during a menstrual period, she was suddenly attacked by very violent pelvic pain, and coincidently with this the discharge ceased. This pain had continued ever since, and had of late increased in severity. Menstruation had occurred at two irregular intervals since the beginning of her illness with great profuseness, and during these periods her pain had been much easier. A pelvic tumour had been discovered by Dr. Millington some weeks before my visit, and this he had regarded as an effusion of blood. She had suffered for about three weeks before I saw her from night sweats, almost constant sickness, utter loss of appetite, intense thirst, with various other symptoms of pronounced hectic. The tumour, when I examined it, involved all the pelvic organs in a fixed mass of cartilaginous hardness, with the uterus embedded in it; the bladder spread over it in front, and the rectum encircled by a ring of hard effusion. The mass could be felt above the pelvis as a round and non-fluctuating tumour, with intestine in front of it. The patient had reached almost the final stage of exhaustion and emaciation. There was

no difficulty in diagnosing the case as one of suppurating hæmatocele. With Dr. Blackford's concurrence we had her removed to Birmingham, and on the 21st I opened the abdomen and found matters quite as I had anticipated. The posterior layer of the broad ligament was lifted completely up out of the pelvis, and so was the anterior layer, as far as I could make out; at least, the only structure I could identify was the base of the bladder, and that seemed to form the anterior boundary of the tumour. From this point it spread backwards, on a level with the brim of the true pelvis, and its posterior boundary was the bifurcation of the aorta. The contents were clearly fluid, and therefore I tapped it with an aspirator needle, and evacuated about half a quart bottle full of curdy blood-coloured pus. I then laid the cyst open from the point of puncture, in a direction from before backwards, and found its floor to consist of a thick layer of laminated clot, hard and rigid. I could make out the uterus rising out of this mass, but I could not discover the rectum.

I stitched the edge of the opening into the abscess to the edges of the parietal wound, and then closed the rest of the peritoneal opening, and fastened in a wide drainage-tube of glass. After the operation the patient's temperature never rose above 37°, she had no more night sweats nor sickness, and her appetite was really keen on the third day. A small-sized wire drainage-tube replaced the glass one on the twelfth day, as the discharge had become healthily purulent and free from clot *débris*. The smaller drainage-tube was removed on the fifteenth day after the operation, and on the twenty-fourth the sinus was quite healed, she had gained greatly in flesh and colour, she was able to walk about, and on the twenty-seventh day she went home perfectly well, the uterus, however, being still quite fixed, as I expect it will remain for years. I have just had a letter from her (March 30th), in which she reports herself as being in perfect health.

In all of these cases I am satisfied that vaginal tapping

would have been useless. In most of the cases, if the abscess had been opened by natural processes, it would have been into the rectum. In the last case it would probably have been in one of the groins; but I think in every one of the cases, unless it be in the second, death would have occurred long before a natural outlet could have been established.

My general conclusion from these cases is that the opening of such abscesses by abdominal section is neither a difficult nor a dangerous operation; that recovery is made in this way more certain and rapid than in any other; and that in future I shall always advise an exploratory incision where I am satisfied there is an abscess, which cannot be reached nor emptied satisfactorily from below.

A CASE

OF

SUB-CORACOID DISLOCATION OF THE HUMERUS,

WITH THE

FORMATION OF AN INDENTATION ON THE POSTERIOR SURFACE OF THE HEAD, THE JOINT BEING UNOPENED;

WITH REMARKS ON THE MODE OF PRODUCTION OF FRACTURE OF THE ANATOMICAL NECK, WITH DISLOCATION.

BY

FREDERIC S. EVE, F.R.C.S.,

CURATOR OF THE MUSEUM, ST. BARTHOLOMEW'S HOSPITAL.

(Received March 23rd—Read May 11th, 1880.)

Thomas B—, æt. 36, was knocked down by a train while at work on the line. He was admitted to St. Bartholomew's Hospital under the care of Mr. Holden, and was found to have sustained a sub-coracoid dislocation of the right humerus; the head of the bone was distinctly felt beneath the coracoid process, and the axis of the limb was directed considerably outwards and backwards.

The dislocation was extremely easily reduced by traction on the limb with the heel in the axilla.

The patient died twelve hours after the accident from injuries to the thorax.

Dissection.—The deltoid muscle was bruised, and the upper portion of its insertion separated from the bone. The muscles around the shoulder-joint were carefully dissected, but no laceration of their substance was observed; there was, however, some extravasation of blood beneath the subscapularis near the joint. On exposing the capsule it appeared quite uninjured.

On opening the joint it was observed that the capsule had been stripped off from the anterior border of the glenoid cavity, but had remained continuous with the periosteum, which was also detached to a small extent from the adjacent anterior surface of the scapula; the joint had not, therefore, been opened by the injury. On the posterior surface of the head of the humerus, at the margin of the articular cartilage, there was a deep vertical indentation or groove, into which the anterior margin of the glenoid cavity accurately fitted.

Remarks.—The dislocation appears to have been of the true sub-coracoid variety, as described by Professor Flower.[1] The bruising of the deltoid, and the mode of occurrence of the injury, indicate that the dislocation was produced by direct violence, that is, by a blow on the upper and outer part of the upper arm.

The condition of the capsule in this case appears to be entirely without precedent. I have been unable to find on record a single case of traumatic dislocation of the humerus, in which the joint was not opened.

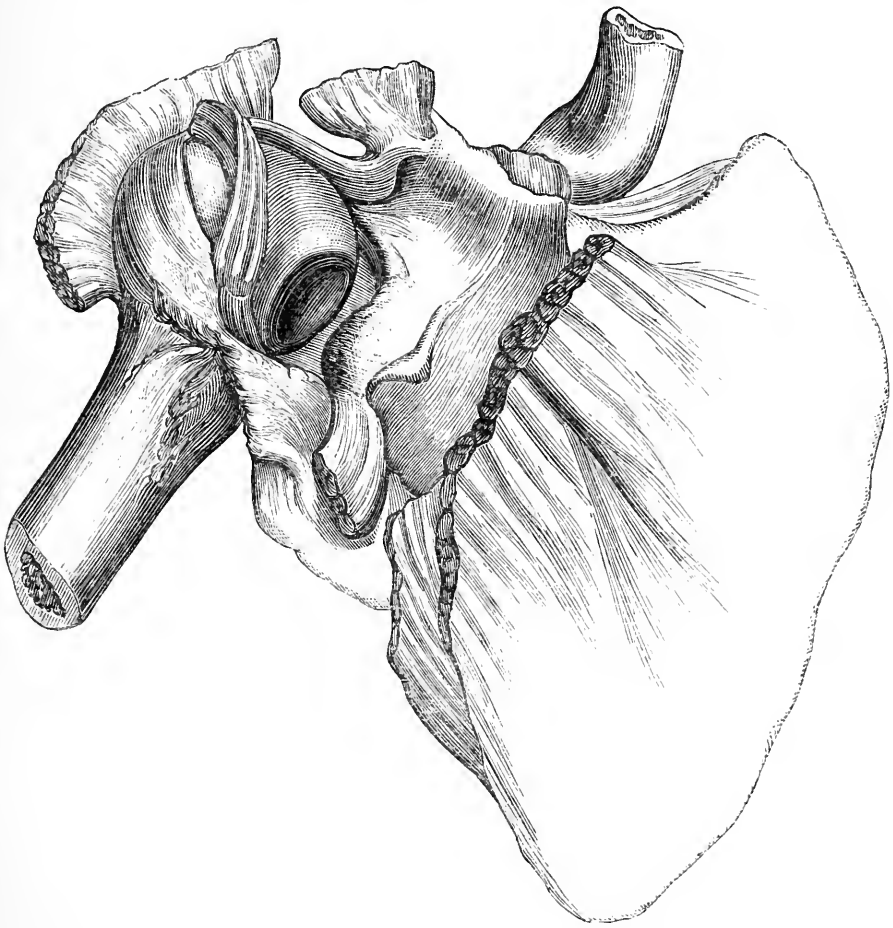
It cannot be ascribed to any abnormal change in the capsule, such as enlargement from distension or unusual elasticity, since room was made for the displaced head of the humerus by the detachment of the capsule and periosteum from the scapula, as described. The capsule and other tissues of the joint also appeared perfectly healthy.

