PROCEEDINGS

OF

THE MEDICAL SOCIETY

OF

LONDON.

VOLUME THE SEVENTH.

EDITED BY

ALFRED PEARCE GOULD, M.S.,

AND

JAMES KINGSTON FOWLER, M.D.

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1884.

ADVERTISEMENT.

The present volume comprises the Proceedings of the Society during its One hundred and eleventh Session, from October the 29th, 1883, to April the 28th, 1884.

The communications are published irrespective of their having appeared in print elsewhere. The Council deems it right to state that the authors of the various communications and remarks alone are responsible for the opinions with which their names are respectively associated.

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FOR MARCH, 1885.

"ON THE PATHOLOGY AND SURGICAL TREATMENT OF JOINTS STIFFENED BY INJURY, OR OTHER CAUSES."

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Each dissertation shall be written in English or Latin, and shall be delivered to the Registrar, on or before the 1st day of November, in the year subsequent to that in which the subject is announced. With the Dissertation shall be delivered a sealed packet, having on the outside a motto or device, and within the author's name and designation, the same motto or device shall be written on the dissertation.

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1847 Silas Stedman.
1849 John Milligan.
1851 Richard Payne Cotton, M.D.
1851 Richard Hodges.
1852 Frederick William Headland.
1853 Alfred William Poland.
1854 Benjamin Ward Richardson, M.D.
1856 William Burke Ryan.
1857 Edwin Canton.
1858 Thomas Herbert Barker, M.D.
1859 Alderman Thomas Houghton Waters.
1868 John Clay.
1870 Thomas Smith Clouston, M.D.
1872 Edwards Crisp, M.D.
1873 John Kent Spender, M.D.
1877 Peter Murray Braidwood, M.D.
1878 John Milner Fothergill, M.D.
1882 Thomas Michael Dolan.
1883 Norman Porritt.

THE HONORARY FELLOWS.

1881 Bamberger, Henry, M.D., Professor of Medicine, University of Vienna.

1878 Barker, Fordyce, M.D., 85, Madison-avenue, New York, Professor of Clinical Midwifery and Diseases of Women at the Bellevue Hospital Medical College.

1876 Barnes, J. M., M.D., Surgeon-General U.S. Army, Washington.

1837 Bennett, Sir James Risdon, LL.D., M.D., F.R.S., 22, Cavendish-square, W., late President of the Royal College of Physicians; Consulting Physician to St. Thomas's Hospital and to the Victoria-park Hospital. P 2, VP 2, C 7, O, S 3, FM.

1881 Billings, John S., M.D., Washington, Surgeon to the United States Army; Librarian of the Surgeon-General's Library, Washington.

1881 Bigelow, Henry J., M.D., 52, Beacon Street, Boston, U.S.A., late Professor of Surgery at the University of Harvard.

1881 Billroth, Theodore, M.D., Professor of Surgery, University of Vienna.

1873 Burrows, Sir George, Bart., M.D., F.R.S., 18, Cavendish-square, W., Physician to in Ordinary H.M. the Queen; late President of the Royal College of Physicians; Consulting Physician to St. Bartholomew's Hospital.

1881 Charcot, Professor J. M., M.D., 17, Quai Malaquais, Paris.

1873 Chauveau, Professor, Lyons.

1881 Da Costa, J. M., M.D., Professor of Medicine in the Jefferson Medical College, Philadelphia.

1881 Emmet, Thomas Addis, M.D., 89, Madison Avenue, Surgeon to the Woman's Hospital of the State of New York.

1873 Farre, Arthur, M.D., F.R.S., 18, Albert Mansions, S.W., Physician-Extraordinary to H.M. the Queen; Physician-Accoucheur to H.R.H. the Princess of Wales and H.I.R.H. the Duchess of Edinburgh.

1876 Flint, Austin, M.D., Professor of Medicine in the Bellevue Hospital Medical College, New York, U.S.A.

1873 Gueneau de Mussy, Noel, M.D., No. 4, Rue St. Arnaud, Paris, Member of the Academy of Medicine; Physician to the Hôtel Dieu.

1881 Halla, Joseph, Professor of Medicine in the University of Prague.

1869 Hare, Charles John, M.D., Manchester-square, W., Emeritus Professor of Clinical Medicine in University College; Consulting Physician to University College Hospital. P, VP 2, C 8, LL. (*Trustee.*)

1873 Helmholtz, Hermann Ludwig Ferdinand, M.D., Professor of Physics and Physiological Optics in the University of Berlin.

1883 Humphry, George Murray, M.D., F.R.S., Professor of Surgery in the University of Cambridge. *Orator.*

1873 Huxley, Thomas Henry, LL.D., Pres. R.S., 4, Marlborough-place, St. John's Wood, N.W., Professor of Biology in the Normal School of Science, and in the Royal School of Mines.

1875 Jenner, Sir William, Bart., K.C.B., D.C.L., LL.D., M.D., F.R.S., 63, Brook-street, W., Physician-in-Ordinary to H.M. the Queen and to H.R.H. the Prince of Wales; President of the Royal College of Physicians; Emeritus Professor of Clinical Medicine in University College, London; Consulting Physician to University College Hospital.

1843 Johnstone, Henry James Wolfenden, High View, St. Lawrence, Thanet, formerly Senior Assistant Surgeon and Lecturer on Anatomy and Physiology, St. George's Hospital.

1873 Langenbeck, Bernhard von, M.D., late Professor of Surgery in the University of Berlin.

1884 Larrey, Baron, M.D., Paris.

1883 Le Roy de Méricourt, A., M.D., Paris.

1876 Mercier, Louis Auguste, M.D., Paris.

1832 Mitchell, S. Weir, M.D., Walnut-street, Philadelphia.

1881 Nussbaum, John Nepomuk Ritter von, M.D., Professor of Surgery in the University of Munich.

1875 Ollier, Professor, Lyons.

1873 Owen, Sir Richard, K.C.B., F.R.S., Sheen Lodge, Richmond Park, Superintendent of the Natural History Departments of the British Museum.

1873 Paget, Sir James, Bart., D.C.L., LL.D., F.R.S., 1, Harewood-place, Hanover-square, W., Serjeant-Surgeon to H.M. the Queen; Surgeon to H.R.H. the Prince of Wales; Consulting Surgeon to St. Bartholomew's Hospital.

1883 Paget, George Edward, D.C.L., L.L.D., M.D., F.R.S., Regius Professor of Physic in the University of Cambridge.

1876 Pancoast, Joseph, M.D., 1030, Chesnut-street, Philadelphia, Professor of Anatomy in the Jeffreson Medical College.

1837 Quain, Richard, F.R.S., 32, Cavendish-square, W., Surgeon-Extraordinary to the Queen; Emeritus Professor of Clinical Surgery in University College, and Consulting Surgeon to University College Hospital. VP, C.

1877 Sanne, A., 11, Rue Cambacérès, Paris.

1873 Scanzoni, Friedrich Wilhelm von, M.D., Royal Bavarian Privy Councillor, and Professor of Medicine in the University of Wurzburg.

1835 Seaone, M., M.D., Salamanca.

1835 Smith, John Gregory, F.R.C.S., 23, Gloucester-place, Greenwich, S.E. VP, C 4.

1881 Tarnier, Stephanie, M.D., Professor of Obstetric Medicine in the School of Medicine, Paris.

1873 Tyndall, John, F.R.S., Professor of Natural Philosophy in the Royal Institution.

1881 Verneuil, Auguste Aristide, M.D., Professor of Medicine in the School of Medicine, Paris.

1873 Virchow, Rudolph, M.D., Professor of Pathological Anatomy in the University of Berlin.

1881 Volkmann, Richard, M.D., Professor of Surgery in the University of Halle.

1838 Williams, Charles James Blasius, M.D., F.R.S., 47, Upper Brook-street, W., Physician-Extraordinary to H.M. the Queen; Emeritus Professor of Medicine and Clinical Medicine in University College; Consulting Physician to University College Hospital and to the Brompton Hospital for Consumption.

CORRESPONDING FELLOWS.

1851 Albaro, J. Mendez, Madrid.
1861 Alvarenga, Pedro Francisco da Costa, M.D., Lisbon.
1882 Badaloni, Giuseppe, M.D., Frosinone, Prov. di Roma, Italy.
1856 Baker, Albert, M.D., Dawlish, Devonshire.
1855 Beardsley, Amos, Bay Villa, Grange, Lancashire.
1850 Benavente, Mariano, Madrid.
Beneke, F. W., M.D., New York.
1850 Böhm, Professor, M.D., Vienna.
Bottani, Giuseppe, M.D., Milan.
1865 Braun, Carl, M.D., Professor of Midwifery in the University of Vienna.
1837 Bühring, J. J., M.D., Berlin.
1874 Burness, Alexander George, M.D., Port Elizabeth, Cape of Good Hope.
Cade, Thomas Charles, Spondon, Derby.
1855 Clark, Alfred, Twickenham.
1855 Coates, Charles, M.D., F.R.C.P., 10, Circus, Bath, Consulting Physician to the Bath Royal United Hospital. c 3.
1850 Cox, William Isidore, Hawkesbury Upton, Gloucestershire. c.
Curtis, George, Dorking.
1876 Dawes, Richard St. Mark, Gawler, South Australia.
De Muynck, J., M.D., Ghent.
1865 Diday, Paul, M.D., Lyons.
1836 Ecstein, Sigismund, M.D., Vienna.
Eylandt, Johann Emil, M.D., Curland, Russia.
1853 Fallot, R., M.D., St. Laurant d'Aigonze, Montpelier, France
1864 Fideli, G., M.D., Rome.
1876 Griffith, Richard Glyn, Dinapore, India.
1864 Hasenfeld, Emmanuel, M.D., Pesth.
1867 Hughes, John Stannus, M.D., Dublin, Professor of Surgery in the Royal College of Surgeons of Ireland.
Hyman, —, M.D., Antwerp.
1851 Izguierdo, Sebastian Obtega, Madrid.
1875 Jones, Philip Sydney, M.D., F.R.C.S., Examiner in Medicine in the University of Sydney, Australia, Hon. Consulting Surgeon to the Sydney Infirmary.

1861 Journez, Henri, M.D., 43, Rue de la Charité, Bruxelles, Belgique.
1852 Junod, Theodore, M.D., Geneva.
Kitching, George, M.D., Enfield.
1851 Kölliker, Albert, M.D., Professor of Anatomy and Physiology at the University of Wurzburg.
Lax, William, Ormskirk, Lancashire.
1876 Leighton, Walter H., M.D., Lowell, Massachusetts, U.S.
Leon, Jose, Madrid.
1851 Llanos, Antonio Campo, Madrid.
Lovera, Jose, Madrid.
Marino, Bonifacio Matreos, Madrid.
Mendez, Bartholome, Madrid.
Molina, M. M., Madrid.
Negri, Gaetano, M.D., Pisa.
Ortega, J. R., Madrid.
1865 Peruzzi, Domenico, M.D., Sinegaglia.
1882 Restrepo, Alessando Eduardo, M.D., Medillin, Columbia, U.S.A.
1860 Roussel, M.D., Dean of the Faculty of Medicine, Montpelier.
Scharla, Gus. W., M.D., Stettin, Prussia.
1876 Schmitz, Richard, M.D., Neuenahr.
1874 Schutgowsky, J., St. Petersburg.
1852 Seaton, Joseph, M.D., Halliford House, Sunbury.
1851 Sesse, M., Mesqui, Madrid.
Stockwell, Thomas Goldesbrough, F.R.C.S., 9, Alfred-street, Bath, Surgeon to the Bath Royal United Hospital.
Tereza, Felix Garcia, Madrid.
Valdez, Franco Cortigo, Madrid.
Williams, Charles, F.R.C.S. Edin., 9, Prince of Wales-road, Norwich; Surgeon to the Norfolk and Norwich Hospital.
Wilson, Charles, M.D., Dalrymple-crescent, Grange, Edinburgh.

THE FELLOWS

OF

THE MEDICAL SOCIETY OF LONDON.

Explanation of Abbreviations.

P.—President.	LL.—Lettsomian Lecturer.
VP.—Vice-President.	FM.—Fothergillian Gold Medallist.
T.—Treasurer.	SM.—Silver Medallist.
L.—Librarian.	O.—Orator.
S.—Secretary.	§—Sec. for Foreign Correspondence.
C.—Councillor.	*—Life Members.

NS.—Non-subscribing.

The number prefixed signifies the date of election. The figures appended indicate the number of Sessions served, and refer to past appointments ONLY.

1883 Acland, Theodore Dyke, M.A., M.B., 79, Lambeth Palace-road, S.E., Demonstrator of Practical Physiology and Morbid Histology at St. Thomas's Hospital.

1878 Adams, Josiah Oake, M.D., F.R.C.S., Brook House, Upper Clapton, E.

1852 *Adams, William, F.R.C.S., 5, Henrietta-street, Cavendish-square, W., Surgeon to the Great Northern Hospital. P, C 8, O, VP 3, LL.

1868 Aitken, William, M.D., F.R.S., Woolston, Southampton, Professor of Pathology, Army Medical School, Netley. NS.

1878 Allchin, William Henry, M.B., F.R.C.P., F.R.S.E., 5, Chandos-street, Cavendish-square, W., Physician to, and Lecturer on Medicine at, Westminster Hospital. *Librarian.*

1873 Allen, Henry Marcus, F.R.C.P. Edin., 20, Regency-square, Brighton.

1873 Allfrey, Charles Henry, M.D., F.R.C.S., Sanit. Sci. Certif. Camb., St. Mary Cray, Kent.

1883 Allingham, Herbeet, W., 25, Grosvenor-street, W.

1872 Allingham, William, F.R.C.S., 25, Grosvenor-street, Grosvenor-square, W., Surgeon to St. Mark's Hospital. C.

1860 Althaus, Julius, M.D., M.R.C.P., Knt. Ord. Crown of Italy, 48, Harley-street, W., Senior Physician to the Hospital for Epilepsy and Paralysis, Regent's Park. C 5, §.

1871 Ambler, Vincent, Colville House, Colville-square, Bayswater, W.
1869 Armitage, Samuel Harris Tatham, M.D., 23, Brook-street, W.
1868 Armstrong, John, M.D., Dartford, Kent.
1868 Armstrong, John Christopher, 196, Parrock-street, Gravesend. NS.
1871 Arnold, Wilberforce, J.P., Crescent House, Belfast, Physician to the Rescue Hospital, Belfast. NS.
1882 Ashton, Charles Ernest, General Hospital, Wolverhampton.
1873 Atkinson, Edward, 36, Albion-street, Leeds, Surgeon to the Leeds General Infirmary; Lecturer on Surgery in the Leeds School of Medicine.
1872 Aveling, James Hobson, M.D., 1, Upper Wimpole-street, W., Senior Physician to the Chelsea Hospital for Women.

1873 Bagshawe, Frederic, M.A., M.D., F.R.C.P., 5, Warrior-square, St. Leonards, Physician to the East Sussex Infirmary.
1871 Bailey, George Hewlett, 9, Cavendish-place, W., Chloroformist to the Charing Cross Hospital and to the Dental Hospital.
1873 Bailey, James Johnson, M.D., F.R.C.S., Marple, Cheshire.
1878 Baker, Henry Francis, F.R.C.S. Edin., 15, Hanover-square, W. C.
1878 Baldock, Alfred, M.B., C.M., 180, Earl's Court-road, S.W.
1879 Balkwill, William Edward, 9, Old Cavendish-street, Cavendish-square, W., Surgeon to the Royal Orthopædic Hospital.
1881 Ballance, Charles Alfred, M.B., M.S., F.R.C.S., 56, Harley-street, W., Assistant Surgeon to the West London Hospital; Demonstrator of Anatomy at St. Thomas' Hospital.
1859 Barnes, John Wickham, F.R.C.S., 3, Bolt-court, E.C. VP, C 3, S 2.
1883 Barnes, Robert, M.D., F.R.C.P., 15, Harley-street, W., Obstetric Physician to, and Lecturer on, Midwifery and Diseases of Women at St. George's Hospital.
1876 Barnes, Robert Sydenham Fancourt, M.D., C.M., M.R.C.P., 7, Queen Anne-street, W., Physician to the Chelsea Hospital for Women; Physician to the British Lying-in Hospital; Assistant Obstetric Physician to the Great Northern Hospital. C.
1874 Barrett, Howard, 3, Tavistock-square, W.C.
1868 Bateman, Frederic, M.D., F.R.C.P., J.P., Upper St. Giles-street, Norwich, Physician to the Norwich and Norfolk Hospital. NS.
1882 Beach, Fletcher, M.B., M.R.C.P., Medical Superintendent to Darenth Asylum, Dartford, Kent.
1868 Beatty, Thomas Carlyle, Seaham Harbour, Durham. NS.
1880 Beevor, Charles Edward, M.D., M.R.C.P., 33, Harley-street, W., Assistant Physician to the National Hospital for the Paralysed and Epileptic.
1868 Bell, The Rev. David, M.A., M.D., C.M., Goole, Yorkshire. C 3.
1867 Bell, Hutchinson Royes, F.R.C.S., 12, Queen Anne-street, W., Surgeon to, and Demonstrator of Operative Surgery at, King's College Hospital. VP, C 3, LS, LL, SM.
1872 Bell, John Hougham, M.D., Downside, Ventnor, Isle of Wight. NS.

1881 BENNET, ROBERT, M.D., Tankerville House, Park-place, Buxton, Senior Physician to the Devonshire Hospital; Coroner for High and Low Peak.

1883 BENNETT, WILLIAM HENRY, F.R.C.S., 1, Chesterfield-street, Mayfair, W., Assistant Surgeon to, and Lecturer on Anatomy at, St. George's Hospital.

1878 BENTON, SAMUEL, 2, Bennett-street, St. James, S.W.; Surgeon to the North West London Hospital. *Councillor.*

1873 BEVERIDGE, JAMES SPOWART, M.R.C.P. Edin., 8, Eildon-street, Edinburgh.

1879 BINDON, WILLIAM JOHN VEREKER, M.D., D.Sc., C.M., *Travelling.*

1868 BIRD, GEORGE, M.D., 49, Welbeck-street.

1850 *BIRKETT, JOHN, F.R.C.S., 59, Green-street, Grosvenor-square, W., Consulting Surgeon to Guy's Hospital. VP, C 6.

1883 BISS, CECIL YATES, M.A., M.B., M.R.C.P., 65, Harley-street, W., Assistant Physician to the Middlesex Hospital, and to the Hospital for Consumption, Brompton; Lecturer on Botany at the Middlesex Hospital.

1881 BLACK, JAMES, B.A., F.R.C.S., 16, Wimpole-street, W., Lecturer on Anatomy to the Westminster Hospital.

1881 BLAKER, WALTER CAMPBELL, Lyndhurst, Hants.

1868 BLOWER, WILLIAM, Bedford. NS.

1871 BLOXAM, JOHN ASTLEY, F.R.C.S., 8, George-street, Hanover-square, W., Surgeon to the Charing Cross Hospital, and to the Lock Hospital. S 2, C 2.

1867 BOND, THOMAS, M.B., B.S., F.R.C.S., 7, The Sanctuary, Westminster, S.W., Assistant Surgeon to, and Lecturer on Forensic Medicine at, Westminster Hospital.

1883 BOON, ALFRED, Basse Terre, St. Kitts, W.I.

1871 BOTHWELL, GEORGE GRANVILLE, Topsham, Devon.

1879 BOTT, HENRY, Brentford.

1872 BOULTON, PERCY, M.D., M.R.C.P., 6, Seymour-street, Portman-square, W., Physician to the Samaritan Hospital. C.

1883 BRADSHAW, JAMES DIXON, M.A., M.B., M.R.C.P., 30, George-street, Hanover-square, W.

1868 BRADY, JOHN, D.L., M.R.C.P., Hon. F R.C.S.I., Bridport House, Rugby.

1868 BRAIDWOOD, PETER MURRAY, M.D., 2, Delamere-terrace, Birkenhead, Examiner in the University of Edinburgh. FM 1877, NS.

1884 BRAILEY, WILLIAM ARTHUR, M.A., M.D., 16, Orchard-street, Portman-square, Assistant Ophthalmic Surgeon to Guy's Hospital.

1869 BRAINE, FRANCIS WOODHOUSE, F.R.C.S., 56, Maddox-street, W., Lecturer on Anæsthetics to Charing Cross Hospital. VP 2, S 2, C 3, SM.

1876 BREWER, ALEXANDER HAMPTON, 136, Richmond-road, Dalston, E.

1873 BRIDGWATER, THOMAS, M.B., Harrow.

1862 BROADBENT, WILLIAM HENRY, M.D., F.R.C.P., 34, Seymour-street, Portman-square, W., Physician to, and Lecturer on Medicine at, St. Mary's Hospital. P, VP, O, LL, C 4.

1879 Brookfield, John Storrs, B.A., M.D., 2, Devonshire Villas, Brondesbury, N.W.

1878 Brooks, Job Edwin, 54, Mill-street, Ludlow, Salop.

1878 Brown, Andrew, M.D., Elton-villa, 1, Bartholomew-road, Kentish Town, N.W.

1882 Brown, Alexander Stewart, Beaconsfield Club, W.

1871 Brown, John, Belmont Lodge, St. John's-hill, New Wandsworth, S.W.

1871 Browne, James Crichton, LL.D., M.D., F.R.S., 7, Cumberland-terrace, Regent's-park, N.W., Lord Chancellor's Visitor. c.

1873 Browne, Lennox, 36, Weymouth-street, W., Senior Surgeon to the Central London Throat and Ear Hospital.

1873 Brunjes, Martin, 9, York Street, Portman-square, W.

1861 Brunton, John, M.A., M.D., 21, Euston-road, N.W., Examiner in Midwifery and Forensic Medicine in the University of Glasgow; Surgeon to the Royal Maternity Charity. c, 2, *Vice-President.*

1874 *Brunton, Thomas Lauder, M.D., D.Sc., F.R.C.P., F.R.S., 50, Welbeck-street, W., Assistant Physician to, and Lecturer on Materia Medica at, St. Bartholomew's Hospital; Examiner in Materia Medica in the University of London. *Councillor. Lettsomian Lecturer.*

1850 Bryant, Thomas, F.R.C.S., 53, Upper Brook-street, W., Surgeon to, and Lecturer on Surgery at, Guy's Hospital. p, vp 2, ll, s 2, c 4. *Trustee.*

1848 Bryant, Walter John, F.R.C.S., 23a, Sussex-square, W.

1858 Buchanan, George, M.D., F.R.C.P., F.R.S., 24, Nottingham-place, W.; Medical Officer to the Local Government Board. p, ll, vp, o, c 3.

1868 Buckle, Fleetwood, M.D., Staff Surgeon R.N. ns.

1883 Bull, William Henry, St. Oswald's House, Stony Stratford.

1884 Bullock, Joseph Ernest, M.D., 87, Ladbroke-grove, W.

1873 Bunny, Joseph, M.D., Newbury, Berks.

1872 Burger, Alexander, M.D., 49, Finsbury-square, E.C., Honorary Surgeon to the German Hospital.

1850 Burnie, William, M.D., Houghton House, Bradford. ns.

1872 Byas, Edward Hegley, Grove Hall, Bow.

1850 *Camps, William, M.D., F.R.C.S., 53, Radnor-street, Chelsea, S.W. c 5.

1839 Canton, Edwin, F.R.C.S., 30, Montague-place, Russell-square, W.C., Consulting Surgeon to Charing Cross Hospital. p, vp 2, o, s, c 6, fm 1857.

1869 Carpenter, Alfred, M.D., J.P., 5, Grosvenor-street, W. o, c 3.

1882 Carpenter, Arthur Bristowe, M.A., M.B., Croydon.

1871 Carter, Robert Brudenell, F.R.C.S., 27, Queen Anne-street, W., Ophthalmic Surgeon to, and Lecturer on Ophthalmic Surgery at, St. George's Hospital. vp, o, ll. *Councillor.*

1848 Cartwright, Samuel, F.R.C.S., 32, Old Burlington-street, W., late Professor of Dental Surgery in King's College; Consulting Surgeon to the Dental Hospital. *Councillor.*

1876 CARTWRIGHT, S. HAMILTON, 32, Old Burlington-street, W., Professor of Dental Surgery in King's College and Dental Surgeon to the Hospital.

1881. CASE, PERKINS WILLIAM PERKINS, M.B., C.M., Queen's-road, West Croydon, Surrey.

1878 CASSIDY, JOSEPH LAMONT, M.D., 82, Guildford-street, W.C., Assistant Physician to the Hospital for Consumption, Hampstead.

1876 CATHCART, SAMUEL, M.R.C.P. Edin., Prudhoe House, High-road, Tottenham, E.

1882 CAVAFY, JOHN, M.D., F.R.C.P., 2, Upper Berkeley-street, W., Physician to St. George's Hospital. *Councillor.*

1867 CHAPMAN, JOHN, M.D., 224, Rue de Rivoli, Paris.

1868 CHESSALL, WILLIAM, M.D., Horley, Surrey. NS.

1868 CHILD, EDWIN, New Malden, Surrey. NS.

1877 *CHISHOLM, EDWIN, M.D., Sydney, New South Wales.

1861 CHOLMELEY, WILLIAM, M.D., F.R.C.P., 63, Grosvenor-street, W., Senior Physician to the Great Northern Hospital. VP, O, C 5, SM.

1870 CHRISTIE, THOMAS BEATH, M.D., F.R.C.P., F.R.S.E., Medical Superintendent, Royal India Asylum, Ealing, W.

1871 CHURTON, THOMAS, M.D., 35, Park-square, Leeds, Physician to the Leeds General Infirmary, and Lecturer on Clinical Medicine and Materia Medica in the Leeds School of Medicine.

1854 CLARK, Sir ANDREW, Bart., LL.D., M.D., F.R.C.P., 16, Cavendish-square, W., Physician to, and Lecturer on Clinical Medicine at, the London Hospital. P, VP, LL, C 5, § 6.

1875 CLARK, ANDREW, F.R.C.S., 19, Cavendish-place, W., Assistant Surgeon to, and Lecturer on Practical Surgery at, the Middlesex Hospital.

1883 CLARK, WILLIAM BRUCE, M.A., M.B., F.R.C.S., 46, Harley-street, W., Assistant Surgeon to the West London Hospital and Demonstrator of Anatomy at St. Bartholomew's Hospital.

1873 CLARKE, THOMAS KILNER, M.A., M.D., F.R.C.S., 66, John William-street, Huddersfield, Surgeon to the Huddersfield Infirmary.

1870 CLOUSTON, THOMAS SMITH, M.D., Superintendent Royal Asylum, Morningside, Edinburgh; Lecturer on Mental Diseases in the University of Edinburgh. FM. 1870. NS.

1873 CLUBBE, WILLIAM HENCHMAN, Grove House, Lowestoft, Surgeon to the Lowestoft Infirmary.

1879 *CLUTTON, HENRY HUGH, M.A., M.B., F.R.C.S., 2, Portland Place, W., Assistant Surgeon and Aural Surgeon and Lecturer on Forensic Medicine to St. Thomas' Hospital.

1869 COATES, WILLIAM MARTIN, F.R.C.S., Salisbury, Surgeon to the Salisbury Infirmary.

1849 COCKLE, JOHN, M.A., M.D., F.R.C.P., F.R.C.S., 13, Spring-gardens, S.W., Senior Physician to the Royal Free Hospital. P, VP, O, L 3, C 3, SM.

1848 COGSWELL, CHARLES, M.D., F.L.S., 47, York-terrace, Regent's-park, N.W. S 4. *Trustee.*

1872 Coles, J. Oakley, 18, Wimpole-street, W., Lecturer on Dental Surgery at the National Dental College; Dental Surgeon to the National Dental Hospital.

1853 Collambell, Charles, F.R.C.S., J.P., The Terrace, 148, Lambeth-road, S.E.

1883 Compton, Francis Charles, 38, Hans-place, Sloane-square, S.W., Microscopical Pathologist and Lecturer on Histology at St. George's Hospital.

1871 Cook, John, M.D., 1, Nottingham-terrace, Regent's-park, N.W., Physician to the Great Northern Hospital.

1862 Cooper, Alfred, F.R.C.S., 9, Henrietta-street, Cavendish-square, W., Senior Surgeon to the West London Hospital and Surgeon to the Lock Hospital. c 3. *Vice-President.*

1873 Cooper, Sir Henry, M.D., F.R.C.P., F.R.C.S., 5, The Avenue, Upper Norwood, S.E., Consulting Physician to the Hull General Infirmary. c 2.

1872 Corfield, William Henry, M.A., M.D., F.R.C.P., 10, Bolton-row, Mayfair, Professor of Hygiene in University College; Medical Officer of Health for St. George's, Hanover-square. c.

1861 Coulson, Walter John, F.R.C.S., 17, Harley-street, W., Senior Surgeon to the Lock Hospital and to St. Peter's Hospital. o, s 2, c 4.

1879 Coupland, Sidney, M.D., F.R.C.P., 14, Weymouth-street, W., Physician to, and Lecturer on Pathological Anatomy at, the Middlesex Hospital; Examiner in Pathology in the University of Edinburgh. c.

1883 Coxwell, Charles Fillingham, M.A., M.B., M.R.C.P., Medical Registrar to the London Hospital; Assistant Physician to the Royal Hospital for Diseases of the Chest.

1874 Craigie, John Hamilton, 13, Savile-row, W., Surgeon Dentist to the Chelsea Hospital for Women. c.

1873 Craven, Robert Martin, J.P., 14, Albion-street, Hull, Surgeon to the Hull General Infirmary.

1881 Cripps, William Harrison, F.R.C.S., 2, Stratford-place, W., Assistant Surgeon and Surgical Registrar to St. Bartholomew's Hospital.

1880 Critchett, George Anderson, M.A., 21, Harley-street, W., Ophthalmic Surgeon, and Lecturer on Ophthalmic Surgery, to St. Mary's Hospital.

1880 Crocker, Henry Radcliffe, M.D., B.S., M.R.C.P., 28, Welbeck-street, W., Physician to the Skin Department at University College Hospital. *Councillor.*

1881 Cross, Francis Richardson, M.B., F.R.C.S., Chandos Villa, Clifton, Bristol, Surgeon to the Bristol Royal Infirmary; Lecturer on Anatomy in the Bristol Medical School.

1855 Cross, Robert, M.D., 42, Craven-street, Strand, W.

1881 Cullimore, Daniel Henry, M.D., F.R.C.S.I., 27, Welbeck-street, W., Senior Physician to the North-West London Hospital.

1874 Cumberbatch, Alphonso Elkin, M.B., F.R.C.S., 17, Queen Anne-street, W., Aural Surgeon to St. Bartholomew's Hospital. c 2.

1871 DALBY, WILLIAM BARTLETT, B.A., M.B., F.R.C.S., 18, Savile-row, W., Aural Surgeon to, and Lecturer on Aural Surgery at, St. George's Hospital. C.

1864 DALE, GEORGE CORNELIUS, M.D., F.R.C.S., Ivy Lodge, Upper Tooting, S.W.

1881 DALLAWAY, DENNIS JOSEPH WILLIAM, Salisbury Club, St. James'-square, S.W.

1873 DALY, OWEN, M.D., F.R.C.P., J.P., 23, Albion-street, Hull, Physician to the Hull General Infirmary.

1880 DAVSON, SMITH HOUSTON, M.D., Campden Villa, 203, Maida-vale, W.

1868 *DAVY, RICHARD, M.B., F.R.C.S., F.R.S.E., 33, Welbeck-street, W., Surgeon to, and Lecturer on Practical Surgery at, the Westminster Hospital. VP, S 2, SM, § 2.

1880 DAWSON, YELVERTON, M.D., 28, Hyde-park-street, W.

1882 DAY, EDWARD JOSEPH, Dorchester.

1867 DAY, WILLIAM HENRY, M.D., M.R.C.P., 10, Manchester-square, W., Physician to the Samaritan Free Hospital. C 3.

1883 DENT, CLINTON THOMAS, F.R.C.S., 19, Savile-row, W., Assistant Surgeon to, and Lecturer on Practical Surgery at, St. Thomas's Hospital.

1879 DEWAR, JOHN, 132, Sloane-street, S.W.

1844 *DIAMOND, HUGH WELCH, Twickenham House, Twickenham, Middlesex.

1881 DICKSON, FRANCIS KENNEDY, F.R.C.P. Edin., Wye House Lunatic Asylum, Buxton, Derbyshire.

1882 DOLAN, THOMAS MICHAEL, 1882, Horton House, Halifax. FM 1882.

1881 DORAN, ALBAN HENRY GRIFFITHS, F.R.C.S., 51, Seymour-street, Portman-square, W., Surgeon to Out Patients at the Samaritan Free Hospital.

1872 DOWN, JOHN LANGDON HAYDON, M.D., F.R.C.P., 81, Harley-street, W., Physician to, and Lecturer on Clinical Medicine at, the London Hospital.

1871 DOWSE, THOMAS STRETCH, M.D., 14, Welbeck-street, W., Physician to the North London Hospital for Consumption. S, C 3.

1877 DREW, JOHN HENRY, 2, Cambridge-terrace, Hyde-park, W., Consulting Surgeon to the Metropolitan Ear and Throat Infirmary. *Councillor.*

1881 DREWITT, FREDERIC GEORGE DAWTREY, M.A., M.D., M.R.C.P., 52, Brook-street, W., Assistant Physician to the West London Hospital, and to the Victoria Hospital for Children.

1874 DRYSDALE, CHARLES ROBERT, M.D., M.R.C.P., 65, Regent-street, W.C., Senior Physician to the Metropolitan Free Hospital.

1874 DUNCAN, WILLIAM ARCEDECKNE, M.D., F.R.C.S., 29, Wimpole-street, W.

1848 DUNCAN, JAMES, M.B., 8, Henrietta-street, Covent-garden, W.C.

1878 DUNCAN, JAMES MATTHEWS, LL.D., M.D., F.R.C.P., F.R.S., 71, Brook-street, W., Physician-Accoucheur to, and Lecturer on Midwifery at, St. Bartholomew's Hospital.

1881 DUNCAN, JOHN THORNTON, Stamford, Lincolnshire.

1857 DUNN, ROBERT WILLIAM, 13, Surrey-street, Strand, W.C. C 3.

1873 DURANTY, E. NICHOLAS, M.D., Marseilles. NS.

1873 DURHAM, ARTHUR EDWARD, F.R.C.S., 82, Brook-street, W., Surgeon to, and Lecturer on Surgery at, Guy's Hospital. O, C. *President.*

1884 Durham, Frederick, F.R.C.S., 38, Brook-street, W.

1878 Edis, Arthur Wellesley, M.D., F.R.C.P., 22, Wimpole-street, W., Assistant Obstetric Physician to, and Lecturer on Obstetric Medicine at, the Middlesex Hospital.

1860 Edmunds, James, M.D., M.R.C.P., 8, Grafton-street, W., Senior Physician to the London Temperance Hospital; Medical Officer of Health and Public Analyst for St. James's, London.

1880 Edwards, Frederick Swinford, F.R.C.S., 93, Wimpole-street, W., Demonstrator of Anatomy and Operative Surgery at St. Bartholomew's Hospital; Surgeon to the West London Hospital.

1868 Elliott, George Frederick, M.D., F.R.C.P., 3, Albion-street, Hull, Physician to the Hull General Infirmary.

1882 Elliott, Thomas, M.D., Monson-place, Tunbridge Wells.

1872 Ellis, James, Calcutta. ns.

1883 Emond, E., Mont Doré les Bains, France.

1883 English, Edgar, 1 Manor-road, Stoke Newington, N.

1880 English, Thomas Johnston, M.D., 128, Fulham-road, S.W.

1883 Ewart, Joseph, M.D., F.R.C.P., J.P., Montpellier Hall, Brighton, Retired Dep. Surgeon General, Bengal Army; late Principal, Professor of Medicine, and Senior Physician, Calcutta Medical College.

1877 Ewart, William, M.D. Cantab., F.R.C.P., 33, Curzon-street, Mayfair, W., Assistant Physician to, and Lecturer on Physiology at, St. George's Hospital. c.

1869 Farquharson, Robert, M.D., F.R.C.P., M.P., Reform Club, S.W. c.

1873 Fayrer, Sir Joseph, K.C.S.I., LL.D., M.D., F.R.C.P., F.R.C.S., F.R.S., 53, Wimpole-street, W., Honorary Physician to H.M. the Queen and to H.R.H. the Prince of Wales; Physician to H.R.H. the Duke of Edinburgh; Physician to the Secretary of State for India in Council; President of the Medical Board, India Office; Consulting Physician to Charing Cross Hospital. vp, ll, sm, p. *Councillor.*

1884 Fenton, Frederick Enos, Macquarie House, Ealing, W.

1878 Field, George Purdey, 31, Lower Seymour-street, Portman-square, W., Aural Surgeon to, and Lecturer on Aural Surgery at, St. Mary's Hospital.

1883 Finlay, David White, M.D., M.R.C.P., 21, Montagu-street, W., Assistant Physician to, and Lecturer on Forensic Medicine and Public Health at, the Middlesex Hospital.

1876 Fisher, Frederic Richard, F.R.C.S., 79, Grosvenor-street, W., Senior Surgeon to the National Orthopædic Hospital.

1868 Fletcher, Thomas Bell Elcock, M.D., F.R.C.P., J.P., 43, Clarendon-square, Leamington, Consulting Physician to the Birmingham General Hospital. ns.

1884 Flint, Arthur, Westgate Lodge, Westgate-on-Sea.

1868 Folker, William Henry, F.R.C.S., Hanley, Staffordshire, Senior Surgeon to the North Stafford Infirmary. ns.

1878 *Fonmartin, Henry de, M.D., B.Sc., B.C.L., M.R.C.P., Parkhurst, Isle of Wight.

1869 Foster, Balthazar, M.D., F.R.C.P., 14, Temple-row, Birmingham, Physician to the General Hospital, Birmingham; Professor of Physic in the Queen's College, Birmingham. ns.

1872 Fothergill, John Milner, M.D., M.R.C.P., 110, Park-street, W., Physician to the City of London Hospital for Diseases of the Chest. c.

1879 Fowler, James Kingston, M.D., M.R.C.P., 35, Clarges-street, Mayfair, W., Assistant Physician to the Middlesex Hospital, and to the Hospital for Consumption, Brompton. *Secretary.*

1873 Fox, Arthur Edward Wellington, M.B., C.M., 16, Gay-street, Bath, Physician to the Royal United Hospital, Bath.

1868 Fox, Charles Henry, M.D., Brislington House, near Bristol. ns.

1871 Fox, Francis, 68, Wimpole-street, W.

1868 Fox, John Makinson, The Grove, Lymm, Cheshire. ns.

1879 Fox, Thomas Colcott, B.A., M.B., M.R.C.P., 14, Harley-street, W., Physician in charge of the Skin Department, Westminster Hospital.

1868 Freer, Alfred, J.P., Stourbridge. ns.

1884 Frost, William Adams, F.R.C.S., 77, Wimpole-street, W., Assistant Ophthalmic Surgeon to St. George's Hospital.

1883 Gabbett, Henry Singer, M.A., M.D., M.R.C.P., 57, Queen Anne-street, Cavendish-square, W.

1868 Gaine, Charles, 30, Gay-street, Bath, Dental Surgeon to the Royal United Hospital. ns.

1882 Gamgee, J. Sampson, F.R.S.E., 22, Broad-street, Birmingham, Consulting Surgeon to the Queen's Hospital, Birmingham.

1862 Gant, Frederick James, F.R.C.S., 16, Connaught-square, W., Senior Surgeon to the Royal Free Hospital. p, vp 2, ll, o, c 3.

1882 Gardiner, Gideon George, M.D., 47, Wimpole-street, W.

1847 Garrod, Alfred Baring, M.D., F.R.C.P., F.R.S., 10, Harley-street, W., Consulting Physician to King's College Hospital. p, vp 2, ll, o, c 9.

1881 Gawith, James Jackson, 23, Westbourne-park-terrace, W.

1849 Gay, John, F.R.C.S., 34, Finsbury-pavement, E.C., Senior Surgeon to the Great Northern Hospital. p, vp, ll, o, c 3, t 6.

1873 Gee, Robert, M.D., M.R.C.P., 5, Abercromby-square, Liverpool, Consulting Physician to the Hospital for Diseases of the Chest Liverpool.

1878 Gell, Thomas Sylvester, M.D., 24, Fitzroy-square, W. Travelling.

1879 Gibbes, Heneage, M.D., C.M., 94, Gower-street, W.C., Lecturer on Physiology and Histology at the Westminster Hospital. *Councillor.*

1856 Gibbon, Septimus, B.A., M.B., M.R.C.P., 39, Oxford-terrace, W., Medical Officer of Health, Holborn.

1882 Gibbons, Robert Alexander, M.D., C.M., M.R.C.P., 32, Cadogan-place, S.W.

1881 Giffard, Douglas W., 5, Pavilion-parade, Brighton.

1883 Gilbert, Philip Francis, Cripplegate Vicarage, E.C.

1867 Gill, William, 11, Russell-square, W.C.

1871 Glynn, Thomas Robinson, M.D., F.R.C.P., 62, Rodney-street, Liverpool, Physician to the Royal Infirmary, Liverpool. ns.

1857 Godfrey, John Blennerhasset, M.D., F.R.C.P. Edin., Ormonde House, North-gate, Regent's-park, N.W.

1869 Godson, Clement, M.D., M.R.C.P., 9, Grosvenor-street, W., Assistant Physician-Accoucheur to St. Bartholomew's Hospital. vp, c 3, s 2, sm.

1872 Godson, Charles, F.R.C.S., 1, Astwood-road, Cromwell-road, S.W.

1873 Goodsall, David Henry, F.R.C.S., 17, Devonshire-place, Portland place, W., Surgeon to the Metropolitan Free Hospital; Assistant Surgeon to St. Mark's Hospital. s 2, sm. *Councillor.*

1880 Goude, Herbert, F.R.C.S. Edin., Smallpox Hospital, Highgate-hill, N.

1878 Gould, Alfred Pearce, M.S., F.R.C.S., 16, Queen Anne-street, W., Assistant Surgeon to the Middlesex Hospital. c 3. *Hon. Secretary.*

1876 Gowers, William Richard, M.D., F.R.C.P., 50, Queen Anne-street, W., Physician to, and Assistant Professor of Clinical Medicine at, University College Hospital; Physician to the National Hospital for the Paralysed and Epileptic. c, sm.

1874 Gowland, Peter Yeames, F.R.C.S., 34, Finsbury-square, E.C., Senior Surgeon to St. Mark's Hospital.

1867 Grasemann, Christian Edward, M.D., 46, Albany-street, Regent's-park, N.W.

1881 Green, Thomas Henry, M.D., F.R.C.P., 74, Wimpole-street, W., Physician to, and Lecturer on Pathological Anatomy at, Charing Cross Hospital; Senior Assistant Physician to the Hospital for Consumption, Brompton.

1841 *Greenhalgh, Robert, M.D., 35, Cavendish-square, W., late Physician-Accoucheur to St. Bartholomew's Hospital. p, vp, o, s 3, c 5.

1868 Gregson, George, 63, Harley-street, Dental Surgeon to the Dental Hospital of London.

1873 Grieve, Robert, M.D., British Guiana.

1875 Griffith, G. de Gorrequer, 34, St. George's-square, S.W.

1884 Griffiths, David Charles Ballinger, 3, Lansdowne-place, Brighton.

1880 Gristock, William, M.D. Lond., 6, Finchley-road, N.W.

1859 Habershon, Samuel Osborne, M.D., F.R.C.P., 70, Brook-street, W., late Senior Physician to, and Lecturer on Medicine at, Guy's Hospital. p, ll, o, c 3.