The indentation or groove on the posterior surface of the head of the humerus is another unusual feature in the case.

From its position, shape, and the mode of occurrence of

[1] 'Holmes' System,' 2nd ed., vol. ii, p. 814.

the injury (presumably by direct violence), I conclude that the groove was produced by the forcible impact of the humerus against the anterior margin of the glenoid cavity.



Sub-coracoid dislocation of humerus reduced during life. The capsule was not lacerated, but was stripped up with the periosteum from the anterior margin of the glenoid cavity. A deep indentation is seen on the posterior surface of the head, produced by forcible impaction on the margin of the glenoid cavity. Specimen preserved in St. Bartholomew's Hospital Museum, No. 1019.

The formation of the groove partially accounts for the slight damage to the capsule, as the head of the bone, having lodged on the margin of the glenoid cavity, was thus prevented from passing further inwards on the surface of the scapula. The groove was situated at the extreme margin of the articular surface, showing that the head was entirely

separated from the glenoid cavity; the case cannot, therefore, be regarded as an instance of incomplete dislocation.

There are two dried specimens of shoulder-joints in the museum at St. Bartholomew's[1] Hospital, showing dislocation of the humerus forwards with the formation of a groove or trochlear surface on the posterior portion of the head, evidently produced by attrition against the anterior margin of the glenoid cavity, which has itself been considerably worn away. Malgaigne[2] has noticed the occasional occurrence of grooves on the head of the humerus after dislocation. He mentions two cases described by Sédillot, which presented much the same appearances as the above. In these specimens, as in the two former, the furrows were hollowed out by the movements of the head upon the glenoid margin, but Malgaigne remarks[3] that he thinks it is a question if they are not sometimes produced, "at the moment of dislocation, by the crushing of the head of the humerus upon the border of the glenoid cavity," a conjecture which is proved correct by the case related. It is also probable that the commencement of the groove might, in some of the specimens mentioned, have been formed in a similar manner.

The occurrence of such a groove as that observed in the case related is of considerable interest, since it may explain the mode of production of fracture of the anatomical neck with dislocation of the head of the humerus forwards. If the blow had been sufficiently forcible the head of the humerus would probably have been chipped off by the anterior margin of the glenoid cavity, instead of simply an indentation being produced by it, and the head would then have slipped inwards on the anterior surface of the scapula, with the articular surface directed forwards, as usually happens in cases of fracture of the anatomical neck with dislocation of the head. Such a condition is well known in a specimen[4] of fracture of the

[1] Sub-series C, 27, 34.
[2] 'Fractures et Luxations,' p. 496.
[3] Op. cit., p. 497.
[4] Specimen shown. Sub-series C, No 103.

anatomical neck with dislocation, in the museum of St. Bartholomew's Hospital, also by Sir Astley Cooper's specimen in St. Thomas's Hospital Museum, in which the parts are dried *in sitû*.

Further, the mode of production of the hitherto unaccountable cases of impacted fracture of the anatomical neck may, perhaps, *in some instances*, be explained in a similar manner. If the posterior surface of the neck of the humerus were driven against the anterior margin of the glenoid cavity and the head thus broken off, it appears possible that a continuation of the force might drive the broken extremity of the shaft upon the articular surface of the head (now directed forwards), producing a more or less complete impaction. There would necessarily be a forward displacement of the head, and this was precisely what was observed in the two specimens described by Mr. R. W. Smith.[1] In both of these the head of the humerus was displaced forwards beneath the coracoid process, and rotated on its axis, so that the articular surface looked forwards; only the outer part of the articular hemisphere of the head was driven into the upper extremity of the shaft, the inner part being free. The bones, in fact, occupied much the same relative positions as in fracture of the anatomical neck with dislocation of the head, except that the outer part of the articular surface of the head was driven into the shaft.

In both specimens a fracture passed through the tubercles; in neither had the capsule been torn.

The rare peculiarities which the case described presents, rendered it, I thought, of sufficient interest and importance to bring before this Society. I am indebted to the kindness of Mr. Holden for permission to do so.

[1] "Fractures and Dislocations."

ON A CASE

OF

EPITHELIOMA OF THE NECK

FOLLOWING A PATCH OF CHRONIC SKIN DISEASE,

IN WHICH

THE CANCER WAS TWICE EXCISED AND THE EXTERNAL AND INTERNAL JUGULARS WERE LIGATURED.

BY

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(Received April 10th—Read May 25th, 1880.)

Eliza H—, æt. 60, an emaciated grey-haired woman, was sent to the Middlesex Hospital by Dr. Langmore, on account of an ulcerated growth on the right side of the neck. She was admitted into Queen Ward on September 8th, 1876.

History.—Nine years previously she had a small circular red and scaly patch about the size of a sixpence on the right side of the neck, which remained in much the same condition for about five years, shedding fine light scales. At the end of this time the surface of the patch was knocked off by the elbow of a child, and the resulting sore was soon covered by small scabs; it slowly but

gradually increased in size. Five months later the sore was cauterised in Petersfield Infirmary, where she remained six weeks; and on leaving, it was about the size of the original patch, and apparently healing. A few weeks later the ulcer began to enlarge and the discharge from it to increase. She placed herself again under medical treatment; and, for fourteen months prior to admission under me, had undergone a course of treatment by "burning plasters;" but still the disease continued to extend. Of late she had suffered great pain during the application of these plasters as well as between the times of their application. She had been losing flesh for eight months and had for a long while been subject to palpitation of the heart. She had also a large bilateral solid bronchocele, the posterior limits of the right lobe of which abutted upon the anterior border of the ulcer.

State on admission.—At the time of admission there was in the central and upper part of the right posterior triangle of the neck an epitheliomatous ulcer covering an area as large as half the palm of a man's hand. This ulcer had the usual raised, everted, and hard edges, and was of a greyish-yellow colour, prone to bleed, not very painful when covered, but apt to smart when exposed to the air. In the centre was a deeper part containing a yellowish slough, and the margin of this part was of a red colour and slightly everted. At the circumference the growth was free of the deeper tissues, so that the finger tips could be dipped between it and them. This was especially the case in front and below; but behind, where the ulcer spread over the anterior border of the trapezius, the margin of the growth was less capable of being raised. The central sloughy portion seemed firmly fixed to the subjacent structures of the posterior triangle. The carotid artery of each side was displaced outwards by the bronchocele, beyond the outer edge of the sterno-mastoid; whilst the right carotid communicated visible pulsation to the *anterior part* of the ulcer, and to the skin above it. Two hospital surgeons who had seen her

refused to operate, because it was thought impossible that the diseased parts could be removed without interfering with the continuity of the carotid artery; but the artery was proved, convincingly, to be free, by making the patient, whilst lying on her back, strive to raise her head, which was tightly held down upon the pillow by an assistant. The resisted effort to bring the chin towards the sternum brings into forcible action the sterno-mastoids, and thus any superficial growth is raised away from the influence of the carotid pulsations. The patient was very anxious to have the disease removed, so on September 20th ether was administered, and an incision was made through the skin about one eighth of an inch from the margin of the ulcer all around it, and the less fixed parts were dissected off. As much as possible of the *central part*, which was now seen to be incorporated with the *scalenus anticus* muscle, and with the lymphatic glands at the back of the sterno-mastoid, was then cut away; the rest was scraped away with a raspatory, and finally the suspicious points were seared with the galvano-cautery. The cautery served also to arrest the bleeding, which was not severe and chiefly venous. A ligature was applied early in the operation to the external jugular vein. The wound was dressed with terebene oil on lint, and covered with a figure-of-eight bandage. A sleeping draught was required at night.

On September 21st she was in no pain, but had had a restless night, and her pulse was 172 per minute, and very small. Throughout the pulse was very rapid and small; the wound healed slowly and was grafted.

On November 3rd it was quite healed, but subsequently a portion of the thin cicatrix gave way once or twice.

By November 27th it was however quite sound, and on the following day she left the hospital recovered.

The parts removed were examined microscopically and were seen to have the characters of an ordinary epithelial cancer of the skin.

In August, 1877, I heard by letter that she remained

quite well, and had been working in the fields all the summer.

In January, 1878, a lump showed itself on the neck in the old spot, which she was anxious to have removed; and for this purpose she went, in March, 1878, to the Petersfield Cottage Hospital. From Petersfield she came again to the Middlesex Hospital, and was admitted under me on March 21st, 1878. At this time there was a large irregularly circular sessile growth behind, but also slightly over the line of the cervical vessels, and in the cicatrix of the former operation; but again the artery was found to be uninvolved. On March 27th this recurrent growth was removed by means of a circular incision surrounding it, and then by dissecting it off the deeper structures from the periphery inwards. In this manner it was all removed, excepting the deeper stratum of the growth over the line of the great vessels, which was left to be separately and more scrutinizingly cut away. It was now seen that the sheath of these vessels was diseased, and on gently touching what remained of the growth with the dissecting forceps some of it immediately broke away, followed by a large gush of venous blood. It clearly came from the internal jugular. There was no possibility of doubting its source.[1] Instantaneously I pressed upon the vein below the bleeding point so as to prevent the entrance of air, which otherwise would have been all the more likely aspirated owing to the rigidity of the diseased walls of the vein. Mr. Hulke, who had been watching the operation, kindly assisted me at once, and arrested the hæmorrhage by keeping his fingers over the opening in the vein. As quickly as possible I exposed the vessel above and below its wounded part, and keeping wide of the disease, placed a silk ligature around it in both situations. All bleeding being stopped, I scraped away the remainder of the growth, excepting the diseased part of the vein between the ligatures, with the raspatory; the wound was dressed

[1] The lumen of the vein was in no way occluded by the disease, but was of quite large diameter both above and below the opening.

with terebene oil, and a compress was retained by a figure-of-eight bandage passed round the neck and opposite axilla. The diseased part of the vein assumed in a few days a peculiar cheesy yellow granular appearance and quickly sloughed away in little flakes and masses.

On April 9th, both ligatures having separated from the internal jugular, some chloride of zinc paste was applied to a small spot, the rest of the wound being protected by zinc ointment. The caustic was reapplied once or twice afterwards, but on April 21st the terebene oil dressing was resumed, and from this time the wound healed progressively, and she left the hospital well on May 23rd, 1878. The patient bore each of the operations, and the after-treatment remarkably well, her calm temperament assisting greatly her recovery.

On February 9th, 1880, I heard by letter that she had had no recurrence, that the neck was quite well, and that her only ailment was weakness and palpitation of the heart from which she still suffers a good deal.

In July, 1880, she was known to be alive and well.

Remarks.—The importance of this case is two-fold; first, as regards the origin of cancer; and, secondly, on account of the ligation of the internal jugular vein.

As to the origin of the cancer. The epithelioma was preceded by a superficial, circumscribed skin affection of several years' standing, and limited to the self-same spot at which the cancer commenced. It is thus closely allied to those cases in which a chronic sore has long preceded epithelioma of the lip; to those of epithelioma of the tongue engrafted upon ichthyosis glossæ, to which the attention of surgeons was first specially directed by Mr. Hulke; and to those other cases of epithelial or duct cancer of the breast preceded by so-called eczema of the nipple and areola, for the first account of which the profession is indebted to Sir James Paget. I have not ventured to apply any name to the antecedent skin disease, which is said to have consisted of the desquamation of

thin light scales from a slightly reddened but otherwise unaltered surface; but though it had not been preceded or accompanied at any time by any appreciable moisture or discharge, it would doubtless be considered a form of chronic eczema.

But apart from the name most appropriate for it, the fact to which I wish to draw attention is the supervention of the epithelial cancer upon the diseased patch.

An abnormal condition of the epidermis had existed for five years, when the part became subjected to fresh and increased irritation, first by a blow, and afterwards by frequent cauterisation. The sore which formed was at first limited to the spot previously affected with the skin disease, and from this it spread wider and deeper, at first slowly, afterwards more rapidly.

The case supplies another illustration, although a somewhat different one from those already published, of cancer excited by long-standing irritation of epithelial surfaces, and spreading from those surfaces to the tissues beyond; it also points to the truth of Sir J. Paget's suggestion, "that a superficial disease induces in the structures beneath, in the course of many months, such degeneracy as makes them apt to become the seats of cancer" ('St. Bartholomew's Hospital Reports,' vol. x, 1874). And, further, it gives, I think, support, like the other cases to which I refer, to the hypothesis of the local as against the constitutional origin of cancer.

As to ligation of the internal jugular vein. The ligation of this, the largest vein in the body which the surgeon is liable to be called upon to ligature, did not in this case cause the slightest untoward symptom, notwithstanding the external jugular of the same side had been ligatured also. The extent to which the vein was involved in the disease, and the friable tissue which had taken the place of the normal structure of its coats, made it impossible to employ the lateral ligature; or, indeed, to rely on any hæmostatic measure short of the ligation of the whole circumference of the vein above and below.

From the knowledge that apoplexy occasionally results from obstruction to the return of blood from the head; having, also, seen patients suffering from cancer of the breast die in a state of coma after the supra-clavicular glands have enlarged sufficiently to resist the flow of blood through the cervical veins; and knowing that the external jugular in this patient was obliterated, I had some fears, during the first twenty-four hours, lest she should suffer from any of the effects of cerebral congestion; and I was, for a time, in doubt as to whether it would not have been better to have followed the advice of Langenbeck, who recommended that the corresponding artery should be tied for wounds of the internal jugular, axillary, external iliac, and femoral veins; or, as he also recommended for wounds of the internal jugular, to have tied artery and vein simultaneously. The sequel of the case shows, however, that there was no need for any such anxiety, and proves that through the agency of the various sinuses of the dura mater and opposite internal jugular vein, of the veins piercing the bones of the skull, and of the inosculations between the cerebral and spinal veins, the collateral circulation can soon adapt itself, even to the entire obliteration of both jugulars of one side. It will, I think, be granted that, though success has attended the procedure advised by Langenbeck when applied to the axillary, external iliac, and femoral vessels, the ligation of one common carotid can have very little effect in diminishing the danger in wounds of the internal jugular vein, either of cerebral gangrene and softening, or of hæmorrhage. The freedom of the anastomosis between the arteries within the cranium minimises the effects of the ligature in checking hæmorrhage, whilst experience has shown that the chances of cerebral gangrene are increased by the simultaneous ligation of the common carotid and its accompanying internal jugular. Professor S. W. Gross, in an admirable paper ('American Journal of the Medical Sciences,' January and April, 1867) on "Wounds of the Internal Jugular Vein," has collected thirty cases in

which the vein alone was tied; in twenty-five cases the ligature was applied circumferentially, and in five laterally.

In only one—Dr. Wattmann's case—were there any symptoms whatever of disturbance of the brain, and in this air had been aspirated, and the vein which was ligatured laterally remained pervious. The patient recovered, and there seems no reason for thinking that the symptoms were due to the ligature. On the other hand, out of *eleven* cases (two of which for good reasons should be left out of the calculation) in which the common carotid or one of its primary branches was tied at the same time as the vein, four (*i.e.* 44·4 per cent.) had brain affection; death ensuing in one case from acute softening, in one from chronic softening, and in one from chronic inflammation; the fourth case recovered, the symptoms being limited to impairment of power of the opposite arm and leg "not exactly paralysis."