1884 Hadden, Walter Baugh, M.D., 21, Welbeck-street, Cavendish-square, W.

1881 Hall, Charles Ross, Hatfield, Herts.

1874 *Hall, Francis de Havilland, M.D., F.R.C.P., 46, Queen Anne-street, W., Assistant Physician to, and Lecturer on Medical Jurisprudence at, the Westminster Hospital. c 3, s 2, sm. *Councillor.*

1881 Hames, George Henry, F.R.C.S., 2, Queenborough-terrace, Kensington-gardens, W.

1880 Hamilton, Francis George, Abchurch House, Sherbourne-lane, E.C. Surgeon to the Central Throat and Ear Hospital.

1878 Hamilton, Julius Lawrence, 34, Gloucester-terrace, Hyde-park, W.

1871 Hamilton, Robert, M.D., Travelling.

1879 Hamilton, Seton Guthrie, Surgeon 1st Royal Life Guards.

1834 Harding, William, F.R.C.S., 4, Percy-street, Bedford-square, W. vp 2, c 3.

1875 Hardwicke, Herbert Junius, M.D., C.M., Purton Lodge, Sharrow, Sheffield.

1873 Hardwicke, Junius, F.R.C.S., Rotherham, Senior Consulting Surgeon to the Rotherham Hospital.

1882 Harper, Gerald Samuel, M.B., 5, Hertford-street, Mayfair.

1871 Harris, Charles James, 11, Kilburn Priory, N.W.

1872 Harris, Henry, LL.D., M.D., F.R.C.S., Redruth, Cornwall. ns.

1873 Harris, William John, 26, Marine Parade, Worthing, Senior Medical Officer to the Worthing Infirmary.

1851 Harrison, Charles Henry Rogers-, F.R.C.S., Vine House, 55, Stockwell-road, S.W., Consulting Surgeon to St. Pancras Infirmary. vp 2, t 6, s 5, c 3, sm. *Trustee.*

1871 Harrison, Reginald, F.R.C.S., 38, Rodney-street, Liverpool, Surgeon to the Royal Infirmary. ns.

1883 Hartridge, Gustavus, F.R.C.S., 47, Kensington-park-gardens, W. Assistant Surgeon to the Royal Westminster Hospital.

1864 Harvey, John Alexander, 35, Princes-square, Bayswater, W.

1882 Harvey, John Stephenson, 6, Rue de la Coupe, Boulogne-sur-Mer.

1882 Haslam, William Frederick, F.R.C.S., The Minories, Bull-street, Birmingham, Assistant Surgeon to the Birmingham General Hospital.

1852 Haward, Edwin, M.D., M.R.C.P., F.R.C.S., 9, Harley-street, W., Physician to the North London Consumption Hospital.

1883 Hawken, Charles St. Aubyn, 20, North-terrace, Wandsworth, S.E.

1871 Hemming, William Benjamin, 26, Notting-hill-terrace, W.

1849 Henry, Alexander, M.D., 132, Highbury-hill, N., Hon. Secretary to the Metropolitan Counties Branch of the British Medical Association. c 7.

1878 Henry, Louis, M.D., Melbourne, Australia.

1883 Herman, George Ernest, M.B., M.R.C.P., F.R.C.S., 7, West-street, Finsbury Circus, E.C., Obstetric Physician to, and Lecturer on Obstetric Medicine at, the London Hospital.

1879 Heron, George Allan, M.D., M.R.C.P., 40, Margaret-street, Cavendish-square, W., Physician to the City of London Hospital for Diseases of the Chest.

1883 Herschel, George A., M.D., 29, Moorgate-street, E.C.

1883 Hewitt, Frederick William, M.A., M.B., 1, St. George's-place, S.W.

1876 Heycock, Francis Raworth, M.D., C.M., 26, Upper Wimpole-street, W., Surgeon to the North-West London Hospital; Assistant Surgeon to St. Peter's Hospital.

1872 Hicks, John Braxton, M.D., F.R.C.P., F.R.S., 24, George-street, Hanover-square, Consulting Physician-Accoucheur to Guy's Hospital. C.

1884 Hill, Berkeley, F.R.C.S., M.B., 55, Wimpole-street, W., Surgeon to University College Hospital and Professor of Clinical Surgery in University College.

1881 Hill, James, M.D., Thatched House Club, St. James's, SW.

1867 Hill, Thomas Harvey, 4, Stanhope-terrace, Bayswater. T, C 3.

1840 Hird, Francis, F.R.C.S., 13, Old Burlington-street, W., Consulting Surgeon to the Charing Cross Hospital. P 2, VP 6, LL, O 2, C 12. *Trustee.*

1873 Hobson, William Henry, Great Berkhamstead, Herts, Honorary Surgeon to the West Herts Infirmary.

1879 Hogg, Arthur John, Westbourne Villa, Ealing, W.

1848 Hogg, Jabez, 1, Bedford-square, W.C., Consulting Surgeon to the Royal Westminster Ophthalmic Hospital. VP, C 2.

1868 Holman, Constantine, M.D., Reigate. C 4.

1881 Hood, Donald William Charles, M.D., M.R.C.P., 43, Green-street, W., Physician to the West London Hospital.

1879 Hookham, Paul, 7, Bloomsbury-place, W.C.

1875 Hope, William, M.D., M.R.C.P., 56, Curzon-street, Mayfair, W., Senior Physician to Queen Charlotte's Lying-in Hospital, and Senior Physician to the Belgrave Hospital for Children.

1883 *Hovell, T. Mark, F.R.C.S. Edin., 3, Mansfield-street, W., Aural Surgeon to the London Hospital.

1879 Huggard, William Richard, M.A., M.D., M.Ch., M.R.C.P., Hammersmith.

1864 Hume, Frederick Henry, 53, Devonshire-street, Islington, N.

1884 Hunter, Sir William Guyer, M.D., F.R.C.P., 21, Norfolk-crescent, Hyde-park, W., Honorary Surgeon to H.M. the Queen.

1881 Hutchinson, Jonathan, F.R.C.S., F.R.S., 15, Cavendish-square, Consulting Surgeon to the London Hospital; late Professor of Pathology and Surgery at the Royal College of Surgeons.

1875 Hutchinson, Samuel John, 44, Brook-street, W., Dental Surgeon to, and Clinical Lecturer at, the University College Hospital, and Surgeon to the Dental Hospital of London. *Councillor.*

1884 Jackson, Frederick William, M.D., 7, Hyde-park-gardens, W.

1868 Jackson, John Hughlings-, M.D., F.R.C.P., F.R.S., 3, Manchester-square, W., Physician to the London Hospital. O, C 2, VP.

1853 Jackson, Robert, M.D., 53, Notting-hill-square, W.

1874 JAGIELSKI, VICTOR APOLLINARIS, M.D., M.R.C.P., York-place, Regent's Park, W., Physician to the Infirmary for Consumption, Margaret-street, W.

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1883 JESSOP, WALTER HAMILTON HYLTON, B.A., M.B., 73, Harley-street, W., Assistant Surgeon to the Central London Ophthalmic Hospital.

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1875 JONES, SYDNEY, M.B., F.R.C.S., 16, George-street, Hanover-square, W., Senior Surgeon to, and Lecturer on Surgery at, St. Thomas's Hospital. *Vice-President.*

1881 JONES, THOMAS WILLIAM CARMALT, M.A., 6, Westbourne-street, W.

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1884 KEETLEY, CHARLES BELL, F.R.C.S., 20, Princes-street, Hanover-square, W.

1847 KELLOCK, WILLIAM BERRY, M.D., F.R.C.S., Stamford-hill, N.

1883 KEMP, JOHN ROBERT, 101, Jermyn-street, S.W.

1873 KEMPTHORNE, JOHN, F.R.C.S., Callington, Cornwall.

1876 KEY, AUGUSTUS COOPER, M.R.C.P. Edin., 30, Wilton-place, S.W.

1869 *KING, KELBURNE, M.D., F.R.C.S., J.P., 6, Albion-street, Hull, Surgeon to the Hull General Infirmary.

1868 KIRKMAN, WILLIAM PHILLIPS, M.D., St. Leonard's-on-Sea. NS.

1868 KNAGGS, SAMUEL, Ebor Mount, Huddersfield, Surgeon to the Huddersfield and Upper Agbrigg Infirmary. NS.

1883 KNAPTON, GEORGE, Southampton.

1875 KNOX, JOHN, M.D., M.C., Resident Medical Officer, Bethnal Green Infirmary, E.

1868 LAKE, WILLIAM CHARLES, M.D., Teignmouth, Devon, Surgeon to the Teignmouth Infirmary.

1883 LANGFORD, PHINEAS PITTS, M.D., Park Lodge, East Finchley, N.

1881 LANGTON, JOHN, F.R.C.S., 2, Harley Street, W., Surgeon to, and Lecturer on Anatomy at, St. Bartholomew's Hospital.

1882 LARKIN, FRANK COLET, M.B., C.M., 8, Duchess-street, Portland-place.

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1873 LEE, ROBERT JAMES, M.A., M.D., F.R.C.P., 6, Savile-row, W., Senior Assistant Physician to the Hospital for Sick Children. C.

1869 LEES, CHARLES ALEXANDER, M.D., Fleet Surgeon, R.N. NS.

1858 Lemon, Oliver, Kings Langley, Herts.

1867 Lichtenberg, George, M.D., M.R.C.P., 47, Finsbury-square, Surgeon to the German Hospital. c 2.

1869 Lipscomb, John Thomas Nicholson, M.D., F.R.C.S., St. Albans, Herts. ns.

1878 Lister, Sir Joseph, Bart., D.C.L., LL.D., M.D., F.R.C.S., F.R.S., 12 Park-crescent, Portland-place, Surgeon Extraordinary to H.M. the Queen, Surgeon to, and Professor of Clinical Surgery at, King's College Hospital.

1870 Lloyd, Ridgway Robert Syers Christian Codner, St. Albans, Senior Surgeon to St. Albans Hospital.

1878 Lockwood, Charles Barrett, F.R.C.S., 8, Serjeants' Inn, E.C., Surgeon to the Great Northern Hospital.

1873 Loe, James Scarborough, 96, Woodhouse-lane, Leeds, Surgeon to the Leeds Fever Hospital.

1881 Lorimer, G., M.D., Buxton, Derbyshire.

1868 Lowe, John, M.D., J.P., Kings Lynn, Medical Attendant to their Royal Highnesses the Prince and Princess of Wales at Sandringham, Consulting Surgeon to the West Norfolk and Lynn Hospital.

1868 *Lund, Edward, F.R.C.S., 22, St. John's-street, Manchester, Consulting Surgeon to the Manchester Royal Infirmary; Professor of Surgery in Owens College; Examiner at the Royal College of Surgeons. o. *Councillor.*

1869 Lunn, William Joseph, M.D., F.R.C.S., Hull, Senior Surgeon to the Hull General Infirmary. ns.

1881 Lyons, Isidor, 19, Queen Anne-street, W., Assistant Dental Surgeon to St. Bartholomew's Hospital.

1879 Lyons, Richard Thomas, Surgeon-Major Bengal Army.

1871 MacCormac, Sir William, M.A., D.Sc., M.Ch., F.R.C.S., 13, Harley-street, W., Surgeon to, and Lecturer on Surgery at, St. Thomas's Hospital. c 3. *Hon. Secretary for Foreign Correspondence.*

1882 MacKellar, Alexander Oberlin, M.D., M.Ch., F.R.C.S., Senior Assistant Surgeon to, and Lecturer on Practical Surgery at, St. Thomas's Hospital.

1862 Mackenzie, Morell, M.D., 19 Harley-street, W., Physician to the Hospital for Diseases of the Throat; Lecturer on Diseases of the Throat at the London Hospital.

1880 Mackenzie, Stephen, M.D., F.R.C.P., 26, Finsbury-square, E.C., Physician to, and Lecturer on Medicine at, the London Hospital.

1880 Mackrell, Alfred Sextus, 59, Queen Anne-street, W.

1881 Maclagan, Thomas John, M.D., M.R.C.P., 9, Cadogan-place, S.W., Physician in Ordinary to T.R.H. the Prince and Princess Christian of Schleswig-Holstein.

1861 MacLaren, Alexander Connell, 60, Harley-street, W.

1864 Macpherson, John, M.A., M.D., 35, Curzon-street, Mayfair, W., Physician to the Scottish Hospital.

1883 Maddick, Edmund Distin, F.R.C.S. Edin., 17, Upper Wimpole-street, W.

1869 Magill, Martin, M.D., F.R.C.S., 6, Westbourne Park-road, W.

1878 Mair, Robert Slater, M.D., 28, Ledbury-road, Bayswater, W.

1876 Manville, Benjamin Ephraim, 38, Sutherland-road, W., Surgeon-Dentist to the Metropolitan Free Hospital.

1883 Margerison, Richard, B.A., F.R.C.S., 15, Gloucester-street, Belgrave-road, S.W., Surgical Registrar to St. George's Hospital.

1873 Marshall, Edward, Mitcham, Surrey.

1859 Marshall, James, M.D., 6, Rubislaw-place, Aberdeen. ns.

1869 Marshall, William, M.D., Torrieburn, Barnes, S.W.

1864 Marshall, William Gurslave, F.R.C.S., Medical Superintendent Female Department, Asylum, Colney Hatch, N.

1874 Martin, Adam Rae, "The Precincts," Rochester.

1862 Mason, Francis, F.R.C.S., 5, Brook-street, Grosvenor-square, W., Surgeon to, and Lecturer on Practical Surgery at, St. Thomas' Hospital. p, t, ll, o, vp, s 2, c 4, sm. *Councillor.*

1869 Mathews, Robert, Bickley, Kent. ns.

1871 Maurice, Oliver Calley, Reading, Surgeon to the Royal Berks Hospital. ns.

1850 May, William Costall, 52, Tregunter-road, South Kensington, S.W.

1862 Maybury, Augustus Kingston, M.D., Holly Lodge, Richmond, Surrey, Consulting Physician to the Richmond Hospital. ns.

1869 McDonagh, James Armstrong, F.R.C.S., 211, Hampstead-road, N.W.

1884 McGann, Terence Joseph, Surgeon-Major, Madras Army.

1873 McHardy, Malcolm Macdonald, F.R.C.S. Edin., 5, Savile-row, W., Professor of Ophthalmology in King's College, and Ophthalmic Surgeon to King's College Hospital.

1868 McIntyre, John, M.D., Odiham, Hants. ns.

1884 Meredith, William Appleton, M.D., C.M., 6, Queen Anne-street, W., Surgeon to the Samaritan Free Hospital.

1874 Méric, Henry de, 25, Duke-street, St. James, S.W., Assistant Surgeon to the French Hospital.

1864 Middlemist, Robert Percy, 10, Bedford-place, Russell-square, W.C.

1858 Millar, John, Medical Superintendent to the Bethnal House Asylum, Cambridge-road, E.

1881 Millican, Kenneth William, B.A., North Lodge, Kineton, Warwick.

1878 Milner, Edward, 32, New Cavendish-street, W., Surgeon to the Lock Hospital.

1882 Mills, Joseph, 15, Henrietta-street, Cavendish-square, W., Administrator and Teacher of Anæsthetics to St. Bartholomew's Hospital.

1882 Mivart, Frederick St. George, 62, Green-street, Grosvenor-square, W., Surgeon to the Western General Dispensary.

1877 Monckton, Marshall, West Dene, Maidstone.

1883 Money, Angel, M.D., 14, Langham-place, W.

1875 Moore, Joseph, M.D., Hard Deane, Thornton-heath.

1883 Moore, Thomas, F.R.C.S., 6, Lee-terrace, Blackheath, S.E.

1883 Morgan, John Hammond, M.A., F.R.C.S., 68, Grosvenor-street, W., Assistant Surgeon to Charing Cross Hospital.

1871 Morley, Alexander, 42, Albemarle-street, W.

1881 Morris, Henry, M.A., M.B., F.R.C.S., 2, Mansfield Street, Portland-place, W., Surgeon to, and Lecturer on Surgery at, the Middlesex Hospital. c.

1878 Morris, Malcolm Alexander, F.R.C.S. Edin., 63, Montagu-square, W., Surgeon to the Skin Department of, and Lecturer on Dermatology at, St. Mary's Hospital. c 2.

1883 Mortimer, John Desmond Ernest, 59, Mount-st., Grosvenor-square, W.

1882 Morton, Andrew Stanford, M.B., C.M., 57, Welbeck-street, W., Senior Assistant Surgeon to the Royal South London Ophthalmic Hospital.

1884 Moullin, Charles William Mansell, M.A., M.D., F.R.C.S., 69, Wimpole-street, W., Medical Fellow, Pembroke College, Oxford; Assistant Surgeon to, and Lecturer on Comparative Anatomy at, the London Hospital.

1883 Mowat, George, M.R.C.P. Edin., St. Alban's.

1878 Mumford, William Lugar, M.D., 1, Bartlett's-passage, Holborn, E.C.

1868 Murray, John Carrick, M.D., 44, Newgate-street, Newcastle-on-Tyne. ns.

1883 Murray, James, M.D., 21, Weymouth-street, W.

1879 Murrell, William, M.D., F.R.C.P., 38, Weymouth-street, W., Assistant Physician to, and Lecturer on Materia Medica and Therapeutics at, the Westminster Hospital.

1873 Myers, Henry Reynolds, 30, Euston-square, N.W.

1868 Nankivell, Charles Benjamin, M.D., Ashley Lodge, Torquay, Senior Physician to the Consumption Hospital, Torquay. ns.

1877 Nesbitt, Dawson, M.D., 34, Cambridge-place, Hyde-park, W.

1880 Netherclift, William Henry, F.R.C.S., Junior Athenæum Club, Piccadilly, W.

1868 Nevins, John Birkbeck, M.D., 3, Abercromby-square, Liverpool, Consulting Surgeon to the Ear and Eye Infirmary. ns.

1876 Newham, James, 16, Princes-street, Cavendish-square, W.

1871 Nicholls, John Frederick, M.D., M.R.C.P., Devizes. ns.

1880 Nix, Edward James, M.D., 143, Great Portland-street, W.

1868 Noble, Daniel, M.A., M.D., F.R.C.P., 258, Oxford-road, Manchester. ns.

1884 Ogle, Charles John, 36, Great Marlborough-street, W.

1871 Ogle, William, M.A., M.D., F.R.C.P., Derby, Physician to the Derbyshire General Infirmary. ns.

1873 Ogston, Alexander, M.D., C.M., 252, Union-street, Aberdeen, Regius Professor of Surgery in the University of Aberdeen; Surgeon to the Aberdeen Royal Infirmary.

1872 Ogston, Francis, M.D., 13, Albyn-terrace, Aberdeen, Emeritus Professor of Medical Logic and Medical Jurisprudence in the University of Aberdeen. NS.

1884 Oliver, George, M.D., West End Park, Harrogate.

1881 Oram, Arthur Murray, M.D., C.M., Sydney, N. S. Wales.

1875 Ord, William Miller, M.D., F.R.C.P., 7, Brook-street, W., Physician to, and Lecturer on Medicine at, St. Thomas's Hospital. C.

1884 Orwin, Arthur, Wiggelsworth, M.D., 2, Ospringe-road, Brecknock-road, N.

1880 Osborn, Samuel, F.R.C.S., 10, Maddox-street, Regent-street, W.

1880 Oswald, James Waddell Jeffreys, M.D., 245, Kennington-road, S.E.

1883 Owen, Charles J. Rayley, 61, Cleveland-square, W.

1878 *Owen, Edmund, M.B., F.R.C.S., 49, Seymour-street, Portman-square, Surgeon to, and Lecturer on Anatomy at, St. Mary's Hospital; Surgeon to the Hospital for Sick Children. C 2, S 2, SM. *Councillor.*

1881 Owen, Isambard, M.A., M.D., M.R.C.P., 5, Hertford-street, Mayfair, W., Assistant Physician to, and Lecturer on Materia Medica and Therapeutics at, St. George's Hospital. S. 2. *Chairman of Council.*

1880 Palmer, Frederick Steven, M.D., Compton Lodge, East Sheen, S.W.

1882 Palmer, William Pitt, 6, Claude Villa, East Dulwich, S.E.

1877 Paramore, Richard, 18, Hunter-street, W.C.

1883 Parker, Robert William, 8, Old Cavendish-street, W., Surgeon to the East London Children's Hospital.

1867 Parkinson, George, 50, Brook-street, Grosvenor-square, W., late Surgeon Dentist to, and Lecturer on Dental Surgery at, Charing Cross Hospital.

1880 Parkinson, George William, 36, Sackville-street, Piccadilly, W., Assistant Surgeon to the Dental Hospital of London.

1881 Parrott, Edward John, Hayes, Uxbridge, Middlesex.

1871 Parsons, Francis Henry, M.D., C.M., "The Hurst," West Worthing.

1872 Patten, Charles Arthur, Marpool House, Ealing, W.

1861 Paul, John Hayball, M.D., M.R.C.P., F.R.C.S., Camberwell House, Camberwell, S.E. C 6.

1854 Pavy, Frederick William, M.D., F.R.C.P., F.R.S., 35, Grosvenor-street, W., Physician to, and Lecturer on Medicine at, Guy's Hospital. VP, LL, C.

1881 *Peacey, William, M.B., C.M., 214, Lewisham High-road, S.E.

1883 Peck, Edward George, M.A., 5, Hertford-street, Mayfair, W.

1871 Pedler, George Henry, 6, Trevor-terrace, Knightsbridge, S.W.

1880 Peel, Robert, 120, Collins-street, East Melbourne, Australia.

1869 Pemberton, Oliver, F.R.C.S., J.P., 12, Temple-row, Birmingham, Senior Surgeon to the Birmingham General Hospital; Professor of Surgery in Queen's College. NS.

1883 Penny, George Town, M.D., Stanley House, 3, Oakfield-road, Upper Tollington-park, N.

1883 Perrigal, Arthur, C.M., M.D., New Barnet.

1883 Phillips, Sidney Philip, M.D., M.R.C.P., 12, Radnor-place, Hyde-park, W., Demonstrator of Anatomy at St. Mary's Hospital.

1878 Philipps, Sutherland Rees, M.D., M.Ch., St. Ann's Heath, Virginia Water.

1869 Philipson, George Hare, D.C.L., M.D., M.A., F.R.C.P., J.P., 7, Eldon-square, Newcastle-on-Tyne, Professor of Medicine in the University of Durham, and Senior Physician to the Newcastle Infirmary. NS.

1876 Phillips, Charles Douglas Fergusson, M.D., M.R.C.P., 10, Henrietta-street, Cavendish-square, W., late Lecturer on Materia Medica and Therapeutics at the Westminster Hospital.

1873 Phillips, George Richard Turner, 24, Leinster-square, Hyde-park, W.

1875 Philpot, Harvey John, 55, Warwick-road, Maida-vale, W.

1883 Pick, Thomas Pickering, F.R.C.S., 18, Portman-street, W., Surgeon to, and Lecturer on Surgery at, St. George's Hospital.

1883 Pitts, Bernard, M.B., M.C., F.R.C.S., 31, Harley-street, W., Assistant Surgeon to St. Thomas' Hospital, S.E.

1873 Port, Heinrich, M.D., M.R.C.P., 31, Harley-street, W., Physician to the German Hospital.

1850 Potts, William, F.R.C.S., 2, Albert Terrace, Regent's-park, N.W. C 3.

1868 Powell, Josiah Taylor, M.D., 347, City-road, E.C.

1871 Powell, Richard Douglas, M.D., F.R.C.P., 62, Wimpole-street, W., Physician to the Middlesex Hospital and to the Hospital for Consumption, Brompton. C 2. *Vice-President.*

1869 Price, William Preston, M.D., 1, Ethelbert-crescent, Margate, Surgeon to the Royal Margate Infirmary. NS.

1869 Prior, Charles Edward, M.D., F.R.C.S., St. Peter's, Bedford, Coroner for Bedford. NS.

1884 Protheroe, John, Travelling.

1873 Purcell, Ferdinand Albert, M.D., M.Ch., 7, Manchester-square, W., Surgeon to the Cancer Hospital, Brompton.

1878 Pye, Walter, F.R.C.S., 4, Sackville-street, Piccadilly, W., Surgeon to, and Lecturer on Physiology at, St. Mary's Hospital.

1882 Pyle, Thomas Thompson, M.D., J.P., 5, Lower Seymour-street, W.

1870 Quain, Richard, M.D., F.R.C.P., F.R.S., 67, Harley-street, W., Consulting Physician to the Hospital for Consumption, Brompton. VP, C 3.

1883 Ralfe, Charles Henry, M.A., M.D., F.R.C.P., 26, Queen Anne-street, W., Assistant Physician to the London Hospital.

1861 Ramskill, Jabez Spence, M.D., M.R.C.P., 5, St. Helen's-place, E.C., Consulting Physician to the London Hospital.

1881 Ranking, John Ebenezer, M.A., M.D., M.R.C.P., 18, Mount Ephraim-road, Tunbridge Wells, Physician to the Tunbridge Wells Infirmary.

1859 *Rayner, John, M.R.C.P. Edin., Swaledale House, Highbury-quadrant.

1850 Read, Reginald, F.R.C.P. Edin., 1, Guilford-place, W.C. c 2.

1850 Rees, George Owen, M.D., F.R.C.P., F.R.S., 26, Albemarle-street, W., Consulting Physician to Guy's Hospital. vp 2, ll.

1879 Reeves, Henry Albert, F.R.C.S. Edin., 6, Grosvenor-street, W., Assistant Surgeon to, and Teacher of Practical Surgery at, the London Hospital.

1882 Reid, Robert W., M.D., C.M., F.R.C.S., 75, Lambeth Palace-road, S.E., Lecturer on Anatomy at St. Thomas's Hospital.

1882 Reid, Thomas Whitehead, F.R.C.P. Edin., 34, St. George's-place, Canterbury, Surgeon to the Kent and Canterbury Hospital.

1872 Reynolds, John Russell, M.D., F.R.C.P., F.R.S., 38, Grosvenor-street, W., Physician in Ordinary to H.M.'s Household; Emeritus Professor of the Principles and Practice of Medicine in University College; Consulting Physician to University College Hospital. c 3.

1872 Richards, Joseph Peake, Medical Superintendent, Female Department, County Asylum, Hanwell, W.

1850 Richardson, Benjamin Ward, M.A., M.D., LL.D., F.R.C.P., F.R.S., 25, Manchester-square, W. p, vp, ll, c 5, o, fm 1854.

1882 Ring, Edmund Cuthbert, Salisbury Club, 10, St. James's-square, S.W.

1830 Robarts, Henry Pratt, F.R.C.S., 31, Great Coram-street, W.C. vp 2, s 9, c 10, fm 1844, sm.

1869 Roberts, Bransby, M.D., Badlesmere House, Eastbourne. ns.

1868 *Roberts, David Lloyd, M.D., F.R.C.P., F.R.S.E., 11, St. John's-street, Manchester, Physician to St. Mary's Hospital, Manchester.

1857 Roberts, David Watkin, M.D., 56, Manchester-street, W.

1874 Roberts, Frederick Thomas, M.D., B.Sc., F.R.C.P., 53, Harley-street, W., Physician to University College Hospital; Professor of Materia Medica and Therapeutics in University College; Physician to the Hospital for Consumption, Brompton.

1880 Roberts, William. Travelling.

1873 Robertson, William Henry, M.D., F.R.C.P., J.P., Buxton, Derbyshire, Consulting Physician to the Devonshire Hospital and Buxton Bath Charity.

1884 Robinson, Arthur Henry, M.D., 12, Albion-street, Hull, Surgeon to the Hull General Infirmary.

1877 Roeckel, Waldemar Joseph, M.B., B.S., F.R.C.S., 7, Grosvenor-street, Grosvenor-square, W., Surgeon to the National Orthopædic Hospital; Surgical Registrar to the Charing Cross Hospital.

1869 Roden, William, M.D., F.R.C.S., Kidderminster. ns.

1869 Rogers, Charles Edward Heron, Retford, Notts. ns.

1856 Rogers, Joseph, M.D., 33, Soho-square, W.

1847 Rogers, William Richard, M.D., C.M., M.R.C.P., 56, Berners-street, W., Consulting Physician to the Samaritan Free Hospital. VP, C 6.

1874 Rose, William, M.B., B.S., F.R.C.S., 50, Harley-street, Surgeon to King's College Hospital.

1883 *Ross, Daniel McClure, F.R.C.S.E., 54, Upper Berkeley-street, W., Curator of the Museum, St. George's Hospital.

1876 Routh, Alfred Curtis, 33, Marina, St. Leonard's-on-Sea.

1881 Routh, Amand Jules McConnel, M.D., B.S., M.R.C.P., 6, Upper Montagu-street, W., Assistant Obstetric Physician to the Charing Cross Hospital; Physician to the Samaritan Free Hospital.

1848 Routh, Charles Henry Felix, M.D., M.R.C.P., 52, Montagu-square, W., Consulting Physician to the Samaritan Free Hospital. P, VP 2, S 4, C 6, SM. *Trustee.*

1879 Ryley, James Beresford, M.D., 24, Finsbury-square, E.C., Physician to the Finsbury Hospital for Women.

1884 Salter, Thomas Knight, 23, Lower Seymour-street, W.

1869 Sandwell, Edward, 10, Charles-street, Soho-square, W.

1863 *Sansom, Arthur Ernest, M.D., F.R.C.P., 84, Harley-street, W., Physician to, and Lecturer on Medical Jurisprudence at, the London Hospital; Senior Physician to the North Eastern Hospital for Children. VP, S 2, C 4, SM, §. *Councillor.*

1873 Scott, William, M.D., M.R.C.P., Waverley House, Huddersfield, Senior Physician to the Huddersfield Infirmary.‡

1873 Sedgwick, James, M.D., Boroughbridge, Yorkshire.

1868 Sedgwick, Leonard William, M.D., 2, Gloucester-terrace, Hyde-park, W. VP 2, C 4, § 3.

1883 Semon, Felix, M.D., M.R.C.P., 59, Welbeck-street, W., Assistant Physician in charge of the Throat Department, St. Thomas' Hospital.

1869 Semple, Robert Hunter, M.D., F.R.C.P., 8, Torrington-square, W.C., Physician to the Hospital for Diseases of the Throat. C.

1876 Sewell, Charles Brodie, M.D., 21, Cavendish-square, W. *Chairman of Council.*

1864 Sewill, Henry Ezekiel, 6, Wimpole-street, W. C 2.

1882 Sheppard, Charles Edward, M.D., B.S., F.R.C.S., Rotherwood, Oakhill-road, Putney, S.W.

1875 Sheppard, Edgar, M.D., F.R.C.S., M.R.C.P., 42, Gloucester-square, Hyde Park, W., Professor of Psychological Medicine in King's College.

1871 Shettle, Richard Charles, M.D., 73, London-street, Reading, Physician to the Royal Berkshire Hospital. NS.

1881 Shipton, Arthur, Buxton, Derbyshire, Hon. Medical Officer to the Devonshire Hospital.

1871 Shipton, William Parker, Buxton, Derbyshire, Consulting Surgeon to the Devonshire Hospital.

1867 *Simms, Frederick, M.B., M.R.C.P., 6, Mandeville-place, Manchester-square, W. C 2.

1883 Skelding, Joseph, 16, Euston-square, N.W.

1883 Skerritt, Edward Markham, M.D., B.S., M.R.C.P., Richmond Hill, Clifton, Physician to the Bristol General Hospital; Lecturer on Medicine and Pathology in the Bristol Medical School.

1862 Slight, George, M.D., 3, Clifford-street, Bond-street, W. c 2.

1871 Sloman, Samuel George, Farnham. ns.

1845 Smiles, William, M.D., St. Martha's Lodge, Guildford. vp 2, s 4, c 9, sm.

1880 Smith, Noble, F.R.C.S. Edin., 24, Queen Anne-street, W.

1848 *Smith, Henry, F.R.C.S., 82, Wimpole-street, W., Professor of Surgery in King's College, and Surgeon to King's College Hospital. p, vp, ll, o c 3.

1882 Smith, Herbert, Westbourne Villa, Uxbridge-road, Ealing, W.

1873 Smith, Heywood, M.A., M.D., M.R.C.P., 18, Harley-street, W., Physician to the Hospital for Women and the British Lying-in Hospital. c 3.

1877 Smith, Sydney Lloyd, 32, Argyle-square, King's Cross, W.C.

1882 Smith, Thomas Frederick Hugh, F.R.C.S., Farningham, Kent.

1873 *Smith, Thomas Gilbart-, M.A., M.D., M.R.C.P., 68, Harley-street, W., Assistant Physician to the London Hospital; Physician to the Royal Hospital for Diseases of the Chest. s 2, sm. *Councillor.*

1872 Smith, Walter, M.R.C.P. Edin., 2, Stanhope-terrace, Gloucester-gate, Regent's-park, N.W.

1874 Smyth, William Woods, Maidstone.

1884 Snow, Herbert Lumley, M.D., 40, Norfolk-crescent, Hyde-park, W.

1869 Spender, John Kent, M.D., 17, Circus, Bath, Physician to the Mineral Water Hospital. fm 1874.

1883 Spitta, Edmund Johnson, Ivy House, Clapham-common, S.E.

1881 Springthorpe, John William, M.A., M.D., M.R.C.P., 49, Manor-road, Lee, S.E.

1864 Squire, Alexander John Balmanno, M.B., 24, Weymouth-street, Portland-place, Surgeon to the British Hospital for Diseases of the Skin.

1881 Startin, James, 16a, Sackville-street, W., Surgeon to the St. John's Hospital for Diseases of the Skin.

1869 Stear, Henry, Saffron Walden, Essex, Senior Surgeon to the Saffron Walden Hospital. ns.

1869 Stedman, James Remington, M.D., F.R.C.S., J.P., Guildford, Surrey, Consulting Surgeon to the Royal Surrey County Hospital. ns.

1882 Stewart, James, B.A., M.R.C.P. Edin., Dunmurry, Sneyd-park, Clifton.

1883 Stewart, William Robert Henry, F.R.C.S., 16, Harley-street, W., Surgeon to the North-west London Hospital.

1884 Stiven, Edward Winnan Fleming, M.D., C.M., Lincoln House, Harrow, Middlesex.

1848 *Stocker, John Sherwood, M.D., M.R.C.P., 2, Montagu-square, W. c 10, s 2.

1884 Stoker, George, 8, Cadogan-terrace, S.W.

1877 Stowers, James Herbert, M.D., 23, Finsbury-circus, E.C.

1873 Strange, William Heath, M.D., C.M., 5, Grosvenor-street, W. *Councillor.*

1881 Sturge, William Allen, M.D., M.R.C.P., 9, Rue Longchamp, Nice. SM

1876 *Sutherland, Henry, M.A., M.D., M.R.C.P., 6, Richmond-terrace, Whitehall, S.W., Lecturer on Insanity at the Westminster Hospital.

1874 Sutro, Sigismund, M.D., F.R.C.P., 37A, Finsbury square, E.C., Consulting Physician to the German Hospital.

1880 Sweeting, Richard Deane Roker, Medical Superintendent Western District Fever Hospital, Fulham, S.W.

1881 Sykes, Edwin John, M.B., C.M., Buxton, Derbyshire.

1883 Sykes, John Frederick Joseph, B.Sc., M.B., C.M., 7, Thayer-street. Manchester-square, W.

1864 Tait, Edward Wilmshurst, 54, Highbury-park, N.

1870 *Tait, Lawson, F.R.C.S., 7, The Crescent, Birmingham, Surgeon to the Birmingham and Midland Hospital for Women.

1875 Tamplin, Charles Harris, 44, Royal-road, Ramsgate.

1869 Taylor, Charles Bell, M.D., Nottingham, Honorary Surgeon to the Nottingham and Midland Eye Infirmary. NS.

1882 Taylor, Seymour, M.D., C.M., M.R.C.P., 222, Taviton-street, Gordon-square, W.C., Physician to the North London Hospital; Demonstrator of Anatomy at St. Thomas's Hospital.

1863 Teevan, William Frederick, B.A., F.R.C.S., Mostyn Villa, Brockman-road, Folkestone. VP, LL, O, C 3.

1858 Thane, George Dancer, M.D., 15, Montague-street, Russell-square, W.C.

1874 Thomas, Llewellyn Morgan, M.D., 15, Weymouth-street, Portland-place, W., Surgeon to the Central London Throat and Ear Hospital.

1860 Thompson, Edmund Symes, M.D., F.R.C.P., 33, Cavendish-square, W., Senior Physician to the Hospital for Consumption, Brompton; Gresham Professor of Medicine. VP, O, S 3, C 3, SM.

1884 Thompson, Sir Henry, M.B., F.R.C.S., 35, Wimpole-street, W., Consulting Surgeon to University College Hospital; Emeritus Professor of Clinical Surgery in University College.

1873 Thomson, John Roberts, M.D., F.R.C.P., Bournemouth, Hants.

1876 Thornton, John Knowsley, M.B., M.C., 22, Portman-street, W., Surgeon to the Samaritan Free Hospital.

1867 Thorowgood, John Charles, M.D., F.R.C.P., 61, Welbeck-street, W., Physician to the City of London Hospital for Chest Diseases; Lecturer on Materia Medica at the Middlesex Hospital. LL, L, S 2, SM, C 3.

1854 Thudichum, John William Louis, M.D., F.R.C.P., 11, Pembroke-gardens, Kensington, W. VP, LL, O, C.

1876 Tibbits, Herbert, M.D., 68, Wimpole-street, W., Senior Physician to the West End Hospital for Diseases of the Nervous System.

1867 Timms, Godwin William, M.D., M.R.C.P., 9, Wimpole-street, W., Senior Physician to the North London Hospital for Consumption.

1836 Townley, James, F.R.C.S., 302, Kennington-park-road, S.E. c 3.

1865 Travers, William, M.D., F.R.C.S., 2, Phillimore-gardens, Kensington, Assistant Physician to the Chelsea Hospital for Women.

1884 *Treves, Frederick, F.R.C.S., 18, Gordon-square, W.C., Surgeon to, and Lecturer on Anatomy at, the London Hospital.

1882 *Tuke, Charles Molesworth, 37, Albemarle-street, W.

1868 Tuke, Thomas Harrington, M.D., F.R.C.P., 37, Albemarle-street, W. c 2.

1884 Turner, G. R., F.R.C.S., 49, Green-street, Grosvenor-square, W.

1883 Tweedy, John, F.R.C.S., 24, Harley-street, W., Assistant Ophthalmic Surgeon to University College Hospital.

1878 Vasey, Charles, 5, Cavendish-place, W., late Dental Surgeon to, and Lecturer on Dental Surgery at, St. George's Hospital.

1883 Venning, Edgcombe, F.R.C.S., Cadogan-place, Belgrave-square, S.W., late Surgeon 1st Life Guards.

1874 Verley, Reginald Louis, F.R.C.P. Edin., 28B, Devonshire-street, Portland-place, W.

1850 Waggett, John, M.D., F.R.C.S., 2, New-square, Lincoln's Inn, W.C.

1884 Wakley, Thomas, 96, Redcliffe-gardens, South Kensington, S.W.

1850 Wakley, Thomas Henry, F.R.C.S., 96, Redcliffe-gardens, South Kensington, S.W., Consulting Surgeon to the Royal Free Hospital.

1877 Walker, George, 12, Lingfield-road, Wimbledon, Surrey, W.

1869 Walker, John Swift, M.D., Hanley, Staffordshire. ns.

1869 Walker, Joseph, M.D., 22, Grosvenor-street, W., Dental Surgeon to Westminster Hospital; Lecturer on Mechanical Dentistry at the Dental Hospital of London.

1880 Walsham, William Johnson, M.B., C.M., F.R.C.S., 27, Weymouth-street, W., Assistant Surgeon to, and Demonstrator of Practical Surgery at, St. Bartholomew's Hospital. c.

1881 Warner, Francis, M.D., F.R.C.P., F.R.C.S., 24, Harley-street, W., Assistant Physician to, and Lecturer on Botany at, the London Hospital; Assistant Physician to the East London Hospital for Children.

1883 Waterhouse, William Dakin, LL.D., 18, Woodchurch-road, West Hampstead, N.W.

1872 Waters, John, 41, Bloomsbury-square, W.C.

1868 Watkins, Charles Stuart, 16, King William-street, Strand, W.C.

1863 Watson, William Spencer, M.B., F.R.C.S., 7, Henrietta-street, Cavendish-square, W., Surgeon to the Royal South London Ophthalmic Hospital, and to the Great Northern Hospital. c.

1881 Watteville, Armand de, M.D., M.A., B.Sc., 30, Welbeck-street, W., Electro-Therapeutist to St. Mary's Hospital.

1884 WEBB, F. ERNEST, 113, Maida-vale, W.

1869 WEBSTER, FREDERICK RICHARD, St. Albans, Herts. NS.

1878 WEISS, HUBERT FOVEAUX, F.R.C.S., 30A, George-street, Hanover-square, W.

1882 WELLS, CHARLES, M.D., 13, College-crescent, Belsize-park, N.W.

1838 WELLS, JOHN ROBINSON, F.R.C.S., 20, Fitzroy-street, W. C 2.

1873 WELSH, JOSEPH, Knighton, Radnorshire.

1884 WEST, SAMUEL, M.A., M.D., 15, Wimpole-street, W.

1880 WHARRY, ROBERT, M.D., C.M., 6, Gordon-square, W.C.

1882 WHIPHAM, THOMAS TILLYER, M.A., M.B., F.R.C.P., 11, Grosvenor-street, W., Physician to, and Lecturer on Pathology and Practical Medicine at, St. George's Hospital. SM.

1884 WHISTLER, WILLIAM MACNEILL, M.D., M.R.C.P., 28, Wimpole-street, W., Physician to the Hospital for Diseases of the Throat and Chest.

1868 *WHITE, JOSEPH, F.R.C.S. Edin., Oxford-street, Nottingham, Consulting Surgeon to the Nottingham General Hospital.

1880 WHITE, WILLIAM HENRY, M.A., M.D., M.Ch., M.R.C.P., 43, Weymouth-street, W., Assistant Physician to the Royal Hospital for Diseases of the Chest. *Councillor.*

1883 WHITEHEAD, WALTER, F.R.S.E., 499, Oxford-road, Manchester, Surgeon to the Manchester Royal Infirmary.

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

1868 WIBLIN, JOHN, F.R.C.S., Southampton. NS.

1871 WILKINSON, JOHN SEBASTIAN, F.R.C.S., Sydney, N. S. W.

1862 WILLETT, EDMUND SPARSHALL, M.D., M.R.C.P., 4, Suffolk-place, Pall Mall, S.W. C 3.

1872 WILLIAMS, ARTHUR WYNN, M.D., 1, Montagu-square, W., Physician to the Samaritan Free Hospital.

1872 WILLIAMS, CHARLES THEODORE, M.A., M.D., F.R.C.P., 47, Upper Brook-street, Grosvenor-square, W., Physician to the Hospital for Consumption, Brompton. VP, LL, S 2, SM, O.

1876 WILLIAMS, HENRY WILLIAM, M.D., C.M., 168, Fulham-road, S.W.

1883 WILLIAMS, JOHN, M.D., F.R.C.P., 11, Queen Anne-street, W.

1883 WILLIS, ARTHUR KEITH, Gascony House, West End-lane, N.W.

1881 WILLS, CALEB SHERA, C.B., Surgeon-Major, Army Medical Department.

1873 WILLS, THOMAS MUNNS, F.R.C.S.I., 44, Merton-road, Bootle, Liverpool, Honorary Surgeon to the Bootle Hospital.

1870 WILTSHIRE, ALFRED, M.D., F.R.C.P., "Torridon," Somers-road, Reigate, Physician-Accoucheur to, and Joint Lecturer on Midwifery at, St. Mary's Hospital. VP, LL, S 2, C 3. *Treasurer.*

1884 WINSLOW, H. FORBES, M.D., 14, York-place, Portman-square, W.

1873 WINSLOW, LYTTLETON STEWART FORBES, D.C.L., LL.M., M.B., M.R.C.P., Hammersmith, W., Lecturer on Mental Diseases at Charing Cross Hospital. C.

1876 Woakes, Edward, M.D., 80, Harley-street, W., Senior Aural Surgeon to, and Lecturer on Aural Surgery at, the London Hospital.

1882 Wolfenden, Richard Norris, B.A., M.D., 19, Upper Wimpole-street, W., Lecturer on Physiology at Charing Cross Hospital.

1873 Woodhouse, Robert Hall, 1, Hanover-square, W., Surgeon to the Dental Hospital.

1879 Woodman, Samuel, F.R.C.S., 5, Prospect-terrace, Ramsgate.

1880 Woollett, Sidney Winslow, Kessingland, Suffolk.

1873 Wordsworth, John Cawood, F.R.C.S., 20, Harley-street, W., Consulting Surgeon to the Royal London Ophthalmic Hospital. c 3 vp.

1884 Yeo, T. Burney, M.D., F.R.C.P., 44, Hertford-street, Mayfair, W.

1870 Ziffo, Jean E., Constantinople. ns.

GENERAL MEETINGS.

November 5th, 1883.

SIR JOSEPH FAYRER, LL.D., M.D., President, in the Chair.

THE minutes of the last general meeting were read and confirmed.
No further business.

March 3rd, 1884.

SIR JOSEPH FAYRER, LL.D., M.D., President, in the Chair.

THE ballot for the election of Officers and Council for the year 1883 was declared open, and Mr. Heycock, Mr. Parker, and Mr. J. Peake Richards were appointed scrutineers.

The following report of the Council having been read was received and unanimously adopted.

The Annual Report of the Council presented to the General Meeting, March 3rd, 1884.

The Council of the Medical Society of London, in meeting the Fellows at the conclusion of a busy and eventful year, has much pleasure in congratulating them upon the progress which the Society has made, and upon the success which has attended its work.

The reconstruction of the Society's premises, commenced in September, 1882, was pushed forward with such celerity that the new suite of rooms was formally opened by the President on Monday, the 2nd of July last, to which date the annual oration and conversazione were postponed by a special vote.

It afforded the Council much gratification that the Society was honoured by the presence of H.R.H. the Prince of Wales at this its inaugural entertainment.

It was also the source of great satisfaction to the Council that it was represented on this occasion by so able an orator as Professor Lund, whose masterly address on the "Present Bearing of the Antiseptic Question" took a worthy place as the first paper read within these walls.

The Council is pleased to find that the acoustic properties of the new meeting room are the subject of universal commendation; and considers that in this respect, as well in its artistic qualities, the design, which was not without difficulties, reflects the highest credit upon the architect, Mr. Henry Cowell Boyes.

The total cost of the reconstruction of the whole block of buildings,

Nos. 11 and 12, Chandos Street, including fittings, has amounted to £3858.

The architect's commission amounted to £192 18*s.* 6*d.*, and the legal charges in respect of the agreement, lease, temporary loan and mortgage, to £255 1*s.* 4*d*—£4306 in all.

Of this sum, £4000 was provided by a mortgage upon the property, the temporary advance of £2300 so kindly made by Sir Erasmus Wilson being repaid; and the remainder was defrayed from the funds in hand of the Society.

Interest at the rate of 5 per cent. per annum, *i.e.* to say £200 a year, is paid upon the mortgage above mentioned, making, with the ground rent of £300, a total annual rental of £500 a year.

Per contra, portions of the premises, which had to be constructed on a scale in excess of the Society's requirements, and the use of the Society's rooms on specified occasions, have been let to the following tenants, viz.: the Entomological Society, the English Cart Horse Society, the Ophthalmological Society, the Epidemiological Society, the Dermatological Society, the Royal Historical Society, the St. Andrew's Graduates Society, the International Exhibition Co-operative Wine Society, Mr. Henry Mills, and Mr. John Henry Phillips, at an aggregate rental of £622 16*s.* a year.

From minor lettings of the meeting room for the use of scientific and literary Societies, the Council estimates that an additional £40 or £50 per annum will be realised, making the gross receipts about £670 in all.

It will be seen, therefore, that the Society may expect to obtain, after deducting £120 for rates, insurance, and repairs, a *net* profit of £50 a year from the reconstructed premises, a sum which, if applied to the purposes of a sinking fund, will more than balance the capital expenditure at the end of the lease, besides holding its own suite of rooms and the Registrar's chambers, the rental of which may be reckoned at £400 a year, without further charge.

No less than sixty-two new Fellows, an unprecedented number, have been elected during the year; and in addition, fifteen of the "Non-Subscribers" admitted during the years 1868—72 have become Subscribing Fellows under Law XI, making a gross addition of seventy-seven to the Subscribing Fellows of the Society.

The Society has, on the recommendation of the Council, conferred the Honorary Fellowship on the following distinguished members of the profession:—George Edward Paget, D.C.L., LL.D., M.D., F.R.S., Regius Professor of Physic in the University of Cambridge; George Murray Humphry, M.D., F.R.S., Professor of Surgery in the University of Cambridge; David Yandell, M.D., Professor of Surgery in the Louisville University, Kentucky, U.S.A.; A. Le Roy de Méricourt, M.D., of Paris; and begs to recommend that a similar honour be conferred on Baron Larrey.

We have to deplore the loss of the following Fellows, who have been removed by death:—Sir Robert Christison, Hon. Fellow; Dr. Boyd, Dr. Grindrod, Mr. Hodge, Mr. Montefiore, Mr. Keene, Dr. Meredyth, and Dr. McOscar, Subscribing Fellows; Mr. Jardine Murray, Dr. Crossley Irwin, Dr. John Osborn, and Dr. Marion Sims, Non-Subscribing Fellows.

Five Fellows have resigned their Fellowship during the year. The net increase in the number of Fellows is therefore sixty-two; in the number of Subscribing Fellows, sixty-five.

The scientific work of the Society, even during the months of March and April last, when the Fellows met at the cost of considerable personal discomfort, showed no lack of vigour; and since the opening of the new rooms has afforded fresh facilities for such work, the interest and importance of the Society's proceedings have been patent to the whole medical world.

The Council is pleased to learn from the President and Secretaries that the arrangements made for the remaining part of the Session promise a series of meetings not inferior in interest to those already past.

It is satisfactory to note that even during the discomfort of the latter part of last Session the attendance only once fell below thirty, and averaged thirty-six. Since the opening of the new rooms the attendance has averaged seventy, the lowest number being thirty-six, the highest one hundred and thirty-two.

The Society was peculiarly fortunate in securing, in the person of Mr. Brudenell Carter, a Lettsomian Lecturer who could place the details of his subject before the Fellows in a manner which for completeness, scientific insight, and perfection of style may well serve as a model to all future medical speakers or writers. The Council is pleased to say that Mr. Carter has kindly acceded to the request of the Fellows that he would publish his Lectures in a separate form.

The Hon. Secretaries regret that the strain of work which the reconstruction laid upon them prevented their commencing the task of editing Vol. VI of the 'Proceedings' until the autumn. The whole is now in the press and will very shortly be issued to the Fellows.

In view of the importance of a question which is occupying a prominent place in public thought the Council has authorised the President to make arrangements for holding a conference on "The Influence of Modern Systems of Compulsory Education and Competitive Examination on the Mental and Physical Health of the Community" in connection with the forthcoming International Health Exhibition some time during the summer months. It is hoped that the Society will thereby tend to elucidate a matter gravely concerning the public welfare.

No essays have been sent in in competition for the Fothergillian Medal this year. Possibly this was due to the wide extent of the subject proposed.

Considering that the custom of annually conferring silver medals was no longer conducive to the interests of the Society the Council resolved to make no award of such medals this year.

Having regard to the complex nature of the business connected with the Leasehold property in Chandos Street, and the need for some experience of its details in the officers called upon to deal with it, the Council at its meeting on February 4th passed the following resolutions:

"1. That there shall be a permanent House and Finance Committee, consisting of the following *ex officio* Members, viz. the President, the Trustees of the Personal Property, the Treasurer, and the Hon. Secretaries for the time being, and of not less than five other Fellows of the Society, who shall be appointed by the Council, and may hold office for a period not exceeding five years.

"2. That to this Committee shall be entrusted the Management, subject to the control of the Council, of all matters relating to the Society's Leasehold Property and Buildings in Chandos Street.

"3. That the Committee shall at its first meeting in each year elect a Chairman from among its members, and that such Chairman shall be its Executive Officer.

"4. That such Chairman shall become on election *ex officio* a Member of the Council.

"5. That this Committee shall inspect the buildings under its charge at least once a year, and shall make a report to the Council at its regular Meeting in February.

"6. That the minutes of the proceedings of the Committee shall be regularly kept by the Hon. Secretaries, and shall be read at the Meetings of the Council next following the Meetings of the Committee.

"7. That the following amendments to the laws be read from the Chair at the four next ordinary Meetings, and suspended in the Library, and become law unless challenged.

"In Law 17. After 'Trustees,' to insert 'and the Chairman of the House and Finance Committee, if not otherwise a Member.'

"In Law 19. After 'Trustees,' to insert 'and the Members of the House and Finance Committee.'

"8. That the following Fellows, viz. Dr. de Havilland Hall, Mr. D. H. Goodsall, Dr. T. Gilbart-Smith, Mr. Edmund Owen, and Dr. Isambard Owen be appointed to constitute the House and Finance Committee, together with the *ex officio* Members above named, and that the Committee thus constituted shall date from March 3rd, 1884, on which day the present Lease and Building Committee shall cease to exist."

The above-mentioned alterations in the Laws were read from the Chair at the ordinary Meetings on Feb. 4th, 11th, 19th, and 25th, before nine o'clock, and, being unchallenged on any of these occasions, forthwith became law.

The Council begs to express its deep sense of the Society's obligations to its President, Sir Joseph Fayrer, not merely for the tact and courtesy with which he has conducted its deliberations, and the numerous contributions which he has made to its 'Proceedings,' but even more for his unsparing personal efforts in advancing the welfare, extending the operations, and ensuring the success of the Society, in every department of whose work his influence has been powerfully felt.

To the unostentatious but arduous labours of Dr. Allchin, the Hon. Librarian, in rearranging and recataloguing the Society's valuable collection of ancient and modern books, the gratitude of the Fellows is also especially due.

Also to the late President, Mr. Francis Mason, who so kindly undertook the Treasurer's duties during Dr. Wiltshire's temporary retirement on account of illness. The Council is pleased to announce Dr. Wiltshire's restoration to health and his approaching return to London.

The Council feels that it would be failing in its duty if it did not take especial notice of the services rendered to the Society by its retiring Secretary. The conduct of the Society through the troubles and difficulties of the past year to its present satisfactory position is mainly due to the arduous and unremitting exertions of Dr. Isambard Owen. The Council feels that the election of Dr. Owen to the Secretariat at a time of so much anxiety has been a most fortunate event of unusual importance to it. Before and during the reconstruction of the premises Dr. Owen's architectural knowledge and talent was of the

utmost service, and in the conduct of the arrangements connected with the lease, mortgage, and agreements of the Society, Dr. Owen's familiarity with legal proceedings stood the Society in the best stead. In addition to these extraordinary duties, which have been freely and successfully undertaken by him, Dr. Owen has also transacted all the other duties of his office with precision, courtesy, and success, unsurpassed by any of his predecessors; and in now retiring from office he carries with him the congratulations, good wishes, and gratitude of every Member of the Council.

Nor can the Council overlook the invaluable services of those Fellows who, with the Trustees and Officers of the Society, carried on the heavy work of the Lease and Building, and of the Conversazione Committees. To these gentlemen, viz. Dr. Theodore Williams, Dr. Brodie Sewell, Dr. de Havilland Hall, Dr. Gilbart-Smith, Dr. White, Mr. Bryant, Mr. Francis Mason, Mr. Goodsall, Mr. Edmund Owen, and Mr. Henry Morris, the cordial thanks of the Society are due.

To Mr. Goodsall, who arranged the financial portion of the Society's building scheme at the cost of much time and trouble, the Fellows are especially indebted.

Nor can the Council forget the generosity of those Fellows of the Society who so liberally subscribed to defray the cost of the Conversazione in July last, and to carry it out in a manner worthy of the occasion.

And, in conclusion, the Council must once again express its high appreciation of the untiring zeal of its old and trusted friend, Mr. W. E. Poole.

The Report of the Honorary Librarian.

Mr. President and Gentlemen,—I have the honour to present my report as to the condition of your Library during the past year.

The admirable and extensive changes which the Society has entered upon in its building have benefited no department of its work so much and so satisfactorily as the Library.

For the first time for some years at least, the valuable books have been collected together from the various corners, in which from cellar to garret of the old quarters they had been hidden, and are now arranged on the walls of a suitable and commodious room.

For their arrangement substantial book-cases have been provided, and on the shelves the volumes have been placed in such a manner that they are grouped under the main divisions of subjects which ordinarily constitute a medical library, leaving space for additions in the several divisions.

The labour entailed in removing the books from place to place, many of them being lifted four times before being finally deposited, was considerable, but unavoidable in the cramped and inconvenient space available whilst the building was in progress. In this work I was ably assisted by Mr. Poole, and for part of the time by his son, whose services the Council have acknowledged.

I regret to inform the Society that the general state of the books is very far from satisfactory; fire, water, and, chief of all, dust, have wrought a disastrous effect upon the condition of many of the volumes, an effect which can only be partially remedied. The Council have placed at my disposal a small sum to do the best in the way of repairs with some of the volumes as they are catalogued, and I trust to be able

to report next year that the progress of damage is at least arrested, and as far as possible remedied.

Although every volume on the shelves has passed through my hands I am not in a position yet to furnish the Society with anything like a report as to the general character of their literary possessions. But this I may say, that whilst present day works are but scanty, there is a very fair representative collection of the works of the last and early part of the present century; but the strength of the Library especially lies in the sixteenth and seventeenth century books, which constitute a collection of which the Society has just reason to be proud. Some additional book-cases lately supplied to the library are intended to accommodate these volumes amounting to about 2500, and the work of arranging them will be commenced forthwith.

The journals and periodical literature are disposed in the smaller room, and for the present sufficient accommodation exists for them.

The Council has sanctioned the expenditure of a certain sum for the glazing of two of the sections, a proceeding that sooner or later must be carried out completely, the heavy cost alone at present standing in the way of so doing.

Of necessity during the changes and rearrangement the lending of volumes to members has been much restricted, and I can make no report of the extent to which the Library has been used.

The existing Catalogue being obviously inapplicable to the present rearrangement the work of preparing a new one has had to be undertaken, and I am busily engaged upon that work at the present time. The preliminary preparation of the book before the actual cataloguing takes place demands much more care and occupies far more time than I had anticipated, and I regret that I cannot make faster progress with it. Meanwhile the general systematic arrangement of the Library permits of such of the volumes required being found with but little trouble.

The fund at the disposal of the Society for the purchase of new books has not yet been touched, and I have not felt justified at present in advising that it should be used, and this for the reason that all our energies are required in dealing with the volumes we have until the new Catalogue is ready. So soon as possible the much needed additions shall be purchased so far as the limited sum at our command is available.

The Library Committee has met four times.

W. H. Allchin.

The subject for the Fothergillian Essay for the year 1886 was then announced to be: "On the Nature of the Fevers usually termed in this country 'Febricula,' 'Simple Continued,' and 'Modified Typhoid.'"

THE MEDICAL SOCIETY OF LONDON.—BALANCE SHEET, 1883-84.

1883	RECEIPTS.	£	s.	d.	1883	PAYMENTS.	£	s.	d.
	To Balance from last Account	148	0	7		By Rent, Gas, Coals, Rates, Taxes, and Insurance	387	3	0
	„ Subscriptions and Arrears	403	14	6		„ Library Expenses	33	3	4
	„ Entrance Fees	47	15	4		„ Furniture and Repairs	100	3	11
	„ Life Compositions	47	5	0		„ Builder, Balance of Account	158	15	11
	„ Rents	534	2	2		„ Architect's Commission and House Agent's ditto	198	3	6
	„ Contributions for use of Rooms	31	10	0		„ Grants, Conversazione, &c.	47	5	5
	„ Interest	3	0	8		„ Salary and extra attendance of Registrar	107	17	0
	„ Loan from Bankers	200	0	0		„ Printing	17	18	6
						„ Interest on Loans	102	10	8
						„ Law Expenses	3	15	6
						„ Collector's Poundage	24	18	8
						„ Current Expenses (including Wages, Postage, Stationery, Advertisements, Refreshments at Meetings, and Bankers' Charges	100	16	0
						Balance	132	16	10
		£1415	8	3			£1415	8	3

Feb. 4th, 1884. (Signed) FRANCIS MASON, *Treasurer, pro. tem.*

Examined, compared with the Vouchers, and found correct,

Feb. 11th, 1884. (Signed) WM. HENRY WHITE, M.D., WM. EWART, M.D., } *Auditors.*

PROCEEDINGS

OF THE

MEDICAL SOCIETY OF LONDON.

111TH SESSION.

October 29th, 1883.

OPENING ADDRESS.

By the President, Sir JOSEPH FAYRER, K.C.S.I., M.D., F.R.S.

GENTLEMEN,—Before we proceed to the regular business of this evening's meeting, I desire, with your permission, to make a few introductory remarks on the occasion of our assembling after the recess to resume the work of the 111th session of this ancient Society, which though venerable in years and experience is always, I trust, vigorous, energetic, and flourishing as its many and more youthful compeers. I am sure you will agree with me that the present is an eventful period in the annals of the Medical Society, and one of such importance as to merit special notice, and whilst reminding you of this I would invite you, at the commencement of a new and important era in its history, to determine to do all in your power to infuse new energy and life into its work, so that it may not only continue to maintain the high position it has so long enjoyed, but that it may still further advance, keeping pace with the progress of scientific and intellectual culture which characterise the age in which we live generally, and so eminently distinguishes the profession of medicine in particular, thus extending the sphere of its influence and utility, and vindicating its title to the high place to which from

its traditions, its ancient renown, its present work, and its relation to other societies which have sprung from it, it is so justly entitled. It is needless to recount to you the various events which have marked the origin, rise, and progress of the Medical Society of London. You probably all know that it was founded by a physician of great eminence when George III was king, that it is the oldest medical society in the metropolis, that from it other societies have taken rise and have so prospered as well-nigh to outgrow and overshadow their parent; and that a long roll of names distinguished in various departments of medicine enumerates its presidents, lecturers, orators, and prizemen, whilst its 'Transactions' contain numerous contributions to medicine of the greatest value.

Dr. Routh, a former President, and Dr. Symes Thompson, a former Orator, have told us in their interesting addresses of the ups and downs, the vicissitudes and changes of fortune it has undergone; how it was amalgamated with other societies, and from time to time changed its local habitation, though not its name; how, from small beginnings in the east, it gradually grew and migrated to the west of London, always tending upwards to a higher standard of practical excellence and utility, until it finally settled in its present home, where, we trust, a renewed career of usefulness will follow the recent step in onward progress which was so happily inaugurated last July by the presence of H.R.H. the Prince of Wales, whose interest in the Society on that occasion was so warmly manifested and so graciously expressed. Now, all this has only been achieved by much labour and perseverance; and it is to the indomitable energy of the Fellows, the untiring labour and interest in its welfare which have been evinced by its officers, and to the firm determination of all concerned that the Society should prosper, that we owe the present happy position in which it is placed, and to which we turn with confidence as an augury of future success. And here, gentlemen, I am sure that I express the sentiments of the whole body of Fellows when I say how deeply we are indebted to our late President, our Council, Secretaries, Librarians, and Registrar, for all they have done, and for having secured for us the great advantages we now enjoy. It is for you to maintain the high prestige, scientific value, and practical utility of the Medical Society and to develope it to a further pitch of excellence that shall be commensurate with the aims of an institution which claims, in true catholicity of purpose,

to represent the work of all branches or departments of medical science.

I am not going to weary you with a long address, and merely propose to occupy your attention for a brief space with a few observations germane to the present occasion and having reference to matters which concern the work of the session before us. I shall then make way for one to whom we shall all eagerly listen, for I am happy to think that our first meeting will be devoted to a subject which must be of deep interest when treated by the great surgeon who has already placed his name as high on the roll of benefactors of the human race as on that of men of science.

But let me first say a few words on the actual condition of the Society, which, I rejoice to think, is altogether satisfactory. The Fellows now number as follows:—Honorary Fellows 39, Corresponding 60, Ordinary 376, Non-subscribing 70, making a total of 545. Of these 47 Ordinary Fellows were added between October, 1882, and May, 1883, and 18 since then are candidates for admission. I trust we shall add largely to that list during the present year.

Our financial position is satisfactory; we certainly are not rich, but we *are* in a position that may be considered as encouraging. The official reports have given you details of receipts and expenditure, and you would learn from them that our disbursements on account of building, furniture, &c., have been heavy.

The Treasurer's report will have informed you how we have met, or are going to meet, the heavy demands accruing from the building of the new rooms, and you probably have heard how much we have been indebted to a late President, Sir E. Wilson, for his generous assistance in this respect. We must all regret that he is not here to see the changes which he has so liberally aided in effecting, and still more regret that ill-health has deprived us of his presence, whilst we express a hope that at no very distant period we may again welcome him among us.

So far, gentlemen, I have had to speak of prosperity and success. I must now turn to the reverse, for we have that also to consider. Still I am thankful to say that, considering our numbers, the proportion of losses by death is not numerically large, though very severe when measured by the value of some of those removed. We have to deplore the death of three of our Fellows, and in one case under circumstances of so sad a nature as greatly to enhance the grief felt

for his loss by the Society, the profession generally, and by his own family and numerous friends. It is with peculiar sorrow that I refer to the death of an old and distinguished Fellow, Dr. R. Boyd, who lost his life in company with his son and three other persons when endeavouring to save the lives of others during the disastrous fire which, at one fell swoop, deprived his family of a much-loved father and brother (and of their valuable property), the profession of an able and accomplished physician, and many, of a much-loved and respected friend.

Dr. R. Boyd was an M.D. of Edinburgh, F.R.C.P. of London, and a Fellow of the Medical and other Societies. He had reached the mature age of seventy-five, but was full of physical and intellectual vigour. He had long been known and eminent as an alienist physician, and had contributed largely to physchological and pathological science; on these subjects his communications were numerous and valuable. At the time of his death he was superintendent and, I believe, proprietor of the Southall Park Asylum which, on the 14th August last, was totally destroyed by fire. It was in his efforts to save the lives of those committed to his care that he and his gallant son, Mr. W. B Boyd, met with their deaths, and thus sadly though nobly terminated a life which had been devoted to good works. The Fellows of the Medical Society will join with me in an expression of deep sorrow for his loss and sympathy with his bereaved family and sorrowing friends, whose deep and lasting regret must, like ours, be mingled with admiration for the heroism with which he and his brave son gave up their lives in the attempt to save those of others.

Mr. Alfred Ebsworth, a Fellow of the Royal College of Surgeons, and Licentiate of the Apothecaries Society, and who resided in Henrietta Street, became a Fellow of the Society in 1864. He read a paper on nursing institutions before the Society, but does not appear to have been a very frequent attendant at the meetings. Probably his professional avocations left him little leisure for other work. From all I have been able to learn he was a man of much general information, and was well known among the brethren of the mason's craft.

Mr. William Field, of Kingsbury, Middlesex, who died during the past year, was one of the oldest Fellows of the Society, having been elected in 1847. He was not a frequent attendant at the meetings, and does not appear to have made any communications,

but showed his interest in it by liberally contributing to the funds of the Society.

The last and not the least of the losses we have sustained, though happily not by death, is that of our Treasurer, Dr. A. Wiltshire, and in alluding to him I feel sure that I shall enlist your warm sympathy and concurrence when I express the deep regret we all feel for the serious illness which has deprived us of a much-esteemed colleague. He joined the Society in 1870, and has been one of its most active supporters. He has filled the office of Vice-President, Secretary, Councillor, Lettsomian Lecturer, and was Treasurer until a few weeks ago, when serious illness compelled him temporarily to resign that office. He had worked hard and written many valuable papers for the journals and societies, on midwifery, on tetanus, delirium tremens, and other subjects, and was, I believe, engaged on a new book when attacked by the disease which has deprived us of his valuable services. It seems but the other day that he presented all the appearance of vigorous health and intellectual activity, and to have been struck down just at that period of life and of his professional career when his energy and merit had attained for him a position which promised the realisation of his natural ambition and the well-earned reward of his lifelong devotion to his work. You will all join with me in condolence and sympathy for his affliction, and in the hope that he may soon be restored to health, to the Society of which he was a brilliant ornament, and to the family and friends who are in anxiety on his account.

Let me now briefly refer to the work of the session before us. I congratulate the Society on the prospect of receiving many communications on subjects of practical interest from men of the greatest eminence in various departments of medicine. I have already in a short circular letter which I had the honour of addressing to the Fellows alluded to these, and it is needless for me to repeat what I then said, but I may just remark that I think we are especially fortunate in the selection of the subject with which our first meeting is to be occupied, and that the Society will have the opportunity of enriching its annals with a paper of great interest. As with all other original investigators, Sir Joseph Lister's doctrines on the antiseptic system have been subjected to much criticism and severe experimental test, and, like them also, on the one hand they have been received and adopted generally with enthusiasm,

whilst on the other, by a few with doubt and even distrust. Of the validity of the theory by which the distinguished author of the antiseptic system, explains its good effects there may perhaps be question, but of the valuable character of its results there can be, I think, but one opinion. Time and experience may modify the explanation of the *modus operandi* and the nature of the details of its practical application, but they can hardly detract from the value of the system itself. I trust that Professor Lister's paper may deal with the subject; that it will be fully discussed; and that your views and experience with regard both to the theory and its practical working may be freely expressed, and also that Sir Joseph Lister will tell us not only the actual state of the question from his own point of view, but how far he may be disposed to modify any pre-existing views and any details of application which his more recent experience may have induced him to adopt. The list of papers in possession of our Secretaries certainly affords prospect of a most interesting session, for the subjects are especially suited to the Medical Society, which above all things desires to be practical, and I am happy to find that many of the promised communications come from Fellows or others who practise in different parts of the United Kingdom. Needless to add that I have no intention in saying this of appearing to undervalue those from our brethren in London. I mean that I am glad to find that our Society is sufficiently appreciated by non-resident Fellows and others at a distance to induce them to communicate to us their experience. I cannot now attempt to comment in detail on special papers in the programme, but I would merely say in anticipation that personally I look forward with unusual pleasure and prospect of profit to some of the subjects, and especially I would refer to one on "Cathetic Fever" by Sir A. Clark, and about which I have written not a little, as it will probably throw light on a subject that came much under my notice in India; and to that by Dr. S. Mackenzie on "Paroxysmal Hæmoglobinuria" which will be to me as to all most acceptable. And here, if I might be permitted to make a suggestion, it would be that the subject of the diagnostic and prognostic significance of albuminuria may be brought before this Society: it is one on which I think much of practical clinical value has yet to be said.

Among other communications one from Surgeon-General Sir Guyer Hunter (who has recently returned from Egypt), on the epidemic of cholera which has prevailed during the last six months in

the delta of the Nile, promises to be of special interest. I am sure the Medical Society will join with me in congratulating this distinguished medical officer on his safe return from an arduous and difficult duty, which has been performed with the ability, judgment, and success which were to be expected from an officer whose high professional character and administrative capacity were so conspicuously manifested during the tenure of his high office as Surgeon-General of the Bombay Presidency.

Sir Guyer Hunter's paper no doubt will give us valuable information as to the origin and diffusion of cholera in Egypt, and, if I mistake not, will go far to controvert certain views which have been advanced thereon and add further proof, were any needed, of the futility of that incubus of commerce and international communication, *quarantine*, which still has so firm a hold on the belief of other European powers and which is still practised to the detriment alike of trade, of commerce, and of health.

The etiology of cholera is indeed a subject of vast interest in its practical bearing. To know the *vera causa* of any disease is of great importance, for it is not until we really have this knowledge that any really rational mode of dealing with its prevention can be postulated, and to ascertain it no effort in research should be spared. We are far from possessing that knowledge of it as yet in respect of cholera, and men of equal experience of the disease still differ totally as to the etiology. In dealing with it as sanitarians we are bound to act on ascertained facts and not on theories, and albeit we know much regarding the laws of its movement, I venture to think we are not yet in a position to declare that we have discovered either its real cause or the mode of its diffusion. It is satisfactory to know that the subject is occupying the attention of several great micro-pathologists at present, and let us hope that their efforts may meet with success.

Experience in India has certainly taught us that in the ordinary sense of the term cholera is not contagious, and has demonstrated the futility of all *quarantine measures*. Experience has also taught us the inestimable value of sanitation both in preventing its occurrence and limiting its propagation and diffusion, and there is no evidence to show that it has *ever* been conveyed to Egypt by ships during all the years that that have elapsed since the opening of the Suez Canal and the consequent continuous stream of traffic from Indian ports which are never free from the disease. And yet

it has been attempted to show that the recent epidemic was so brought to Egypt until clear proof was demonstrated that such was not the case. I think it will be found that not the least valuable result of Dr. Hunter's mission is the light he has been able to throw on this aspect of the subject.

Happily, though different views are held both here and in India as to the etiology and the diffusion of cholera, all are agreed that quarantine is useless. According to those who hold the non-contagionist view, quarantine is as irrational as it is mischievous. Whilst those who believe in its transmissibility by human agency, in ships' cargoes, think quarantine is useless because it can never be practically and efficiently carried out. Our continental friends argue, being firmly impressed with the contagionist view, that there is reason logically why quarantine should be made more strict, and they look with disfavour on our substitutes of inspection and segregation of those who are affected. The subject is one of vast importance not only from its medical, or rather I should say epidemiological point of view, but also as a political and international question of interest, and any information that can bear practically on it is of value. But this is neither the time nor the occasion for dwelling on the subject, interesting though it be; and I shall not trespass on your indulgence further, or anticipate what Dr. Hunter may have to say in regard to it. It only remains for me to say that I am delighted to meet you all again; that I trust the session will be a very prosperous one, that the attendance will be numerous, the discussions vigorous and critical as usual, and that everything we may do will be for the advancement of medicine and the furtherance of all the best interests of our noble profession.

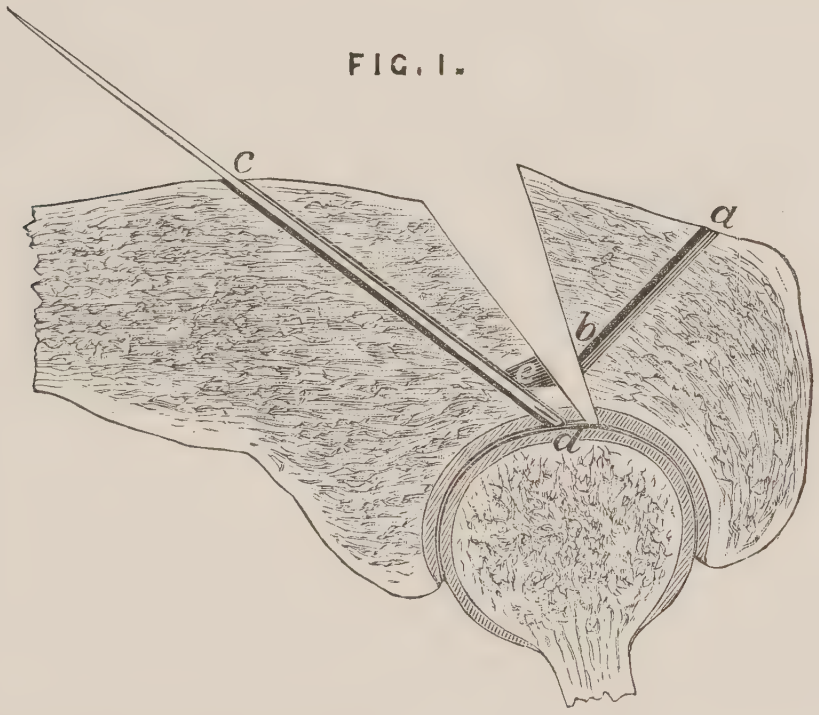
ON THE TREATMENT OF FRACTURE OF THE PATELLA.

By Sir Joseph Lister, Bart., F.R.S.

Sir Joseph Fayrer and Gentlemen,—Some time ago Mr. Holmes remarked to me that it would be well for me to place before the profession statistics of the operations which I had performed for fracture of the patella; and when you, Sir, did me the honour to request that I should open this session of the Medical Society with a paper, it occurred to me that I could hardly

do better than act on Mr. Holmes's suggestion. But before entering on the strict subject of the communication which I have the honour to bring before you, it will be advisable to make some prefatory remarks regarding the circumstances that led me to it. In March, 1873, my friend Dr. Hector Cameron, of Glasgow, recommended to my care, in the Edinburgh Infirmary, a case of ununited fracture of the olecranon. Dr. Cameron had formerly been my house surgeon in the Glasgow Royal Infirmary, and I had

FIG. 1.



afterwards for several years the great advantage of his assistance in private practice; and he reminds me that I had often expressed to him the opinion that the use of a metallic suture, antiseptically applied, which we had employed in ununited fracture of the shafts of the long bones, ought to be extended to the olecranon and patella.

The patient to whom I refer presented himself to Dr. Cameron as an out-patient, and as he could not at that time operate himself, he sent him to me. He was a man thirty-four years of age, who five months previously had received a blow from a policeman's *baton* on the left elbow. This occasioned great swelling, which seems to have concealed the true nature of the case from the surgeon whom he first consulted. On admission, there was a considerable interval between the olecranon and the shaft of the bone, and although the limb was muscular, it was comparatively helpless, as he could not extend the forearm at all without the aid of the other hand. On March 28th, I made a longitudinal

incision, exposing the site of the fracture; and having pared away the fibrous material from the fractured surfaces, I proceeded to drill the fragments in order to apply the suture. The fracture was oblique from before backwards, as shown in Fig. 1. I found no difficulty with the proximal fragment in making the drill appear upon the fractured surface at a little distance from the cartilage (see *b*, Fig. 1); but with the other fragment the obliquity of the position in which the drill had to be placed was so great that, instead of emerging at the fractured surface, as I had intended, I found it had entered into the substance of the humerus (*d*, Fig. 1). I therefore withdrew the drill, and substituted for it a needle (*c*, *d*). Then with a gouge I excavated an opening (*e*) upon the fractured surface opposite to the drill-hole (*b*) on the other surface, until the needle was exposed. Withdrawing the needle, I introduced a silver wire in its place, and had no difficulty, by means of forceps passed into the excavation made by the gouge, in drawing out the wire. I was then able to pass it on through the other drilled opening, and thus the two fragments were brought into apposition. The ends of the wire were twisted together, and left projecting at the wound. Healing took place without suppuration or fever, and the wire was removed on May 19th, seven weeks after the operation. The wound made for its extraction soon healed, and the patient returned to Glasgow; and I afterwards had the satisfaction of learning that he was wielding the hammer in an iron ship-building yard with his former energy.

I have had two other cases of united fracture of the olecranon; and as these are closely allied to the subject of my paper I may refer briefly to them.

One was a man, forty-five years of age, incapacitated for his occupation as a plasterer by inability to extend the right elbow completely. The ununited fracture of nine weeks' standing was oblique laterally. It was treated as in the last case, the operation presenting no difficulty. It was performed on March 20th, 1878. The wound healed without suppuration, but the wire was not completely removed; for, the loop having given way near the twist, the twisted part alone was taken away, and the loop left behind. It never caused any inconvenience, and he afterwards wrote to us from his home at Bristol that he was able to follow his old employment.

The third case was that of a gentleman, thirty-three years old, who had consulted no fewer than eighteen surgeons on account of the weakness of his left arm, caused by ununited fracture of the olecranon. I operated on July 28th, 1881, paring the broken surfaces as in the other cases, using a chisel and hammer for the purpose; and having drilled the fragments with a common bradawl, I brought them together with moderately stout silver wire. In this case, however, I did not leave the ends of the wire projecting from the wound; but having given them one complete twist (or two half twists), cut the ends off short and with a small hammer hammered the twisted part down flat upon the ulna. The advantage of this practice was strikingly exemplified by the difference in the course of this case from its predecessors. Instead of keeping him under treatment for several weeks until the wire could be removed, I was enabled to allow him to return fifteen days after the operation to his home in Wales with a sound cicatrix, and, trusting to

the connecting loop of wire, permitted him to use the elbow freely. I afterwards learned that he was able to drive a four-in-hand as well as ever.

The practice of cutting the ends of the wire short and hammering down the twist upon the bone is one to which I shall have to refer again in connection with my later cases of fracture of the patella. It is in every respect an advantage. The hammering down of the twist renders it more secure than if it is left projecting, to be moved by every shifting of the dressing, and perhaps broken, as in the second of the cases just referred to. We also get rid of a source of disturbance, and sometimes of considerable uneasiness, in the wound. The time of healing is greatly shortened; and the knowledge that the loop of wire securely holds the fragments in position allows the use of the joint to be commenced much earlier than when we have only the organic band of union to trust to. The practice is also of the highest value in ununited fracture of the shafts of the long bones. The thickness of the wire must be proportioned to the force to which it is to be subjected. For the olecranon, only about one twenty-fifth of an inch in diameter is amply sufficient. For the shaft of a femur of an adult male a piece of wire, about one tenth of an inch in diameter, is requisite in order to resist with certainty the enormous force of the great muscles of the thigh. The pieces of bones which I hold in my hand were removed in August, 1881, from a case of badly united fracture of the femur.

The patient was a gentleman from Rio de Janeiro. The fracture had occurred about the junction of the middle and upper thirds of the bone; it had been so badly united that the fragments overlapped very much, and also were at a considerable angle with each other, so that the limb was extremely distorted as well as much shortened. Bloodlessness of the operation having been provided for by elevation of the limb and the application of an elastic tourniquet, I cut down from the outer aspect of the thigh upon the seat of fracture. Then with a periosteum detacher I separated the soft parts from the place of junction of the fragments, and in the next place I went through the extremely laborious process of cutting through the osseous union (which was of almost ivory hardness) parallel to the axes of the two overlapping fragments. This having been at length accomplished, and the soft parts still further detached, I found I was able not only to get the limb into a straight position, but also by a very moderate extension so to reduce the riding of the fragments that by sawing off comparatively small portions I was able to bring their extremities into apposition and apply my suture. This was done with wire of about this same thickness. The limb was put up at first with a long splint. Being a weakly man, it was some months before absolutely firm union

was obtained. It would have been extremely embarrassing to have had the ends of the wire sticking out from the wound all that time. On the contrary, it was a very great comfort to have no occasion to think about the wire; and ultimately he left for South America with a perfectly straight limb, almost of the same length as the other, and with thoroughly firm union established.

But to return to the immediate subject of my communication. Ever after my first case of ununited fracture of the olecranon I was on the look out for a fracture of the patella to treat on the same principle. Dr. Cameron, however, anticipated me.

In October, 1876, being now full surgeon in the Glasgow Infirmary, he admitted a man with transverse fracture of the patella. He treated him in the first instance in the ordinary way, and dismissed him at the end of eight weeks with a pretty short and strong ligamentous union. Eleven days later, however, he reappeared, having ruptured the fibrous band by a violent movement during a state of intoxication. The fragments were then found widely separated. Dr. Cameron again treated him on ordinary principles for eight weeks, at the end of which time the fragments were still so widely apart, and the limb so feeble, that Dr. Cameron determined to cut down and apply the wire suture antiseptically. This he did on March 5th, 1877. On making a longitudinal incision he exposed a condition of the parts of which, through his kindness, I am able to show you a sketch, viz. a ligamentous union one inch in length, connecting pretty equal-sized fragments with nipple-like projections extending from their attenuated margins, much thinned by absorption. He cut away the fibrous material, and having pared the edges of the fragments and drilled them in two situations with a bradawl, he connected them with two sutures of stout silver wire, the ends of the wires being left projecting at the wound. At the same time he introduced an independent drain into the joint. The wound healed without suppuration or fever; and though osseous union was not obtained, which, as Dr. Cameron remarks in his report of the case, was not to be wondered at, considering the thinned state of the surfaces, yet he had the satisfaction of discharging the patient with close approximation of the fragments and a thoroughly useful limb.

In October of the same year (1877) a patient with transverse fracture of the patella was admitted under my care into King's College Hospital. He was a man, forty years of age, who, while riding, was thrown over the horse's head, and fell on the right knee. He could not rise, and was brought to the hospital. In the first instance, I attempted with this patient to bring the upper fragment down so that it should be in contact with the lower. For this purpose I applied an apparatus, which was so arranged that the upper fragment could be drawn down by means of weights and pulleys. Four days later, however, I found that there was still a quarter of an inch interval between the fragments, and I suggested the operation of cutting down and applying the wire suture. This, however, he would not then consent to, and preferred returning home to be under the care of his ordinary medical attendant. Eight days later, or fourteen days after the accident, he was readmitted, expressing a wish to be operated upon. On October 26th I accordingly proceeded to operate, making a vertical incision about two inches in length over the patella, exposing the fragments, which were then one

inch apart. My inability to bring down the upper fragment into contact with the lower became explained when the parts were exposed; for there were found between the fragments exceedingly firm coagula, with fibrous tissue, fascial and periosteal, mingled with them. The clots having been cleared away from between the fragments and from the interior of the joint, I applied a common bradawl in the middle line of the patella, drilling each fragment obliquely so as to bring out the drill upon the broken surface a little distance from the cartilage. Pretty stout silver wire was then passed through the drilled openings and the fragments brought accurately into apposition. Before they were brought together, however, an arrangement was made for the drainage of the joint. This was done on the same principle in all the cases that I have to record, and I may therefore describe the matter once for all. A pair of dressing forceps with the blades closed were passed through the wound to the most dependent part of the joint at its outer aspect. The instrument was then forcibly thrust through the synovial membrane, the fibrous capsule, and the fascia, until the point of the forceps was felt under the skin. An incision was next made with a knife through the skin upon the end of the dressing forceps so as to allow it to protrude. The blades of the forceps were then expanded so as to enlarge the opening which they had made in the deeper structures without risk of causing hæmorrhage. The drain was then seized in the forceps that protruded through the wound and drawn into the joint. The ends of the wire were now twisted together and the twisted ends brought out at the wound, which was closed with sutures and a small drain inserted. The limb, enveloped in an antiseptic dressing, was placed in a trough of Gooch's splint, with the upper end oblique, corresponding to the line from the tuberosity of the ischium to the great trochanter, and the lower end excavated in the form of a horseshoe, while the horns of the horseshoe were well padded to support the sides of the foot. It is unnecessary for me to enter into details as to the progress of this case. The temperature chart indicates, after a little temporary disturbance immediately after the operation, an entirely a-febrile condition. The wounds healed without any suppuration. At the end of eight weeks the wire was removed by an incision through the cicatrix. Eight days later the wound made for the removal of the wire had healed. At the end of ten weeks from the operation the patient was allowed to get up; and though no passive motion had been employed he could move the limb freely through an angle of about 30°. Two days later he was discharged, and, unfortunately, nothing has been heard of him since.

This, I believe, is the first instance of a recent case of fracture of the patella being treated by wire suture antiseptically applied.

My next case occurred two years later.