Finally, in two cases, Fleming's and Guthrie's, in which the common carotid was ligatured nine days and eight days respectively after the internal jugular, there was no cerebral affection. The first recovered, but Guthrie's patient died of arterial hæmorrhage the day following the ligation of the artery.

It thus appears that the simultaneous ligation of the common carotid and internal jugular, contrary to the opinion of Langenbeck, is nearly *fourteen* times more likely to excite brain mischief than the ligation of the internal jugular alone; and that in no case in which the ligature has been *circumferentially* applied has either apoplexy, softening of the brain, or other cerebral disorders ensued. If we confine our attention to the cases collected by Professor Gross, which are analogous to the one forming the text of this communication, viz. *where the injury to the vein has occurred during the removal of tumours of the neck*, it will be seen that of twenty-two cases of ligation of the vein alone, fourteen recovered and eight died. Of the eight deaths only four were attributable to the ligature, viz. one from pyæmia, and three from secondary hæmor-

rhage; the other four were directly due to the *injury*, viz. in two to asphyxia from aspiration of air, and in two from exhaustion from hæmorrhage. Thus we have eighteen operations with fourteen recoveries and four deaths, three of which were from secondary hæmorrhage the consequence of the ligature having been applied laterally. Out of seven cases in which the vein has been injured during the removal of tumours, and in which the vein and artery were ligatured simultaneously, there were five recoveries and two deaths, but neither due to the ligature: in one the patient died from bronchitis, in the other from shock.

Next, if we examine Professor Gross's article with a view of ascertaining the general results of all cases in which the ligature has been applied to the internal jugular, we find sixteen deaths out of a total of forty-two cases. But, if we deduct the five deaths, which were the immediate result of the *injury* and the one from bronchitis, all of which it is obviously unfair to include amongst the results of ligation of the vein, we have a total of thirty-six cases, with ten deaths, *i.e.* a percentage of 27·7. This is nearly 16 per cent. less than the mortality after ligature of the common carotid for all causes, and just about $6\frac{1}{2}$ per cent. less than that after ligature of this artery for tumours, judging by the statistics of 600 cases published by Dr. Pilz, of Breslau, in the 9th volume of Langenbeck's 'Archiv.'

Whilst it may be concluded that incised and punctured wounds of the internal jugular, if not treated by the ligature, are almost invariably fatal, yet the surgeon who may be unfortunate enough to injure this vein during any surgical operation, may take comfort from the thought that the chances of a good recovery, after the *complete* ligation of the internal jugular, are very considerable, provided the carotid artery is not interfered with, and that neither severe hæmorrhage nor the aspiration of air into the vein has occurred before the application of the ligature.

The mortality of cases hitherto reported, in which the ligature has been applied around the whole circumference

of the vein, and not laterally, is not more than 10 per cent. But if the artery be ligatured at the same time as the vein, the chances of recovery are about equal; or, in other words, the mortality is about 50 per cent., being 7 per cent. higher than after ligature of the common carotid alone.

ON A CASE

OF

BASIC CAVITY OF THE LUNG

TREATED BY

PARACENTESIS.

BY

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(Received April 13th—Read June 8th, 1880.)

Samuel P—, æt. 49, a carpenter, was admitted into the Middlesex Hospital, under the care of Dr. Powell, on the 29th August, 1879, suffering from severe cough, pain in the right chest, and profuse and fœtid expectoration. His family health history was good, and up to the commencement of his present illness he had enjoyed good health. He had been intemperate as regards stimulants, but had never had syphilis.

The patient's illness began with bronchitis in December, 1878, followed by pleuritic pains in the right side, and copious and fœtid expectoration, for which antiseptic inhalations were ordered in February, 1879. Some slight

hæmoptysis occurred during this attack. He considerably improved, however, and in May went to Torquay, the fœtor of expectoration having ceased.

In July, the patient having again lost ground, first came under Dr. Powell's notice with a certificate from Dr. Saul, of Bloomsbury Street, under whose care the patient had been, to the effect that he was suffering from *pneumonic softening of the right lung of seven months' duration.* On August 19th, the patient was found much prostrated by pleuritic pains, severe cough, profuse discoloured and very fœtid expectoration, consisting of nummulated masses contained in a tenacious frothy mucus. The temperature was raised, the tongue thickly coated, and complete anorexia and profuse night sweats were complained of, with relaxation of bowels. Antiseptic inhalations and bismuth mixture with quinine and opium at night, gave some relief, but the patient was advised to have the cavity, of which the physical signs described below gave evidence, in the base of the right lung tapped, and he was admitted into the Middlesex Hospital for that purpose on August 29th.

On admission it was noted that the patient was a large framed man (weight previous to illness 16 stone), with large features, but now sallow, anæmic, flabby looking, and somewhat emaciated. His complaint was of severe cough and pains in the right side. His breath was fœtid, and the expectoration, of gangrenous odour, amounted to about one pint in the twenty-four hours. Pulse 84, temp. 99°. He lay in the dorsal position with slight inclination to the right side, and with the head tolerably low.

Chest broad, with wide costal angle, slight flattening below the right clavicle. The normal region of cardiac dulness was covered by lung, and the heart's apex beat could not be felt, but by stethoscopy was judged to be half an inch to the left of the ensiform cartilage. The whole front of the chest was resonant. Hepatic dulness commenced between the fifth and sixth ribs in the nipple line. Very slight respiratory murmur was audible over the right front, but on the left side there was good vesicular breath sound.

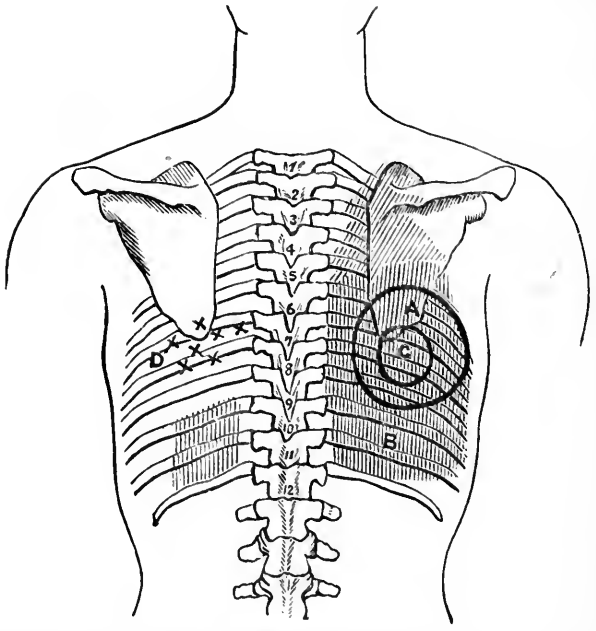
The right posterior base was quite dull on percussion up to the level of the spine of the scapula, above this point a slight degree of resonance was elicited. The superior line of dulness slanted downwards from the scapular spine, towards the lower axillary region, thus marking off the limit of the lower lobe. Over a hand's breadth at the extreme base, posteriorly, the breath sounds were absent, above that level they became tubular, and a point at the level of the seventh dorsal spine, in the line of the angle of the scapula, corresponded with the centre of an area about four inches square, over which the breath sounds were amphoric and attended with gurgle, and the voice sounds pectoriloquous (see Fig. 1). In the upper scapular region the respiration was weak and bronchial, and a few large clicks were audible. On the left side, posteriorly, there was some dulness at the extreme base, and about the angle of the scapula was a small area, over which the breathing was bronchial, and accompanied with some large crackling sounds. The upper part of the lung was clear. The tongue was still much furred, the bowels confined. Urine acid, specific gravity 1020, contained no albumen.