William T—, a coal porter, thirty-seven years of age, a muscular man, was admitted on December 13th, 1879. He slipped, on December 9th, while carrying a sack of coals, and felt something give way in one knee. On endeavouring to rise he found himself unable to do so. The right patella was found fractured transversely, the interval between the fragments being about an inch. There was a considerable amount of effusion into the joint. On December 15th—that is to say, six days after the accident—I proceeded to operate, making a longitudinal

incision as in the last case, about two inches long, over the patella. The lips of the wound being held apart with blunt hooks, a hole was drilled in each fragment in the median line; stout silver wire was passed and secured by four half turns. A drainage-tube was introduced, as in the last case. For the first fortnight after the operation there was what we may call an absolutely afebrile state; during the rest of the time of his residence in the hospital there were two accidental elevations to 100·5°, but nothing to indicate anything serious. The wound healed, as in the last case, without any suppuration. Six weeks after the operation he was allowed to get up. Eight weeks after the operation an incision was made through the cicatrix for the removal of the wire, the loop of which was cut and the wire withdrawn. On February 23rd—that is, a fortnight later—the bandages which had been previously applied to the legs were removed, and it was found that he could bend the limb to a right angle, he could walk well, and was able to kick. On February 22nd last this patient showed himself. We then took the following notes:—"The patella seems perfectly natural, except a trifling irregularity of outline on one side. It is evidently osseously united. The movements of the joint are perfect, from complete flexion to extension. He can kick vigorously. He says the joint is just as trustworthy as ever. He frequently carries a weight of 220 pounds for upwards of 100 yards, and he walks without the slightest limp." [The patient was introduced and exhibited to the Society. He said his limb was as good as ever, and that it never failed him.] Except for the linear cicatrix, no one would be able to tell that this patient had had anything wrong with his knee at all.

The next case was one of ununited fracture.

The patient, Joseph R—, aged twenty-two, was admitted into the hospital on September 27th, 1880. He is a soldier, and stated that on June 3rd of that year, while running across a green, he slipped upon a piece of turf, and in trying to recover his balance he snapped his knee-pan and fell. He was taken to a military hospital, and the limb was put in splints, which were kept on for seven weeks, a starched bandage being applied after their removal. On admission the fragments were found separated by an interval of three quarters of an inch. There was a firm fibrous band of union between them. The knee, however, was quite stiff, in consequence apparently of the rigidity of the extremely atrophied quadriceps extensor. He was quite unable to walk; if he attempted to do so he held the leg in his hand, and he complained of uneasy feelings in the joint. On October 22nd I cut down over the patella as in the former case, and having cut away the fibrous tissue between the fragments, refreshed the osseous surfaces, and then, having provided for drainage as before, drilled the fragments and drew them together with stout silver wire. Nothing occurred worthy of any notice until four weeks after the operation, when I proceeded to attempt passive motion. Without chloroform this proved impossible, but under chloroform I used considerable force, when, the rigid quadriceps refusing to yield, the twist of the wire gave way; the cicatrix, which had quite healed except where the wire projected, opened, and there was heard a sound of air being sucked into the joint, the fragments becoming at the same time widely separated. I injected the joint with an antiseptic solution, a procedure which I candidly confess I should now regard as probably superfluous. However, such was done; and six days later

when all disturbance caused by this second injury and the irritation of the antiseptic injection had passed off, I operated upon him a second time. Chloroform was again administered, and the cicatrix was laid open. I then found that the ends of the wire were lying in place ready to be twisted together. I found at the same time a considerable mass of coagula present. These I cleared away from between the fragments and from the joint, twisted the wire again, and thus we had a second operation in one patient. The after-progress was as in the other cases. Here we have a doubly long temperature chart, because we have two cases in one, so to speak; but it is seen that here there has been no deviation whatever from the normal state; neither as the result of one operation nor the other has any febrile condition as regards temperature been produced. In due time the wire was removed, and the wound made through the cicatrix having healed, he was discharged, eight weeks after the second operation. He was then able to walk, but with a stiff knee, with scarcely any mobility. I did not attempt any more passive motion after previous experience, and, while I was well pleased to see that he could walk with a stiff knee, whereas he could not walk at all on admission, I hardly ventured to hope for anything better. However, on February 22nd, 1883, he presented himself, when we made the following report:—"The patella is perfectly natural, except a little irregularity of the two borders opposite the seat of the fracture. The surface is quite smooth. There is no interval between the fragments. There is evident osseous union. The degree of flexion is increasing. He can bend to an angle of sixty degrees and extend again completely. He laid aside his stick last summer and returned to his work as a gardener seven months ago. He can do anything except kneel. In getting over a paling he has to throw the leg over in a partially extended position. He walks with a barely perceptible limp." We may now say that he walks with no limp at all. [The patient was introduced and exhibited. He said he could work very well, that he was on his legs all day and felt no uneasiness.] The very great interest in this case seems to me to lie in the improvement that has occurred without any passive motion on the part of the surgeon merely from the natural actions of the limb.

The next case was also one of ununited fracture.

The patient, Martha F—, aged forty-three, was admitted November 5th, 1880, on account of an accident which had happened eight weeks before, when she felt something snap in her knee while trying to save herself from falling. She was unable to walk or move the joint, which quickly swelled to a large size. No splints had been used, and no treatment of any kind employed. On admission there was fulness over the knee, and a transverse fracture of the patella was discovered in the middle of the bone. The fragments were separated one inch. There was fluid in the joint, as indicated by fluctuation. On November 12th I operated as in the last case, paring away the fibrous material between the fragments and refreshing the osseous surfaces with cutting pliers. The fragments were then drilled obliquely in the middle line, and brought into apposition by means of stout silver wire. This, however, could only be done when the limb was raised high into the air, so as to relax the quadriceps, she being a stout woman with powerful muscles. With regard to this patient, again, I have to show a temperature chart free from any febrile indication. In eight weeks after the operation the

patient was allowed to get up, and she walked about on crutches. She was discharged three weeks later, able to bend the knee slightly, lowering and raising the foot three or four inches. On the 14th of February the patient was readmitted to have the wire extracted; and five days later she was finally discharged. It will be remembered this was a case of ununited fracture, and that when the patient left the hospital she could only move the foot for a distance of about four inches. But on February 22nd of this year, when we had an opportunity of seeing her, the following note was taken:—"There is perfect union of the fragments. She can walk from Drury Lane to Billingsgate and back, and walks without a limp. She can bend the knee to a right angle, and says the mobility increases every day. From the position at a right angle she can raise the leg in a perfectly natural manner to the extended position. She is a stout, heavy woman. She cannot kneel." [The patient was introduced and exhibited. There seemed to be a perfect patella; nothing abnormal to be felt about it. She said she could do everything except kneel.]

The next case was one of recent fracture.

Wm. G—, sixty-two years of age, was admitted June 21st, 1881. He is a healthy man, but a pretty hard drinker. On the morning of his admission he slipped, put out his right leg to save himself, fell, and could not rise. His leg doubled under him. On admission the patella was found fractured transversely, with one inch of interval between the fragments, which could be brought together with difficulty. The knee was considerably swollen. On the 24th, that is, three days after the accident, I operated upon him. The operation was conducted precisely as in the recent cases that have been before described, except that in this patient, for the first time, the ends of the wire were cut short, and the twist hammered down. A few hours after the operation he became delirious, and we were apprehensive of delirium tremens. However, he was quieted with a dose of opium, and gave no further trouble. The temperature chart here, as in the last case, exhibits entire absence of febrile indication. The wounds healed without suppuration. Exactly six weeks after the operation he was allowed to get up, and I had the satisfaction of exhibiting him before some of the members of the International Medical Congress, and showed them that he could raise his limb from a position of flexion almost at a right angle to the completely extended posture. I have not seen him from that time until to-day. Having learned that he was employed in a Birmingham establishment I wrote to my friend Mr. Chavasse, of that city, and I received from him this letter: "I saw G— this morning, and have made arrangements for him to leave by the two o'clock train to-morrow afternoon. In case he should not keep his appointment there is a faint linear cicatrix present over the joint. The knee can be flexed as well as the other. There is bony union of the patella, a faint ridge being felt at the point of union. No suture is to be detected by the touch. At his work as a stamper all day long he works a pulley with the affected limb, which raises a weight of 60 lb. In damp weather he feels very slight inconvenience in the site of the old fracture." Happily our patient has kept his appointment, so that we are able to see him to-day. [The patient was introduced and shown. He said he could do anything with the limb, the same as with the other. He worked a stamp hammer, and he could work that hammer, weighing 1 cwt., all

day long. There was no difference whatever between the movements of the two knees. There was a barely perceptible cicatrix, and a perfect patella.]

My sixth patient was a woman.

Elizabeth C—, fifty-seven years of age. She also was a recent case, admitted on October 21st, 1881. Going downstairs she fell, with her left leg bent under her, striking the knee against the ground. She was brought at once to the hospital. The left knee was very much swollen from effusion, hot and tender; the fragments of the patella were felt separated from each other about one inch. I believe it is generally wise to let any distinct inflammatory appearances that exist as the immediate result of the accident pass off before the operation. In the former case there were no such or scarcely any; therefore we operated three days after the accident. In this case they were manifest, and we allowed a week to pass. The operation was performed just as in the last case the ends of the wire were twisted with two half turns—that is to say, one complete turn; the ends were cut short, and then with the small hammer the twist was hammered flat down upon the patella. On the evening of the first day there was a depression of temperature; on the following day a little rise—to 101°; after that there was nothing to indicate anything febrile. Two weeks after the operation passive motion was commenced; the wounds were then so very nearly healed that the milder antiseptic boracic lint was used for the superficial sore which remained. Three weeks after the operation the limb was bent to a right angle; this was painful to her, but caused no disturbance of the fragments. Four weeks after the operation passive motion was again employed; the wounds were completely healed. Six weeks after the operation the patient was allowed to get up and could already walk fairly. Three days later she was discharged; she could then walk very well. On February 23rd last we took this note regarding her:—"The patella appears perfectly natural with the exception that the wire is felt, causing the slightest projection, under the skin, which, however, moves freely over it. The movements of the joint are perfect from complete flexion at a very acute angle to perfect extension. She kneels as she used to do, and only occasionally the wire comes into contact with anything, and then she feels the skin over it tender. She can walk any distance, as before the accident, and does so without a limp." [The patient was introduced to the Society, and fully confirmed the previous report.]

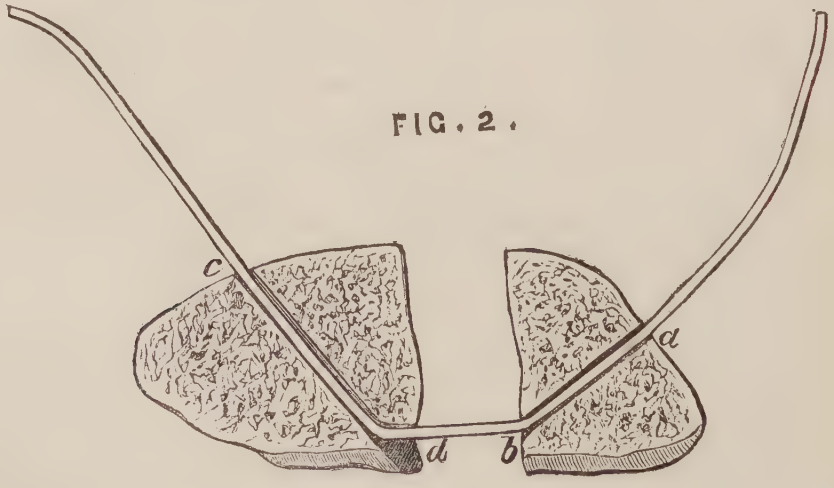
My last case is a man sixty-seven years of age.

John P—, admitted into King's College Hospital on January 6th of the present year. He had fallen from an omnibus on the previous day, striking his knee upon the ground. There was transverse fracture of the patella and great swelling of the joint. Six days after the accident I proceeded to operate. On exposing the seat of fracture, I found a condition of things which possibly, had I known it, might have induced me to abstain from operating. The lower fragment was very small, and was comminuted. There was one entirely loose piece as large as a filbert, and another about half that size, which of course had to be extracted. Such a condition, taken in connection with the advanced age of the patient, did not promise well for satisfactory union. Having sponged the clots out from between the fragments and from the joint,

and established a drain, I drilled; but as the lower fragment was so exceedingly small, I was obliged to drill, not through it, but through the ligamentum patellæ, and by this means was able to bring the large upper fragment into contact with the raw surface below. You will see that his temperature chart is free from any febrile indication; and the wounds healed without any suppuration. Here, as in the last two cases, the ends of the wire were cut short, and the twist hammered down upon the patella. Three weeks and four days after the operation the wound was nearly healed. The knee was bent to an angle of 30°, and from that position the patient could himself raise it to complete extension—a thing which without the wire, and without our feeling that the wire was in a very secure form, we should not have thought of permitting. Four weeks after the operation the wound was healed. The knee was then bent to an angle of 40°. Eight weeks after the operation the patient was discharged, having been for a considerable time before allowed to walk about the ward, and walking well. You will observe, gentlemen, that this patient has not had the same time for improvement that the others have had. [The patient was introduced and examined. He said he was getting on "first rate;" he found the leg continue to get stronger, and he was able to move it better; he noticed that it was getting stronger every week—he might say every day.] I confess I am surprised to see how complete a patella this man has, considering how extremely small the lower fragment was.

Gentlemen, these are all the cases which I have operated upon, and I consider it fortunate that I am able to bring before you six out of the seven. I should like now to say a few words as to the method of operating. There is very little indeed to be added to what I have already said, except this: It is very desirable that the lower surface of the patella should be left quite smooth. We cannot be perfectly sure when we drill that the bradawl will come out exactly at a corresponding point on the two surfaces. Supposing that on

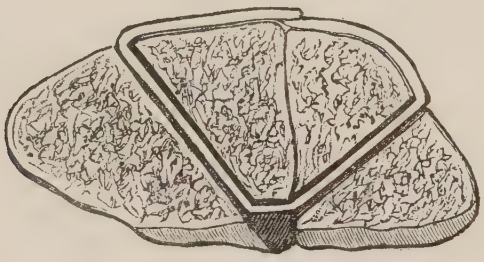
FIG. 2.



one side the instrument should have come out too far down, it may be into the cartilage (as at *d* in Fig. 2); we do not regard that at

first, but pass the wire through the two drill-holes, and then on that side on which the hole has come too far down, by means of the bradawl we simply chip away a little of the material that is above the wire until the wire comes to be in a position exactly opposite to the hole on the other side, leaving a gap below, as indicated by the dark shade at *d*. This is a perfectly simple matter; at the same time it might possibly not occur to anyone doing the operation. Here we have the wire represented twisted and the twist hammered down. The twist always goes to one side, and being on the other side in this instance is not shown in Fig. 3.

FIG. 3.



I think it must be admitted that these cases show that the mode of treatment which I have recommended, when applied to recent transverse fractures of the patella, affords a means of restoring the joint to practically a perfectly natural condition, provided only that no disaster occurs. But that is a tremendous proviso, and no one is more conscious of it than myself. Before I made the incision in the first case that I have recorded to-night I remarked to those who were assembled in the theatre that I considered no man justified in performing such an operation unless he could say with a clear conscience that he considered himself morally certain of avoiding the entrance of any septic mischief into the wound. Supposing, on the other hand, that a man can say that with a good conscience, then I conceive that he is not only justified but bound to give his patient the advantages that we see are to be derived from this method of procedure. We know, of course, quite well that by ordinary means of treatment patients often recover with very useful limbs. Every now and then osseous union is obtained. It is a thing which I used to pride myself formerly on striving to get, and I have achieved such a thing, but it was rare, and it was obtained by a very tedious process; and if ligamentous union occurred, we never felt sure that what was a very short ligamentous

union when the patient was discharged might not be a long ligamentous union at a later period. A gentleman consulted me with transverse fracture of the patella a couple of years ago. He happened to come to me just as I was about to start for my autumn holiday. I did not care to operate upon him and throw the responsibility of the after-treatment upon another, and it happened to be a case especially favorably circumstanced for osseous union. There was nothing between the fragments; the two could be brought into apposition with the utmost facility. I applied an apparatus which seemed to maintain perfect immobility of the fragments, and when I came back from my holiday I found them as I had left them, with no interval between them, and I hoped there might be osseous union. Eight weeks after the accident the patient was allowed to get up and walk, and walked exceedingly well. Six weeks later I asked him to come and report himself, and I was sorry to find that there was already half an inch of separation of the fragments. He was then about to start for India. What the separation may be now I do not know. It is true that there may be a very useful knee-joint with a very considerable length of fibrous union. Still, when there is a great length of fibrous union, the knee is never equal to the original. Some of our experience with these cases, where we could inspect the actual state of things, will serve to explain the uncertainty of the results of ordinary treatment. We have found, for instance, such dense masses of clot so mixed with fibrous tissue as to make it quite impossible that the fragments should be brought into apposition. I also found in one case such a tilting up of one of the fragments that it would have been perfectly impossible to get the osseous surfaces in contact, except by direct operative procedure. Considering therefore the great inconvenience which results in many cases when the treatment is conducted on ordinary principles, I believe that if we can really say that we are morally certain that we do not subject the patient to risk, we are in duty bound to give him the benefit of this method. It has been said it is not justifiable in recent cases, though it is justifiable in ununited cases where there is a useless limb. I must confess that if I believed I was subjecting a patient to serious risk to life I should not feel justified in operating on the ununited case, and the ununited case is in every respect worse as a subject of operation than the recent. The ununited case has the fragments probably dwindled by absorption,

and these fragments, already dwindled, have to be pared. There is a separation, it may be a very considerable separation, and this has to be overcome, it may be by dividing the quadriceps extensor, an operation of difficulty; and in proportion to the length and difficulty of the operation is the chance that the surgeon may forget some point of importance with regard to the antiseptic element. Then, again, when you come to divide the quadriceps extensor, you divide a very vascular structure, and you may have hæmorrhage; and further, when the fragments are brought into apposition, they are very likely at considerable tension, and the tension may be apt to cause through the nervous system an inflammatory disturbance, and this tends to weaken the parts, and to diminish the power of resistance by which the natural tissues are able to combat the entrance of septic agencies, even though they be in contact with the part. In the recent case, on the other hand, everything is favorable. We have a wound involving no bleeding, and there is no need to pare the fragments. All we have to do is to sponge away clots, and the surfaces are ready for coaptation. The drilling is a matter of no difficulty; it does not take long; it does not cause anxiety; there is no shock to the system, and no tension. In every respect the circumstances are favorable as regards the operative procedure. And then, when we come to consider the chances of successful antiseptic management, if there is in the whole body a situation which is well adapted for antiseptic treatment, it is this; for of all the conditions requisite according to our present methods of procedure, that which is most important is that the skin on all sides round about the wound should be able to be amply overlapped by the antiseptic dressings. Here we have the wound in the middle of a long limb; from the groin to the foot we may have our antiseptic envelope. Then, again, we have this envelope surrounded with a secure bandage, and the bandaged dressing encased in a splint; and even if you come to have the patient delirious, as one of my patients was, or supposing a patient to be very curious, as some patients will be, it would puzzle him to get the wound exposed under these circumstances. Now, there are wounds so placed that you cannot well guard against this risk. I had a gentleman lately under my care with a psoas abscess, who seemed duly impressed with the importance of the antiseptic management, and yet his brother, who was a medical man, coming in one day saw him drawing the dressing aside and peeping at the wound.

Now, a man cannot peep at a wound in connection with a fracture of the patella; it is so circumstanced mechanically that he cannot do it; and I believe that if we use the means that we have now at our disposal we may say with a safe conscience, if we use them aright, that we do not subject the patient to anything like so great a risk as people used to be subjected to, not many years ago, when they had fatty tumours removed in general hospitals. We must all of us remember cases in which, after such operations, erysipelas or diffuse suppuration came on, and the patient—nobody's fault, of course—died. I have referred to a case of ununited fracture of the olecranon where eighteen surgeons had been previously consulted. I trust no one here will suppose that I mentioned this circumstance for the purpose of glorifying myself. I mentioned it in order to emphasise what I believe to be the truth, that by antiseptic means we can safely do, and are therefore bound to do, operations of the greatest importance for our patients' advantage which without strict antiseptic means the best surgeon would not be justified in recommending. How wise those eighteen gentlemen were in counselling against any operative interference, provided they were not prepared to operate strictly antiseptically, I think we must be all agreed. As regards the operative procedure in that case, it was of the most simple character possible; any first year's student could have done the operation exactly as well as myself. The reason that I felt justified in performing it was simply and solely that I knew that strict antiseptic treatment would convert serious risk into complete safety. As regards antiseptic treatment, I should just like to make this remark: that nowadays it is not a very complicated business, either in theory or in practice. First, as to theory. We do not require any scientific theory in order to believe in antiseptic treatment. You need not believe in the germ theory at all; if you are not convinced of the truth of the germ theory of putrefaction and of septic agencies generally, no matter whatsoever, with reference to antiseptic practice, all you have to believe is that there are such things as putrefaction and other septic agencies, that our wounds are liable to these, that they are very pernicious, that these things come from without, and that we have the means of preventing them by various chemical agencies. That is all that we require, and I think anybody who knows the present state of surgical practice must admit these to be truisms. It has sometimes been a great grief to me to think that because

gentlemen are not convinced of the truth of the germ theory out-and-out, therefore they say, Lay aside antiseptic treatment altogether. And then as to practice. It is not a very difficult thing to wash your hands in a carbolic solution and have your instruments in this carbolic solution for a quarter of an hour before you operate. It is not a very difficult thing to wrap round the limb a suitable envelope of antiseptic material. What I believe to be one of the most important things of all is strictly to maintain this rule inviolate, which I insist upon with my dressers, and which I confess I have insisted upon more of late years than I used, and that is always, when we change a dressing, invariably first to cover the wound with something pure;—not to wash the surrounding parts with antiseptic solution, and then, after this has been done, put a dressing on the wound, but before we begin to defile our lotion at all, put on the wound what is pure, and last thing of all wash the surrounding parts, which, though they look the same to our eyes, are different, *toto cœlo*. The edges of the dressing are septic—the wound, if it is as it ought to be, is aseptic. I have known such a thing, for instance, as for a gentleman in dressing a stump after amputation of the thigh to wash the perineum with a rag dipped in the carbolic lotion (1 in 40) and then, having washed the perineum, immediately to squeeze this rag over the wound. Gentlemen, that makes you laugh; but I assure you these are the kind of things that are constantly occurring; and disasters happen in consequence; and then gentlemen with whom things go wrong invariably say that with them everything has been perfectly done—a thing which for my part I am always loth to say. I am not likely to have many more years of active surgical work, and I felt that when you, Sir, gave me this opportunity it was my duty to speak what I believe to be the truth; for I feel it to be a grievous thing that patients should be hurried out of their lives or deprived of usefulness of limbs, simply for want of sufficient earnestness with regard to the endeavour to obtain complete exclusion of septic agencies from wounds, according to our present lights and our present knowledge. (Applause.) Gentlemen, I thank you most heartily for that cheer; for there was a time when such remarks proceeding from me might have met with a different reception.

November 5th, 1883.

DISCUSSION ON THE TREATMENT OF FRACTURE OF THE PATELLA.

Mr. Bryant spoke of the great surgical importance of the subject. He had no hesitation in accepting the principles of the treatment adopted by Prof. Lister; and, indeed, was of opinion that no room for criticism in the matter existed. He was pleased also to learn that one suture was enough to hold the fragments together. But he felt himself at issue with the author when he came to consider to what cases the treatment was applicable. Prof. Lister had spoken in no uncertain words on this point. He had distinctly indicated that he preferred to operate on recent as compared with old cases. The atrophy of the fragments, the prolongation of the operation, and the separation of fragments were the chief difficulties in the old cases; the treatment was more likely to be successful in recent instances because these difficulties were absent. Mr. Bryant then proceeded to say that he did not understand some of Prof. Lister's expressions. What was the meaning, for instance, of being morally certain that all septic influences could be avoided? He confessed that he was unable to fully comprehend that expression. What was it to be morally certain? Did Prof. Lister mean that his method was so good that it makes him certain? Mr. Bryant thought that if that were Prof. Lister's interpretation, then it seemed to him as though such statements represented what was beyond human power. And he expressed himself in similar terms of the notion that "serious risk" could be changed into "absolute safety." He asked Prof. Lister whether he literally meant that? Was he able to provide against the dangers that lurk in the background, dangers which arose from the possible existence, prior to the accident, of organic visceral disease? By no stretch of his imagination could he fall in with such expressions. He ventured to say, with all feeling of respect for Prof. Lister's great authority, that the use of such terms instilled over-confidence in his juniors and disciples. But although Prof. Lister had made use of these words, Mr. Bryant could not believe that he meant what he said, for in another part of the paper allusion is made to certain "disasters" which may occur. Under what conditions should the operation be performed? He believed he interpreted Prof. Lister aright when he said that Prof. Lister only insisted upon the grand principles of antisepticism. It was not now construed that the Listerian method was the only mode of applying the principles. Twenty years ago there can be no doubt the operation would have been scouted by us all as outside the field of legitimate surgery. What an interesting study was a surgeon's deathbook! There we should find disasters recorded due to causes quite beyond the ken of all surgeons. In many instances, though the surgeon has done his very best, yet how untoward the result! He felt sure that what was true of his own deathbook was applicable to the deathbook of other surgeons. With all that could be done our principles will fail us sometimes. If, then, disasters and bad results must occur, in what cases is this operation justifiable? Now, since good results are the rule in the ordinary treatment of fractures of the patella, why risk even in

the least the life of the patient? Mr. Bryant then instanced four or five cases which had come under his notice during the past two years. One was a woman, aged fifty-seven, who broke her left patella in 1863 and her right in 1867. In 1883 she again fractured the left patella through the lower fragment, but up to that time she had had a perfectly useful limb, although the fragments were separated an inch and a quarter. A man, aged forty-one, fractured both patellæ in January, 1882, and although there was a separation of half an inch yet he could go up a ladder as well as ever. Another man, aged twenty-nine, refractured the patella through the lower fragment, and although there had been separation of an inch and a half, he could do what he liked till January, 1882, when the second fracture occurred. In another case a man, aged thirty-one, sustained a second fracture, again through the lower fragment, after three years, during which his limb had been fully useful. In all his experience Mr. Bryant had only seen once a useless limb, and that was in a nervous lady. In that case he had performed Mr. Lister's operation with a good result. Another case of fracture of both patellæ with very wide separation was instanced as showing how well union may take place under the old-fashioned treatment. To sum up, then, Mr. Bryant felt that he should hesitate to accept the operation as one to be applied generally; the bulk of cases of simple fracture do well without the operation, and a very small percentage do badly. As a primary mode of treatment he should not recommend it, but as a sign of the value of the antiseptic method he praised most warmly the surgeon who recommended it.

Mr. Adams spoke in terms of great praise of the wonderful cases shown by Professor Lister. It was a marvel to him to see bony union with perfectly free motion, and he felt that a new and valuable operation had been added to practical surgery; but he rather agreed with Mr. Bryant in actual practice. He had recorded in the 'Pathological Transactions,' vol. ii, 1860, many cases which went to prove that the rule in fractures of the patella is to get non-union. From his experience he would divide cases into four series. The first, with separation of half an inch, would do well with the ordinary treatment; the eversion of the edges of the fragments was a feature of importance and had to be overcome. In the second class of cases, with separation of fragments of about an inch, he would use Malgaigne's hooks. In the third series a question would arise whether Malgaigne's hooks or Professor Lister's operation would be best, and here he thought the skill of the surgeon would settle the point. Finally, there were cases of ununited fracture, and for these, in an experienced surgeon's hands, Professor Lister's method was no doubt the correct treatment.

Mr. Astley Bloxam showed three cases to the Society, all of which had done well under the operation recommended by Prof. Lister. In none was there any febrile movement or untoward accident, and in all the final result was excellent. He thought the operation was one which ought to be done after all recent fractures.

Mr. Sydney Jones thought that the operation was not desirable in all cases of fracture of the patella, because the old-fashioned treatment gave us all that could be desired, and why should we have a wound into the joint in such cases? In later stages, or when the limb was useless, the operation might be performed with success and justification.

Mr. Royes Bell read notes of four cases in which the limb previous to operation was useless in a greater or less degree. In all he had per-

formed the Listerian operation with final success in three. In the fourth case there was partial ankylosis. He was rather in favour of free incision—say of six inches—through which the quadriceps might be divided if necessary. No harm seemed to follow from such extensive procedures under antiseptics.

Mr. GANT in the main concurred with the views of Mr. Bryant. He thought the operation ought to be tested by its results. We had to regard the operation from two points of view,—first, as to the state of the limb, and secondly, as to the life of the patient. In this respect he felt sure that in ordinary cases the old-fashioned mode of treatment gave results with which no operation could compare, all things being considered. He believed that the Listerian operation was of great value in dealing with cases of compound fracture of the patella uncomplicated with fracture of the other bones of the limb.

Mr. HENRY MORRIS offered his humble tribute of honour to Professor Lister for the introduction of the antiseptic system, but he could not agree with him that the operation under discussion was one which ought to be carried out in every case of fracture of the patella. He questioned much whether bony union was a necessity. Did not the very accident prove the friability of the patellar bone? Hamilton's statistics showed that the fresh tear occurred, if at all, generally after a period of five months. Mr. Morris would recommend restriction of movement for a year at least. Mention was made of a case of fracture of the patella treated by sutures in which, after some time, a fresh accident ruptured the union of the affected patella. This case was under the care of Professor Thompson, of Dublin, and bore witness to the singular friability of the patella. He must dissent from Professor Lister in regard to the simplicity of the operation. From his experience of third year students he argued that no first year's student could be considered competent to perform the operation. Rather, he thought, it required the skill of a good surgeon. Although he fully admitted the value of antiseptic surgery, he by no means considered the Listerian mode the only way of practising it. He thought that the mode in which the operation was performed, the hand that did it, and the condition of the patient, were all of much importance, for a successful issue.

Mr. W. ROSE had done the operation three times with undoubted success. He said it was not a difficult procedure. He had every faith in the antiseptic treatment, and would continue to treat all cases of simple fracture by this method. He wished it to be known that in one of his cases there had been slight suppuration, but Mr. Cheyne had been unable to detect bacteria in the discharge.

Mr. EDMUND OWEN hoped that it would be a pertinent question if he asked Professor Lister whether, in the event of a possible fracture of his patella, he would submit to the operation he recommended at the hands of another surgeon. He thought fixation of the joint for six months was of the greatest value in the treatment. He did not think the future of these cases was so disastrous as had been made out. Although he believed in the antiseptic method, he refused to accept this operation, concluding that it was "magnifique, mais ce n'est pas la chirurgie."

Mr. MORRANT BAKER agreed with Mr. Bryant in nearly all that he had said. He thought the operation was most valuable when the limb was useless. But had not anchylosis, amputation, and death been heard of after its performance?

Mr. G. R. Turner had collected the results of fifty cases, and the success which had attended the operation in recent cases was very pronounced.

Page 26, line 20,

For "Hamilton's statistics showed that the fresh tear occurred, if at all, generally after a period of five months,"

Read, "Out of a very large number of cases of fracture of the patella examined, Prof. Hamilton had found that in two only of twenty-seven refractured had the fibrous uniting medium given way after a period of five months."

union and perfect motion were obtained after four months.* Professor Lister then went on to say that it gave him great satisfaction to learn how universally the antiseptic principle had been recognised. So long as the grand principle of antisepticism remained, it mattered not whether carbolic gauze, eucalyptus gauze, salicylic cotton-wool, or iodoform were used, provided they were used effectively, though iodoform does not seem to be protective against erysipelas. Since Koch and others had shown that corrosive sublimate was destructive not only of bacilli but their spores, some surgeons had employed that agent. If the truth must be spoken, he would say that in his paper he alluded not to disasters which might happen to himself, but to such accidents which might befall those surgeons who did not feel fully confident of their powers to exclude septic influences. He alluded to the disorder which he had witnessed in out-patient rooms, where every kind of dressing was mixed together without regard to their nature. If volatile antiseptic dressings were freely exposed, and if dressings impregnated with boracic or salicylic acids, which were non-volatile, were yet allowed to remain where all sorts of dirt might be picked up, he could not wonder at the bad results which would ensue upon their use. But we ought to be sure that we use them so that we may be certain that their antiseptic principles are not exhausted before the dressing is changed. Corrosive sublimate was very soluble in water and easily washed away by discharge, and so it was necessary to use it in large mass. In Germany he had lately seen an absorbent wool made of wood shavings which were imbued with the sublimate, and he believed that used thus in large mass such dressings were very efficacious. He protested strongly against the use of antiseptic agents in the random fashion in which he knew they were still employed. He knew that Mr. Bryant used iodine, and he (Professor Lister) would not be in the least disconcerted if that agent alone remained to him, though it did rust the instruments. With regard to the justification of the operation in recent cases, what he said was that if one had not a thorough belief in the powers of the antiseptic treatment, the operation should not be performed. He could not agree that the ordinary results of the treatment of fracture of the patella were so good as some gentlemen had depicted them. He referred to the cases read by Mr. Royes Bell. He

* *Vide* the 'Lancet,' Jan. 3rd, 1880.

had seen great stiffness last a whole lifetime under such treatment. Moreover, he thought that laying the patient up for six months, or restricting the movement of the joint for a year or more, as Mr. Morris recommended, had positive disadvantages as compared with a treatment which required six weeks merely. It was a very large question, indeed, when we came to the subject of what would justify a surgeon in performing a given operation. Who is to decide what risk may be run in operations, speaking generally? Even in simple operations like the removal of fatty tumours, or atheromatous cysts of the head, considerable risk of erysipelas was run under the old system. So with cases of removal of loose cartilages from joints, great risks were incurred unless surgeons were very sure indeed of their antiseptic arrangements. In his early life he was told that it was justifiable to operate in a case of ununited fracture of the humerus, but not in one of ununited fracture of the femur, because the patient might die of pyæmia; but not very long ago he had known an operation for ununited fracture of the humerus prove fatal from pyæmia. Since last Monday he had performed an operation on a case of ununited fracture of the femur, and dovetailed the fragments together. The patient suffered considerably from the immediate effects of the operation. He had calculated on that; but now, so long as the wound was aseptic, he considered the patient practically safe. So, then, he considered that the justifiability of an operation was a very large question, dependent on many factors. And speaking for himself, he would repeat that in many cases strict antiseptic treatment effects a conversion of serious risk into complete safety. But there is a great difference between different cases. The difference between the surgical treatment of fracture of the olecranon and that of the femur was enormous; in the former there was no shock and no loss of blood. He ventured to say that Mr. Bryant would perhaps be surprised at his (Professor Lister's) deathlist. Sir Joseph Lister had operated on a number of cases of ununited fracture of the femur and of loose cartilages in joints, on a multitude of cases of disease of the knee-joint, and without any fatal result. Alluding to Mr. Owen's quotation, he could but remark that he considered that was "chirurgie" which saved people's lives. He would by no means encourage all surgeons to operate in cases of recent fracture of the patella; though he would say that for himself he was firmly convinced of the strength of his position when he said that he regarded the operation as easy and without risk so long as antiseptic principles were strictly adhered to. But he had brought the cases of suture of the patella before the Society principally with the object of illustrating what could be done, more than to advocate its employment under all circumstances. Sir Joseph Lister concluded by appealing to his surgical hearers not to reject the advantages of antiseptic surgery in such necessary operations as ligature of arteries and removal of tumours.

November 12th, 1883.

CASE OF MALPOSITION OF TOOTH.

By F. de Havilland Hall, M.D., F.R.C.P.

The tooth which I hand round was removed from the right nostril of the girl whom I exhibited on the 19th March, 1883, and about whose case I undertook to make some remarks. The tooth is evidently a permanent canine.

M. S—, aged fifteen, was admitted into the Westminster Hospital March 18th, 1883. Her mother and father died of consumption, one brother died in infancy. She has one brother and one sister alive, aged respectively seventeen and twelve. The patient presents the muddy aspect characteristic of hereditary syphilis. The nose is flat and broad, the corneæ somewhat cloudy.

She states that she has had a fœtid discharge from the right nostril for six years. She has also suffered from "bad eyes," but cannot remember any other ailment.

The septum of the nose was found to be destroyed. In the right nostril projecting through the mucous membrane was seen the crown of a tooth. The temporary canine on the right side is still firmly fixed in the dental arch, also several other deciduous teeth.

My colleagues Dr. Walker and Mr. Morton Smale kindly examined the patient for me. The former in a report he made to me on the case writes, "I have not seen a case with eruption so abnormal; the right temporary canine is still *in situ* in right upper maxilla. The tooth in nostril I take to be the permanent canine." Mr. Smale coincided with this view. As the girl was not in a very satisfactory state of health Dr. Walker feared that a permanent fissure might result if the tooth were removed at once, so the girl was treated for some weeks with the hypophosphite of lime and cod-liver oil. On July 11th the tooth was extracted and the socket soon filled in. This condition must be a very rare one as I have not been able to discover many cases. Mr. Salter* says, "Inversion of the teeth is another very rare form of irregularity. I have seen both the superior lateral incisors completely inverted and growing upside

* 'Dental Pathology and Surgery.'

down; the crowns of the teeth appeared in the nostrils, from which I removed them."

Mr. Charles Tomes uses the term inversion to describe a condition in which the anterior teeth are directed inwards and pass behind those of the lower jaw. He says, "Of all teeth, none are so frequently out of the normal position at the time of eruption as the canines." In saying this Mr. Tomes is referring only to such slight degrees of displacement as may be rectified, for he goes on to remark "that no other members of the set so frequently fall from an objectionable into the proper position without mechanical assistance."*

Mr. Tomes gives a woodcut of a case "in which the right canine is placed across the dental arch, the root being directed towards the median line of the palate, and the crown towards the cheek. The point of the crown was the only part which was not completely buried in bone." And he goes on to say that "a horizontal position in the base of the alveolar ridge is sometimes taken by the canine, the apex of the crown being exposed to view, or covered only by gum or imbedde in bone."† In my case the crown was distinctly visible in the nostril.

The patient is still under treatment for the ozœna, but this has been greatly benefited by the insufflation at night of a powder consisting of one part iodoform and three starch, followed in the morning by the use of carbolic acid lotion with the addition of ten grains of biborate of soda and ten of bicarbonate of soda to the ounce.

Since the preceding was written there has appeared in the 'Lancet' of Nov. 3rd an account of a case of malposition of a tooth.

A Hindoo lad, aged fourteen, was admitted for what he considered a tumour growing within the nasal cavity. This was seized with dressing forceps and extracted. It proved to be a tooth of the shape of a canine. The boy had got his set of permanent teeth, with the incisors and canines entire on each side.

Mr. Sewill doubted whether the malplaced tooth could be regarded as the cause of the ozœna, for it was quite healthy, and in most examples of misplacement of teeth no inflammation was excited.

Mr. Black thought the mode of development of the upper jaw might tend to explain why the canine tooth became so altered in position. He asked whether the notion that a third tooth might appear was well founded.

The President said that he had seen teeth frequently in abnormal situations, and in his experience they might give rise to inflammation

* 'A System of Dental Surgery,' 2nd edit., p. 128.

† Op. cit., p. 179.

and cysts, as in the antrum, where suppuration was known to occur, and be relieved by operation, so that both pus and tooth may be discharged.

Dr. HALL, in reply, agreed that the tooth did not set up the ozœna, which was no doubt the result of the syphilitic taint. He said also that a third tooth had been described by many writers.

ON PAROXYSMAL HÆMOGLOBINURIA.

By STEPHEN MACKENZIE, M.D., F.R.C.P.

IN a disease of decided infrequency, and concerning which much less is known than remains to be learned, every case placed on record is of value—of far greater value, probably, than any speculations as to its nature. I shall, therefore, commence by narrating three new cases as a contribution to the study of the disease, and then review what is known concerning it.

CASE 1.—R. M—, aged thirteen, has just left school, and not yet followed any occupation; has always lived in the east-end of London, and has always enjoyed good health; he has had chilblains on his toes each winter, and his face and hands get blue in cold weather. About one week before Christmas, 1881, he had been out in the cold without an overcoat, and felt chilly. When he passed urine after this he noticed it had the colour of porter. At the same time he experienced a pain in the left hypochondrium, but this soon passed off. The same evening he passed more urine like porter. He was taken to his medical attendant, who said he had inflammation of the kidneys. He remained in bed the whole of the next day, by the end of which time the urine had regained its natural appearance. On the following day he went out, and on his return he passed urine of a port-wine colour, and later, resembling porter. From that time until August, 1882, with an exception to be mentioned, whenever he has been out in the open air when it was at all cold, his urine has had the dark colour, varying in intensity with the degree of cold and duration of exposure. When out a short time, or the temperature not very low, the urine has had a deep red colour; when out for a longer time, or the temperature low, the urine has been as dark as London porter. In March, 1882, he went for a month to Hampshire, and during the time he was away the urine was perfectly natural, never once having the peculiar colour; when he returned to town the coloured urine reappeared. It was colder on his return to town than it had been when he was in Hampshire. At first he said he had never shivered in the attacks, but on questioning and explaining what was meant, he stated he had had shivering when he had been out in the cold, each attack lasting about a quarter of an hour. It appears that when the external temperature has been very cold, he has distinctly shivered. He has never had nettle-rash. When out in the cold, if his arms are exposed, he gets "goose-skin," but only under such conditions. The patient came under my care at the London Hospital on May 3rd, 1882. He then, as now, presented the appearance of perfect health.

He is tall and well grown. There has never been a trace of a yellow tint in his skin or conjunctivæ. There have been no abnormal physical signs in the chest. The spleen and liver have not been notably enlarged at any time when I have seen him in an attack. He has had no pain in any attack subsequent to the first. The following account of the urine gives a fair idea of its behaviour:—When he came to the hospital on the afternoon of Wednesday, May 31st, he said his urine had been quite natural from the Tuesday morning. On making him pass urine in my presence it was of a dull brownish-red colour, like weak porter. Examined spectroscopically it gave two well-marked bands between D and E. The guaiacum test also showed the presence of blood. Microscopical examination failed to detect a single blood-corpuscle, only granular or amorphous pigment being found. Examination of a drop of blood showed no abnormal appearances. No changes were detected in the ocular fundus. The same evening the urine was a little darker than natural. The following morning it had quite a natural appearance. He went out of doors at 11 a.m., and returned at 1 p.m., and then passed urine as black as porter. At 4 p.m. he passed urine a little deeper in colour than natural. At 6.30 p.m. he walked to my house, and then passed urine of a rather bright red colour. I got him to enter in a note-book the character of the urine each time he passed it, and the conditions he had been under. The urine has been examined chemically, spectroscopically, and microscopically on many occasions. It generally has a high specific gravity when containing colouring matter. This ranges from 1026 to 1037. Its reaction has been acid on all occasions, sometimes faintly, sometimes moderately so. It always contains albumen when colouring matter is present, proportionate to depth of tint. Albumen has not been present in the specimens I have examined, which were free from blood-colouring matter. The spectroscopic appearances have depended on the amount of colouring matter. When of a bright port-wine colour the two characteristic absorption bands of hæmoglobin between D and E of Frauenhofer's lines have been well marked. When of a deeper colour it shuts out all the spectrum except red (June 15th), stronger still, only a trace of red may be seen (June 17th), or all light may be stopped when the urine has the black or porter colour (June 25th). On diluting those more deeply coloured specimens of urine with a suitable quantity of water the characteristic absorption bands of oxyhæmoglobin are revealed. Microscopically all that is noted is granular and amorphous pigment. No blood-corpuscles; no casts; no oxalates or other crystals. Microscopic examinations have been made twenty or thirty times, always with the same result. Such were the features presented in this case up to August, 1882. Since coming under my care he had been taking for the most part quinine in five-grain doses twice a day, and during the latter part of the treatment one-drachm doses of liquid extract of ergot. On August 8th, in the evening, the urine was port-wine coloured, and since that evening its colour has been perfectly natural, the peculiarity ceasing as abruptly as it made its appearance. Since August 8th the patient has been out at all times, warm or cold, without experiencing any return of his complaint. Throughout his disease his general health has been unimpaired, his appearance has been perfectly healthy, and he has grown considerably. I examined his urine passed in my room on November 1st, 1882. It was of a pale straw colour, sp. gr 1033, no albumen; no blood by guaiacum test. He is now employed as an out-

door messenger. He comes of healthy parents, and no instance is known of a similar affection occurring in his family, nor is there a history of unusual liability to bleed in the patient or his family. No gout in family. At my request the patient came to see me on November 5th, 1883. He appeared in perfect health, and had had no recurrence of his symptoms. He went through last winter without experiencing any inconvenience from the cold. He passed urine in my room. It was of a faint yellow colour, clear, acid, sp. gr. 1029, free from albumen, as shown by the action of nitric and picric acids and heat. The latter caused a precipitation of phosphates. No blood by guaiacum test. The only circumstance to which the patient can attribute any influence in the production of his disease is the fact that previously to it he was employed in tending a boiler, and was exposed to rapid transitions from warmth to cold.

Case 2.—William P—, aged fifty-five, a rigger. The patient was born in Ireland, and went to sea at the age of ten. He was only engaged in the North American trade, and never crossed the equator. He never had ague, and never suffered from want. He left the sea in 1848, and has since followed his present employment, and lived at Poplar or Bow. He had "running" thirty-five years ago, but no sore on the penis or lumps in the groin. No sore-throat or general eruption then or since. He had good health until eighteen years ago, when he was living at Poplar. One very cold day in the winter he was engaged in the forenoon at his work when he shivered all over. He came down from aloft, and felt a desire to micturate. There was a great deal of snow on the deck, and when he passed urine he noticed it looked like blood. He continued his employment until his dinner hour, when he went home. He then passed urine in a chamber utensil and found it was quite dark, like blood; it had the same appearance at night. He sought medical advice, and when the gentleman he consulted was shown the urine he asked (the patient said) whether he was a teetotaller, to which he gave an affirmative reply. The doctor, after examining him all over, told him the condition was due to "poorness of blood." Some medicine was given him, and the weather at the time becoming mild and fine, he got rid of his illness in about a week. When the weather became cold again he had attacks of shivering and black urine. This continued on and off for about four weeks, and then he got quite well of it and remained well until the present attack, eighteen years later. During the attack he noticed that when he got warm after passing the bloody urine it became of a natural colour. For the last eighteen years he has continued his occupation, necessitating constant exposure, without interruption, in all seasons, winter and summer, without experiencing any inconvenience from the cold, and without passing bloody urine until the beginning of the present year. About twelve months after his attack of bloody urine he had ulceration of the skin of the thorax. After lasting about a year it got well without any treatment. It broke out again four years ago, and has continued since. He has used ointment for it, but has not sought medical advice. At the beginning of January, 1883, about five weeks before coming under my observation, he was engaged in rigging in the forenoon of a very cold day. He first felt chilly, and this increased to a regular shivering fit, in which his teeth chattered. He felt that he wanted to micturate, and when he came down from the rigging and went to the urinal, the urine he passed was bloody. He went home, and remained indoors the rest of that day and the day fol-

lowing. The remainder of the day on which the urine became bloody it was dark each time it was passed, but the next day it acquired its natural appearance. Between then and coming under observation he had two attacks. Each occurred on cold days in the forenoon, lasted some hours, and passed off when be became warm. He had worked during the intervals, and the attacks only occurred on especially cold days. He came to the London Hospital on February 3rd, when he was seen by my colleague, Mr. Mansell Moullin, who, recognising the nature of the case, kindly transferred him to my care. The patient, a dark-haired and dark-eyed man, fifty-five years of age, looked very anæmic and ill, but had no distinct icteric tinge in the skin or conjunctivæ. Examination of his body showed no changes in the thoracic organs, no enlargement of the liver or spleen, and no changes in the fundi. There was a large irregular ulcerated surface of skin in the upper part of the front of the chest, with scars in the surrounding skin left by previous ulceration. His urine was clear, and free from albumen and blood-colouring matter. He came again on February 10th, not having had any attack in the interim. He was made to pass urine; it was clear, acid, sp. gr. 1022, free from albumen or trace of blood-colouring matter by guaiacum test. I got him to place his feet in a bucket of quite cold water, to which, after a few minutes, a large lump of ice was added. He kept his feet in the water for fully half an hour. At the end of this time his legs and feet felt very cold, but the sensation of coldness did not extend above the legs. His temperature remained, as it was previously, normal. At the end of the half hour he was made to pass urine again. He himself judged from his feelings that it would not be bloody. It was clear, of a faint yellow colour, acid, sp. gr. 1018, free from albumen, and the guaiacum test showed it free from blood-colouring matter. It gave, as well as a specimen he brought with him, passed at home, and that he passed before immersing his feet, a violet tint when in contact with strong nitric acid. Judging the ulceration of the skin to be syphilitic (this was before I had carefully gone into the history on this point) I prescribed five-grain doses of iodide of potassium three times a day, and two grains of quinine night and morning. The next day (February 11th) he was determined to bring on an attack to show me the kind of urine he passed. For this purpose, at about 7.30 a.m. he went on to the roof of some sheds, took off his pilot coat, and set to work to tar the sheds. It was a bright cold day, with a strong easterly wind blowing. After being engaged in this way for about a couple of hours he noticed his feet getting numb with cold. He kept on, however, and felt the cold creeping up his legs into his body. When it reached the upper part of the chest he felt sick, and had a taste as of blood in his mouth. He always had this sensation, he said, when the cold reached the upper part of the chest, and then he knew he would pass blood. At this time he was shivering all over most fearfully, so much so, he said, that he stood with difficulty and had to sit down. He felt a desire to pass urine, and when he did so it had a deep blood or porter colour (specimen brought, passed at noon). He then went into the warm kitchen and the shivering soon passed off. He ate a hearty dinner, and did not pass urine until 6 p.m., when it had, he says, quite a natural appearance. After tea he came to my residence in an omnibus, and at about 8.30 p.m. passed urine in my consulting room. He was then free from all chilliness. Urine passed at noon of a deep reddish-brown or porter colour, acid, sp. gr. 1025. On boiling, copious brown flocculent preci-

pitate of albumen, undissolved by nitric acid (in moderate quantity). Spectroscopic examination: All the spectrum except red and a trace of green shut out. Diluted with about an equal quantity of water, there were two well-marked bands between D and E. After standing some time a copious brown sediment was deposited, the supernatant liquid remaining of a deep dirty-red colour. Microscopic examination: No blood-corpuscles; numerous casts, of various sizes, blood pigment, and much amorphous brown pigment; a few leucocytes, and a little round (bladder) epithelium; no oxalates. Urine passed at 8.30 p.m.: Of pale yellow colour, acid, insufficient to take sp. gr.; very faint trace of albumen; nitric acid gave the violet tint seen on previous occasions. Spectroscopic examination: No trace of blood-colouring matter (by gaslight); guaiacum at first gave no trace of blue colour, but after it had stood for some minutes a blue tint appeared, and in a few more minutes a deep blue colour was present. Microscopic examination: After standing twenty-four hours, a slight brown flocculent deposit, which was found to be due mainly to casts containing blood pigment, a few leucocytes and round epithelium. Free amorphous pigment, small in quantity, was present, nearly all the pigment being in casts. No crystalline deposit. On February 14th he reported himself at the hospital and had had no further attack. The mixture containing iodide of potassium was replaced by one containing perchloride of iron, as I was then convinced the eruption was not syphilitic. On February 17th and 21st he attended, and on each occasion his urine was free from albumen and blood-colouring matter. Cod-liver oil ordered on latter occasion. When he attended on February 24th he stated he had been on the pier for a couple of days. On the 23rd he had shivered, and subsequently passed urine dark brown in colour, and turbid. He brought it with him. It had sp. gr. 1030, contained albumen, and the guaiacum test showed the presence of much blood. The urine was clear the same night. The urine passed when at hospital on the 24th was a little dark in colour, contained a little albumen, and by guaiacum test a faint trace of blood. His general condition was improved; he looked and felt better. February 28th, March 3rd and 7th: No fresh attack. On the latter date his urine was free from blood-colouring matter and albumen, the eruption was healing, and he felt so much better that he thought he might resume work. I dissuaded him from doing so. He came again on March 10th, feeling very weak, and stated that on the previous day—which was a bitterly cold frosty day with a north-east wind—he had been out for half an hour or so. At the end of that time he became very chilly and had to return home, and then passed urine, which he brought with him. It was porter-coloured, almost black by reflected light, and of a deep red by transmitted light. It had a sp. gr. of 1030, was acid, and contained albumen. Guaiacum test gave a deep blue reaction. Microscopic examination: No blood-corpuscles, hæmoglobin casts, no crystals. Diluted with an equal volume of water, the spectroscope showed two bands between D and E. The same evening the urine had acquired nearly its natural colour. To-day, after coming out into the cold, nearly of equal degree to that of the preceding day, he passed water at the hospital. It had a sp. gr. of 1020, was of a slightly deeper tint than natural. The guaiacum test showed a trace of blood-colouring matter, and on boiling a trace of albumen. After standing there were a few hæmoglobin casts; no blood-corpuscles or crystals. When he came on the 17th he said he had had one attack on the 15th after exposure. His teeth did not chatter,

but he felt chilly. On the 16th he had two attacks, first time at 9 a.m. after exposure. At 6 p.m. he passed porter-coloured urine, not having been out since the previous attack. He brought this urine with him. It had a sp. gr. of 1015, and gave blue colour with guaiacum and the spectrum of oxyhæmoglobin. That passed at the hospital on the 17th at 2 p.m. was of natural colour, sp. gr. 1016, free from albumen and hæmoglobin, and gave a violet tint when boiled with nitric acid. March 24th: No blood or albumen in urine passed at hospital, sp. gr. 1020. No fresh attack. He reported himself again on March 31st and April 14th and 28th. He had no attack subsequent to March 16th. He had gained flesh, his colour had returned, and his eruption was healed. As already stated, he took five grains of iodide of potassium three times a day for one week. With this exception the treatment consisted throughout of perchloride of iron, cod-liver oil, and two grains of quinine three times a day. The patient at my request reported himself on November 5th. He had remained quite free from an attack; his health, he said, was better than it had been for years. He passed water in my room; it was acid, clear, faint yellow, and insufficient in quantity to take sp. gr. No albumen with heat and picric acid; when boiled with nitric acid it became slightly darker.

Case 3.—For an opportunity of seeing this patient I am indebted to Dr. Anderson, of Richmond. It is valuable, as the disease has persisted about twenty-three years, and as an intelligent person's own description of his case. The patient is a gentleman of independent means in his fifty-first year. The following is his own account:—"On or about the 8th of August, 1860, I had my first attack while on a journey to the Highlands, in consequence of getting thoroughly chilled through sitting for six hours on the outside of a coach, and only clad in light summer things and a very thin mackintosh. The day was cold, and it rained incessantly the whole journey. I was so very ill during the last stage that I had to be supported by a fellow-passenger. On arriving at my destination I was only able to shake my head in response to my host's offer of hot brandy-and-water, tea, a hot bath, &c. I tumbled into bed somehow or other, and shivered so that the clothes shook and my teeth chattered. However, I at length fell asleep, but recollect awakening once during the night and fancying I was dying, as my heart seemed as if it had stopped. I again went to sleep, and awoke in the morning 'all right.' I noticed then that my urine was perfectly black, but thought nothing more about the matter. I was in good health when I started on my journey, and attributed my illness to the cold and wet, and to having had nothing since breakfast but some weak brandy-and-water. I was twenty-seven years and a half old then. Every subsequent attack has invariably occurred through exposure to cold, and in no other way, the intensity of the attack only varying according to the time of exposure. Symptoms: The first symptom is only what I can describe as an uncomfortable feeling about the region of the wind—just below the breast. Then, as the chill goes on, yawning and stretching out one's arms, and then a drowsy fit. If the attack is a bad one, there is besides an intense shivering, and the jaws rattle so that the mouth cannot be shut. These symptoms are accompanied by intense thirst, and at the same time a loathing of anything in the shape of beer, wine, or spirits. I generally take milk when procurable, if not, a lemon squash. If the attack comes on before dinner, the appetite disappears, however hungry I may have been. After going to bed and getting a good sweat, and

passing black urine, I am all right again, though I feel slightly shaky for an hour or so afterwards. As a general rule my health is good between the attacks, and nothing in the shape of food or drink seems to hurt me. The blackness of the urine depends on the severity of the attack, and so do the above symptoms. The attack can never be, or rather has never been, brought on by the exposure of the previous day. The attacks come on in the summer just the same if I am exposed to cold, but of course the winter is the worst time. The chills never occur without exposure to cold. None of my family have ever had these chills, or even symptoms of them ; the same with regard to my two girls, aged sixteen and eighteen. I have never been exposed to the risk of getting ague; have never been abroad, except a short visit to Belgium. It is difficult to name the various causes of the chills, as their name is legion. In general, as before remarked, any exposure to cold brings on an attack. One was brought on by trying to stop a leaky pipe in winter (the hands and arms were immersed in the cistern), and which so benumbed the hands that, as a matter of course, a chill came on." This patient contracted primary syphilis as a young man, but had no subsequent manifestations.

These cases serve to illustrate most of the important features of this curious condition. All three were males. In each the disease began abruptly on exposure to conditions which had not previously excited an attack. In one the disease ceased under treatment, and he has remained free from symptoms for fifteen months; it is impossible to say whether the condition will return. In the second case the patient has had two transitory attacks with an interval of eighteen years between them, though through all those years he was exposed to the same conditions as evoked the two attacks from which he has suffered. In the third case, the disease once excited has persisted for twenty-three years, in spite of much treatment. The disease we are considering presents, as several writers have remarked, considerable practical difficulties in its study. The patients are often not sufficiently ill to voluntarily become inmates of hospital wards; whilst if they do so, they, as a rule, owing to the uniform temperature, remain free from attacks. Hence it is that few opportunities are afforded of studying all the features of an attack *ab initio*. The same difficulties are experienced in private practice. In spite of these obstacles, our knowledge, though manifestly incomplete, is gradually increasing, and I propose to review what is the present state of our information concerning the disease.

First, as the most obvious and important matter, with regard to the condition of the urine. A patient, whose urine has previously manifested no observed peculiarity, on exposure to cold, or, in rare

cases, to some other exciting cause, notices that it has the colour of blood or of porter. In a few hours usually it regains its natural appearance, to become blood-coloured again on exposure to the same exciting cause. It is convenient therefore to consider the urine passed in the paroxysm and that passed between the paroxysms separately.

The paroxysmal urine.—When freshly passed it sometimes has a colour as deep as porter, or it may be of a deep port-wine colour, or of any tint from these to a little deeper than the natural straw colour of health. The tint is dependent, as a rule, on the degree of cold to which the patient is exposed, and on its duration. In ordinary well-marked attacks it is port-wine or porter coloured. The urine when freshly passed is usually slightly turbid from suspended urates and hæmoglobin granules. On standing usually a copious sediment falls to the bottom of the vessel, and the supernatant fluid becomes clear and blood-coloured to a varying degree.

That the colour is due to blood is shown by the guaiacum and Heller's tests, and by spectroscopic examination. In most cases the spectroscope gives two well-marked absorption bands between the orange and green of the spectrum, or between D and E of Frauenhofer's lines. In not a few cases, in addition to those bands which are characteristic of oxyhæmoglobin, an additional band is seen in the red portion of the spectrum near its middle, and is due to the presence of methæmoglobin. There are certain discrepancies in the statements of different writers as to the significance of the various absorption bands. The matter is ably discussed by Drs. Forrest and Finlayson,* and by Dr. Ralfe, in his recent 'Clinical Chemistry.'† There is no doubt that, in many cases, or Dr. M'Munn would go so far as to say most cases,‡ methæmoglobin is present, but it certainly is not in others. I had numerous opportunities of examining the blood urine of Case 1, and several opportunities of examining that of Case 2, passed in my presence, and therefore quite fresh, but in each of these only the two bands between D and E were present. On one occasion Dr. Meymott Tidy found hæmatin in the urine of Case 1, which had been kept for more than a day, and he suggested as an explanation

* 'Glasgow Medical Journal,' June, 1879.

† 'Clinical Chemistry.' By C. H. Ralfe. Cassell & Co. 1883.

‡ 'The Spectroscope in Medicine.'

that it had been placed in a dirty bottle. Albumen is always present when blood-colouring matter is in the urine, and usually in proportionate amount. It behaves to reagents much as albumen does in ordinary circumstances, only it is, when precipitated, coloured of a dirty brown or chocolate colour by the dissolved hæmoglobin. Some have described special peculiarities of the albumen: that it floats on the surface (Lichtheim*), and that it is readily dissolved by slight excess of acid (G. Harley†); but neither of these peculiarities is constantly observed, and Dr. Saundby‡ has shown that serum albumen is present in addition to paraglobulin. An important point on which further information is needed is whether, in the paroxysm, the albumen always precedes, is exactly contemporaneous with, or follows the appearance of the colouring matter. Rosenbach§ has shown that in the beginning of a paroxysm albumen may precede the appearance of blood-colouring matter. It must be remembered that urine may have a colour scarcely exceeding the natural, and yet give (as in Case 2), after a few minutes' interval, the blue colour with guaiacum, and on standing deposit a few hæmoglobin casts. As a rule, however, the albumen and blood-colouring matter appear to go hand in hand.

There is nearly always an abundant coffee-ground-like deposit. The most remarkable feature of this is that, in spite of the proved blood origin of the colour of the urine, no blood-corpuscles are, as a rule, present. They are not entirely absent in all cases, but in none in which they are found do they bear any relation to the amount of colour. Drs. George Harley, George Johnson,|| Rosenback¶ (in experimentally induced attacks), and others, have found a few red corpuscles. Dr. Wickham Legg, in his valuable article on this disease,** writes that a gentleman had an attack while waiting to see him, and the urine passed in his presence was immediately examined. "There was no difficulty in recognising many red blood-corpuscles. The urine was set aside till the next day, when it was again examined. This time I could not discover a single corpuscle." I had repeated opportunities in Case 1, and

* 'Volkmann's Sammlung Klin. Vorträge,' No. 134.
† 'Med.-Chir. Trans.,' vol. xlviii, p. 170.
‡ 'Birmingham Med. Rev.,' vol. xi (reprint).
§ 'Berlin klin. Woch.,' 1880, No. 10, p. 132.
|| 'Med.-Chir. Trans.,' vol. xlviii, p. 181. ¶ Op. cit.
** "On Paroxysmal Hæmaturia," 'St. Barth. Hosp. Rep.,' 1874, p. 77.

several opportunities in Case 2, of examining the freshly-passed urine when bloody, but on no occasion did either contain blood-corpuscles; nor did that of a case previously published by me.* All are agreed that the presence of red blood-corpuscles is occasional, but exceptional, and this gives the disease one of its peculiarities. The copious deposit consists in most cases of amorphous urates, and amorphous or granular pigment-matter, hæmoglobin granules. In some cases casts, hyaline, granular, and pigmentary, are present. They were depicted by Dr. Harley in 1865.† In some cases casts containing blood pigment are always found, as in Case 2, and in others, as Case 1, I have never found a single cast. Dr. Beale has stated‡ that the granular appearance of the casts is due to urates, and may be made to vanish by heat. I have never tried this, but it will be seen that the appearance of the casts in Case 2, which I hand round, corresponds very closely with that in the tubules of Dr. Dreschfeld's sections of the kidney from his case of hæmoglobinuria from chlorate of potash, and which by his kindness I have the opportunity of showing. These correspond very closely with the "peculiar brownish cylinders" described by Ponfick in the urine after transfusion.§ In many cases crystals of oxalate of lime have been found in the urine (Harley, Greenhow,‖ Adam,¶ my own previous case on one occasion, and in several other cases). They were never present in Cases 1 and 2, nor in many other recorded cases. Van Rossem** has founded a theory on their assumed constant occurrence. He believes that blood, as such, was poured out by the kidneys, and dissolved in the bladder by the oxalates. The fact that, in many cases, they are never present refutes this hypothesis. Uric acid has also been found in several cases, but more often it is absent. Blood-crystals have been found in two cases; once by Sir William Gull;†† and in a case under Dr. Grainger Stewart, one hæmatin crystal was found on a single occasion by Dr. Strang.‡‡ Epithelium from the kidney and

* 'The Lancet,' July 26th and Aug. 2nd, 1879. † Op. cit.
‡ 'Practitioner,' 1868, vol. i, p. 73.
§ 'Virchow's Archiv,' 62.
‖ 'Trans. Clinical Soc.,' 1868, p. 40.
¶ 'Glasgow Med. Journ.,' June, 1879.
** 'Dissert.,' Amsterdam, 1877.
†† 'Guy's Hosp. Rep.,' 1866, p. 381.
‡‡ 'Brit. Med. Journ.,' 1878, vol. ii, p. 103.

bladder is found in greater or less abundance in many cases. Sometimes the nuclei of the cells have imbibed the blood pigment and are of a ruby-red colour. A few leucocytes are also often met with. The reaction of the urine is usually acid, and in my experience, as well as in that of Boas, often remains so for many days. It may, however, be neutral or alkaline when freshly passed, as in a case recorded by Rosenbach. The specific gravity is usually high,* but it may be low, as Lichtheim, Rosenbach, and Boas have shown.† Probably the specific gravity, reaction, and some other points vary in different cases in relation to food and personal peculiarities, some of them, perhaps, independent of the disease. The urea has been found increased in some cases, diminished in others. The urine remains blood-coloured, with its accompanying peculiarities, for a varying time in different cases, probably depending in the main on the degree and duration of the exposure and the individual susceptibility. In some cases that voided after the chill has passed off is quite natural in appearance, if the bladder has been emptied previously of black urine caused by the chill. In other cases the second, third, or even later evacuations of the bladder are more or less coloured, the colour lessening with each succeeding micturition. It has been known to remain bloody for a fortnight or three weeks.‡

Inter-paroxysmal urine.—This in the majority of cases appears natural, and that it is so practically is shown by the fact that cases have been watched for periods of many years without marked changes in the inter-paroxysmal urine being detected, or evidence arising as to any organic affection of the kidney. The inter-paroxysmal urine may be of a high or low density, the urea equal to or less than the normal. Albumen may persist after all traces of blood-colouring matter have disappeared,§ and traces of hæmoglobin may be present in inter-paroxysmal urine free from albumen (Saundby). In Case 2 a violet tint was produced on boiling the urine free from obvious colouring matter with nitric acid, and Dr. Wickham Legg "noticed a deep violet colour arise when some of the urine passed between the paroxysms was treated with strong

* G. Harley and Dickinson.

† Boas, "Paroxysmale Hämoglobinuria," 'Deutsch. Archiv für klin. Med.,' vol. xxxii, 1883, p. 355.

‡ Dr. Cock's case, quoted by Dickinson.

§ Forrest, op. cit.

hydrochloric acid." Indican has been found in several cases (Gee, Virchow, &c.). Though such a circumstance appears to be unusual, it may be expected that organic kidney disease will sometimes supervene upon this affection, for the ordinary causes acting in this direction must be in operation to a greater or less degree. Thus, perhaps, may be explained some cases in which albumen is found apart from the attacks. Dr. Sutton has recorded the occurrence of uric acid renal calculi occurring in a patient who had previously suffered from characteristic attacks of paroxysmal hæmoglobinuria following exposure to cold.†

Other phenomena of the paroxysm.—In most cases a chill precedes the occurrence of blood in the urine. In some cases, as in those recorded by Dickinson, Lichtheim, Rosenbach, and Boas, and in Case 3, the initial symptoms consist of immoderate yawning and stretching of the limbs. The chill may be slight, or it may go on to a regular rigor. The patients are nearly always conscious of the sensation of cold, and they often know by experience whether the sensation of cold is sufficiently intense to be followed by bloody urine. The sensation of coldness may be limited to exposed parts or to the extremities, but often it is general. In artificially induced attacks blood has appeared in the urine without outward manifestations of a chill.† There is often a condition of cutis anserina excited, and urticaria has been present in a fair number of recorded cases; the urticaria concurring with the bloody urine following cold, and often limited to exposed parts (as in my previous case, and as observed by Forrest, Lichtheim, Küssner, and others). In some cases in the cold stage, the patient's nose, ears, and hands become dusky or cyanotic; and occasionally this condition may go so far as to assume the appearance described by Raynaud‡ as local asphyxia or symmetrical gangrene. Conversely, in cases of Raynaud's disease, hæmoglobinuria has been shown to exist (Wilks,§ Southey,|| Barlow, and Mahomed¶). Accompanying the chill and other symptoms there is sometimes great thirst, as in Case 3. Boas mentions in one of the cases in which he induced attacks that the

* 'Hunterian Society Report,' 1878-79, p. 53.

† Rosenbach, op. cit., p. 134.

‡ 'De l'Asphyxie Locale' (Paris, 1862).

§ 'Med. Times and Gazette,' 1879, vol. ii, p. 207.

|| 'Clin. Soc. Trans.,' 1883, vol. xvi, p. 167; ibid., vol. xvi, p. 179.

¶ Discussion at the Clinical Soc., 'The Lancet,' May 5th, 1883.

patient drank about two litres of water in a short time. Drowsiness often supervenes. In some cases headache is a marked symptom. Pains are sometimes present in different cases, in various positions. Some patients have severe colicky pains in the abdomen, some have pain in the back. There is sometimes tenderness on deep pressure over the kidneys in the back. The testicles are sometimes retracted (Dickinson). The temperature in some cases remains normal throughout; in others there is a marked rise (as high as 105·2° F., Saundby), and the rise may be preceded by a fall of temperature which may be general, or limited to the exposed parts. A valuable case giving records of the temperature in intentionally, but naturally, induced attacks is given by Dr. Matthew Charteris.* Sweating often accompanies the reaction from the chill, and it may be general or local. Nausea and vomiting are sometimes observed, both in the spontaneous and induced attacks. The patient sometimes has a feeling of intense prostration in the attack, and there may be collapse.† In some an icteric tint in the skin and conjunctivæ follows an attack, or, if previously present, becomes intensified. The liver and spleen have been found enlarged in one or two cases, an increase taking place during the attacks; but these conditions are too inconstant to have any bearing on the essential nature of the disease. The duration of the attack varies in different cases, and depends probably on the duration of the exciting cause and the individual's susceptibility to its influence.

Such are, in general, the phenomena of a paroxysm. Between the attacks the patient may feel and appear quite well, or he may feel weak and prostrate, and have an anæmic or faint icteric tint. That the patient should become weak and pale when we consider the amount of blood destroyed is not surprising. Laache‡ has shown that in a case of Heiberg's the corpuscular richness fell to 46, and the hæmoglobin to 52, per cent. of the normal. A more remarkable fact is that in some patients, as in Case 1, the appearance of health is preserved throughout, and it must be taken to show how quickly blood losses are repaired in such. Working men as a rule return to work the day following an attack. Case 3 went out shooting the day following his severe initial paroxysm.

What is the pathology of this curious disease? Two things are

* 'The Lancet,' Aug. 30th, 1879, vol. ii, p. 306.

† Rosenbach, 'Berl. klin. Wochenschr.,' 1880, No. 10, p. 434.

‡ 'Die Anæmie,' p. 45, Christiania, 1883.

certain from the observed phenomena of the paroxysm. 1. That a blood dissolution takes place. 2. That in the majority of cases this is brought about by the influence of cold. We shall presently see that probably there is a second factor contributing to the blood dissolution. I think there can be no question that a blood dissolution or destruction takes place. In the overwhelming majority of cases blood-colouring matter only is found in the urine, and the occurrence of blood-corpuscles must be regarded as infrequent, unessential, and accidental. We must examine into the evidence as to where the blood dissolution takes place. I formerly thought that the kidney itself was its seat; that in the attack, owing to chilling of the surface and contraction of the cutaneous vessels, blood was driven into the renal glomeruli under such pressure that the hæmoglobin was expressed from the corpuscles and excreted by the kidneys.* Drs. Roberts† and Pavy‡ also believe that the blood escape is produced by altered vascular conditions in the kidney produced by cold through the influence of the nervous system. Murri formerly advanced a view identical with that I ventured to suggest, but he has since discarded it. I based my opinion chiefly on the fact that the blood serum had not been found to contain dissolved hæmoglobin. Since then, however, valuable additions have been made to our knowledge, and it is necessary to reconsider the question. Hayem, Küssner, Boas, Strübing,§ and Fleischer‖, have shown that the serum of the blood, obtained by cupping the patient, does contain dissolved hæmoglobin, and that it varies in tint from a yellow to a ruby red. Whether this is true of all cases or not it is certain then that it occurs in some. It is well known that when blood dissolution takes place, and the colouring matter circulates in the blood stream ("Hæmoglobinæmia," as Ponfick calls it), when it reaches the kidneys they separate and excrete it. Blood dissolution is brought about in a variety of ways, by the injection into the blood or tissues of diluted glycerine, naphthol, arseniuretted hydrogen, chlorate of potash, phosphorus, bile acids, ether, blood dissolved by freezing, &c., and it occurs in connection with typhus, yellow fever, and scarlet fever, occasionally, and after severe burns. The hæmo-

* 'The Lancet,' 1879, vol. ii, pp. 116 and 155.

† 'On Urinary and Renal Diseases,' 3rd edit., p. 152.

‡ 'The Lancet,' 1866, vol. ii, p. 34.

§ 'Deut. med. Woch.,' 1882, No. 1.

‖ 'Berl. klin. Woch.,' 1881, No. 47.

globinuria of cattle and horses I have not time to discuss; in that of the latter hæmoglobin has been shown to be dissolved in the serum. It is important to clearly discriminate between these causes of hæmoglobinuria and the paroxysmal disease excited by cold, but these conditions may throw a side-light on some of its phenomena. As already stated, when hæmoglobin dissolved in the blood serum reaches the kidneys, they excrete it as long as their powers remain intact. Bridges Adams has shown* that the hæmoglobin is separated both in the glomeruli and tubes, but in Dr. Dreschfeld's case brought before the International Medical Congress in London, in conjunction with Mr. Stocks,† it will be seen that the blood-colouring matter is confined to the convoluted and straight tubes, none being found within the glomeruli or Bowman's capsules. Moreover, as Ponfick has shown,‡ when the blood of an animal of a different species is transfused into the circulation, the foreign elements part with their hæmoglobin, and perish. Coincidently with this circulation of dissolved hæmoglobin there occur in the human subject, when lamb's blood is transfused, rigors, high temperature, and later sweats; and the phenomena of paroxysmal hæmoglobinuria may, as Lichtheim has pointed out, be so far imitated as to induce an attack of nettlerash. If the amount of hæmoglobin in the blood is very great, the secreting power of the kidneys is injured, and if sufficiently prolonged, it may cause death by pathological changes induced in them (Ponfick).

It is clear from these considerations that dissolved hæmoglobin does circulate in the general blood stream, in some cases at least; that some of the symptoms observed in these paroxysmal cases may be due to this cause; and that the kidneys may be passive, merely eliminating the hæmoglobin. Lepine distinguishes two forms of the disease—in one of which destruction of blood-corpuscles takes place in the blood, whilst in the other it occurs in the kidney.

If the blood dissolution occurs in some part of the circulation apart from the kidney where does this take place? To the solution of this question Boas has made some observations of much importance. He made a patient subject to the attacks breathe for half an hour air cooled by passing over iced water. The experiment was repeated several times without the patient experiencing the subjec-

* 'Centralblatt f. d. med. Wissenschaft,' 1881, s. 240.

† 'Trans. Intern. Med. Congress,' 1881, vol. i, p. 398.

‡ "Exper. Beiträge," 'Virchow's Archiv,' Bd. 52.

tive symptoms of an attack, or alterations taking place in the urine; but during the shivering stage of an attack induced by the patient holding the hands in iced water, the serum obtained by cupping was found to contain hæmoglobin, and the urine to be bloody. On pricking a finger and examining the blood, he found an absence of the natural rouleaux; the red corpuscles were paler than natural, they appeared larger and more translucent than normal, their shapes were altered so that many were spindle-shaped, three-cornered, and oval, and, what was especially noteworthy, there was an alteration (Moderabilität) of their contour. These appearances were compared with those of the blood of a healthy man, drawn and examined at the same time. In a second case, in a well-marked experimentally induced attack, the blood showed no abnormality, except a marked increase of white corpuscles, which he considered physiological. A third patient, a subject of the disease, placed his middle finger in a glass of broken ice. After about a quarter of an hour blood drawn from the cooled finger was examined. It showed no alteration in the number of red blood-corpuscles, but rouleaux formation had quite disappeared, and great alterations had taken place in the shapes of the corpuscles. In addition to these changes larger and smaller polymorphous, opaque, dark-brown blood-flakes (Schollen) were seen. Such blood-flakes he had observed in normal blood, but not in such numbers. Still more evident were the changes in another finger of the patient's same hand, which finger had been shut off from the general circulation by means of an elastic ligature. (He had assured himself by observations on his own and the patient's blood when so shut off that no alterations were so produced). After this bound finger had been in iced water for ten minutes the blood showed the same alterations as in the former examination, but much more marked, with a greatly increased number of blood-flakes. A third finger constricted, but not cooled, showed only scattered blood-flakes, no alterations in the blood-corpuscles. These experiments were repeated on another occasion. He found in a constricted finger cooled in iced water a great tendency to alterations in shape of the red blood-corpuscles, some corpuscles devoid of colour (Ponfick's phantom corpuscles), others showing a granular clouding, besides blood-flakes between the masses of corpuscles which did not form rouleaux. Examination of the patients' fingers not so cooled showed no alteration in the number of red or white corpuscles. The colour of the red corpuscles was quite natural, but they showed an

extraordinarily delicate condition, appearing to have a tendency to dissolution and form alterations.* Under the transparent cloak of the initial D, a writer in the 'Medical Times and Gazette,'† who had himself at that time suffered from the disease eleven years, says, "If a drop of blood from the pricked finger be examined under the microscope, the globules appear to be deficient in outline, soft, and soon melt down into an irregular network. In fact, I should describe the essence of the disease as a hurried and imperfect formation of blood-corpuscles, which readily stagnate in the capillaries under cold and quickly perish, and so are eliminated by the kidneys as a broken-down mass of blood stuff, whereas, under healthier circumstances, they would be discharged as bile and urinary pigment." Murri has also depicted marked alterations in the shape of the red blood-corpuscles. J. Wolff‡ reported pœcilocytosis, microcytes, pale colour of red blood-corpuscles, diminished tendency to rouleaux formation, but no phantom corpuscles. He noticed no differences between the paroxysmal and inter-paroxysmal blood, nor in the blood of the ligatured finger immersed in cold water. On the other hand, Rosenbach,§ in an artificially induced attack, found no alteration in the blood during the paroxysm, beyond an increase of the white corpuscles (six to eight in the field). Hayem found no alterations except the appearances of anæmia. In Dr. Wilks' important case there was only noticed an increase in size of the white corpuscles and excess of fibrine.‖ In Dr. Grainger Stewart's case the only peculiarity noticed was that the corpuscles did not form rouleaux, some of the corpuscles sticking together by their edges, but the bulk of them formed into irregular masses. The colourless corpuscles were not increased in number, but some of them were pigmented. The patient had had ague in India.¶ I examined the blood of my previous case and of Case 1 (I think, also, that of Case 2), when they were passing black urine, but not in the cold stage, but could not find any alterations in the appearances. Dr. Adam examined the blood three

* Boas, "Paroxysmale Hæmoglobinuria," 'Deutsch. Archiv. für klin. Med.,' 1883, p. 374.

† Vol. i, p. 215, 1879.

‡ 'Centralblatt für die med. Wissenschaft,' 1883, p. 820.

§ 'Berlin. klin. Wochensch.,' 1880, p. 134.

‖ Op. cit.

¶ 'Brit. Med. Journ.,' 1878, vol. ii, p. 103.

days after an attack, but the only noticeable peculiarity was an absence of rouleaux formation.* The examination of the blood in different parts of the body seems to be a direction in which good work may be done, and cases of local asphyxia appear especially deserving of study. Boas concludes from his own observations that in each attack the primary condition is a destruction of the red blood-corpuscles, taking place locally in the parts exposed to cold, and that the general symptoms are secondary to this blood destruction and the circulation of the dissolved hæmoglobin. No evidence exists as to whether bile acids are present in the blood in this disease, paroxysmally or inter-paroxysmally.

So far we have only considered the blood dissolution and elimination. The circumstances of the attack indicate an exaggerated sensibility of the cutaneous nerves or their centres. Raynaud and Bernhardt believe that local asphyxia depends on increased irritability of the vaso-motor centre in the medulla oblongata or spinal cord, kept up by peripheral stimulation, in consequence of which slight depressions of temperature are sufficient to excite changes that, in healthy persons, would only be induced by exposure to a very low temperature, or exposure for a long time. This is an essential factor in the development of the paroxysms. On what it depends we do not know. Healthy persons exposed to the same degree of cold do not get the chills. The concurrent urticaria of some cases indicates the neurosal nature of the disease. There have been recorded, however, some cases in which severe muscular efforts have been the exciting causes of the attacks. Fleischer† and Strübing‡ have recorded cases in which the attacks followed prolonged marches. In Sir Wm. Gull's case the blood in the urine was increased by lifting a heavy weight or walking much; and in one of Dr. Wickham Legg's cases the first attack was thought to follow a long ride. Dr. Druitt, moreover, mentions that psychical disturbances are sufficient to induce attacks in a very sensitive subject, whose attacks were usually caused by cold.§ In the majority of cases the exciting cause is cold acting on a hypersensitive reflex nervous system. Whether the blood destruction occurs in all cases in the exposed parts, or in others in the kidneys or elsewhere, I consider not quite determinable at present. Boas's observations have

* 'Glasgow Med. Journ.,' 1879.

† Op. cit. ‡ Op. cit.

§ 'Medical Times and Gazette,' 1873, vol. i, pp. 408, *et seq.*

evidently been made with great care and completeness, and are entitled to careful consideration and repetition. It is interesting in connection with paroxymal hæmoglobinuria to notice the occasional association of temporary albuminuria from exposure to cold. Dr. George Johnson has recorded some cases* following cold bathing; and Dr. Hermann Weber has mentioned† a case of hæmaturia and albuminuria following immersion in water. I have a case under observation where albuminuria, and occasionally hæmaturia, follow unusual exposure to cold.

There remains to consider whether there is not another factor in the production of the blood dissolution, which has been foreshadowed in the observations as to the appearances of the blood-corpuscles. Have the blood-corpuscles in the subjects of this disease a lessened resistance to the action of the conditions to which they are exposed (cold and possibly carbonic acid), so that they part with their hæmoglobin more readily than in the circumstances of health? The evidence supplied by the appearances of the blood-corpuscles is suggestive, but at present inconclusive. But there is other evidence. A considerable number of the recorded cases have suffered from syphilis, and some of these have been cured of their hæmoglobinuria by antisyphilitic treatment.‡ Others have undoubtedly previously suffered from ague, though certainly in many cases there is no evidence of this agency being in operation. Some again appear to have been rheumatic, as in a series of cases recorded by Dr. Fenn, of Richmond.§ It is possible that in these conditions a certain imperfection of blood-formation takes place, and that the connection between the stroma and hæmoglobin of the red blood-corpuscles is less tenacious than in healthy corpuscles. In this way it may be that constitutional states are an element or factor in the production of some cases. Thus possibly may be explained the second outbreak of the disease in Case 2, when his health was deteriorated, probably in connection with the long-standing ulceration of the skin (strumous?). In this way may possibly be explained the other fact that whilst in some cases the disease persists for years, in others it is a temporary and evanescent condition. Morbid anatomy has thrown no light on the

* 'Clinical Society's Transactions,' vol. vii, p. 42.

† 'British Medical Journal,' 1873, vol. ii, p. 664.

‡ Murri.

§ 'British Medical Journal,' 1878, vol. i, p. 465.

disease. Murri found in one case both kidneys disseminated with tubercle, and displacement of the right kidney (with two renal arteries). There were large collections of pigment in the cortical tubes. He thinks that the post-mortem appearances in this case, and in another he mentions, throw no light on the nature of the disease.* Otto has published another case. "Soon after the attack recorded the patient died of an intercurrent disease, the urine remaining normal in colour till death. The left kidney was found twice as large as the right, which was of normal size. Both kidneys are said to have been normal in structure, but it is not stated if they were examined microscopically."† It is not likely that morbid anatomy would reveal any pronounced lesion in a disease that may last upwards of twenty years without seriously disturbing the health.

It is possible that under this disease there are conditions varying in their causation. It occurs at all ages, with a very marked predominance in young adult males. It may, as in an instance recorded by Dr. Saundby, be hereditary; a father and at least two children having suffered. It often begins quite abruptly, under conditions apparently similar to those to which the patient has been repeatedly exposed without evoking an attack. It may prove transitory, lasting a few months or less, or it may prove a life long companion. When apparently cured it is very prone to recur. The disease exists in various degrees of intensity, and it is possible that minor degrees of it occur in untrained observers without recognition. In some cases immersing a finger or two in iced water will provoke an attack; in others, continuous exposure to a considerable degree of cold is necessary to call it forth, and even then the symptoms may be exceedingly slight. The disease is, as a rule, more frequent in winter than in summer, simply because the opportunities to exposure to cold are more frequent then. The disease occurs in summer, as is shown in Cases 1 and 3, and it has indeed been shown in one case that the attacks were more frequent in summer,

* 'Dell' Emoglobinuria da freddo Revista Clinica di Bologna,' 1880. Murri believes now that the essence of the disease is—(1) in a diseased condition of the blood-forming organs, which imparts to a number of the red blood-corpuscles a diminished resistance against cold, and perhaps against CO_2; (2) in an increased irritability of the vaso-motor reflex centre.

† 'Berlin. klin. Woch.,' 1882, No. 39, and 'Medical Times,' 1882, vol. ii, p. 474.

owing to the patient remaining indoors in the winter. The disease is stated to be not uncommon in India. Of course, in hot climates opportunities are afforded of getting chills, while at the same time they can be avoided. Probably malaria is an important factor in these tropical cases.

In conclusion, one or two words only on treatment. The uncertainty which underlies the pathology of the disease must be reflected in the treatment. We have seen that there is some evidence that constitutional conditions are a factor in the production of the disease. Whenever, therefore, any dyscrasic condition is discoverable, our treatment must be directed against it. Where syphilis has been an etiological influence, the disease has in several cases, it is asserted, been cured by mercury and iodide of potassium. Syphilis should certainly be always inquired for, and, when there is positive or presumptive evidence of its existence, antisyphilitic treatment tried. In some obviously malarial cases benefit has followed the administration of quinine in full doses. Quinine has, moreover, proved serviceable in a great many cases in which there has been no evidence whatever of malarial infection. Indeed, with the exception of some syphilitic cases and one case treated by the late Dr. Warburton Begbie by ammonium chloride, most of the recorded cases that have improved have done so when taking quinine. It is possible that some of them would have got better without it. Most patients find avoidance of cold, the usual exciting cause, the only effectual treatment, and just in proportion as they are able to protect themselves from cold by clothing and habits do they escape attacks. In this way some have sought immunity, with varying success, in warmer climes. Whilst experience teaches nearly all patients that cold is to be especially avoided, it is possible that a gradual accustoming the hypersensitive integument to cold may be at once the most philosophical and most successful treatment. Dr. Barlow has mentioned a case* in which marked benefit had occurred by washing a child suffering from this disease with cold instead of warm water, and another patient of Dr. Barlow's reported benefit from this treatment. In a case published by Küssner, and supplemented by Boas,† the patient was benefited so much by cold water treatment (Kaltwassercur) that on leaving the hospital he was able to go out into the cold air without

* 'The Lancet,' May 5th, 1883.

† Op. cit.

an attack being produced. Only when he went back to work, which involved continuous exposure to cold, did the paroxysms again make their appearance. I have not tried this treatment myself, but I think Dr. Barlow's suggestion well worthy of trial, and shall certainly employ it in the next suitable case I have to treat.