The residue of expectoration, examined on three occasions, after boiling with caustic soda, was found once only to contain some elastic tissue.

September 10th.—The limits of the auscultatory signs of excavation were marked out as indicated in the diagram annexed (Fig. 1), viz. the lower margin of excavation corresponded with the level between eighth and ninth spinous processes. Its diameter measured three and a half inches, the central point being five inches from the spine, and the outer margin reaching the posterior axillary line.

11th. *Operation by Mr. Lyell.*—The patient being placed under the influence of ether was conveyed to the operating theatre. Having been turned well over to the left side so as to expose the right side of the back for operation, a medium sized aspiratory trocar was thrust horizontally inwards, through the eighth intercostal space, immediately above the margin of the ninth rib, in the

FIG. 1.



A. Marks area of different excavation signs.

B. Dulness very marked; breath sounds very feeble and distant.

C. More restricted area over which cavity signs were most distinctly developed.

D. Some large crackles, re-echoed from opposite side.

line of the angle of the scapula; this spot having been previously marked as corresponding to the lowest point of the cavity. The trocar penetrated to a depth of from two to three inches.

On withdrawing the trocar, no fluid followed, and from immobility of the instrument it was considered that the cavity had not been struck, and the canula was accordingly removed. The patient spat up a little sputum brightly tinged with blood, and after coughing, some fine emphysema crackling was apparent in the tissues immediately about the puncture.

The instrument was reintroduced in a similar manner in a somewhat higher point in the same vertical line.

The canula was felt to be grasped as firmly as before, but there was more blood-stained expectoration, and the subcutaneous emphysema was more marked.

After waiting a few moments, without any fluid escaping from the canula, the instrument was thrust somewhat more deeply into the lung, after which a greyish purulent fluid welled sluggishly up through the canula, and clearly demonstrated that the cavity had been reached.

A transverse incision was made for about one inch on either side of the canula through the skin, dividing all the soft parts down to the inner costal wall.

The small canula was now withdrawn, and in its place a largest size hydrocele trocar was inserted, through the canula of which a small dark clot and some fœtid fluid escaped. The puncture wound in the intercostal space was then enlarged by incision, with a probe-pointed bistoury, of the tissues surrounding the trocar. This incision was limited in extent from fear of hæmorrhage, but it was sufficient to permit, on withdrawal of trocar, an india-rubber drainage-tube, of the size of the little finger, to be passed into the cavity of the lung to a depth of three inches from the external surface. The outer extremity of the tube was then split and secured to the margin of the wound by a couple of silk sutures. The skin, which had been drawn somewhat downwards before making the external incision, on being released regained its natural position, and in this way the superficial and deep openings were placed on a somewhat different level.[1]

The wound and drainage-tube were then covered by cotton wool wrung out of solution of carbolic acid, and the dressing secured by a bandage round the chest. During the operation, about two ounces of sanious fœtid sputa were expectorated, but comparatively little escaped by the wound. The hæmorrhage was very trifling. Towards the completion of the operation the patient's respiration became much embarrassed and the features livid, but these symptoms quickly passed off when the anæsthetic was discontinued and he was placed on his back.

Half an hour after the operation a severe rigor occurred,

[1] It was thought at the time that this would be rectified on the patient's arm and shoulder being lowered when he resumed the dorsal posture.

in which the breathing became rapid and the pulse very small and feeble. The patient, however, soon rallied well after an enema of brandy. The wound was dressed on the evening of the operation, and disinfected by the use of the carbolised spray. A moderate quantity of offensive purulent discharge, mixed with blood, was found upon the dressings. Evening temp. 99·6°. Morning temp. 98·4°. Pulse, 100.

12th.—During the night the patient had expectorated about ʒij of muco-purulent sputa, slightly tinged with blood, and there had been a fair but not abundant discharge of pus through the tube. The margins of the wound were somewhat reddened, and the surface had a greyish aspect. The drainage-tube having been withdrawn, the whole surface of the wound was washed over with diluted Condy's fluid, a little of which was allowed to flow into the lung, being expelled again by the spasmodic cough it occasioned. The drainage-tube, having been thoroughly disinfected by carbolic-acid solution, was reinserted without difficulty, and again secured by a suture.

The pus which escaped was thick, yellowish, and very fœtid. To diminish the fœtor, a spray of carbolic acid was employed with the object of some being inhaled through the wound with respiratory movements. Cotton wool, soaked in carbolic lotion, was then again applied, and over and around it a layer of oakum. Evening temp. 100·2°.

13th.—Patient was disturbed by cough during the night, which he attributed to the taste of carbolic acid coming into the throat. The expectoration was, however, very slight. Pulse soft, 80 in the minute. Appetite still bad. Evening temp. 99°. Pulse 112. Ext. Opii, gr. j, o. n.

14th.—Morning temp. 100°. Pulse 72. Slept well and feeling easier. Evening temp. 100·4°.

15th.—Morning temp. 100°. Pulse 96. There was free escape of purulent fluid through the wound and tube, but in no considerable amount. *No expectoration, the profuse and extremely fœtid sputa present before the operation having quite ceased.* The wound still covered with an

adherent grey slough, part of which was removed by scissors. The antiseptic dressings had not materially diminished the fœtor of the discharge, and, owing to distinct darkening of the urine from carbolic-acid absorption, a dressing of thymol solution was substituted. There was some tendency for the discharge to collect in the external wound. The tube was removed and replaced by a new one. Evening temp. 102·4°.

16th.—Morning temp. 97°. Pulse 108. Tongue furred but less so than yesterday. In consequence of the external wound being on a somewhat higher level than that through the intercostal space, there was a tendency for the discharge to collect in a kind of pouch, and the drainage-tube on its escape through the intercostal space was bent at an angle. To remedy this, the patient having been again anæsthetised, an incision was carried through the whole thickness of the tissues of the back from the centre of the original incision, vertically downwards for one inch and a half, so that its lower end was on a level with the opening of the intercostal space. The tip of the little finger could now be easily passed between the ribs into the opening in the lung, and the drainage-tube was again directly inserted and fixed by sutures to the margins of the wound, A small portion of the eighth rib was found denuded. During these procedures the breathing of the patient became alarmingly embarrassed from the combined effects of the anæsthetic and the prone position necessary for the operation. Evening temp. 101°. Pulse 116.

17th.—Patient rallied well from the second operation, the benefits of which were manifest in the freer escape of the discharge from the wound. There was still a complete absence of expectoration by the mouth, and although complaining of dry cough, the patient slept fairly well. Appetite returning. Urine acid, 1025, no albumen.

On the 19th dry absorbent cotton-wool dressings were employed. The temperature had continued low, and the patient's general condition was decidedly improved.

On the 25th exuberant granulations in the wound gave

some trouble, and the channel for the drainage-tube had to be dilated daily with the little finger. The tube had slipped out during the night, and the patient had been disturbed by irritable cough, not attended, however, with any expectoration.

29th.—Breath highly fœtid, although no expectoration.

October 2nd.—Morning temp. 98·4°. Tube again found in dressings. A slight return of the expectoration. A tube provided with a large external india-rubber shield substituted for that before used and retained in position by strapping. It was also now noticed that in introducing the tube its inner extremity appeared to meet with some obstruction, or to abut against a solid tissue, shortly after passing into the lung, so as to prevent its further penetration. The discharge continued to escape readily, although in somewhat lessened quantity.

During the past few days an attempt had been made to reduce the stench of the discharge, and so diminish the fœtor in mouth and breath, by injecting disinfectants into the interior of the cavity. Two ounces of permanganate solution (ʒij Condy's fluid to the ℥j), with m. x of Tinct. Opii, were injected at each dressing. The injection caused a good deal of cough, continuing for some time afterwards. The patient complained that he tasted the injection through the remainder of the day. Evening temp. 99·6°.