It will be seen that great uncertainties still underlie our knowledge of the disease. The following seem directions in which careful observations are likely to throw light on the pathology of the disease.

A. Microscopical examination of the blood. 1. Between the paroxysms. 2. During the paroxysms. (*a*) In exposed parts, or in parts artificially cooled, with and without previous ligaturing. (*b*) Simultaneous observation at other and unexposed parts.

B. Examination during the paroxysms (by spectroscope or guaiacum test) of the blood-serum obtained by cupping or a blister.

C. Observations as to whether albumen precedes the appearance of blood-colouring matter in the urine, in the paroxysms, and whether it remains for a time after all trace of blood-colouring matter has disappeared.

The PRESIDENT said that he had seen cases in India which he could not doubt were examples of paroxysmal hæmatinuria, and some of these had happened in the course of intermittent fevers.

Dr. DICKINSON had had large experience of such cases. Sixteen cases had been under his own care, and for one more he was indebted to Sir William Jenner. One fact which came out very prominently from this collection of cases was the remarkable influence which ague or malarial disease seemed to play in the causation. Five of the cases had had ague, and three had resided in a malarial district. In two, other members of the family had suffered from ague; and except in three cases, a strong presumption of malarial influence existed. It must be remembered that malarial conditions of body are very tenacious and come out whenever they have a chance, thereby earning the title of an insidious disease. Paroxysmal hæmatinuria resembled an attack of ague in many ways. It might be said metaphorically that the disease eliminated itself by the kidney rather than through the skin, as in ordinary ague. Then the association of ague and hæmatinuria had been noted, as in a case recorded by Dr. Mackenzie. Dr. Dickinson had had under his care a young woman who had contracted malaria in India. She had undoubted attacks of ague associated with hæmaglobinuria, hæmorrhages in various parts of the body, and purpura. Under the influence of quinine the patient got nearly well, but died some time later from perforation of a typhoid ulcer. At the autopsy, the kidneys showed signs of inflammation, in the overgrowth of fibroid tissue and in the blocking of tubes. Besides, there were many areas of interstitial hæmorrhages to be seen around, but not in, the Malpighian bodies. That cases get well of their own accord there could be no doubt. And of the value of large doses of quinine continued for a long time Dr. Dickinson had no doubt.

Dr. THEODORE WILLIAMS had seen one case in the person of a young officer who had had ague. The malady disappeared without treatment.

The PRESIDENT remarked that the fact was that a paroxysm of intermittent fever was the least frequent expression of malarial disease. The affection soon tends to lose all periodicity.

Dr. MACKENZIE, in reply, said he did not ignore ague as an element in the causation of the disease. But there were cases, as in one of his own, where no suspicion of malaria could be entertained.

November 19th, 1883.

ON POSTURES INDICATIVE OF MENTAL STATES.*

By FRANCIS WARNER, M.D., F.R.C.P.

A POSTURE may be defined as the relative position of the members. Postures, being the results of the last movements, are indications of the nerve mechanism which produces such movements. Positions of the parts of the body resulting from the action of opposing muscles, as seen in the limbs and face, may be called postures of these parts. The results of movement produced by the brain in various mental conditions may be thus studied; the positions being looked upon as the motor action accompanying that kind of brain action which is called mentation. In making clinical observations, children in various nervous conditions were the principal subjects; subsequently similar postures were seen in statues and other art works. The "nervous hand" of a neurotic child presents the wrist drooped, the metacarpo-phalangeal joints hyper-extended, the fingers slightly bent, the thumb drawn backwards—such a posture is seen in either hand of the Venus de Medicis. In the Diana of the British Museum is the representation of a strong woman, and the free hand is in an exactly antithetical posture to that of the Venus; wrist extended, fingers and thumb flexed. This posture is often seen in energetic conditions of the mind—*i.e.*, it is a common motor outcome of that brain condition whose mental state is called "energy." It is not thought that such facts are accounted for by assuming that they are due to heredity; inquiry and experiment are necessary to explain the causation of spontaneous postures. The Cain of the Pitti Gallery, Florence, was cited as giving the posture of a hand in

* This paper is published *in extenso*, with illustrations in 'The Journal of Mental Science,' April, 1884.

fright. Here, as in the Venus, both hands are in a similar posture, indicating a similar condition of both cerebral hemispheres. Symmetry of postures is often observed in clinical experience; on the other hand, it is very common to observe the "nervous hand" on the left side only. In the Dying Gladiator an example is seen of postures due to organic conditions; the urgent dyspnœa here determined the postures, not the action of the brain. In the statue of Hercules at rest gravity determines the posture of the arms. From an examination of many works of art the conclusion may be drawn that frequently artists do not consider it necessary to represent the mind of their subjects by an exhibition of nerve-muscle action, but trust chiefly to physiognomy in the representation of the outcome of brain action.

Dr. Milner Fothergill said that he had been in the habit of observing hands for many years, and was convinced of one positive fact. This was that the thumb in energetic females was large and capable, whilst the opposite obtained in females who might "be pleasant, but had no mind of their own."

Dr. Money referred to a generalisation made in 1880 by Dr. Ferrier —viz., that the flexor and adductor groups of muscles were more powerful than the extensors and abductors. The latter groups were more prone to disease. It was conceivable that in "energetic" constitutions the weaker muscles, speaking physiologically, might be more powerfully energised, whilst the reverse would obtain in those states indicative of or associated with "devitalisation."

Dr. Francis Warner replied that Dr. Fothergill's remarks, though true, had to deal rather with physiognomy than with the question which was under discussion. He agreed with the remarks made by Dr. Money; but he had seen the energetic hand in cases of organic disease of the brain. The tendency of weak muscles to overact in states of abnormality he could also testify to.

SOME NERVOUS DERANGEMENTS OF THE HEART.

By J. Milner Fothergill, M.D.

The diseases of the heart proper are (1) valvular, (2) muscular, and (3) nervous. With the first division the profession at large is fairly intimate. Their essential feature is the murmur found therewith,—each its own. But a murmur when heard is not always the outcome of valvulitis; nor, on the other hand, is the characteristic, or indeed any, murmur heard always where there can exist no moral doubt about the presence of disease, especially in the sigmoid valves. Nor does the experience of the deadhouse conflict with this clinical observation. The diseases of the heart associated with a

murmur are now generally recognised. The second division includes hypertrophy, dilatation, and mural decay, otherwise fatty degeneration. With the first two forms of mural change, and the circumstances under which they are met, all are familiar; while the relations of fatty degeneration to pre-existing hypertrophy with atheromatous changes in the arteries, otherwise the condition known as "the gouty heart," are now fast being generally realised. With the nervous affections of the heart—the third division,—however, less general familiarity exists. Yet they constitute a large division, are frequently encountered, and are certainly on the increase at the present time. Consequently intimate acquaintance with them and their features is highly desirable. Of the first two divisions of heart-affections it may be said broadly, they force themselves upon the patient's consciousness rather by their consequences—some outcome of their existence—than directly. The one exception to this statement is the palpitation induced by effort in structural change in the heart, involving essentially the condition of dilatation. When the muscular wall of the heart is dilated, so that the individual fibrillæ are stretched and elongated, then demand upon the heart is followed by palpitation, or, in other words, violent systolic contractions. But when the person is quiet and the body at rest this palpitation does not occur. Its great characteristic is that it is essentially associated with *effort*. This distinguishes muscular failure from those conditions of cardiac derangement which are truly neurosal. Of course, a neurosal affection of the heart may co-exist with the condition of dilatation; and then some pains may be requisite to separate the different component factors of the case.

Putting aside such complex conditions, and also the other complex conditions where attacks of palpitation are set up by spasm of the arterioles (very common with the gouty heart), otherwise angina pectoris (vaso-motoria), as not included in the present consideration, I propose to draw attention to certain purely nervous derangements of the heart. The first of these is pure intermittency—a halt in the usual rhythmic stroke of the ventricles. It may occur at short intervals, or only be found once in one hundred beats. This is a mere disturbance of rhythm. It is very commonly met with in old or elderly men, where organic changes may readily be suspected. As so associated it is apt to occasion groundless alarm. It may possibly be linked with some degenerative change, some defective nutrition of the cardiac ganglia,

co-existent with fatty degeneration of the muscular fibrillæ; but of this we know nothing certainly as yet. Perhaps before long the subject may attract the attention of our young friends who study pathological processes and follow their minutest operations under the searching lens of the microscope. At present we can only suspect such an association from clinical observation solely; though the subject is one which promises to repay well the labour the microscopist may bestow upon it. But frequently there are no evidences of any mural decay—any degeneration of the muscular fibrillæ in cases where the halt is well pronounced. In such cases it is without significance; at least in the present stage of our acquaintance with the subject. Some years before his death the late Nestor of our profession, Dr. Archibald Billing, paid me the compliment of consulting me for an intermittent action of his heart, which was of this kind—viz. without significance. When found with other semeia of degenerative change it has a significance,—which, however, is borrowed from them rather than furnished by itself. When intermittency is increased by effort, then it is well to carefully examine the condition of the circulatory organs. When it is found with irregularity of rhythm, and this becomes more pronounced on exertion, you may be pretty certain that there is something more present than a mere "neurosal halt." As a pure neurosis, intermittency is the resultant product of some emotional hurricane, profoundly impressing the nervous mechanism of the heart. Such a case came under my notice in the person of a Canadian who had had his warehouse burnt down. Next day his pulse was distinctly intermittent, but is now almost quite regular. For some three years he carried with him the memory of that night of excitement in the form of a distinct intermittent halt in the action of his heart of which he was disagreeably conscious. Such "neurosal halt" is very frequently encountered in practice. Contrasting with this insignificant halt stands the opposite condition where a halt in the radial pulse is commonly found, yet there exists no corresponding absence of the ventricular systole. Here the cardiac contraction is often too feeble to furnish a pulse-wave extending to the radial artery, at least one perceptible to the finger; and such an asystolic condition is fraught with danger. It is, however, sometimes found in aged persons in very cold weather, where its significance is not quite so ominous.

A very common neurosal disturbance of the heart is palpitation.

As related to the hysterical temperament it is found in attacks of hysteria along with a tight artery, and the free secretion of aqueous urine of low specific gravity,—the physiological outcome of high arterial tension. At other times it is strictly emotional, and here its causal relations usually demonstrate its nature. In some cases, mostly females, it comes on during the night, awaking the patient in a fright, and causing great alarm. Especially alarming is it if accompanied by distinct halts, when the patient thinks her hour of dissolution has come—"like a thief in the night." Such nocturnal palpitation has various relationships. It is common in women at the menopause where there is a suspicion of gout; it is found in others after coitus, or after some correlative discharge from the generative organs, the *ébranlement épileptiforme* of the French. It may only be experienced in sleep, "the period *par excellence* of reflex excitability;" or may be felt also in waking moments, according to the nature of the case. In other cases it is experienced immediately after the generative act, and may be so violent and distressing as to cause the spousal relations to be entirely suspended. In one case known to me such suspension had existed for a period of seven years; but after a course of treatment the heart improved so much that coition could be resumed without discomfort. The attacks of palpitation, so related causally, may occur at other times. Some years ago a stalwart gentleman from the West of England consulted me for palpitation of the heart, and after pursuing several lines of inquiry without result, at last its relations to his spousal duties were investigated, and were found to be unmistakable. About the same time I was consulted by a widower whose wife had died of acute disease. Here violent paroxysms of palpitation were developed, relieved at once and effectually by the sexual act; but only temporarily.

The palpitation found with chorea in children is well known; and so is that found in adults with the enlarged thyroid gland and prominent eyes of exophthalmic goitre, or Graves' disease. In both cases there is some derangement of the sympathetic nerve in which the cardiac nerves are involved; and in the latter malady the disturbance is very intractable to treatment.

Then, palpitation of the heart may be set up by some abnormal condition existing elsewhere. Quite frequently in the out-patient department of Victoria Park Hospital I encountered cases of palpitation in women in which a displaced uterus was the provoking

cause. The conclusive proof of the relationship is furnished by the fact that though little or no relief ensues from treatment before the offending uterus is replaced, so soon as that organ is once more in its normal position the attacks of palpitation disappear,—as if dispelled by an enchanter's wand. When then, palpitation of the heart is found with evidences of uterine displacement, it is well to call in the aid of the gynæcologist, when a satisfactory result quickly follows. Quite commonly palpitation is linked with ovarian irritation. Here there is also very frequently "pain in the side;" or, to speak more correctly, in ovarian irritation there is "pain in the side" often accompanied by palpitation. Here the irritation set up in the offending ovary traverses a series of nerve-fibrils and passes out ultimately at the peripheral cutaneous branches of the intercostal nerves, where it is felt in gusts of neuralgic pain; or it takes the direction of the cardiac nerves and is felt in paroxysms of palpitation. In either case the origin of the neurosal disturbance may usually, with a little care, be traced to an ovary, generally the left. Not rarely the irritation disturbs the stomach, too, by nausea or vomiting such as follows pregnancy, orchitis, a blow on the testicle, or a calculus in the pelvis of the kidney. Palpitation in young, or even in adult, females is quite commonly reflex in its nature and the consequence of irritation elsewhere, and especially in the reproductive organs. Such "reflex palpitation" is most frequent in the female sex, because their emotional or sympathetic nerve-centres are less under the control of the inhibitory or governing centres than is the case in the opposite sex. In cases where males possess the feminine temperament—that is, great mobility of the nervous system—such reflex disturbances are not uncommonly manifested.

But it is not with disturbance of the reproductive organs alone such "reflex palpitation" is found. Professor Botkin, of St. Petersburg, has related a case where the palpitation was due to a floating kidney. Some time ago a gentleman came under my care whose heart-disturbance was clearly linked with irritation in his prostate gland; while in the case of a young gentleman sent up to me from Liverpool the most patient investigation could find nothing to account for the sudden onset of violent attacks of palpitation, until it was quite accidentally discovered that there had also recently been developed some irritation at the anus. They came together, and, what is more, they departed together.

There are, too, hearts whose action is persistently and continuously tumultuous; where there is a great deal of actual palpitation at times, with intervals when the heart's action is quieter, but never calm. This form of neurosal derangement, for the want of a better term, I have called "the badly behaved heart;" and very bad its behaviour is in some cases. Though most frequently met with in women, it is not confined to the female sex. Recently a Mexican consulted me with such a heart. To say that it was a perfect nuisance to him is to give a feeble description of the annoyance it caused him. In this case the disturbance was clearly related to mental worry. Beyond this it had no distinct relations; it had nothing to do with effort; it preserved no times nor seasons, and stood clear of indigestion. The only matter with which it was in any way linked was "flatulence;" and in that it only obeyed the law that when elastic gas in the alimentary canal presses upon the heart, through the thin diaphraghm, it embarrasses its action. A similar heart existed in a Jewess who attended Victoria Park Hospital, who was sorely troubled by her unquiet circulatory centre. Here the disturbance also had no definite relations: sometimes it was worse, sometimes it was better, but always tumultuous and uncomfortable, and resisted treatment for a long time; but yielded at last.

The strictly gastric relations of the arhythmic heart have not yet been clearly defined. There is, of course, the embarrassment caused to the heart by gas in the stomach, but that is not strictly a neurosal derangement. Then there is the disturbance caused by eating indigestible food, which somehow produces an intermittent halt, or other irregular action, often of the character of impaired energy, which is explained by some by the vague phrase "due to the fibres of the vagus." But beyond the two facts that there are unquestionably gastric as well as cardiac fibres in the vagus, and that the cardiac disturbance is co-existent with times of dyspeptic trouble, it is impossible to say anything. It is just as likely that the link lies in the fibres of the sympathetic as in those of the vagus nerve. From the fact that the cardiac disturbance occurs with periods of indigestion, either due to taking improper or indigestible food, or coming on when the system is wearied (the two circumstances when this blended condition is evinced), it is just as likely, and may be more likely, that the causal condition is often exhaustion of the organic ganglia. Of course I am not prepared to

deny that amidst the varied strands of the vagus there may be gastric fibrils which transmit an impression originating in the stomach to cardiac fibrils, whether those of the inhibitory or accelerator strands. In the case of a surgeon in the Indian service who suffered from intermittency at times ever since he could remember his trouble was greatly aggravated by his residence in India. Further, it seemed to follow very certainly upon a breakfast of ham and eggs with coffee during a winter stay in the hills of the north-west. In this case the trouble was greatly relieved by severe and prolonged exertion, and if the exertion were sufficiently great it disappeared altogether. A return to an easy life invariably brought back the intermissions. (This case illustrates well the great fact that while exertion reveals the incapacity of actual disease in the valves and muscular walls of the heart, it has no effect upon neurosal conditions—indeed, in some cases improves them.) I spoke a minute ago of the various strands of the vagus nerve. Among these are "inhibitory" fibres which hold the heart back, which "rein it in" (the coachman's reins), and "accelerator" fibres which hasten its action, corresponding to the coachman's whip. The first hold back the heart when their roots need blood. Disturbances of the heart's action may be set up through either set of fibres. When any irritation, direct or reflex, affects the inhibitory fibres of the vagus, then the heart's action is slow and enfeebled till any exertion is impossible. In fact, the recumbent posture in bed is enforced and movement brings on faintness when these fibres are affected in suppressed gout; and great alarm results. This, indeed, probably constitutes the "gout at the heart" you read of. When the great toe begins to swell and burn these cardiac symptoms disappear. When the accelerator nerves are the seat of irritation, paroxysms of exceedingly rapid action are set up, where the heart will beat some 200 times a minute for several hours, the paroxysm passing away as suddenly as it comes on; but leaving the heart exhausted. Such cases have come under my notice lately. In one, seen at Torquay, the patient was apparently well when quiet, but any excitement was liable to bring on one of these paroxysms of accelerated action, which not only alarmed the patient at the time, but left her very prostrate for some time after. Beyond these conditions there is that known as "the irritable heart." Here there is a certain amount of inability to bear effort in the heart. When perfect quiet is maintained the heart's action is normal; but any effort, even assum-

ing the standing posture, will cause a marked increase in the rapidity of the heart's action, far beyond what is normal. The consequence is that greater effort is only made on necessity, and the sufferer is considerably crippled for work. The circumstances under which the "irritable heart" is induced are physical exertion and mental strain,—combined and sustained for some time. Commonly it is a hardworked doctor, who has quite as much to do ordinarily as is good for him; then comes an anxious case, involving broken rest for several nights, and after that the doctor finds himself with an irritable heart; and only very slowly is this condition recovered from. So Da Costa found among the soldiers in the American civil war; so I find it among civilian patients. Like all neurosal affections of the heart, it only slowly yields to remedies. Beyond the affections outlined here, there are the well-known "smoker's heart" and the "tea-drinker's heart"—conditions of perverted action due to indulgence in these two toxic agents. Some persons cannot smoke at all, others can only smoke a little Turkish tobacco in the evening; while others again merely require to avoid strong tobacco in the early part of the day. If they exceed this, each his own boundary, uncomfortable sensations are experienced, referable to the heart, whose rhythmic action is disturbed. Precisely the same occurs in other persons with tea. These are "toxic derangements" of the heart. Some persons have habitually slow-acting hearts; but this abnormality is scarcely a "derangement."

This summary will enable us to draw some broad distinctions between organic disease and neurosal affections of the heart. In the first place organic change will reveal itself in two ways: (1) by signs discoverable upon physical examination; and (2) by the physiological indications of the effects of effort,—as shortness of breath upon exertion for instance; frequently by a lessened bulk of urine, the outcome of low arterial tension. But in neurosal affections of the heart there are no such evidences; the heart on examination is found normal except there is some perverted action. In the irritable heart, however, the effects of effort are discoverable. In other cases the neurosis has its own peculiar features, which are quite unlike those of disease of the heart—*i.e.* of organic change. It is not necessary for me to lay stress on the fact that a neurosal condition and organic disease may co-exist. It is necessary, however, for me to lay stress on this matter; there is no evidence, at least

that I have been able to find, either from my own observation or the perusal of the works of others, that a neurosal affection of the heart ever develops into organic disease as a process of development. They have no relation to each other. A neurosal affection is a nervous disturbance; an organic disease involves morbid change. Epilepsy may be merely functional, or it may be the outcome of a distinct pathological process. So we may have a "neurosal halt" in a perfectly sound heart, as well as in one undergoing fatty degeneration. Angina pectoris may be found with a heart in full textural integrity; at other times it is found with a fatty heart. The prognostic significance goes with the surroundings of each. When there are co-existent evidences of fatty degeneration, then the neurosal halt is significant, and the paroxysm of angina pectoris may end in death. When the heart walls are sound, neither causes apprehension. It is the associated conditions rather than the neurosis which weigh with us in forming our opinion of the position of the case prognostically.

As to remedial agents, the neurosal affections of the heart once more declare their want of kinship with organic disease. The combination of digitalis and iron—of such priceless value in certain cardiac conditions—is valueless in neurosal affections, often only making matters worse.

There are, then, many and valid reasons for drawing a broad distinction between the nervous derangements of the heart and those maladies which may fairly be spoken of as "heart-disease."

Sir JOSEPH FAYRER thanked the author of the paper for the lucid exposition he had given of the various neurosal diseases of the heart. He could confirm much that had been said, and especially the fact that such states were often relieved by treatment which would have done harm to sufferers from organic cardiac mischief.

Dr. SANSOM testified to the great prevalence of these maladies. He thought there was some other factor than mere dyspepsia in certain cases of cardiac intermittency. Cases illustrating his remarks were quoted showing how even single errors of diet in things not usually considered indigestible might produce the disturbance, which was as readily relieved as originated. An instance of fright leading to apparently acute dilatation of the heart was narrated as affording grounds for the belief that organic disease might be consequent on a primary neurosal affection.

Dr. WYNN WILLIAMS asked whether uric acid circulating in the blood could produce intermittent action of the heart.

Dr. ISAMBARD OWEN thought that those cases where the pulse failed at times to reach the wrist were of very grave significance. He had

seen instances of very frequent pulse unassociated with other signs and lasting for months.

Dr. Braxton Hicks gave some facts observed in his own person. Rapidity of pulse and even intermission seemed to be distinctly related to overwork or some mistake in diet.

Dr. Fothergill replied in the affirmative to Dr. Williams, and agreed with the remarks of Dr. Owen. If Dr. Sansom had seen mural disease of the heart arise from neurosal affection, he (Dr. Fothergill) had witnessed the production of valvular changes; and so there seemed but little doubt that these slight affections might lead to more grave disease. A large number of observations in every direction was what was wanted.

November 26th, 1883.

THE TREATMENT OF THE PREMAXILLARY BONE IN HARELIP.

By Francis Mason, F.R.C.S.

Mr. Francis Mason exhibited a boy, aged seventeen, upon whom an operation for double harelip had been performed (successful as far as the lip was concerned) when the patient was three months old. The object in showing the case was to elicit from the Fellows an expression of opinion as to the best mode of dealing with the projecting intermaxillary or premaxillary portion in double harelip.

Mr. Mason had for many years held that the best plan was to remove the projecting mass, and the case before the meeting tended still further to confirm his views as to this point. He took exception to the method of pushing or bending back the intermaxillary bones. In this instance they were seen to be movable and wedged diagonally in the cleft of the palate, the effect of which was to prevent the gradual apposition of the superior maxillæ. Only the two central incisor teeth projected from the intermaxillary mass. This fact tended to support the observation that the lateral incisors were absent in cases of extensive harelip and cleft palate, or, if not absent they appeared to be very ill-developed, as was the case in this instance. They were to be seen slightly projecting from each superior maxillary bone. Mr. Mason said he intended to remove the intermediate mass, and subsequently would endeavour to close the fissure, but he feared there was more than usual risk of

failure after operation in this case owing to the cleft having been kept widely open for so many years.

A specimen taken from Sir Wm. Fergusson's collection, of a case similar to the present one, was shown.

Sir Joseph Fayrer was of opinion that the removal of the intermaxillary bone would not give so satisfactory a result in the majority of cases. The incisor teeth might be removed if found injurious. It was important to maintain the width and shape of the arch.

Mr. Pickering Pick had always thought it was necessary to preserve the central bone. In this case the result had not been fortunate, because there seemed to have been a twist. Maintenance of the roundness and width of the arch of the mouth was most important. The difficulty of dealing with the flattening of the nose was very great. In his last two cases he left the tongue of skin attached to the præmaxillary bone, and after removal of a wedge-shaped piece of the septum had depressed the bones together and kept them apposed by means of an elastic bandage. The wiring of the bones together was not advocated, as liable to interfere with the development of the teeth.

Mr. Royes Bell said that Sir Wm. Fergusson sometimes removed the projection and at other times scooped out the tooth pulp. His own practice was to remove the intermaxillary bone.

Mr. Mason said he was familiar with the plan of scooping out the teeth and also with the circumstance that the bones tend to come together after the harelip had been treated. He asked Mr. Pick what became of the teeth in his mode of operation.

To this Mr. Pick replied that the teeth were believed generally to right themselves, though this was not always the case, as in the present instance.

ON SOME HITHERTO UNAPPRECIATED FACTS IN THE ANATOMY OF THE VENOUS SYSTEM.

By John Gay, F.R.C.S.

In this paper I wish to draw attention to some hitherto, I believe, unknown anatomical facts that bear upon doctrines with which we have long been familiar, but the foundations of which, being laid during what I may call the transitional period of anatomical research, may very reasonably become subject to modification if rightly transferred to some other and more substantial basis. Since 1837-38, when the Lettsomian Lectures, "On Some Special Diseases of the Veins," were delivered by me, I have devoted considerable attention to the further study of the venous system. This has been more than repaid by the abstract enjoyment I have derived from that source. The facts I am about to relate are the results of a

series of experiments by injections, conducted for a long time past with every effort to render them exact, intelligible, and trustworthy. The conclusions at which I have arrived, and of which I can entertain no doubt or question, relate mainly to the free anastomosis of veins of the trunk even in parts where valves exist and might be supposed to interfere with such freedom of intercourse. I began the series in the human subject, then I took monkeys, and subsequently, to satisfy myself of the inferences they yielded, had again recourse to human anatomy. After trying a variety of injections in monkeys, which failed of their purpose on account of their dying from disease (usually phthisis), I got a macaque from the Zoological Gardens that died apparently without any pathological lesions that could stand in my way, and Mr. William Pearson at the Royal College of Surgeons injected it with wax. This he did with a certain amount of success, enough for my purpose, although my friend Professor Flower was good enough with his critical eye to see defects, and to draw my attention to them. The injected specimen (shown), when fresh, was a very beautiful object; Professor Humphry, Sir James Paget, and other anatomists, including Professor Flower, examined it carefully, and I think admitted the success, as far as it went, of the injection. But it has suffered from the attempts to preserve it; still I hope it contains evidence of the points I desire to establish. Prior to and since that injection I have had opportunities of repeating the experiment in the human subject at Mr. Cooke's admirably contrived and well-stocked anatomical studio in a little burial ground near the Foundling Hospital. To my friend Mr. Cooke, and the means afforded me by the resources of his interesting and comparatively perfect retreat, I am to a very large extent, and indeed, well-nigh exclusively indebted. The injections were made and followed by a sectional display of the vessels and parts by Mr. Montille, recently of Professor Humphry's Museum at Addenbrooke, and now at that of St. Thomas's Hospital, to whom I am much indebted; as well as to Mr. Pearson, to whom I am beholden for the opportunity of showing the macaque before you.

The results of these injections exactly correspond, and will, I trust, help to lay the foundation in some important respects of the new reading of vein anatomy to which I have alluded, and to which I will now direct more particular attention. As it bears considerably on the azygoid and related systems, I will first offer a few

remarks on their historical anatomy, with the hope of showing the excellent work done in that department on the revival of learning in Europe, especially in Italy and farther east. At the end of the second century the learned physician of Pergamus founded anatomy by observation and made it a science. But he imperfectly understood the nature or uses of the azygoid veins. It was not until the time of Eustachius that these veins became known; he published a series of plates on their anatomy, and was followed, as I learn from Professor Huxley, by Volthier Coiter, of Gröningen, who improved our knowledge of them, and at the same time invented that much, and in some respects well, abused practice of vivisection. Fabricius of Acquapendente followed up the inquiry until we come to Canini, who still more thoroughly examined these vessels, and first brought to light the fact of their having valves; and onwards to our immortal Harvey, who gathered up all the fragments of knowledge that had been collected by these great masters, and landed them on the *terra firma* of the great discovery of the circulation.

At the present time, a latent suspicion has been awakened in favour of physiological research as the means of advancing the science of healing and the practice of medicine, rather than in that of consolidating its basis through the elucidation of what is still obscure and has been least explored in our knowledge of anatomy, and an exact record of the results of such researches. To this view I am tempted to demur. Both are good, but perfection in anatomical details must antedate and form the basis and substratum of all physiological advance. The oscillations in the progress of physiological science, as well as all rightful progress in the art of medicine, correspond very much with the fluctuations in the advance of anatomical science. It is not my design to attempt to add to or improve the knowledge we possess of the anastomotical relations of the smaller veins as given in the excellent and trustworthy works of Gray and Quain, well known to everyone. These veins form a network of intercommunication throughout the body. Every venule and vein contributes to blood extradition from its capillary reservoir and its full discharge into the systemic veins, with a certain and co-ordinate amount of free oscillation in order to provide against casual or designed obstruction. In its way to the heart this course of the blood-streams is marvellously provided for by the arrangement of the sinuous and often angular channels which

conduct it. Here I must interpose the statement that all the trunk veins, like the muscular or axial veins of the extremities, appertain to the systemic system, while the collateral are exclusively, and without exception, cutaneous or superficial veins. If you want to get any communication between portions of the systemic veins most remote from each other, you will have to delegate the cavæ at their entrance to the heart and above their chief trunks, and trust to cutaneous channels for its re-establishment.

My first statement bearing upon the facts shown in this monkey is that if you inject a tributary vein in any part of the body, as, for instance, the internal mammary in the trunk, or in the limbs, and in any direction, either backwards or centripetally, the injected fluid (if thin enough for permeation) will fill every vein throughout the body, and extend into its finest ramifications. The macaque exhibited is the proof to which I refer. You will observe that there is no vein or vein twig that is not fully injected, and from that one point—viz. the internal mammary. This I conceive to be a new fact in connection with the vein circulation. I have put its proof to the test by a considerable number of experiments, and in no one instance have I failed, provided the animal experimented upon was healthy. In order to prove the communications, even where these are apparently exposed to valvular interruption, as in the azygoids, its vessels must be injected by two distinct methods and from different points; first, forwards from an iliac, renal, or sacro-lumbar—a systemic vein; and then after tying its trunk in another subject, backwards or in a direction opposed to the natural current, from the orifice of a remote collateral—*e.g.* an internal mammary, or internal jugular vein. I have caused these methods of injecting this department of the vein system to be adopted several times. In the first the injection will be checked by the valves at the junction of the thoracics with the intercostals, whilst in the second it will take a circuitous route and fill the void costal portions. The next point is that the injection in taking this course fills every interposing organic structure. It will traverse the lungs, liver, kidneys, and, as I have every reason to believe, the spleen, supra-renal capsules, the thyroid, and other organic structures. The third is, that it does not follow that because one large organ, such as the liver or even other parts or organs of the body, are thus injected, all should be equally so filled. In the case of a subject injected

by Mr. Montille (at present lying in Mr. Cooke's studio for observation by anyone desiring it), the lobules of the liver were completely injected from the jugular and femoral veins, but no part of the fluid had passed into it by the portal vein. This vein was empty. And this is quite intelligible, for the course of the injection might be first through the superior, which receives the trunk of the inferior, phrenic, and passes by a single conjoint vein into the external hepatic vein and the liver. The phrenics anastomose freely with the lower intercostals, and thus the mammary blood can flow into the heart through the azygoids. The spermatic vein on the right side, as is well known, passes into the superior cava, and on the left into the renal as well. The injection might also fill the inferior hæmorrhoidal. This completes the proof that the whole of the vein blood from the nearest to the most remote parts of the trunk has free access to the hepatic lobules without being dependent on the portal veins for its conduct, so that it would appear that these veins are not exclusively portal to that organ. By the network already alluded to, the liver is accessible to the vein-blood equally from all parts of the system.

The bearing of this inquiry on physiological science is far from being remote as to its consequences. It shows that blood depuration by organic textures does not depend so much upon the means of access to the liver and other depurating organs of the blood from every part of the body, as upon the healthy performance of their functions. Any interruption to either the first or second of these necessary conditions will be followed by marked local evidence, such as skin sallowness, corresponding with the area over which they have failed of being fulfilled.

Mr. Black delineated the course by which the injection would travel, and asked what new fact had been adduced.

Mr. Perkins Case inquired of the relative facility of injection of the veins of the trunk and extremities.

Mr. Thomas Cooke had found that injection of the peripheral veins of the extremities was capable of filling many of the neighbouring veins, although valves were present.

Mr. Pearce Gould referred to a discrepancy in the results of injection of the monkey and the human being; in the former the mesenteric veins were said to have been injected. He regarded the force under which the injection was introduced—a force much beyond the natural—as a material factor in the experiment.

Dr. Thudichum said that the section of the portal vein was like that of a laurel leaf, and thought that the reason of the non-injection of the portal vein was because of the strength of the valves here.

Mr. Gay, in reply, said there was much that was inexplicable. He spoke of the inefficiency of the valves in many parts of the system. The communication between the hæmorrhoidal veins and sacro-lumbar veins would explain some of the apparent anomalies.

INSTRUMENTS USED FOR RHINOSCOPY AND IN THE TREATMENT OF NASAL POLYPI.

Mr. Spencer Watson exhibited a short focus (six inches) mirror for anterior rhinoscopy. It had a central aperture, behind which was a biconvex lens. This enabled the observer to get very good illumination of the parts with a magnified image.

Mr. Watson also exhibited an improved pharyngeal mirror for posterior rhinoscopy, and explained his method of obtaining a view of the posterior nares, the chief point in his method being to avoid contact with the pharynx or soft palate by the use of instruments. It was important to divert the patient's attention from the throat as much as possible; this could often be effected by directing him to make short, shallow inspirations and expirations, as in "panting for breath."

For the removal of the polypi, deeply seated or in the pharynx, Mr. Watson recommended the use of a ring knife set on a long handle, which would be passed through the anterior nares to the posterior aperture, and when there guided by the forefinger of the left hand passed through the mouth. The patient thus operated on should be under an anæsthetic and a gag must be applied. After operations for polypi the application of strong nitric acid, or some similar liquid caustic, to the parts from which the polypi had been removed seemed to delay or prevent their recurrence. In order to apply such liquid caustics a platinum tube was used. The acid could then be applied on some convenient holder passed through the tube without any risk of cauterising the sound parts. If preferred, the galvanic cautery wire could be applied either through this tube or through the dilating ivory tube made for the purpose.

Mr. James Startin showed a patient suffering from elephantiasis of the leg.

December 3rd, 1883.

CASE OF TWO FISTULÆ IN THE PENILE PORTION OF THE URETHRA, SUCCESSFULLY TREATED BY A PLASTIC OPERATION, AFTER OPENING THE URETHRA IN THE PERINÆUM.

By H. H. Clutton, M.B. Camb., F.R.C.S. Eng.

Fistulæ in front of the scrotum, involving as they often do a considerable loss of the floor of the urethra, present exceptional difficulties to the surgeon who attempts to close them. The innumerable plastic operations that have been planned and executed, stand as witnesses in our surgical literature to this difficulty. This must be my apology for bringing before this Society a solitary case such as the one I am about to record. Urethroplasty, with endless variety of flap and suture, has perplexed the surgical mind from the commencement of this century, whilst in most of the cases recorded you hear little about the method of dealing with the urine. Yet this undoubtedly forms the chief obstacle to success.

Sir Henry Thompson in his classical work on stricture of the urethra has strongly insisted on the uselessness of tying a catheter into the bladder for the cure of fistulæ in the penile portion of the urethra. And there are few surgeons who have not seen the urine flowing by the side of the instrument where the catheter has been used in this way. Useful as this treatment is for the simple dilatation of a stricture, it cannot be of much service in keeping out the urine from the wound after a plastic operation on the urethra. The only other plan at all suitable besides the one that I am now about to advocate is that of passing a soft instrument down the urethra into the bladder every time the patient wants to micturate. This has undoubtedly been attended with greater success than the preceding plan, and the same author has most graphically recorded an interesting case of this kind, where the patient was taught to pass the catheter for himself during the treatment, and the result was a most brilliant success. But there are many disadvantages not the least of which is the difficulty of having this method thoroughly carried out. You are dependent in a great measure upon the good sense of your patient, upon the sensitiveness of his urethra, and the

total absence of cystitis. On the other hand, if the urethra is opened behind the scrotum, and a catheter kept constantly in the bladder through this opening, all these difficulties are obviated. The urine flows away without any pain or discomfort to the patient, and a certain amount of cystitis is not incompatible with success. The operation is a very trifling one, as the membranous portion of the urethra is alone incised, and a catheter introduced into the bladder through this opening cannot be attended with greater risk than when it is lying in the whole length of the urethra. In addition to all this there is one great advantage. There is no risk of urethritis in that part of the canal in which you wish to perform the plastic operation. The urethra in front of the scrotum is left entirely free from urine, and there is no constant source of irritation and consequent inflammatory exudation, such as naturally follows upon tying a catheter into the whole length of the urethra, or even such as must in some cases result from passing a catheter as often as may be necessary to relieve the bladder. Constant catheterism must disturb the parts which you are striving to heal, and in some patients would infallibly produce a copious purulent secretion. Again, if there be a certain amount of cystitis, the patient is by no means in a worse condition with a "vent-peg" in his perinæum. If cystitis be present before the operation, he will probably be relieved; if set up in an irritable subject by the operative manipulation, little though it be, it very soon subsides, and one may have, I think, the consolation of believing that he never would have stood the constant catheterism which is the only other alternative.

My patient, whose age was nineteen, was brought to me by Dr. Stone, of Reigate, in the spring of last year. He had two large apertures in his urethra in front of the scrotum, and gave the following account of himself before he came under the care of Dr. Stone. Eight months previously he had had a very sharp attack of gonorrhœa, never having had it before. This was followed by cystitis, and the doctor at that time attending him tied a catheter into his bladder. Two large abscesses formed almost immediately after its removal, one just in front of the scrotum and the other just behind the glans penis. In consequence of this he put himself under the care of Dr. Stone. The fistulæ following these two abscesses continued to secrete a large quantity of pus, and treatment continued for some months appeared to have little or no effect upon

the discharge. He was also in wretched health. Under these circumstances we decided to send our patient to Australia for the voyage. He returned in six months in capital condition, with mucous discharge from the fistulæ instead of pus, and suffering only from the annoyance and inconvenience of too many outlets for his urine. The discomfort, however, was a very serious one; as much water ran out of the fistulæ as out of the meatus, and he was obliged to take down his trousers when he wished to empty his bladder. He had also a very copious mucous discharge requiring the constant application of an abundant dressing. On passing a full-sized metallic bougie, the urethra was found to be of its normal calibre, but the instrument could be seen and felt with a probe in the two places before mentioned. The floor of the urethra was destroyed for about a quarter of an inch in the anterior fistula and for fully three quarters of an inch in the posterior, and the latter also communicated with a mucous secreting cavity in the scrotum. Considering the extensive nature of these lesions and the irritable condition of his urethra, which had been abundantly proved during the previous treatment, we determined to establish an opening in the urethra behind the scrotum before attempting a plastic operation on the fistulæ.

On October 19th he was placed under the influence of an anæsthetic whilst an opening was made into the urethra in the perinæum. Through this an elastic winged catheter was placed in the bladder, and by means of india-rubber tubing the urine was conducted into a vessel beneath the bed. We determined that the plastic operation on the penile fistulæ should not be done till the preliminary opening in the perinæum had been firmly established, and the patient had become accustomed to this condition. For some days he suffered constantly from spasm, with pus, blood, and mucus in his urine. The idea struck Dr. Stone that this was in a great measure due to the column of urine in the tube hanging over the bed. He believed that this drew the mucous membrane of the bladder into the eyes of the catheter, and irritated its walls. The tubing was therefore removed, and a small vessel placed in the bed at the end of the catheter. After this change our patient had much less pain and discomfort, and his symptoms of cystitis abated. On Nov. 3rd, a fortnight after the preliminary opening in the perinæum, he was again placed under the influence of an anæsthetic, and both fistulæ closed by quilled sutures. The edges were of course

thoroughly pared down to the urethra, and in the case of the posterior fistula the lining membrane of a cavity which led from the urethra into the scrotum, and secreted a considerable amount of muco-purulent fluid, was dissected out. Silver sutures were passed down one side of the raw surface across the urethra and out on the other side without appearing in the wound. They were then attached to quills and the flaps brought together, the edges of the skin between the quills being also sutured with catgut. The method employed was therefore similar to the operation for ruptured perinæum, and was of the simplest kind. There was no flap from a distance or sliding operation, but a simple straight incision down the middle brought together with two pairs of quills. It would be tedious to give all the details of the following week. Shortly, it may be said that he suffered very little after the alteration of the tubing connected with the catheter, and that there was scarcely any mucus, blood, or pus after this alteration. The catheter required changing every two or three days, as it quickly became coated with phosphates. The wounds healed rapidly and soundly, and on Nov. 9th, six days after the plastic operation, all the sutures were removed. The anterior fistula was perfectly closed, but the posterior one had two tiny apertures through the new cicatricial tissue and several large suture holes. The dressing was therefore reapplied. On Nov. 16th the suture holes had healed and the cicatricial tissue in the median line was fairly firm. On the 20th, seventeen days after the plastic operation, as the fistulæ were closed and he was again suffering from some irritation of the bladder, the catheter was removed. These symptoms rapidly subsided and he began to pass water in the ordinary way. On the 24th, exactly three weeks after the plastic operation on the penile fistulæ, he was passing water through the urethra without any escaping either by the perinæum or through the fistulæ. Subsequently, on moving about, a pinhole aperture was discovered in the posterior of the two penile fistulæ. This gave him no inconvenience, and produced only a little moisture after micturition. The copious discharge of mucus, which previously annoyed him, entirely ceased, and he had no gleet whatever. A full-sized bougie *à boule* could be easily passed down the whole length of the urethra and no contraction felt. On Dec. 20th there was still the same pinhole aperture, which was being treated by Dr. Stone with occasional applications of caustic. From this time he went about as usual, and declined further treat-

ment, as he felt quite comfortable in every way. Six months afterwards he still had the slightest moisture after micturition from the same minute opening through the cicatrix of the posterior of the two penile fistulæ. He was, however, so happy and comfortable and found so little inconvenience from it that he decided to remain without further treatment..

Sir Joseph Fayrer had had much to do with sinuses and fistulæ of the penis when in India. He had practised the operation as described by Mr. Clutton not only for acquired but also for congenital defects of the penis.

Mr. W. Rose spoke of a case which had come under his care; in this instance he had benefited his patient by the perineal section.

Mr. Edmund Owen referred to the possible difficulty of closing the post-scrotal fistula after affecting the healing of the ante-scrotal. He related a case of incontinence of urine of obscure origin for which he had performed median cystotomy with great relief to the patient. But much trouble had been caused in the endeavour to heal the perineal fistula. Otis had showed what large-sized instruments the urethra could take.

Mr Pickering Pick referred to the importance of the question raised by Mr. Owen. He narrated a case of penile fistula which he had seen many years ago, in which different methods of treatment had been tried. He asked whether some cases might not be best treated by puncture of the bladder per rectum as a means of relieving the fistulæ from the influence of the urine.

Mr. Davy thought there were many ways of attempting to prevent the injurious effect of urine on fistulæ submitted to plastic operation. The urine might be aspirated or pumped away from the bladder, or the perineal section or puncture of the bladder per rectum might be performed. Cases illustrating the value of different procedures were narrated. And the method of connecting the anterior with the posterior fistula by complete division of the scrotum and joining them was recommended as of value in some cases. Mr. Davy had performed this kind of operation in two instances, and both seemed to have done thoroughly well.

Mr. Royes Bell also spoke of the value of a similar method of surgical treatment.

Sir Joseph Fayrer had never met with any difficulty in healing the posterior perineal wound. And he would prefer to use the perineal section rather than puncture the rectum.

Mr. Clutton, in reply, thought there could be no difficulty in getting the perineal fistula healed, provided there was no disease there in the form of cicatricial tissue. He considered that the presence of chronic infiltrated tissues made all the difference in the question of healing.

RECURRENT ANEURISM OF THE SUPERFICIAL FEMORAL ARTERY AFTER LIGATURE OF THE EXTERNAL ILIAC, TREATED BY EXCISION OF THE SAC.

By William Rose, M.B. Lond., F.R.C.S.

The patient, a stableman, aged forty, was admitted into the Royal Free Hospital on Feb. 4th, 1880. Nine days before admission his right leg slipped through a hole in the flooring, and he felt "something give way" in the thigh, together with pain, followed by stiffness. He went on with his work, and two days afterwards noticed a lump in front of the right thigh. There was no history of syphilis, and his general health was good. Three inches below Poupart's ligament, and in the course of the superficial femoral artery of the right thigh, I discovered a hard pulsating tumour, the size of a hen's egg, presenting the usual characteristics of an aneurism. He complained of numbness in the leg, which was slightly œdematous. By means of a relay of dressers digital compression of the external iliac upon the pelvic brim was maintained for twenty-one hours continuously but without result. On Feb. 18th, therefore, I tied the external iliac with carbolised catgut antiseptically. On the operating table the pulsation in the aneurism was completely arrested; but when changing the dressings on the following day I noticed a slight fluttering pulsation, which persisted throughout and was present when he left the hospital on May 15th with the aneurism contracted to the size of a small walnut. He returned to his former occupation and drank freely. In July, 1882, after an interval of two years and two months, he came back to the hospital, the aneurism having increased to the size of a large lemon, and the pulsation and bruit being well marked. He complained of pain and weakness in the limb. The external iliac was completely obliterated, and the beat of the common iliac could hardly be detected. Pressure on the femoral artery, which was pervious in Hunter's canal, had no appreciable effect on the aneurism. The patient was ordered to keep his bed, and Martin's bandages were applied from the toes up to Poupart's ligament as tightly as he could bear it; iodide of potassium was administered internally in gradually increasing doses, from five to forty grains three times a day, and his diet was steadily reduced at the same time to one pint of fluid and fourteen ounces

of solid food daily. This treatment was continued for one month, the only variation being that the aneurism was left free and not included in the bandage the latter part of the time. The effect of all this on the aneurism was not satisfactory; it was perhaps slightly diminished in size and a little softer. Having thus given dietetic, medicinal, and pressure treatment a fair trial, I thought the time had arrived for active operative interference, and the patient himself was anxious that something further should be done. Several courses of treatment presented themselves for consideration. The operation of tying the common iliac appeared to me to hold out a very feeble prospect of success, and although the possibility of gangrene following did not seem so probable in this case, where the anastomotic chain had been so quickly formed, yet, on the other hand, the effect upon the aneurism itself was very problematic, as indeed was shown at a subsequent period. Again, from the negative effect of pressure, I could not place any reliance on distal ligature in Hunter's canal as being alone sufficient. Everything tended therefore to point to the necessity of dealing with the aneurism direct, and after careful and anxious consideration I decided to cut down and attempt to remove the aneurism whole, tying all communicating vessels or laying the sac open if necessary; at the same time, if I encountered difficulties inevitably compromising the future of the limb, I could but fall back upon amputation at the hip as a *dernier ressort.*

For a fortnight previous to operation the patient resumed his ordinary diet with a view to improve his general condition. On Oct. 18th he was placed under the influence of the anæsthetic mixture, and Mr. Davy, who kindly assisted me, introduced his rectal lever and compressed the right common iliac as a precautionary measure in case of the elastic band slipping. Contrary to expectation, the pulsation in the aneurism, though much reduced, was not entirely controlled by this, the probable explanation being that the middle sacral had become a considerable factor in conveying the blood to the limb after ligation of the external iliac. I need hardly point out the significant value of such a demonstration, affording, to my mind a sufficient argument against the ultimate success of applying a ligature to the common iliac, had we contemplated such a line of treatment. The next step consisted in raising the limb and applying an elastic bandage firmly from the toes to the aneurism. Esmarch's elastic torniquet was then wound

tightly round the limb as far above the tumour as possible, close up to the hip-joint, and kept from slipping down over the aneurism by means of a loop of tape passed round the coils of india rubber on the outer side, held taut by an assistant, and drawn upwards. The bandage was then removed, and the limb was thus rendered bloodless. Having washed the parts thoroughly with a 1 in 20 carbolic lotion, the spray was turned on, and I made an incision seven inches in length over the centre of the tumour in the longitudinal axis of the limb, and exposed the sartorius, which was expanded over, and its deeper fibres apparently incorporated with the sac. I reflected the bulk of the muscle to the outer side, and carefully dissecting through the dense and thickened fascia, opened into Hunter's canal and exposed what appeared to be healthy artery about an inch below the aneurism. After placing two catgut ligatures upon the vessel, I divided it between them, and using the fingers and handle of the knife, with an occasional touch of the edge, lifted the aneurism intact from below upwards. During this proceeding the femoral vein was opened, but this could scarcely be avoided, on account of its intimate connection with the sac. The long saphenous nerve lay behind, and, though flattened by the pressure of the aneurism, was easily dissected clear. In this way the upper portion of the neck of the sac was reached, and, having secured it with a strong thick catgut ligature as close up as I could get under the india-rubber bands, I cut through the structures half an inch below, and the tumour was free. The Esmarch tourniquet was loosened slowly and removed, and then the rectal lever. The bleeding was insignificant, one or two small vessels divided in the dissection requiring ligature. The wound was brought together with carbolised silk sutures, a medium-sized drainage-tube inserted, and the usual antiseptic dressings applied, the limb being wrapped in wool and bandaged with flannel. The patient made a rapid recovery. The result is a firm healthy cicatrix, with complete obliteration of the external iliac and superficial femoral arteries. The patient is in good health, the limb is as strong as the other, and he returned to his work twelve months ago.

One of the most interesting points in this case is the explanation of the recurrence of the aneurism after complete occlusion of the external iliac. My belief is that, in consequence of the rapid formation of an anastomotic channel through the branches of the

internal iliac with those of the profunda, a reverse current was established in the latter vessel, causing return of pulsation and rapid increase in size of the aneurism. The aneurism was no doubt of idiopathic origin, the man's account of it being misleading. It must have existed before he fell, but was probably increased by the injury, rendered painful and so attracted his attention. Lastly, I would lay great stress on the immense value of the bloodless method of Esmarch in such a case. By its means the operation was practicable, and, indeed, comparatively easy; without it the difficulties would have been increased by the welling up of blood into the wound, and the sac would most probably have been opened either by accident or design. I believe, therefore, that in cases where we may reasonably consider the neighbouring arterial tissue healthy, we have in the bloodless method, where applicable, a powerful aid in the treatment of aneurism not hitherto sufficiently appreciated.

Sir JOSEPH FAYRER narrated some cases of aneurism which had come within his experience in India. In one instance the aneurism had burst some days after being punctured; the patient almost died from hæmorrhage, but life was saved by cutting down on the sac and ligaturing above and below it.

Mr. THOMAS BRYANT asked whether Mr. Rose regarded the aneurism as of traumatic or "pathological" origin. He also asked why the common femoral artery in preference to the external iliac artery was not ligatured. He referred to a paper by Porter of Dublin for experience of the operation of ligature of the common femoral. In small aneurisms he thought it did not matter whether the sac were removed or not. He considered that in removing the large aneurism Mr. Rose had deviated from the ordinary method. He submitted also that the operation of Antyllus in cases of suppurating aneurism should be thoroughly considered before amputation was resorted to, or in situations where amputation could not be carried out.

Mr. DAVY thought that the application of this treatment to deep-seated aneurisms was not to be recommended.

Sir JOSEPH FAYRER said we could not depend on the artery being sound even in cases of traumatic aneurism.

Mr. W. ROSE, in reply, said that the case was one of idiopathic aneurism. Sir W. Fergusson had taught him that the external iliac was the best artery to deligate. He ascribed his success in the present operation to the use of the bloodless method.

December 10*th*, 1883.

PASSAGE OF A FOREIGN BODY THROUGH THE ALIMENTARY CANAL.

Mr. S. J. Hutchinson showed a piece of vulcanite chain $3\frac{1}{4}$ inches long, the links being $\frac{7}{16}$ths in. by $\frac{9}{16}$ths in., with a steel watch snap at the end, which had been swallowed and passed *per anum* in twenty hours by an infant fifteen months old.

The chain almost caused suffocation by being impacted in the pharynx, but Mr. Hutchinson having been unable to extract it had successfully pushed it down into the stomach; and in showing it, he laid stress on the fact that emetics were never, and purgatives seldom, necessary in these cases, and he said that this very large body had caused very few symptoms during its rapid passage.

ON A NEW METHOD OF EXPOSING THE KNEE-JOINT IN ORDER TO REMOVE PULPY DEGENERATION OF THE SYNOVIAL MEMBRANE.

By H. Royes Bell, F.R.C.S.

The patient, Charles T—, aged twenty-seven, single, a barman, living at Woolwich, was admitted into Fisk Ward on February 7th, 1883, suffering from disease of the right knee-joint. Five years ago he had an attack of pleurisy in his left side. His father died of phthisis, but his mother and three sisters are healthy. In the winter of 1881 he caught cold from having been obliged to work in the wet, as the Thames had overflowed and inundated the public-house where he was employed. Severe pain came on in the right knee-joint, which swelled up, and obliged him to keep his bed. In a few days it became less painful, and he consulted a chemist; but, getting no better, he went to the Plumstead Infirmary in September, 1882. His limb was placed on a back splint, blistered, and eventually fired with the actual cautery. Here he remained for ten months, but finally returned home.

When admitted into King's College Hospital he was a tall, pale, thin, delicate-looking man, in the habit of indulging in much alcohol.

His right knee was swollen, stiff, and painful; he could just manage to walk with the aid of two sticks. There was little or no effusion into the joint, the swelling being due to thickening of the synovial membrane. The joint was painful at night, it started also, and was more painful after examination. Recognising the value of the antiseptic method of operating in these cases, it was resolved if possible to remove the diseased synovial membrane only. In order to do this effectually, and at the same time to be able to convert this, a partial operation, into the more complete and radical one of excision of the knee-joint, on February 24th a transverse incision was made, which passed over the middle of the patella, as for excision of the joint, only a little higher up, through the soft parts, from one condyle to the other. The patella was sawn transversely across its middle, then by dividing the soft parts forming its capsule the interior of the joint was freely exposed. (The lateral ligaments are not necessarily cut, and if care be used to carry the incision above the semilunar cartilages these also may escape injury.) In this case, having cut the external semilunar cartilage in making the incision into the joint, it was removed, and also the internal, although they did not appear to be diseased. A large mass of thickened synovial membrane was found under the ligamentum patellæ, and also under the quadriceps extensor tendon above the patella. There was some soft red-looking material on the crucial ligaments, which was scraped off from them. On examining the articular cartilages no morbid changes were observed. Esmarch's elastic band had been used; this was now relaxed, and all bleeding vessels secured. The sawn patella was brought together by one silver wire suture, the wound closed, antiseptic dressings were applied, and the joint placed in a back splint (McIntyre). The silver wire was brought out through the incision, and subsequently (April 10th) removed, the patella having united.

The patient progressed favorably for about three months and a half, the wound having healed, and the patient getting about very well on crutches, and wearing a back splint. He had quite lost the old gnawing pain and the nightly startings. On one occasion he went out, and unfortunately visited a public-house and got tipsy; the next day he was much upset by his carouse, and from that time the pain and startings of the limb returned. He readily consented to the proposal that the knee-joint should be excised, which was done on June 16th, about four months after the performance of the

first operation for the removal of the pulpy synovial membrane. He has at the present moment a straight, firmly anchylosed knee-joint, with but very little shortening of the limb. The patella is seen on section to have quite united by bone, there being no evidence of a section having been made except at its articular surface, which is a little irregular. The articular cartilage has been in places supplanted by fibrous tissue, which fixed the patella to the femur. The articular surfaces removed from the femur and tibia show extensive ulceration of the cartilages, which has taken place subsequent to the first operation. It is thought that this transverse section of the patella enables the surgeon to examine thoroughly the condition of the joint and to remove more effectually than can be done by the lateral incisions usually made all the diseased synovial and other tissues, and to convert if necessary this, a minor operation, into the major one of excision of the knee-joint. The specimen shows how perfectly a healthy patella will unite when kept in accurate apposition. Unfortunately the first operation failed in its object, it being just possible that this method of operating is more applicable to children than to adults. This man, although doubtless delicate, and from his habits not a good subject for operation, presented on careful examination no disease of his lungs or kidneys contraindicating interference.

Mr. Richardson Cross, surgeon to the Bristol Royal Infirmary, has narrated before the British Medical Association (session 1883) a number of cases of partial operations on the knee and other joints, which have been most satisfactory.

Mr. William Adams said that he should prefer to operate on adults, and not on children. The most remarkable feature of this case was the perfect bony union.

Mr. Pearce Gould mentioned the case of a man, aged forty-seven, lately under his care at the London Temperance Hospital, suffering from fibroid thickening of the synovial membrane of the knee-joint; the man refused amputation and so Mr. Gould performed the operation of removal of the diseased synovial membrane. Unfortunately, there was no reparative action, and the patient died of exhaustion, having refused secondary amputation when it offered a good chance of saving his life.

MORPHIA HABITUÉS AND THEIR TREATMENT.

By B. W. Richardson, M.D., F.R.S.

In my early career I once met one of the most remarkable representatives of opium eaters who ever lived, and from the late Professor Easton, of Glasgow, his medical attendant, I heard the clinical history of that extraordinary man. Later in my life, namely, in 1869, I demonstrated before the Medical Society of London the negative action of opium on birds, in illustration of the then novel discoveries of Dr. Weir Mitchell, of Philadelphia.

The first of the facts here named fixed my attention on the physiological study of the action of opium. The last, in the most unexpected way, brought me into practical communication with the subject to a somewhat unusual degree. Within a week after the report of the experimental demonstration made before the Medical Society, I was waited on by a member of our own profession who had habituated himself to the use of opium, and who had become desirous of being relieved of the infliction. He was soon followed by another person who was under the same condition, and from that day to the present I have rarely, if ever, been without a patient similarly affected.

The appearance of such patients in the consulting room is by no means regular. Sometimes I do not see one for many months; then, in one month, I see three or four. In the week ending November 24th, three such cases appeared; all of them new cases. I have noticed that when a series of new cases begin to appear, some old ones usually present themselves, and I name these facts because they seem to indicate either that there are certain times or seasons when the symptoms produced are more urgent, or when the desire to be freed from the cause of the symptoms is stronger than at other seasons.

I notice also in these general and introductory notes that amongst the habitués of whom I speak there are many who are given to consult by letter, and who decline to be seen personally. Certain of these act on this plan because they are anxious not to be recognised as devotees to the practice; but others follow the same course because they are too nervous to make a personal appearance,

or to hear the opinion which they think may be expressed in regard to the condition in which they are and the mode of life they are to follow for cure. This proceeding is strikingly characteristic of an advanced stage of induced disease, when the nervous system is very severely affected. I have consequently learned to look upon persons who firmly decline to be seen as deserving of special attention and care.

There is another class of habitués which has a distinct peculiarity. The members of it are habitual devotees, but, according to their view, they do not suffer permanent injury. They are not anxious about themselves, but they are communicative. They seem as if they were impelled to tell someone what they are doing, and what they experience, and why they fell into the habit. Some also of the opium habitués are exceedingly intelligent; and I shall have occasion, as I progress, to read a report made by one of them, bearing upon many points connected with the practical subject in hand.

There are three modes by which the opium habitués indulge:

(*a*) By swallowing opium in the solid form or in solution.

(*b*) By smoking opium and inhaling the fumes.

(*c*) By the subcutaneous injection of morphia.

In the beginning of my practical life in medicine, the only mode of opium-taking in this country was by the first of these processes. Such a person as an opium smoker was scarcely known to exist in English society, while the injection of morphia by the subcutaneous method was absolutely unknown.

Of late years these positions have been changed. Opium eating or drinking has become less common; opium smoking has gained some footing; and, since the discovery of the process of subcutaneous injection, dating from about the year 1859, the practice of subcutaneous injection of morphia has increased steadily, until it has now become a formidable enemy to health. Owing to the extent of the subcutaneous method, owing, also, to the fact that I have a wider knowledge of it than of the habits of eating or smoking opium, I shall in this essay confine what I have to say to subcutaneous morphia administration. I will consider the subject under six heads:

1. The circumstances which commonly lead to the habit.
2. The time required to render the habit confirmed.
3. The degree to which the poisoning may be carried.

4. The phenomena produced.
5. The dangers incurred and the ultimate results.
6. The mode of cure of the habit.

The Beginnings of the Practice.

In the larger number of instances of the habit of subcutaneous injection of morphia, the reason assigned for the beginning of the practice is alleviation of pain. There are exceptions to this rule, certain of which are very singular. One person assured me that he began in order simply to ascertain what the effect was like, and that finding it not unpleasant, he went on until he felt as if he could not live without its continuance, Another explained that he resorted to it from imitation, that a friend who lived with him had recourse to it. and that "he followed suit," but could give no definite reason why. Three have been led to the practice through alcoholic excess, under the idea that they might by this method free themselves from the craving for alcohol. Two or three have assigned sleeplessness as the first motive, but much fewer than might have been expected name this special provocative. One, a very distressing example, named "sleeplessness" with "mental torture"—I use the precise terms employed—as the primary causes. A few have stated that the habit was acquired by first having had resort to it for the treatment of disease that was not accompanied by pain but was thought to be fatal. One of these was a sufferer from diabetes, two from persistent intermittency of the heart. But, as I have already intimated, the alleviation of physical pain was the excuse, valid or invalid, which, in the large majority of instances, was adduced as the starting-point of the practice, the pain commonly referred to being neuralgic in character. Sciatica is the form of neuralgia which I have heard most frequently named. The following is a verbatim account from one habitué who has voluntarily sent me a complete history of his case:

"For some years I suffered misery from a morbidly scrupulous conscience, a weak will, and a dyspeptic diathesis. But at thirty I began to educate myself and take pupils, marrying soon afterwards. Ten years later I was completely prostrated by sciatica, and for the next decade could only do light work—honorary—pursuing, however, my studies with what strength I had. This brings me to an age of nearly fifty. Just then my wife died of cancer. For some

months I had been accustomed to give her relief, always with the doctor's direction, by administering morphia hypodermically; and when another attack of sciatica threatened me, I applied the same remedy to myself, beginning with one sixth of a grain once in a day. The effect of the injection was not only to stave off sciatica, but to give me both pleasure and power. Previously to commencing the habit I was *very dyspeptic*, my tongue always moist and loaded. I was not able to study until some hours had elapsed after a meal. I was limited to the same extent after walking. I was much troubled with various conscientious scruples, and often, I fear, vexed my neighbours by my irritable temper. I frequently took a bottle of claret a day and a glass or two of grog at night to make life tolerable and sleep practicable. Sleep was such a difficult thing to me that I might have said, 'Others sleep that they may live, I live that I may sleep.'"

He then goes on to say that from commencing with the small dose named above, one sixth of a grain, he largely increased it, and at last came to an average of three and a half grains per day, at which quantity he steadily sustained the habit for four years.

I shall refer to this gentleman again, in order to show that he defends the practice on the ground that he is happier and better for it. But I adduce him at this moment because he has given, in what I have read, the most common of all histories of the origin of the habit, and because he is a representative type of the class of person who is most susceptible of falling into the habit. It will be seen that he is a man of nervous temperament, with bilious admixture of temperament; thoughtful, anxious, excessively, I must add morbidly, conscientious. He is naturally touched acutely by what other persons less sensitive would consider moderate and endurable pain, and having once tasted relief derived from the narcotic injection, he continues it until the habit becomes, in fact, a second nature. His nervous centres are never free from the presence of an alkaloid, which forms what may be almost considered a new physiological combination with them.

If I adduced any number of further illustrations I should not be doing much more than repeating the one I have cited. His record is peculiar only in that his habit was purely voluntary. He had no advice from any medical man on the subject; the most that can be said on the matter being that he had learned how to perform the subcutaneous operation from the circumstance that his wife,

under most justifiable and, no doubt, necessary conditions, had been subjected to it.

One feature of this case, however, demands attention, because it is a feature which belongs particularly to many other cases. It will be observed that the writer speaks of having been habituated to the use of alcohol before he commenced to inject himself with morphia. He was not what would commonly be called an intemperate man, but he had learned to rely on alcohol to a considerable extent. He often, he says, had to take a bottle of claret a day and a glass or two of grog at night, to make life tolerable and sleep practicable. He suffered, as a result, from constant alcoholic dyspepsia. The state is one which very often proves the starting-point of the morphia practice. It is as if the person moved from one habit to another, and as if the first induced the second. It does not follow that the morphia practice puts out the alcoholic—indeed, that rarely occurs—but no doubt the resort to alcohol is modified by the action of the morphia.

Amongst total abstainers from alcohol the morphia habit, if not unknown altogether, is unknown to me. I have never met with an example, and my knowledge of the abstaining community is not inferior to that of any physician who has the means of inquiry at his command. This fact influences me much in concluding that alcohol, acting like an additional worry and wearer of life, is an important secondary agent in the production of the opium habitué and of subcutaneous injection of morphia as an habitual practice.

Time required for Fixing the Habit.

Opinions need not vary very much on the question, How long a time is required before the practice of injecting morphia becomes a fixed habit demanding particular treatment for its cure—treatment, that is to say, independent of the mere determination of the patient? In other words, how long is it before the will of the patient is rendered altogether subservient to the effects which the morphia induces?

I notice that Dr. Mattison, of New York, who has sent me several essays on the subject now in hand, considers that in a month the habit is completely established. We may readily accept this term, and I think, on the whole, it is correct. I believe the craving is usually established earlier than in a month, if the injection be pursued regu-

larly once or twice in each twenty-four hours, and if it be found pleasant to increase the dose of the narcotic steadily, as is too often the fact.

But while we may consider that the desire for repetition of the morphia is fixed by a month's indulgence, it must not be inferred that the same serious difficulties are then experienced in breaking away from the practice as are encountered when it has been carried out for a longer time. I have had, I confess, great difficulties in persuading the habitué of a month or six weeks to cease, and I have failed altogether with one who was, I believe *bonâ fide*, of this class. They are, however, more amenable to treatment. Much depends in every instance on the tendency of the affected person for alcohol. If the alcoholic tendency be great, the narcotic craving is sure to be intensified. In a word, the first habit often quickens while it lessens the second.

I have more than once met with a case where some other narcotic practice has preceded the morphia intoxication. I had for some time under observation a gentleman who combined chloral drinking with morphia injection. An inability to sleep and an intolerant aching in the temples, with tension and pains in the eyeballs, was assigned as the first provocative to the chloral. In fact, an excess of alcohol, though it was not freely admitted, was the first cause of all the mischief. That led to a persistent alcoholic dyspepsia, to worry of mind, irritability, and restlessness. To obtain sleep chloral was sought as a remedy. Chloral produced a heavy sleep, but it did not relieve the pain, and so morphia injection was the next step; and in a few weeks morphia became the dominant narcotic. The chloral was now suspended, or was only resorted to at intervals, while the alcohol was, perhaps, taken less freely. The morphia was soon so definitely master that it became apparently as necessary as food, and was taken as if it were a meal.

It is impossible to imagine, unless it is witnessed, the hold which the morphia narcotic has upon its victims. One of the habitués, a medical friend of considerable eminence, who began the habit in order to relieve sciatica, who was perfectly aware of all the consequences of the habit, who was constantly coming to me to talk about them, who was never off the statement that he would give it up, would, without waiting in his conversation and protest, take the bottle and needle out of his pocket and charge himself with a dose in my presence.

A lady who was for a while under my care, and who was dotted all over those parts of her body that were within her reach with the marks of the punctures she had inflicted, would beg, while imploring me to get her out of the practice, to allow her to show me how she did it.

I have noticed in these very confirmed examples of the disease that the system becomes so sensitive, or insensitive, to the narcotic, that the dose can, as it were, be weighed by the sense of its action. I have seen the attempt to cure by reducing the quantity of morphia in the solution utterly fail because of this sensitiveness. The intention has been detected at once from the effect not being complete, so that the difference of half a grain would not escape observation and adverse comment.

The Degree to which the Toxic Effect may be Carried.

The dose of morphia commonly resorted to in the first instance is very small compared with what is afterwards used. A sixth, or a quarter of a quarter of a grain at most, is first injected. Very soon, usually within three or four days, this dose is felt to be losing in power, and, as a rule, is steadily increased. I notice, also, as a general fact, that the dose once increased, is maintained, although it may be repeated in the twenty-four hours. I mean by this that if a dose of one sixth of a grain be raised to a quarter of a grain, the quarter of a grain will be the next dose, however soon it may be taken afterwards. "We never retrench," was the observation of one of these habitués, and when they are left to themselves, this, I believe, is a very correct description.

The increase of the dose is ordinarily followed up in consequence of the fixed dose losing its effect; I use the term that is mostly offered, "losing its effect," although it is sometimes a difficult matter to define what is precisely meant by the statement. No two habitués explain in the same terms what effect it is that is lost. One will say that the dose, kept always at the same amount, fails to allay the pain, if pain be complained of. Another will say that the fixed dose fails to give quiet. A third will affirm that the fixed dose begins to produce restlessness, and that it must therefore be increased in order that the remedy may not become worse than the disease. In time, as the new physiological constitution is deve-

loped, a variety of other statements begin to be named which are so incongruous and vapid that they admit of no reasonable interpretation. One patient, for instance, argued that it "was easier to inject a large than a small dose." Another, that "the larger dose caused less local irritation;" and so on, as if any excuse whatever were sufficient without the trouble of thinking over the subject for any better excuse. When the patient is in this frame of mind he affords evidence that the practice has proceeded very far, the listlessness of mind becoming, as it were, an index of a perverted mental state which is never absent in the confirmed sufferer.

The dose is increased step by step, a fact which accounts for the enormous increase which may be attained without direct danger to life from instant toxic action. There have been a few cases in which a large dose has directly killed, but when such an event has occurred it has been due, I believe, either to an accident in measuring the dose, or to an intention to commit suicide. I have known no example myself in which the dose has been suddenly raised to a dangerous point of activity. From a sixth of a grain to a quarter, from a quarter to half, from half to three quarters, from three quarters to a grain, in steady proportion, is the usual method of progress.

Few confirmed habitués stop short at anything less than three grains in twenty-four hours; some go on to five or six, and continue at that quantity for long periods. If they do not exceed the first-named dose, they may continue to follow their usual occupations, and may not be discovered to be habitués. I know one person who takes three grains a day, and who is so ingenious in the administration, and so systematic, that I doubt if anyone suspects him. He takes his dose of a grain on going to bed, the same dose on getting up, and one other similar dose some time in the course of the day, "as he feels to require it." He has learned, so runs his plea, "to know by experience when the morphia is required," though he can fix no precise time. He quotes De Quincey, that his desire is like the Roman centurion's soldiers, it always comes when he calls it, but sometimes it comes when he doesn't call it. If the dose taken exceed much over three grains a day, there is left very little power for very active work, physical or mental. Usually with doses exceeding three grains there are soon some sensations of exhaustion and of mental incompetency, which tell their own story and destroy the regular course of life and duty.

At the same time it must be admitted that the quantity that can be tolerated without destruction of vital action, and tolerated, too, for long intervals of time, may greatly exceed three grains. The largest dose I have ever known of as being taken in the twenty-four hours was eighteen grains; but as this was an exceptional case, which did not come directly under my own observation, I do not vouch for the accurate measurement of dose. From ten to twelve grains is not out of the way in confirmed habitués, who have learned frequently to repeat the administration. Dr. Mattison has published the history of a case, written by the habitué himself, of a physician who took twelve grains a day for a considerable time, and in three years had injected into himself 6000 grains, but now has broken himself from the habit altogether. The patient, part of whose self-written history I have read, has taken, I compute, 4000 grains in smaller doses extended over a rather longer time, and continues in what he considers to be good health. In another instance, which I knew, but which ended fatally, six grains a day were, I calculated, taken for two years, or over 4200 grains in the entire period. It was commonly taken in quantities of a grain and a half four times a day.

In summary on this head, I think it may be assumed safely that a dose of fifteen grains of morphia, divided into several administrations, may be taken daily without immediate toxic effects of a fatal or immediately critical kind, when the administration has been slowly carried up to that amount. I do not think that many morphia habitués exceed six to eight grains a day. The majority, I should say, limit themselves to from one to three grains.

It is of some moment to be conversant with the kind of solution employed by the people for the purpose of injection. I have been more than once told that there is a mixture sold from a prescription, the precise formula of which has not been published. I do not believe there is any truth in the statement. It may be that a few persons have used a prescription, written by some physician, who for a distinct and legitimate reason has written it. It may be, also, that some medical men who have got into the habit of using a solution have prescribed their own form, and by these means, one or other, the rumour may have floated.

As a matter of fact, I find, on inquiry, that the fluid for injection sold by the chemists is the solution of the Pharmacopœia, the dose for which is marked as from one to six minims. The solution, as

we know, is an acetate, and is the same as that which we ourselves ordinarily prescribe for hypodermic use. I have been informed by one first-class dispensing house that sometimes Squire's aqueous solution of the bi-meconate is asked for injection. Beyond that I can discover no evidence of any particular form of solution being popular or in anything like public demand.

The habitués tell us, generally, that they commenced the habit either by being first submitted to the operation by a medical man, or by having seen it properly performed on someone else. They begin, in nine cases out of ten, by injecting five minims. They increase gradually, and they attain at last the largest increases by frequent repetition of as full a quantity as they can get in from one puncture of the needle. They are not commonly afraid of pain from the puncture; but to this an exception is now and then found. One of our brethren, who was an extraordinary habitué, was so sensitive to the needle that he resorted to ether spray in order to produce local insensibility. He froze a small surface from a minute ether jet, and punctured by the side of the frozen part where the skin was insensible but not hard. He was of opinion, that by the plan named he saved the mark which is usually left, as well as the pain; and he was enabled to inject an immense number of times near the same spot, as if he had induced a false absorbing sac or pouch which acted as a receiving cavity.

The Phenomena Produced.

When the injection of morphia is kept within reasonable limits, the phenomena which occur from it, subjective or objective, are often obscure.

The subjective symptoms, when they are marked, are sleeplessness, if the narcotic be not indulged in, and a perturbed sleep when it is; an impaired appetite or an appetite very capricious; a deficiency of mental capacity, especially of power to will; a drowsy melancholy, apt to pass into apathy or moroseness; and, occasionally, a semi-hysterical state, with tendency, particularly in women, to emotional excitement followed by depression. The physical powers are, I think, always reduced, and failure in muscular power for sustained efforts is perhaps one of the earliest serious symptoms. I should characterise the muscular action as slow rather than feeble. It is as if the muscles were not excited by the will to

quick action. There is, however, true feebleness, which is quickly shown after a rather prolonged exercise. There is much dislike to change of life, great talking about the desirability of change, great expression of resolution for change, but no actual carrying out, voluntarily, of that which is so much declared for. The subjective phenomena of chilliness or coldness of surface are rarely, if ever, absent, although they are exchanged, frequently, for passing sensations of a feverish character without thirst.

Some peculiar sensorial nervous phenomena, in the way of deficient or perverted faculty, are often presented. The sense of smell may have become so modified that one odour is undistinguishable from another, or every odour may be resolved into a particular odour which has at some previous time made a special impression. Thus one person had a fixed odour of benzine whenever the olfactory sense was touched by any perfume or smelling substance. Another experienced an odour like that which emanates from a waning wick or candle. Taste is usually affected much in the same way as smell. It is either perverted so that one substance can scarcely be distinguished from another, or the sense is more particularly susceptible to one special thing, or is deficient for everything.

I have not ascertained correctly whether touch is seriously affected, the means of determining the fact not being easily at command; but I have noticed that habitués are clumsy in the way in which they touch objects, and, as it were, undecided and hesitating. How far the senses of hearing and sight are influenced I have also not been able to discover with much exactness. Investigation is here required. Most habitués complain of being oppressed by shrill sounds, which may be due to irritability of mind, and some complain of an aura and of a painful oppression, as if the ear were blocked up. To all any prolonged exercise of the sight is tiring, and for that reason reading is very rarely followed for any length of time. The retina is often intolerant of bright light.

The objective phenomena presented by the morphia habitué are fairly characteristic. During all the time in which the habit is carried on the temperature of the body is irregular. Soon after the administration of the morphia there is a rise in the temperature called by some observers neurotic pyrexia. It has been known for a long time past that the action of opium upon the body is to raise

the temperature, and pyrexia has, therefore, been accepted as one of the diagnostic signs of poisoning by opium. The sign is, I think, never absent. In the most determined habitué there is seen, soon after the dose is taken, a pyrexia which lasts for a period of two to three hours. The temperature may rise to 104° Fah. I have rarely known such an increase, but 100° to 101° is by no means infrequent. The rise in temperature is attended at first with dryness of the mucous and cutaneous surfaces, ending in a secretion more or less excessive. Profuse excretion from the skin may sometimes occur, and free excretion from the bronchial surfaces and salivary glands with copious expectoration is a symptom by no means uncommon.

The circulation in the morphia habitué is slow, as a rule, and the pulse is small and resistant. The resistance is well marked when the patient is fully under the influence of the narcotic. As the effect goes off, the pulse relaxes and the action quickens, but the acceleration passes rapidly into feebleness, and then the motion grows slow again. The period of feebleness is the period of desire for a repetition of the narcotic.

The action of the heart is regular and unattended with any unnatural murmurs due to the morphia *per se*. The natural sounds, while the action of the morphia is in full force, are short and sharp. Later on the sounds are fuller. The motion of the heart is not made irregular by the morphia at the time, and, I believe, never intermittent. But as the effect of the narcotic tones down, there is distinct irregularity and sometimes a reduplicated second sound.

The respiratory sounds are rarely quite natural. At one stage the vesicular murmur is dry, with soft bronchial cooing murmurs; at another stage there is moist bronchial râle.

The tongue of the confirmed habitué is usually dry, and rather more red than natural. It is not often loaded or white. The bowels in almost all cases are constipated, but what we call ordinary dyspeptic symptoms do not give much trouble. In this respect the toxic action of morphia is extremely different from that of alcohol. There is no flatulency, no distension, no colic, no pain in the alimentary canal. Dislike for food or carelessness for food with what is described as a depraved appetite are the dyspeptic phenomena that are chiefly noticeable.

In the commencement of the practice nausea and vomiting are

sometimes severe symptoms. They may continue for many weeks. They may recur at intervals even in the most confirmed habitué, and they may cause much distress. They rarely are sufficiently painful to check the habit. On the contrary, as they subside under the habit, they are feared as symptoms belonging to every attempt at abandonment of it; and not without cause, since, by a strange rule, they nearly always are presented when the attempt to break from the habit is carried out.

The action of the kidneys is irregular, but excessive or frequent excretion of urine is not marked. In nearly every case the urine is not voided until the bladder is largely charged. This symptom deserves special observation, for as the reflex action of the bladder becomes less decisive the bladder may be filled to dangerous distension without the patient being aware of the condition. The bladder may, under these circumstances, become temporarily paralysed, and may require to be relieved by the catheter. The urine is scarcely ever charged with deposit. Even during the neurotic pyrexia it is not very high coloured unless there be some complication. The colour of the urine at nearly all times is pale, the reaction neutral.

The expression of countenance of the morphia habitué is of one older than his years. The expression is careworn. The sallowness which some observers have described I have not particularly recognised. The eye is dull; the pupil is either contracted or considerably dilated according to the stage.

The moral condition of this habitué is very different from that of the confirmed alcoholic. The opium habitué is not *intentionally* untruthful. He makes confession rather than concealment of his habit. He is not violent; he is not humorous; he is not mischief-making. He may, under the additional influence of wine or spirit, be rendered for a short season hilarious and witty, but, apart from such influence, he is under a cloud of depression which never completely leaves him and is usually severe.

Keeping within the strictest bounds of description from nature, I have in the above passages sketched the condition of the morphia habitué who is in the stage during which recovery is fairly possible. That the account may carry with it the strictest impartiality, let me turn once more to my friend who has sent me the latest confessions of an intelligent representative of his class.

This gentleman, be it observed, makes no apology for what he is

doing. On the contrary, he defends the practice. Comparing himself with what he was before he commenced the practice, he declares that, in regard to dyspepsia, power of study, power of memory, power of walking, temper, alcohol drinking, sleep, diet, he is a changed man for the better. None of his recent acquaintances would guess that at one time he had a bad temper.

The practice, adopted with a full knowledge of its dangers on the moral side, and, so far as they are known, on the physical, is attended with all the above-named advantages in his case, as he believes.

The bad results, of which he gives an honest account, are summed up in the subjoined observations:

(*a*) Torpidity of the liver, counteracted by use of podophyllin.

(*b*) Abscesses forming in the neighbourhood of the puncture, twelve of which have occurred, only one of them reaching to any depth, but most of them painful.

(*c*) Tendency to thickening of the outer integument in points; a free secretion, attended on waking in the morning with copious expectoration from the bronchial surface; and an obstruction, at night, in breathing as if from one of the nostrils being choked up.

(*d*) A difficulty in swallowing morsels of solid substance like a pill, as if the object were stopped in its passage by becoming enveloped in a layer of tenacious phlegm or mucus.

(*e*) A tendency at times to violent perspiration, not habitual, but seeming to occur when the drug has almost deserted the body, or when the system has been much weakened, as by an abscess.

(*f*) On attempting to break off the habit a sense of an unpleasant smell, like that of a candle which is just going out, and with a corresponding taste in the saliva, which fluid is, however, quite odourless to independent observers.

(*g*) Occasionally a difficulty of articulation, such as is caused by taking an excess of alcoholic liquor, and once, and once only, although the amount of morphia taken that day was less than usual, distinct signs of intoxication, of which he was quite conscious, but was helpless to suppress or to conceal.

(*h*) Impotency, which is complete.

(*i*) Difficulty of breaking off the habit of injecting the morphia; with diarrhœa and extreme and long-continued debility at every attempt to break it off.

(*k*) Mental exaltation and depression, which would be great did he not arrange for the reaction to synchronise with sleep, so that it is only when he first wakes in the morning that he suffers from weakness and depression.

(*l*) Much excitement in reading works of fiction, the imagination becoming so excited as to lead him to call out, though naturally he could read the same works with perfect coolness.

(*m*) Morally a reduced sensitiveness and adaptability; an inclination to take things more apathetically than he otherwise would; and "less go" than he formerly felt, except during brief periods of exaltation.

In relation to exaltation of mind he has noticed, at times, a tendency to magnify and exaggerate dangers and their sources of feeling. He has detected himself in going through a railway tunnel or in waking up in the dark in a state of great, once or twice almost uncontrollable, alarm. In each case indigestion may have been a partial cause of the alarm.

These are the results of about four years' hypodermic injection on a daily average of three and a half grains, with no increase for two years past except under special circumstances, followed quickly by reduction to the usual amount of three and a half grains in the twenty-four hours.

The history thus rendered supplies very faithfully the minor phenomena produced by the habit of morphia injection. I must now turn to the major phenomena incident to deeper indulgence.

The symptoms in the more determinate form are, subjectively, exaggerations of those which have already been described. The most prominent symptom is the determined appetite for the morphia and the inability to appease it. As this appetite intensifies, the will power, already reduced, is practically destroyed. There is no real care for anything in life, no true repose, no happiness. The ordinary signs of the toxic action of opium are not observable. There is no coma, no special drowsiness. No one particular mental faculty fails, but all faculties are reduced until at last there is general failure, enfeeblement of all the faculties

together, with similar failure of the voluntary and involuntary muscular powers.

The objective symptoms are those of diminished vitality generally. The mind is seen to be uncertain and weak; the limbs are seen to be feeble, and the body is usually brought towards the condition in which it is known in old age. The pyrexia is less intense than in lighter stages, and is more evanescent. The digestive functions are impaired, but emaciation is not by any means a necessary condition. There are no specific local signs, none pointing to any one particular nervous lesion. The life declines altogether, and I should rather compare the final failure, which I have now witnessed in three instances, to death from premature senile decay than to any other mode of dissolution.

There are many incidental points of a diagnostic character which might with advantage be introduced here did not the introduction bid fair to lead away from the subjects which are more immediately under review. But there is one to which I would for one moment refer, in parenthesis, and which relates to the morphia pyrexia as a diagnostic sign between the action of alcohol, chloral, and morphia. Under alcohol and chloral the evanescent flush of surface heat is followed by reduction of temperature. Under morphia the pyrexia is sustained, as so often happens after injury to the brain. In cases where peculiar symptoms of a narcotic kind are manifested, the observation of these different results is most useful in diagnosis. The late Mr. Peter Marshall, a former President of the Medical Society of London, once brought me a patient whose case was an extreme puzzle. The patient had repeated attacks of an intoxication of some kind, but what the producing agent might be was the difficulty. She kept her own counsel, and, in consequence of the strict watch that was sustained, it was confidently felt that alcohol was not the enemy. I took the temperature of this patient during a paroxysm, and found it to be 103°, which of itself excluded alcohol and chloral as the cause of the pyrexia; and, as Mr. Marshall afterwards detected that the same rise of animal temperature regularly accompanied the toxic paroxysm and remained for a long period, I diagnosed opium or morphia as the cause, which diagnosis turned out to be perfectly correct.

Results and Dangers Incident to the Habit.

The results and dangers of the habit of morphia injection are portrayed in the account of the phenomena. It is clear that the greatest danger is the confirmation of the practice by the habitué, and the ever-increasing desire for the poison. There are very few persons who, like my correspondent, have the power to stop at three grains and a half a day when they have once attained to that necessity of desire. Out of thirty such persons whom I have met with in fifteen years, and on the information derived from whom this paper is constructed, he is the single exception, and the trial with him is not yet over.

There are certain results which, if not actual dangers, are serious disadvantages. Setting some of these, which we may call minor, aside, and including even in the minor the pyrexia and occasional excessive perspiration, there is, in moderate examples of the habitué, the slavery of the habit, which alone is sufficiently baneful. There is the perversion of sensory function. There is the impotency which in the male subject seems to be the common result. There is the mental exaltation and depression. There is the apathy, the disinclination to follow any active pursuit. There is the easily-produced muscular exhaustion. In immoderate examples of the habit there are the positive dangers of apathy extending to moral paralysis, of failure of mental capacities, of premature decline.

As a set-off against these bad results in the common run of cases, it is strictly fair to name exceptions in which it may be said, with all truthfulness, that the habit has, in a tentative point of view, been good as a method of treatment. I knew an instance in which a member of our profession, who was suffering from diabetes, found that he could control the excretion of urine, and live an artificial but passable and useful life by injecting into himself one to three grains of morphia in twenty-four hours. I was so sure of the soundness of this practice that I sustained him in it, and for a period of eleven years it went on systematically. The effect of the injection was so definite that the quantity of urine excreted could be determined by the dose of the remedy. Ultimately this patient died from what might justly be called premature old age, but he attained the sixty-sixth year. He was fairly happy up to within a few weeks of death; and I have every reason to believe that his comparatively easy life

was prolonged, by the morphia, many times the period over which it would have extended had the diabetic affection been permitted to run its own course.