3rd.—Drainage-tube kept in position, and no return of expectoration since yesterday morning.

14.—Breath very offensive. Patient had, however, been gaining strength of late, and had sat up for one or two hours each afternoon for the last four or five days. Cough still at times troublesome. Diarrhœa ceased. The discharge through the tube less in quantity and less offensive. The channel had now closely contracted round the drainage-tube, and after removal there was occasionally some difficulty in reinserting the tube. The tube passed also somewhat less deeply into the lung.

20th.—Patient was transferred to the Brompton Hospital on the 16th, and bore the journey well. Discharge

to-day more abundant; cough troublesome; disagreeable taste of Condy in mouth.

3 p.m., note by Dr. Powell. Right chest resonant in front to fifth rib; abdominal resonance extends upwards an inch above cartilage in nipple line; no cardiac dulness. Heart's position judged by stethoscope to be normal, but covered by lung. Exaggerated breathing over left lung. Very weak breath sounds over right lung with bronchial râles. Rough friction in infra-mammary and lower axillary regions. Fœtor with cough very marked. Expectoration scanty and difficult. Sputa purulent. Great difficulty was experienced in maintaining drainage; different tubes were tried but although the channel admitted the large tube now seen in position, the fœtor of breath continued, the cough became troublesome, the expectoration increased to about one ounce daily, and the patient rapidly lost ground until the 29th of October, when he was seized with pleuro-pneumonia on the opposite side, under which he sank on the 31st.

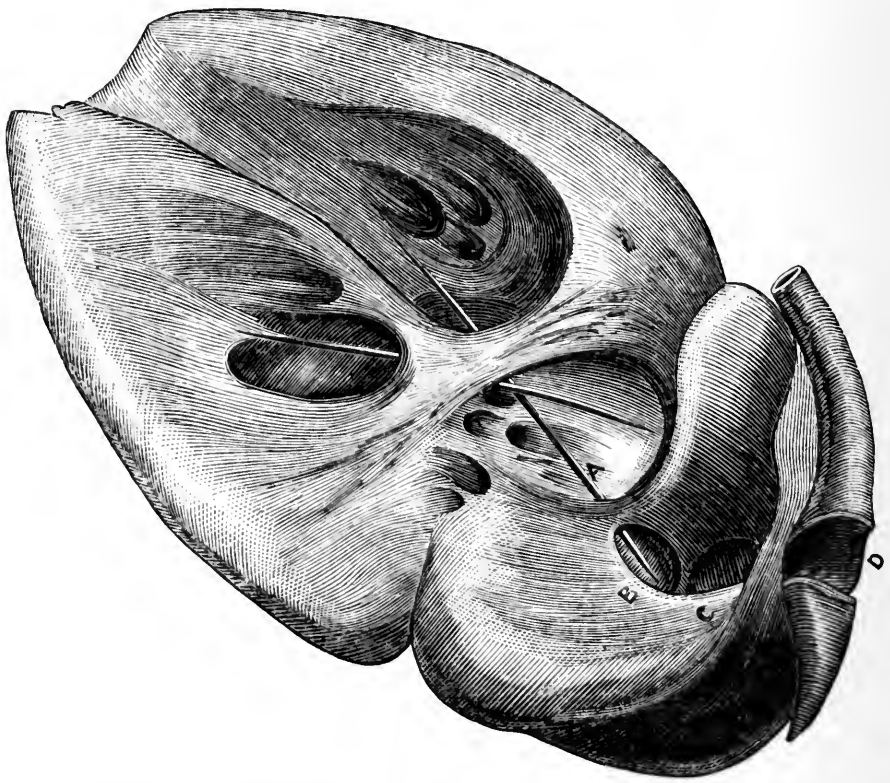
Post-mortem examination (November 1st, 1879).—The position of the opening for the reception of the canula was below the angle of the right scapula in the eighth intercostal space, four inches to the right of the median line. The opening admitted the little finger, and a probe penetrated directly inwards for an inch and a half beyond the surface of the rib. The surface of the eighth rib in the wound was found to be bare and roughened, and some spiculæ of the bone bridged across from one rib to the other on each side of the opening. About four inches of the eighth and ninth ribs were detached by the saw, so as to include the opening between them, and the examination of the chest was then proceeded with.

The left pleura contained about a pint of turbid fluid, with flakes of recent lymph and some recent bands of adhesion. The base of the lung was the seat of broncho-pneumonia.

On the right side the pleura was posteriorly much thickened by œdematous cellular tissue, and the two

surfaces adherent from apex to base. The lung and costal pleura and attached portion of eighth and ninth ribs were removed. On subsequent examination it was found, as will be seen in the annexed drawing, kindly made by Dr. Reginald Thompson, that the channel (Fig. 2 D, B) occupied by the tube traversed the lung in a direction

FIG. 2.



In the above figure, at D, is indicated in section the drainage-tube channel through the thickened intercostal space and pleura above the 9th rib, which is bridged over by a narrow strip of undivided lung, beyond which the remains of a superficial cavity are seen. Continuous with this cavity is seen a dilated bronchus (B), which again communicates with the main cavity (A), and with secondary cavities beyond, as indicated by the bristles.

inwards and slightly downwards for $1\frac{5}{8}$ inches. The portion, about an inch, of this channel, which was intra-pulmonary, had a somewhat corrugated surface and terminated

in a truncated extremity, at the upper portion of which it communicated with another shorter and smaller channel, which was directed obliquely upwards to form with it an elbow-like angle of 50°. This second channel (B), which was $\frac{3}{4}$ inch long and $\frac{3}{10}$ inch in diameter, terminated above in the main cavity. Its smooth lining membrane, and its giving off a small branch, showed it to be a bronchial tube. It terminated by an opening admitting No. 8 catheter in the lowest point of an irregular trabeculated cavity (A), which extended upwards for $2\frac{1}{4}$ inches, and was $1\frac{1}{2}$ inches across, and was connected superiorly, as indicated in the drawing, with several other cavities, with which the whole of the posterior lobe was riddled, and which were occupied with curdled secretion. These secondary cavities had the appearance of intercommunicating bronchiectatic cavities.

The appearance of the inner dilated portion of the channel in which the tube lay justified the belief that it was a cavity outlying from the main cavity (A) of bronchiectatic origin, and which, originally of larger dimensions, had contracted upon the tube. The cavity was situated close under the pleura, and its inner extremity was only separated from the diaphragm by a thickness of $\frac{1}{8}$ inch. The main cavity was situated at a depth of from $\frac{1}{2}$ to $\frac{3}{4}$ inch from the pleural surface and separated from the surface of the diaphragm by one inch only of spongy lung tissue.

The rest of the lower lobe not occupied by cavities was condensed by connective-tissue growth and thickened bronchial and vessel sheaths. The upper lobe of the right lung was emphysematous, with some recent congestion and broncho-pneumonia at the apex, but no cavities.

Remarks.—The operation of tapping a cavity in the base of the lung has been so rarely accomplished that we have not hesitated to bring the present case before the notice of the Fellows of the Society in some detail, as a contribution to experience in the matter. Probably, the

depth of such cavities from the surface and the difficulty of defining their exact seat, as compared with more superficial apex cavities, will account for their having been so rarely dealt with by the surgeon. Within our experience, and so far as we have read, the first instance in which a cavity in the base of the lung, uncomplicated by empyema, has been tapped, occurred in the practice of Dr. Cayley at the Middlesex Hospital, in December, 1878.[1] The case was one of acute pneumonia with gangrenous abscess in the lower lobe of the right lung, in which, after exploratory puncture, a free incision was made through the ninth space into the cavity by Mr. Lawson, four or five ounces of fœtid pus liberated, and a large-sized drainage-tube introduced.

The second case was that related in this paper, and more recently a third case, closely resembling that of Dr. Cayley's, has been reported in the 'Lancet,' by Mr. Solomon Smith, of Halifax,[2] in which a gangrenous abscess in the base of the lung recurring in the course of acute pneumonia was incised. In both Dr. Cayley's and Mr. Smith's cases the patients were already greatly prostrated by acute disease and the gangrenous process, and lived but a few days. Although unsuccessful in saving life, these operations—well planned and admirably performed—seem to have afforded the only chance of recovery.

A fourth case was seen by one of us, in consultation with Dr. Pomian and Dr. Brewer, of Stoke Newington, in July, 1878, in which paracentesis of the lung was performed on August 1st, 1878, by Mr. R. Godlee. In this case, however, there was probably empyema present as a complication, although on the death of the patient, five months later, from disease extending to the other lung, the pleural cavity was closed by spongy adhesion, showing no sign of pyogenic membrane, and a scar on the pleural surface, corresponding with a fibro-caseous centre situated in the base of an indurated lung, were all that remained of what must have been a large abscess.

[1] 'Clinical Society's Transactions,' vol. xii.

[2] 'Lancet,' January 17th, 1880.

Two cases have been reported by Dr. C. T. Williams,[1] in which the base of the chest was tapped for a cavity in the lung. In one of these cases the operation was performed by Mr. Erichsen, with great and long-continued relief to the patient. In both cases, however, the lung disease was complicated by a communicating empyema.

Dr. Barry, in his work on 'The Three Digestions,' published 1743, strongly advocates operative interference in cases of pulmonary abscess, and relates several cases in which the chest had been, under his direction, incised. The boldness and success with which he directed these operations at a time long before the stethoscope was known, must excite our admiration. A careful examination of his account of the cases operated upon, however, renders it extremely probable that they were cases of empyema, and not of pulmonary abscess. Cavities situated at the apex of the lung have been tapped or aspirated on several occasions. A very interesting, and so far as it goes a very successful case, is related by Dr. Hastings and Mr. Storks in the 'London Medical Gazette' for December, 1844, and commented upon by Dr. Hocken in two later numbers of that journal. In this case a large apex cavity on the left side was incised through the second intercostal space, and a drainage-tube introduced. More recently several cases have been reported by Dr. F. Mosler,[2] of Greifswald, Dr. Pepper,[3] of Philadelphia, and others.

Passing, now, to the consideration of the case of basic cavity with which we are especially concerned, the most striking and instructive immediate result of the operation was the almost complete cessation of expectoration and cough, although the amount of discharge from the cavity, when compared with the bulk of the expectoration before the operation was remarkably small, not more than 2 oz.

[1] 'Clinical Society's Transactions,' vol. xii.

[2] 'Ueber locale Behandlung von Lungenkavernen,' von Dr. F. Mosler, Prof. in Greifswald, Berl. Klin. Woch., October, 1873.

[3] 'Philadelphia Medical Times,' March, 1874.

of discharge, as compared with 16 to 20 oz. of previous expectoration. There was no evidence of accumulation of secretion in the lung after the operation, the discharge through the wound, although small, comparatively in amount, was free, and the patient had no difficulty in coughing. But the cough, except when excited by irritating injections and sprays, was almost *nil*.

It would seem, then, that bronchial irritation set up by the passage of the acrid fluids and fœtid gases yielded by the cavities was the source of the greater portion of the expectoration, and when these fluids found another vent the bronchial secretion ceased. The examination of the sputa justified this view, and the conviction that secondary damage in the same and opposite lung was being occasioned by some of the cavity products becoming, in the course of difficult expectoration, inhaled into other parts was the chief reason for performing paracentesis in this case. It is a fact, by no means new to experience, that irritating secretions of pulmonary cavities play an important part in determining in many cases laryngeal ulceration and secondary destructive lung changes. One may often observe post-mortem in cases of phthisis the bronchial tracts highly injected and freely secreting, which lead from cavities which are in a state of activity, and we believe that the present case only illustrates, with unusual clearness, what may be true with regard to most cases of phthisis, viz. that a large proportion of the expectoration arises from bronchial irritation, the degree of which varies according to the acridity and septic qualities of the cavity products, both these qualities being highly marked in the present case.

Dr. Hastings notes the same fact in his case, that immediately after the cavity was tapped cough and expectoration ceased.

The expediency of operating in cases of apex cavity is doubtful. There are many theoretical objections to it, and but little practical experience in its favour.

Apex cavities are always efficiently drained by the

bronchial tubes leading from them at a point or points much lower than any artificial opening could be made. We do not forget the remarkable effect of the operation in ameliorating the symptoms in Dr. Hastings' case, an effect attributed by Dr. Hocken to the cavity being cut off by the operation from performing any part of the respiratory functions (l. c., p. 485). Other cases of operation have not been so successful, and perhaps the best that can be said is that they have been attended with so little trouble as to fully justify the adoption of similar measures in any special cases.

The conditions present in cavities at the base of the lung are very different, however. All secretions from such cavities must, under great difficulties, be projected upwards through the trachea from the depth of the lung. If the cavity be chronic the pleura over the region of the cavity, and probably over the entire lobe affected, are, as a rule, adherent. The cavity walls and surrounding pulmonary tissue are more or less thickened, and indurated; such cavities cannot contract upon their contents, and the escape of fluid from them, not being favoured, as in the case of apex cavities, by gravitation, they are never thoroughly emptied, and sooner or later become fœtid. These adhesions of the pleuræ and dense tissue surroundings of the cavity, however, are circumstances favourable to paracentesis.

We think, therefore, it may be assumed as a sound proposition that chronic basic cavities, and, *à fortiori*, such cavities when sloughing or fœtid, are most suitable for operation, both from the physical conditions and the symptoms they present.

The symptoms of acute abscess or gangrene of the lung are of far greater urgency than those of chronic cases, and our temptation to immediate interference is correspondingly great; but, on the other hand, the dangers of operation are in them very much greater than in chronic cases. Apart from the danger of hæmorrhage very difficult or impossible to control, and the uncertainty of striking

the cavity, there is the danger, from the absence of any strong adhesions, that some of the contents of the cavity may escape into the pleura and a virulent empyema be set up. These considerations and the possible effect of the shock of the operation upon a patient so acutely ill should, save in a few particular instances, turn the balance against operation. It must be remembered, also, that a fair proportion of cases of gangrenous abscess of the lung get well on general stimulating treatment without operation.

In the present case the affected lobe of the lung had evidently become disorganised beyond repair by the extension and multiplication of what may in the first instance have been a single cavity. The failure of other treatment in removing fœtor, lessening expectoration, and improving general health, the continuance of hectic fever, and more especially the signs of extension of damage in the upper part of the same lung, or the signs of broncho-pneumonia on the opposite side, would make one urgent in advising interference. At any time after three or four weeks (judging from other cases of pleurisy) adhesions would be sufficiently firm. In cases of chronic basic cavity, in which gangrene or sloughing supervened, immediate interference would be indicated.

In the present case some doubt was expressed as to whether there was not also empyema present. The absence of displacement of heart, detrusion of liver or enlargement of side, and the low position of the cavity, signs, together with the slanting anterior boundary of dulness in the axilla, rendered the diagnosis of any considerably empyema impossible. The question was, whether there might not be a localised empyema kept partially empty by discharging through a fistulous opening in the lung. The extreme dulness at the base, extending considerably lower than auscultatory signs could be obtained, and the copiousness and fœtor of expectoration favoured such a view. But the presence of flattening rather than bulging of the posterior chest, with retraction rather than planing or distension of the intercostal spaces,

led to the inference that much of this dulness and effacement of breath sounds at the right posterior base was really due to contraction upwards of the base of the lung, uncovering and bringing into contact with the thoracic parietes the posterior curve of the diaphragm and the liver. This view was somewhat startlingly verified *post-mortem*, when it was found that the trocar inserted at the eighth space was barely an inch above the diaphragm, and that the wall of the cavity actually tapped was only separated from the diaphragm by about an eighth of an inch.[1] The expectoration, although very copious, was not suggestive of empyema; the pus expectorated was too freely *mingled*, being, as it were, *encased* with mucus. Upon this latter point, however, much stress could not be laid, since, although thin, diffluent, purulent sputa welling up into the bronchial tubes and expectorated in large quantities at a time, is positively distinctive of empyema with bronchial fistula, still if the fistulous opening be small or sinuous the lung soon becomes damaged secondarily, and the purulent sputa from below more slowly infiltrates its bronchi and cavities and is expectorated mingled with their products. The diagnosis is admittedly very difficult, and yet it is very important that it should if possible be accurately determined in all cases; for whereas in the case of lung cavity our operative measures are directed towards the exact seat of the cavity, in an empyema, on the other hand, our desire is to make the incision at the lowest practicable point of the thorax out of the way of lung signs.

The signs of bronchitis, and even of broncho-pneumonia, from time to time in the left lung increased our anxiety to tap the patient. But at the angle of the left scapula there were signs closely imitating those of a cavity. These signs were not constant, however, and it was suspected that they might be reflected sounds from the cavity on the opposite side. *Post-mortem* examination proved this surmise

[1] It is perhaps well to point out, however, that this cavity had evidently considerably contracted since being tapped.

—founded upon the observations of the same phenomena in other cases—to have been correct.

With regard to the operation, we may observe that the greater thickness of external tissues to be penetrated renders paracentesis of the base of the lung posteriorly more difficult than that of the apex. Other difficulties are met with in retaining the tube and preserving it from pressure during recumbency, but these disadvantages are counterbalanced by the dependent position of the opening facilitating the escape of the contents of the cavity without effort on the part of the patient.

In the present instance the cavity in the lung was opened at its most inferior extremity, chiefly on the ground that the most perfect drainage would be secured. This method of entering the cavity did not answer all expectations, and, indeed, possesses some considerable disadvantages. In the first place, the endeavour to secure the most inferior opening practicable increases the risk of missing the cavity with the trocar at the first exploratory puncture, an accident which happened in this particular instance, and necessitated a reintroduction of the instrument at a somewhat higher level. Secondly, the most inferior extremity of a cavity of similar character to the present is not unfrequently situated at a greater depth from the surface than the centre; before reaching it an increased thickness of lung tissue would have to be penetrated, rendering the operation more difficult, and the risk of hæmorrhage from the lung proportionately greater. Thirdly, the most potent objection of all, and one which in considerable measure neutralises the intention of the operation, is the danger of encroachment upon the artificial opening into the cavity, and a consequent interference with a free escape of its secretions, by contraction and shrinking of the lung tissue after the abscess has been emptied, and when attempts at repair commence. This happened in the present case. During the later weeks it was remarked that the tube did not appear to enter the cavity in the lung with the same freedom as before; its

inner extremity apparently abutted against solid lung tissue, beyond which point no further penetration was possible. This was fully explained at the investigation after death, when it was evident that the large central cavity had been opened at the time of the operation through one of the secondary cavities, and drained through this. As soon as the latter was completely emptied contraction of the walls ensued, and the channel in which the tube was found after death was thus formed. It is readily conceivable that in an irregularly-shaped cavity the more outlying portions could be the first to undergo contraction, and should it have been tapped at one of the points the drainage might in this way be subsequently interfered with.

From these considerations we are inclined to conclude that, in dealing with cavities situated in the lower lobe, the opening should be made, not at its extreme lower end, but rather at a spot where the physical signs point to the greatest extent of excavation and proximity to the surface, for this would not only facilitate the performance of the operation, but also offer the least impediment to a perfect and prolonged drainage.

The method of operating adopted, which is described in detail in the notes of the report of the case, consisted of two parts: a free external incision about three inches long down to the intercostal space, and an opening into the lung cavity through the intervening lung tissue by means of a large trocar and canula, enlarged by a probe-pointed bistoury, and establishing a channel along which the drainage-tube was passed. The external incision was originally horizontal, but was subsequently converted into one of T-shape by a further operation. In operations on the back, where the thickness of tissues down to the thorax is considerable, a free external incision is to be recommended, and if one simply horizontal is found insufficient, a crucial may be employed.

In those cases where the cavity is large and superficial, and where only a thin stratum of lung exists between it and the pleura, no difficulty can be experienced in estab-

lishing a free opening, but according as the cavity is deeply situated, and of small dimensions, the difficulties and dangers of the operation increase—both the difficulty of reaching the cavity and the danger of possible serious hæmorrhage.

With these difficulties in view we deemed it more prudent to adopt the method of puncturing the lung tissue, by means of a large trocar and canula, and the result justified the means employed, an amply sufficient channel being afforded for the passage of a large drainage-tube, and that without the occurrence of any hæmorrhage. This method, however, differs in its performance from that adopted by other operators. In a case, in which the operation was performed by Mr. Storks, and which is reported in the 'Medical Gazette,' for 1844, and also in the case of Dr. Cayley's, the opening into the cavity was effected by the use of the scalpel, whilst in that related by Professor Mosler the opening in the intercostal space was enlarged, the lung penetrated, and the cavity opened by dressing forceps, in a manner resembling the opening of a deep abscess with the same instrument.

The operation by Mr. Storks is described in the following terms:—A vertical incision was made from the middle of clavicle downwards, laying bare the third intercostal space. A fine hydrocele trocar was then thrust into the cavity, and it having been ascertained that air escaped through the canula, a scalpel was passed obliquely upwards into the cavity; a probe being passed along this it was withdrawn, and the wall of the cavity was then divided to the extent of an inch with a probe-pointed bistoury, the wall being extremely dense and almost cartilaginous. Air and blood immediately escaped from the aperture, and the patient spat up about three drachms of blood. On the third day a piece of gum-elastic catheter was introduced, projecting externally about a quarter of an inch, and retained by a ligature passed round the end and secured by strapping.

In a subsequent case related by Mr. Storks, in which

he performed a similar operation, the external incision was made along the upper border of the rib, and about two inches long, with a narrow straight bistoury, and this instrument pushed through the intercostal muscles, and cautiously inwards into the cavity, and the incision prolonged to the extent of an inch. He also states that the opening should be made of sufficient size to admit a tube of half an inch diameter.

The operation described by Professor Mosler is as follows:—Along the upper border of the third rib, about $5\frac{1}{2}$ cm. from the sternal margin, an incision 3 cm. long was made, dividing skin and superior intercostal muscle, after a firm pleural adhesion had been assured by the duration of the disease. The intercostal wound was dilated with dressing forceps, and the abscess cavity then gradually opened by thrusting these into the lung, to an increasing depth until a rush with inspiration and escape of purulent fluid mixed with air demonstrated its opening. There was no hæmorrhage. A silver drainage-tube was inserted and fastened by strapping. In all these cases (with the exception of Dr. Cayley's) the abscess cavity was situated in the upper lobe, and the opening was an anterior one, and the depth from the surface was probably not great. The operation in all was perfectly successful in its performance, but, where the cavity is situated at the base of the lung and deeply in its substance, we prefer the method adopted by us in the present case. The subsequent treatment of the patient was directed chiefly to maintaining as free a discharge as possible through the tube, and the disinfection of its cavity in order to reduce the intense fœtor of its discharge. We were but partially successful in these respects, and did not find antiseptic injections were well tolerated by the patient.

The external dressing should be as light as possible; a layer of dry absorbing cotton wool rendered antiseptic, applied loosely over the drainage-tube and frequently changed, we found to be the simplest, and at the same time the most efficacious dressing.

INDEX

These Indices to the annual volumes are made on the same principle as, and are in continuation of, the General Index to the first fifty-three volumes of the 'Transactions.' They are inserted, as soon as printed, in the Library copy, where the entire Index to the current date may always be consulted.

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