Treatment of the Morphia Habitué.

There are two primary questions to bear in mind when the treatment of the morphia habitué is under consideration:

(1) Is it right or necessary to interfere at all?

(2) If it be necessary to interfere and stop the practice, how is it best to conduct the interference?

Touching the first of these questions, I have learned now always to make the strictest preliminary inquiry into the motive that led to the commencement of the habit, and into the fact whether, if the original motive were reasonable, the cause for it remains.

If the motive were originally sound, and if the cause remain, I endeavour to judge whether the affection which the morphia is used to control is of more importance than the habit. If I think it is, and if I do not see any better method of treating the prime evil than the method by morphia, then I only interfere to the extent of regulating the administration of the morphia itself.

If, for example, I find diabetic symptoms favorably controlled by the morphia, I do not interrupt, necessarily, the reasonable continuance of the administration. I merely suggest regulation. If I find a person suffering from malignant disease which is out of the range of surgical art, and if this patient is relieved of pain and finds life rendered endurable by the injection of morphia as a habit, I only interfere to the extent of systematically regulating the dose.

If I find a person suffering from extreme or permanent intermittency of the pulse, and that relief from such intermittency is secured by the injection of morphia and by nothing else—an experience by no means rare—I only interfere to the extent of systematically regulating the dose.

The practice in my hands changes, however, from this plan if pain alone, pain, I mean, apart from clear signs of organic disease, is presented. If the pain be said to be neuralgic, the most frequent originator of the habit, "tic," or "sciatica," I endeavour to dissuade from the habit, and try to give relief by other means should relief from pain still be required. In such trials it is often

found that when the morphia is suspended there is no pain to combat; there is nothing left to combat except the special desire, the feebleness, and the absence of power of will which have been induced by the remedy.

Touching the second point of practice, namely, the best means of breaking the habit, when it is felt to be necessary to break it, there are three matters which require consideration, and on which medical opinions are widely different.

Some endeavour to break through the habit by giving a substitute, too commonly alcohol or chloral, for the morphia. This practice cannot, I think, be accepted as sound. It is handing the sufferer from one enemy to another. I have never seen anything but evil from this course, and I have twice witnessed results from it so exceedingly bad that I cannot for a moment hesitate to condemn it out and out. I have said that the method hands over the sufferer from one enemy to another; I have known an instance where it cast a man on all three at once, to the rapid destruction, first of his mind, and soon of his life by general paralysis.

Another line of curative practice for the morphia habitué is that which has been quaintly designated "the tapering off plan." This consists in the gradual reduction of the dose until none of the drug is required, coupled with other modes of treatment of a general kind, such as carefully regulated diet, change of scene, and administration of what are still designated as tonic remedies, amongst which bark and quinine hold the first place.

I shall withhold comment for a moment on this mode of treatment in order to bring forward the third as a preliminary to the line of practice I shall myself venture to support.

The third method is that of abrupt withdrawal of the morphia and the actual restraint of the habitué until he has ceased to feel the effects of the loss, whatever his sufferings, nay, torments, may be during the ordeal.

Each of these plans has its advocates, and each advocate seems to be equally assured as to the soundness of his doctrine.

To my mind the sudden withdrawal method would present every possible advantage if one quality were attached to it, namely, its rational possibility. There are, perhaps, some cases in which it is possible, but they are rare. When the habit has not been carried out for more than a few weeks, and when the dose injected is not more than half a grain to a grain in the twenty-four hours, then, I

think, we may make a successful effort by direct withdrawal. Beyond this the method is, in my opinion, impossible. With alcohol it is possible and safe suddenly to withdraw the agent. With chloral it is possible and safe. With morphia the difficulty is all but insuperable. With the morphia habitué the sudden withdrawal causes direst symptoms of disease of the truest objective type. It is not merely what he, the habitué, feels, it is what he is seen to suffer. He vomits; he is purged; he is subject to colicky pains; he secretes saliva profusely; he is at one time bathed in perspiration, at another time he is icy cold with a dry skin; he is restless to an extreme; he refuses food or rejects it if he swallows it; he passes little urine; he goes through long intervals without sleep, and his craving for the narcotic is constant, and I had almost said maniacal.

To such a person something must be administered, and if alcohol, chloral, cannabis indica, henbane, or other substance be given for the morphia they do not satisfy. The symptoms continue until they are relieved by the one thing,—a readministration of morphia. I believe the symptoms have been allowed, under the mode of direct withdrawal, to progress up to collapse; but here I speak out of my own knowledge, because I have on no occasion permitted them to progress towards that extreme condition nor near to it.

The mode of direct withdrawal is, in my view, I repeat, out of the range of safe possibility, except in the limited class of cases to which reference was made a few sentences above, and there is this further objection to it that, when it has been carried out, the patient, having no confidence in the extreme measure and no sympathy or trust on those who have carried it out, relapses so soon as he is free. The patient is, in fact, like a child who with no will of his own to conquer an evil passion treats as enemies, to be disobeyed and distrusted, all who, by sheer force, conquer its desire, and who can only be led by kind and firm reasoning into the right path.

There remains now for consideration the system of treatment which consists in the systematic, firm, and gradual withdrawal of the morphia.

The plan thus named requires, in its turn, to be carried out with strict regard to rule. It is quite useless if it be extended over too long a period; it is also quite useless if it be supplemented by substitutes. I notice that Dr. Mattison allows seven days for the

cure by this method. If he means that period as an average time I am with him; if he means it as applicable to every case that comes for treatment, I differ from him, though not largely. I should differ in lengthening the time according to the dose that has become habitual. I mean that if the dose is large and has been long continued, I do not consider a moderately longer time, say fourteen days, as too long absolutely. It is, without doubt, good to be as rapid as is safe, and if fourteen, or at most twenty-one days are expended in the effort to produce a radical cure, all the time that should be given has been given.

In commencing the abstinence, is it best to reduce the number of administrations in the twenty-four hours, or to retain the same number of administrations and to reduce the dose at each administration? I have tested both methods, and I conclude that it is most practical to continue, at least for some days, the old times of administration and to reduce the dose each time. The dose must be reduced gradually, and no tricks must be played of doing it by injecting some other thing, such as water or tincture, without the morphia. That proceeding destroys confidence. If the quantity were six grains in the twenty-four hours, taken in one-grain doses, I should reduce it by giving five grains, divided into six equal doses, on the first day; four grains, similarly divided, on the second day; three grains on the third and on the fourth days; two grains, again divided into six parts, on the fifth day; one grain on the sixth day, and in like manner as to number of administrations. On the seventh day I should divide half a grain into six doses as before, but should drop one administration; and then day by day continue to drop one administration until there were none. These proceedings would occupy a period of about thirteen days, and if they by themselves, unaided by any surreptitious alliances with "substitutes," do not succeed, I fear that in the large majority of cases, nothing will.

Reviewing, indeed, all that I have seen, I am forced reluctantly to admit that success is quite the exception. At the same time I can consistently and faithfully feel that I have never laboured in any case without doing some good. I have checked a patient from resorting to an increased dose; I have succeeded in getting a dose reduced from a dangerous to a comparatively safe quantity. I have one habitué who from six grains per day has come down to one grain, and who writes to me from time to time reporting that

the one grain suffices. I have another who has given up the habit altogether for nearly twelve months, and who, I have every reason to hope, will remain firm, though the temptation to relapse is often hard with him. On the whole, I have fair reason to be satisfied with the results.

What is now required for successfully combating this habit is united action, united knowledge, united will on the part of all practitioners of the healing art. It is in the endeavour to obtain this desirable object that I have brought the subject before the old and important Medical Society of London with which I have been associated all through my professional life, and from which I have received so many and distinguished favours.

Dr. GEORGE THIN had had considerable experience, and was strongly of opinion that the sudden breaking off was the only safe method of treatment. The gradual reduction of the dose could not be relied upon. The peculiar moral perversity of habitués was well known to Sir Robert Christison, and Dr. Thin considered that their words were not to be relied upon. He read an abstract from the 'Friend of China,' showing that the discontinuance of the habit *in toto* was not followed by disastrous consequences. The resemblance between the effects of opium eating, opium smoking, and morphia injection was illustrated from Dr. Thin's experience. He considered that opium-smoking was quite as injurious as the other forms of the opium habit.

Dr. MACLAGAN had seen nothing but good result from the abrupt termination of the habit. But no matter how treated they were apt to fall back from time to time. No reliance could be placed on their statements. In the convalescence from this state disagreeable cold profuse perspirations were frequently met with.

Dr. HERON spoke of a gentleman who told him that he had been in the habit of taking not more than three grains daily of solid opium for twenty years. In alternate months he took one grain and a half with a view to prevent the habit getting the mastery of him. Dr. Heron also referred to some serious symptoms which he had seen follow upon the hypodermic injection of half a grain of morphia in a case of gall-stones.

Dr. RICHARDSON was inclined to agree with Dr. Thin that the remote effects of opium and morphia in different forms were somewhat similar, though he could not say the same of the immediate effects. He quite believed what Dr. Heron had stated, and spoke of the susceptibility to opium which sufferers from gall-stones seemed to possess, a fact which was known to Home, who introduced the opium treatment of diabetes. When the morphia habit existed in a minor degree he thought the abrupt removal of the drug was the best treatment, but when the major degree of the habit had to be treated, he considered that a gradual reduction extending over a period of some days was the best. He said that habitués did not intentionally tell falsehoods, but they seemed to say anything quite regardless of truth or falsehood.

December 17th, 1883.

ON CATHETER FEVER.

By Sir Andrew Clark, Bart., M.D., F.R.C.P.

Gentlemen,—Somewhere about the year 1850 one of the medical officers of Haslar Hospital, between fifty and sixty years of age, of nervous constitution, but apparently robust in health, requested me to examine his urine. Accordingly I submitted it to as careful an examination as I was then competent to make, and all that I could find against it was that it was too great in quantity, too low in density, and too pale in colour. He then informed me that he had trouble with his bladder, that he meant to consult Sir Benjamin Brodie, and that he wished me to accompany him for that purpose to London. Sir Benjamin examined his patient, drew off a large quantity of urine from the bladder, told him he was suffering from simple enlargement of the prostate, prescribed the regular use of the catheter, and with a few general cautions against a careless diet and exposure to cold, he quickly but kindly dismissed us both. We returned to Haslar. In about a week the patient was free from local discomfort, and without complaint of his general health; but then he began to feel and to look ill. He complained of malaise and weakness and general pains. He lost his appetite, was tormented by thirst, had nausea, became feverish, took to his bed, got daily worse, and, notwithstanding the efforts of his colleagues, who could not agree as to the nature of his malady, he died in about three weeks from the beginning of his illness. No post-mortem examination was made or allowed. The case, here so imperfectly narrated, made upon my mind an impression which has never been effaced; but until about the year 1865 I saw no other case exactly resembling it. In that year I was summoned by Mr. Peter Marshall to visit a gentleman suffering from fever. Certainly he was in what is called vaguely by our elder brethren a typhoid state. He was between fifty and sixty years of age. He was lying on his back in bed, apparently in a state of great prostration. The face was faintly

yellowish and mottled, the lips dry, the pupils dilated, and the breath fœtid. The tongue was small, brownish red, dry, tremulous. There was complete anorexia. The bowels had been imperfectly relieved. The urine, habitually removed by the catheter, was low in density, acid, deposited on standing a little muco-pus, and contained a small quantity of albumen. The heart's action was quick and frequent; the pulse small and compressible. The bases of both lungs were congested. The skin, subicteric and for the most part rough and dry, was here and there, chiefly about the hands and feet, bedewed with watery sweat. The acuteness of all the special senses was blunted; and the patient dull, heavy, and indifferent, could yet be roused to speak and answer clearly questions put to him. The temperature of the body at the time of the examination in the afternoon was 103°. The history of this case resembled that of the case first narrated, and first seen by me. The patient, supposed to be healthy, but suffering from an affection of the bladder, was, a few weeks before my visit, enjoined the daily use of the catheter, did well for about a week, then became ill, and fell suddenly into the condition in which I saw him. Neither Mr. Marshall nor I ventured to form a definite diagnosis of the nature of the patient's malady; but remembering the case at Haslar, I suggested that the fever, which we agreed was not a specific fever, had originated out of the condition begotten by the entrance upon catheter life. I saw the patient only once. The remedies proposed—the food, the alcohol, the quinine, and the aperients—were all of no use to the patient, and he died within a week of our consultation. With great difficulty permission was obtained to make a post-mortem examination, and although it was made with both care and interest (for I was interested in the matter), nothing definite was found outside the bladder, and nothing in it sufficient to necessitate or account for death. The prostate was enlarged, the bladder was dilated and thickened—viewed from the inside it was trabecular and slightly saccular—the mucous lining congested and in part eroded, and everywhere coated with a greyish-white stinking mucus. There was nothing to be detected in the urethra or kidneys, although they were examined carefully. I was accustomed to such examinations, and neither Mr. Marshall nor I could say anything better of the cause of death than that it was due to irritative fever. The study of this case gave birth to the opinion, now in my mind a confirmed belief, that

the entrance upon catheter life occasionally gives rise to a pernicious fever, which, in the majority of instances, destroys life, and sometimes, without the intervention of any sensible structural change, is sufficient to account for death. Since 1866 every year has added to my experience of such cases, and for over ten years at least I have been in the habit of mentioning such cases to surgeons with whom I have had the privilege of consulting. From Sir James Paget, and especially from Sir Henry Thompson, whom I have often met in such cases, I have received at various times much important information; but as I have received from neither of these distinguished surgeons explanations completely in harmony with my own experience and thought of such cases as the one just narrated, and as further information might be now in their possession, or in the possession of others, I ventured upon a recent occasion at the Clinical Society to mention the subject in the way in which it has occupied my thoughts, and to invite from my surgical contemporaries their latest experiences and conclusions concerning it. The constitution of the Clinical Society did not permit abstract questions of this kind to be discussed; therefore, at the request of several persons, I have brought it before you to-night. My remarks finding a place in the medical journals, and being by them widely disseminated, have elicited from practical surgeons in all parts a variety of interesting and instructive communications concerning the nature and causes of the fever which occasionally follows casual or habitual catheterisation. Nevertheless, as the exact character of those remarks which I made has been, doubtless by my own fault, misapprehended, and as the subject is of such importance as not only to justify but to require that statements made respecting it should be accurate and clear, I gladly, as I have said, comply with the request conveyed to me from various quarters to reopen the subject.

Now, it is not my intention on the present occasion to narrate a series of cases, and to build thereon a dissertation upon what, for the sake of provisional convenience, I have called catheter fever. I have not, indeed, at my command the materials necessary for such an undertaking, and if I had, devoid as I feel myself to be of the enlightenment and strength which flow out of the surgical instincts begotten by long surgical experience, I would not attempt it. I have seen many cases of catheter fever, but I have never had charge of one. I have visited my cases only occasionally, and in

consultation with other practitioners, and almost always my colleagues have been too busy to keep and to furnish me with minute and continuous records. Indeed, from the present temper of the public mind, the fear which patients have of being made the subjects of experiment, and the demands so thoughtlessly and even cruelly made upon a practitioner's time, it has become increasingly difficult to keep clinical records of any case for oneself; and the public, in what I must call its vicious stupidity, is thus hindering us from helping it so well as we might otherwise have the power to do. But although my records of individual cases of catheter fever are thus, from the necessity of the case, incomplete, I venture to think that, both by the study of their salient characteristics and by the questioning of those in charge of them, I have learned at least enough of their nature and importance to justify me in making certain propositions and in asking certain questions concerning them. The statements which I purpose making about this catheter fever will be most conveniently embodied in a short series of propositions. But before submitting them to your consideration and criticism, it will be necessary, in order to obtain a clear and comprehensive understanding of the underlying subject, to discover the origin and to follow the historical development of those ideas which have shaped the theories of surgeons and determined their lines of practice. I have placed here a table, which, I regret to say, is imperfect. It is a table prepared by my assistant, Dr. Delépine, who has devoted to it most careful study and conscientious care, and it has, on account of the deficiency of indices to books, occupied an amount of time and caused a degree of labour which you can scarcely imagine. I shall have it for further purposes completed; in the meantime it will enable those who do not follow me closely in reading to determine for themselves the order and development of the ideas which have been promulgated concerning this subject. I shall not in my retrospect follow as minutely as is here followed the development of these ideas, but I shall take the leading ideas, for by holding to these leading ideas alone I think we shall be able to survey the whole field.

In 1800 it was known, but not distinctly expressed, that surgical interference with the urethra and bladder was sometimes and in certain circumstances followed by an irritative fever. It was not, however, until 1810 that Moffait, as quoted by Velpeau, described a case of chronic stricture of the urethra in which simple cathe-

terism was followed by rigors, irregular fever, purulent arthritis (as it is called in France), and death.

In 1832 ideas concerning the causal relationship of catheterism to consecutive fever first found form and expression in the writings and teachings of Brodie and Velpeau, and perhaps of Civiale. Brodie distinctly, and even emphatically, mentions the dangers of catheterism, and describes as occasionally occurring in consequence of it paroxysms of irregular fever like ague, and leading sometimes to prolonged debility, sometimes to continued fever with rheumatic pains, and sometimes even to mania. He further says that in such cases death may follow, but he cites no case of its actual occurrence. Velpeau enters much more minutely into the nature and relations of the fever thus mentioned, and contributes several new ideas to the further development of the subject. He alleges that in some persons perfectly healthy, not exposed to the influence of malaria, even easy catheterism may develop a consecutive and continuous fever, and that this fever has five varieties. In the first it consists of a single paroxysm of fever, ending in malaise and debility, with recovery in a few days. The second consists of recurring paroxysms of fever issuing in continued fever, and oftentimes fatal. The third consists of an inflammatory fever arising out of nephritis, phlebitis, or other local inflammation. The fourth consists of a fever associated with purulent arthritis. The fifth consists of a rapid succession of violent paroxysms of fever, speedily ending in collapse and in death. Velpeau then points out, and very clearly, that in the second and fifth varieties he has never found present at the autopsies any adequate structural cause of death, and in these cases he is disposed to regard the origin of the fever as caused by the absorption of vitiated urinary constituents. But on this, and indeed on similar points of pathogenesis, he is both obscure and vague. Civiale, whose great work is disfigured by passionate claims of priority in this matter (of which claims he furnishes no proof), and by the satirical invective which he launches against the juster and greater Velpeau, gives a full and admirable description of this catheter fever; but whilst admitting that the fever is due sometimes to the urethra and sometimes to the bladder, and contending that the fever of the one differs essentially from the fever of the other, he almost angrily minimises the effects of surgical interference; and, it would seem as if with a judgment disturbed by emotion, contends that in most cases the fever has

existed before the use of the catheter, or that it is due to nephritis. (I owe to Dr. Matthews Duncan my best thanks for a letter in which he tells me that he himself has listened to Velpeau setting forth the characters of this very catheter fever to which I am calling attention.) But in a latter part of Civiale's work, marked by greater sobriety of language and a more judicial tone of argument,* he distinctly qualifies these strong assertions, changes his point of view, and says that the cause of the fever is vague and uncertain, and that in speaking of its nature we can only guess.

In 1858 M. Phillips contributed some further ideas concerning the conditions under which this fever is developed. Describing the fever, and in the main following the classification proposed by Velpeau (who, it may be remarked, is the source whence almost all subsequent writers have derived their inspiration, and sometimes their ideas, without acknowledgment), he asserts first that the simplest as well as the severest catheterism, with the largest or the smallest instrument, may originate the fever; secondly (and this is the most important contribution, if it is a true one, which he has made), that unless the affection of the urinary passage lies behind the bulb, the fever does not follow; and, third, that the predisposition to the fever does not lie in the state of the nervous system at all, but in the existence of certain diatheses and in chronic disorders of the general health.

In 1859 Marx, in a monograph of remarkable merit, reviews the state of the question at his time, and contributes to its elucidation and development certain important facts and ideas expressed with great clearness, and used with judicial ability. He adopts a classification of the varieties of catheter fever similar to that given by Velpeau, recognises uncomplicated cases issuing in death without any discernible structural lesion outside the bladder, quoting eight or nine that occurred in his own experience; asserts that it may occur in persons of perfect health, and that in them as well as in others not healthy, it may follow upon any state of the urinary organs requiring the passage of a catheter; and finally, he declares with emphasis the fever to be positively uræmic, and ascribes its origin to insufficiency of the kidneys arising out of functional or structural disease. In this work, perhaps for the first time, the causal relationship of catheterism to catheter fever is most clearly and comprehensibly set forth, and a logical, coherent,

* Page 612, edit. 1860.

and ingenious, if not accurate, theory of the genesis of the fever is propounded.

In 1867 the practical, and, indeed, also in some respects the theoretical aspects of this subject were most greatly advanced by the publication of the now classical work of Sir Henry Thompson on the urinary organs. In this work, characterised by great force, method, clearness, precision, and knowledge, he mentions the perils of catheterism, gives instructions for averting them, notices the occurrence, in a few exceptional cases, of a low irritative fever, and quotes Sir Benjamin Brodie to show that in a few weeks it might terminate in death. Furthermore, he says (and here he differs from almost every one of his predecessors in this line of inquiry) that in all such cases there will be found old-standing pyelitis, with dilatation and marked degeneration of the renal structures, and that in no circumstances could such patients long survive. Looking at the unrivalled experience of this distinguished surgeon, and remembering how often I myself have consulted with him about cases such as this I am now considering, I confess to a feeling of disappointment that he has not made time to give to the profession a more serious and adequate account of this important matter, which he must know better than almost anyone else.

In 1867 the distinguished President of this Society, Sir Joseph Fayrer, gave an admirable account of the varieties of catheter fever, declared, if I understand him aright, that the predisposition to it lay in the malarious state and its consequences, or else in incipient or advanced disease of the kidneys or other parts of the urinary tract; that it began (and from him we have for the first time a clear and express statement on this point) in reflex disorder of the nervous system; that it was not toxæmic, and that catheterism alone, without injury or sensible irritation, was in these circumstances sufficient to originate the febrile phenomena and process.

In 1868 Sir James Paget notices this fever, and makes some remarkable additions to our knowledge of it. He says that when the urine is of low density and abundant, when the patient is gouty, dyspeptic, or otherwise chronically disordered, when having a stricture it becomes irritable and weakens the health, or when, being old, something has occurred to cause temporary depression of the health, catheterism will be specially dangerous, and may bring about a fever ending in death. He leans to the opinion, but does not distinctly express it, that renal degeneration is the cause

of the fever, but admits that so far as his own experience goes the cause is apparently inadequate. Lastly, he states that the mortality of such cases is from 3 to 4 per cent.; which even with my grave views of the subject is a much higher ratio than my own experience would have led me to expect.

In 1871 Banks, of Liverpool, describes the effects of simple catheterism, and classifies them into three varieties: first, rigor with malaise, recovering speedily; second, rigor with malaise and prostration, followed in a few days by fever and death; and, third, cases of shock, producing death within sometimes a single day. Admitting that renal disease predisposes to such attacks, he denies that they are due to suppression of urine, and he ascribes them to shocks of greater or of less severity propagated through the sympathetic nervous system. Here the nervous theory begins to preponderate over the other.

In 1873 Malherbe, in his work on 'Fever of Diseases of the Urinary Passages,' presents a fairly just account of the state of our knowledge of this subject at the time of publication, and he adds certain important cases of his own. These cases are illustrated by a number of interesting and instructive temperature charts. He avers that the fever may arise without local irritation, describes it as uræmic, alleges that the predisposition to it is renal, and whilst admitting—nay, giving evidence of the admission—that in some cases, not a few, no structural lesions are found after death, he yet holds to the hypothesis that the origin of the fever is in renal imperfection.

In 1877 Mr. Marcus Beck contributed to the further elucidation of this subject two important papers bearing upon them the marks of practical knowledge, accuracy of observation, and careful thought. Describing the fever in which death may occur in from nine to forty-eight hours (and not, as I found, noticing the longer fever of which I speak), he holds that the predisposition to it lies in chronic disorders of the health, in renal imperfections, and in age; that the exciting cause is probably mechanical irritation or lesion of the urethra, and that the fever is begotten through irritation of the spino-cerebral and sympathetic nerves reflected upon the kidney, and bringing about structural and other insufficiency thereof. Beck strongly supports the hypothesis that the fever is uræmic.

Now, without carrying it further, it will be seen from this rough historical retrospect that catheterism is occasionally followed by a

fever, which has received the various names of urethral fever, urethro-vesical fever, urinary fever, uræmic fever, catheter fever, and so forth; that in some cases this fever is dependent upon, or associated with, purulent arthritis, ordinary pyæmia—what is known in England as surgical kidney or interstitial nephritis—and that in a small, but still noticeable, percentage of cases no adequate structural cause of death has been found. Now, it was of this last variety of fever that I spoke upon a recent occasion at the Clinical Society, and it was of this variety alone that I made, or mean to make, the following propositions:—

First, that about middle life in men perfectly healthy, or with no discoverable evidence of disease, except perhaps, and even that not always, a low density of urine, the commencement of the habitual use of the catheter is sometimes followed by fever of the remittent type, which often ends in death, and that for the fatal issue in such cases no adequate structural explanation can be found. *Secondly*, that it is important that such a fever, arising in the midst of apparent health from such a seemingly small cause, and leading so often (as it certainly does) to a fatal issue, should be well and widely known, lest death should take the friends of the patient by surprise, and arrangements necessary to the welfare of a family should be left unmade. *Thirdly*, that although it is well known that in persons affected with renal disease, or with chronic gout, or with grave disorders of the general health, the commencement of habitual catheterism is attended with peril to life from secondary fever, the fact that this fever may arise in what seems to be good health, and without the mediation of any visible structural lesion, issue in death, is not well known—or at least well known only to a few—and has, I repeat, no adequate place in English surgical literature or in the English surgical teaching of this time. Of course this knowledge will be found, as I have in a very imperfect way shown you, in special monographs and papers, but those are the luxuries of the few, and for the most part the luxuries of specialists who work in that direction; but such knowledge should be, as I think, fully imported into all our common text-books, and so made accessible to the whole body of the profession. *Fourthly*, that this fever is neither distinctly uræmic nor distinctly pyæmic; that although having some of the characters of each, it has all the necessary characters of neither; that probably it begins in the nervous system; that probably the disturbance of the nervous

system reacts in the first instance upon the general metabolism of the body, and in the second instance upon the secretory organs, beginning with the kidney; that the effect upon the kidney may consist either in structural alterations of the kidney, invisible by the aid of our finest instruments of research, or (as seems to me much more probable) in alteration of the constitution of the blood, that dynamic condition of its constituents in the renal vessels essential to the elaborative action of the secretory cells thereof; and lastly, that the occurrence of these conditions may, and often is, enforced by septic reabsorption into the blood. Fifthly, that a more complete knowledge of this variety of fever, and of the conditions of its origin, maintenance, and increase may, at least we may hope, lead to a material diminution of its mortality; and that even now, by treating in a serious manner entrance upon catheter life by taking the precautions set forth by Sir Henry Thompson, by great temperance in the use of foods and stimulants, by rest, warmth, and by other general means, upon which I shall not now dwell, such mortality, I repeat, may be possibly considerably diminished. Of these propositions the one at the present moment most open to attack is the fourth, wherein it is asserted that this fever is not distinctive and exclusively uræmic. For in these days it has come to pass that almost every writer of distinction adheres to the view of the uræmic origin and nature of catheter fever, or of the thing known under that and other names, and I am, as it were, left by myself very imperfectly armed to oppose it. I oppose it. I ground my opposition to the exclusively uræmic theory upon the fact that the phenomena of catheter fever, not as they exist at a particular moment, but in their assemblage and in their progress together, are different from those of the ordinarily recognised uræmia. The duration is at once longer and shorter—longer than that of acute uræmia, and wanting its headache, its perversions of sensation, its changes in the urine, its convulsions, its profound coma; shorter than that of chronic uræmia, wanting its neuralgias, its recurring headaches, its defects of sight, its fleeting paralyses, its itchings of the skin, its vomiting, its characteristic breath, its attacks of dyspnœa and palpitations, its painful nervousness, its low temperature. Furthermore, the urine of the catheter fever, of this variety at least of catheter fever, is always loaded with micro-organisms of various kinds, and although it is deficient in urea and contains more or less albumen, it deposits no tube casts and it is

capable of amendment. Lastly, whilst chronic uræmia issues in death, catheter fever may issue, sometimes does not issue, in complete recovery. Sir Henry Thompson may remember that I saw with him a distinguished nobleman not far from Oxford-street, who had to make an entrance upon catheter life, and he very justly and wisely warned the relatives that it was a serious procedure, and that perhaps he might suffer constitutional disturbance after the starting of this mode of life; and it happened that he did suffer this constitutional disturbance. It happened indeed that he had a mild variety of this fever which I am now describing, and that it continued for between a fortnight and three weeks. Eventually, with Sir Henry Thompson's help, he recovered, and I think I may say now, five, six, or seven years afterwards, he is in good health at an advanced age. Except in its long duration, in its occasional rigors of great severity and its exceptional clearness of mind, the phenomena of what is called endocardial fever resemble more nearly those of the fever which I have called for the moment catheter fever than any other malady with which I am acquainted.

Two questions of a practical kind arise out of this study of the history of catheter fever. The first is this: seeing that by almost universal assent the fever originates at least in a disturbance of the nervous system, and seeing furthermore that in the cases accessible to me at least there is no account of the fever following in cases where narcotics or anæsthetics have been used, may it not be that the fever is capable of being cut short by the administration on entering upon habitual catheterism of narcotic or anæsthetic remedies? I remember that my great master, Syme, in Edinburgh, for a reason which his instincts very often knew better than his understanding, invariably gave his patients whom he had to catheterise frequently a grain or two of opium from the very beginning, and I must also add that he was singularly free from catheter accidents. The second question is this: assuming the presence of the fever, and seeing that quinine has signally failed in controlling it, what are the drugs to be employed on such occasions? and what is the sort of hygienic management to be followed, especially in respect of food and alcohol, which are so variously used on such occasions, in order that the fever may be brought, if that be possible, to a successful ending?

Such, then, are the main conclusions which I have drawn from my fragmentary studies of this form of catheter fever. I know

that they are incomplete; I even fear that they may be inaccurate; but, however this may be, I submit them to your consideration, and I console myself with the reflection that they may call forth the ripened experience of practical surgeons, who, in this matter, furnished with a larger knowledge and a more practised judgment, may be able not only to correct me where I am in error and to confirm me in any small point where I may have caught the truth, but to supply us with the very sort of knowledge which at this juncture we need and ask.

Sir Henry Thompson said: Sir Joseph Fayrer and Gentlemen,—I received a very courteous invitation from your Secretary to take upon myself the very responsible task of opening the discussion upon Sir Andrew Clark's paper. I may perhaps be permitted to premise that, in common with the rest of you, I hear this paper for the first time. You must not suppose that the acceptance of the position I have taken has carried with it the slightest information as to the line that Sir Andrew Clark would follow, or of any facts that he has brought before us to-night. Had I had that advantage, I might perhaps have been able to offer you a better *résumé* of what I might desire to say respecting it. You find me, therefore, rising with an enormous mass of matter in my mind which I hear for the first time, admirably arranged, and resulting from the diligence of Sir Andrew Clark, with the careful power of organising what he has to deal with which he so manifestly possesses. I desire in the first place, if I can, to see how I may divide this large amount of matter in an orderly way so as to deal with it in detail. I must thank Sir Andrew Clark in the meantime for the opportunity he has given us of discussing this question. A more important question can scarcely come before us in connexion with this department of surgery, and which, having naturally seen a good deal of, I should be glad to endeavour to throw any light upon in my power. First of all, I desire, with great deference, to object *in limine* to the title itself of "catheter fever." I do not think Sir Andrew Clark holds very keenly to it, for he said "What we may call catheter fever." I must object at present to the term, and I think we shall see before I have finished the ground upon which the objection is made. There is a great deal of information floating in the minds of many of us, without perhaps being distinctly formulated, in relation to this subject, and it is precisely these formulæ which Sir Andrew Clark asks for to-night. He has given

us the terms employed to denote this fever by many writers, commencing with Velpeau's, but I think we should do better to adopt the one simple and familiar term of urinary fever, and afterwards discuss how much of that may belong to the catheter, and may have any right to be called catheter fever. I think it becomes our duty first to inquire what urinary fever may be. Sir Andrew Clark has proposed for discussion catheter fever, which is only one form of urinary fever, and he has asked us to discuss one particular kind of it, evidently suggested to his mind by the two cases which he saw—one in 1850, which made a great impression upon him, and the other in 1865, which he saw with Mr. Peter Marshall. These two cases I take to be types of the kind of fever that he desires us to discuss to-night. But first, in order that there may be no mistake or confusion as to what it is we are discussing, let me say that in my experience—and I believe I am now formulating the views of us all who have had any experience in the matter—that there are, regarded from a clinical point of view, three distinct forms of urinary fever. The first, perhaps, should scarcely be called an acute fever; it is a simple febrile attack. All observers will agree that it may be called an acute transient attack—the most common phenomenon we observe in connection with urinary organs, the urethra particularly, when mechanically disturbed. We are all familiar with it, and it may occur after certain provocation which is not always catheterism. It may happen equally after other disturbing conditions, but inasmuch as the catheter or the bougie is the implement in ninety-nine cases out of a hundred which thus affects the urethra, the febrile symptoms have been called under the circumstances "catheter fever." But it is really a form of urinary fever—a single paroxysm occurring after provocation. We are all familiar with it as occurring within four or five hours after that provocation, commonly after the first passing of the urine. Supposing the instrument had been passed at a certain hour, then in three or four hours, shortly after passing water, not invariably, a severe rigor occurs, followed by dry heat and pains in the back, and that again followed by sweating. These symptoms pass away slowly, no recurrence takes place, and the patient is after two or three days as well as ever. That I would call the acute transient attack. The temperature rises rapidly and very often highly during the cold stage; it continues during part of the hot stage, and it gradually falls and ceases to be elevated through the sweating

attack. The whole series of phenomena may occupy four or six hours, anything up to twenty-four or thirty-six hours, and it may manifest very different degrees of severity. And this attack occurs in persons who have the most healthy renal organs ; but whether it occurs always from one cause I think is very doubtful. Certainly I think it occurs occasionally from a simple absorption into the blood-vessels of some small portion of the urine, and the attack I have described is simply a sign of nature's efforts—a "storm," if I may so term it, produced in the system, while nature is getting rid of the poison. I think there is another kind of attack, which occurs solely as a nervous attack, and that the phenomena described may be caused by the two conditions there can be no doubt. I think it is the first, the typical form, in which urinary fever is presented—the acute transient attack. The second distinct form we meet with is the acute recurring form. We will suppose that the patient who has had such an attack as I have described having become nearly, if not quite well, two or three days afterwards has another attack. This second attack always gives rise to a certain amount of suspicion or anxiety on the part of the attendants ; for mostly some degree of renal implication must be believed to exist. In two or three days, by no means after any regular interval, perhaps he has another, and one's anxieties are increased ; but by no means necessarily should there be any alarm in such cases, because we find that they often recover with rest and care, and in connexion with treatment of the local disease with which they are associated, probably stricture. This will cause, perhaps, some anxiety ; but after a short time, with a good deal of lowered health it comes to an end : and after four or five attacks the fever disappears. Such cases are also quite familiar to us, and I think we must regard them as examples of the second distinct type—that of acute recurring fever. Both of these are, I conceive, excluded from the cases which we are especially considering now. The form of case on which Sir Andrew Clark has laid most stress this evening, and out of which this discussion originally arose, is the third form, entirely distinct from those which I have sketched, and it may be called the chronic or continuous urinary fever. I can scarcely, perhaps, describe it better than it was described by Sir Andrew Clark in reference to those two cases. It comes on very insidiously, not necessarily by any smart rigor, with perhaps a little chilliness, and usually in an old man who has had deficient power of passing water for a very

long time; then when it is absolutely necessary for him to commence catheterism, he begins to feel out of sorts, loses appetite, is thirsty, has a dry tongue, is always feverish, but without distinct attacks; meantime the temperature is not much interfered with necessarily, and sometimes not at all. As an illustration of this, I may say that I have been called to two cases of that kind during the last week, the second of which I saw to day. When first seen there was complete retention; the catheter was absolutely necessary, and forty-five ounces were removed. All possible precautions were taken, and now, on the fifth or sixth day of catheterism, he is somewhat delirious, and in a not very hopeful state. He has a temperature slightly under 98° taken by myself to-day. The marked difference in the character of this third form is that which I call your attention to—sometimes increased temperature, sometimes a wavy line of temperature from day to day, at others rather below the average, and this latter is a condition which I may remark is by no means a hopeful one. Now, I have not mentioned one class of cases which has been referred to by Sir Andrew Clark, and which I think we have no right to call fever at all—I mean those cases in which death will occur from twenty-four to thirty-six or forty-eight hours after the attack. I have referred to such cases, although Sir Andrew Clark possibly has not seen it, in my writings, but purposely have not classified them with fever because I thought that these phenomena did not come in connection with any feverish condition. This is clearly a case of shock of some kind, and when we know that the simple passing of a bougie upon a young man who has never had an instrument passed before in his life very often produces a rapid fainting fit and then a fit of slight convulsion looking more or less frightful for a few minutes, but invariably disappearing before long; when we see this happening on the mere passage of a bougie in a perfectly healthy man upon whom some exploration has been necessary, we cannot but be struck with the close sympathy that is established between the urinary organs and the nervous system,—and it is therefore not surprising that in a very few and exceptional cases, with no obvious disease existing in the organs, the passage of the catheter sometimes may bring about a fatal result in twenty-four hours, no special lesions being found to account for it. I think these have no right to be included in any discussion under the name of fever, and especially of catheter fever. I will now ask you just to consider what I may call the

typical form, in which this third condition of continuous or chronic fever reaches us who are surgeons. But perhaps before I do that I may just advert to one thing which I desire particularly not to forget. Sir Andrew Clark expressed surprise that I should have said that in these fatal cases of urinary fever the autopsy revealed invariably advanced disease of the kidneys and ureters. Now, I have never said anywhere in any part of my writings that such is the case, but only that such advanced signs were found in relation with the chronic continuous form of which I am now speaking associated with long-standing, neglected disease. I have especially limited that remark of mine, and it occupies the last three lines of one of my lectures on this subject. I there state that in no case have I ever seen a fatal event in the circumstances which I am now describing in any other than advanced disease of the kidneys and ureters. Happily, a great many cases do not require autopsy, as in the case, so well referred to by Sir Andrew Clark, of the nobleman whom he and I saw together. That occurred nine years ago, and I believe that gentleman was then sixty-five years of age. I remember the case well. The patient disliked, naturally enough, the restraint necessarily imposed, and was, as all such patients are at the outset, much disturbed on learning that it would probably be essential for him from that time henceforward to use a catheter on every occasion on which he desired to pass water. He was then sixty-five, he is now seventy-four, and that catheter has added that number of years to his existence, which has been passed in comfort and in activity since. Notwithstanding, his case was one of severe and long-standing urinary fever, and I was anxious about the result of it for some time; but the patient has done well, as many, many others have done. Hence, to a certain extent, my inability to supply you with the results of autopsy. Then there is another reason, which is also to the point, that is that in private practice it is not always easy to get autopsies. It is not as in hospital cases. How I miss the enjoyment and the advantage of hospital experience, which, having had for twenty-five years, I was at length compelled to give up! You can imagine that it is not always by any means easy to get the post-mortem examinations we require, nor is it always easy under a very great press of business to find time to make them, but whenever I can make them I always do so. But I repeat, however, what I said in the three lines at the end of my lecture, that I have seen this continuous chronic form appearing in elderly men

only after long-standing obstruction of the urethra, and then not without advanced renal disease. Lastly, let me refer to the case which I said was a typical form of that which often reaches the surgeon. The cases which reach the surgeon have not unfrequently such a history as this: A man of sixty-four or sixty-five tells me that for a long time he has been passing water five or six times or more in the night and every hour and a half or two hours in the day. I ask him to pass some, and he does so with difficulty. I observe a dribbling stream of pale water, and he assures me he has passed all he can. But at the lower part of the abdomen there is an obvious protuberance, and before doing anything further I get his history more completely. He states that he has been in this way for a year. "How much more?" I ask; and then, perhaps, he thinks it may be two. Then, when I ask him, "How long have you been rising two or three times in the night?" he replies, "Well, it may be six or seven years." Then I know full well that I have a case of advanced obstructive disease of the urethra, almost certainly prostatic, and I have to convince him that such is the fact; because, if I simply told him that he had a distended bladder and that it was necessary to use the catheter, he would not believe it; he would probably reply that he wanted help to check the unnatural flow, and not contrivances for withdrawing it. He is told that his bladder, to use a simile he can understand, is like a cask with the tap at the top instead of at the bottom, and that the reason why he makes water so fast is that he can only pass an ounce or so from the top while the vessel is filled below. It is very necessary that patients should understand this. The catheter is then introduced, and to his great astonishment a number of ounces, perhaps twenty or thirty, of urine are drawn off, and, if the quantity is large, leaving some still there. It is necessary to tell him what is the matter, and to make him see it for himself. On inquiry how this state of things has come about, it often appears that he has neglected his symptoms altogether, sometimes indeed that he has had some kind of treatment for years; but that he has avoided all suggestions that further examination is necessary. Perhaps he has been sent to Contrexeville or to Vichy or somewhere else, and without any benefit; that he has undergone a considerable amount of treatment there and elsewhere. Then he says, "Sir, I have the greatest possible objection to the catheter. I have been told that if I once adopt the catheter I shall be in a worse condition than

ever I was, and only add another to my present troubles." The condition of this patient is already damaged irretrievably. What should be said by the surgeon to such a patient is this—and it is a plan from which I have found great advantage during the last few years, as I have especially mentioned in a recent edition of my lectures—"I will have nothing to do with you if you cannot arrange for remaining without any exercise, in a warm room, perfectly quiet, with gentle careful catheterism as often in the day as may be necessary." He is to be told that it is an important event in his life to exchange a distended bladder for one that has to be maintained nearly empty; and that the change to this new life is one that requires a sort of apprenticeship and the closest supervision. Formerly I was less circumspect, but experience has taught me the necessity for this cautious procedure. For after all, it must be evident that a great number of these cases do exceedingly well without any precautions; if the quantity is not too large no care is necessary. But where the residual urine is considerable, it mostly involves serious consequences, unless great care is taken. Nevertheless as a most rare exception to that rule, I may say I once saw a man with five pints of urine withdrawn from his bladder, and commencing what Sir Andrew Clark terms his catheter life, without any precautions, and without a single rigor being occasioned by it. The reflection, then, which I cannot help making is this: that it is not an attack of any catheter fever, but the want of the catheter at an earlier stage of the malady, which has placed these individuals in their dangerous condition. In these cases, where the patients have been long taking medicine and travelling from place to place, in search of relief, had the catheter been used when there were only six or eight ounces left in the bladder we should have heard nothing of catheter fever. So that you will see the ground of my objection to the term, and will understand my belief that the only association that the word has with this fever is the important fact that the catheter was wanted and was not used. Perhaps in the progress of such a case we may hear that a fatal event takes place some two or three or four weeks after catheterism was commenced. I ask what was found at the autopsy, and I am told that it was surgical kidney. Then I reflect, and I say with equal justice, "Surgical kidney!" Surgical kidney only in the sense that it came to pass through want of surgical treatment at the outset. Surgical kidney and catheter fever are two terms

that I do not like to accept in connexion with this matter. I think we had better adopt for the symptoms the term of "urinary fever," and for the pathological condition of the kidney a term which shall describe the form which is present; for these symptoms may arise in connexion with varied forms of disease of a local kind, and even diseases of septicæmic origin. And if in treatment we can carry out the plan I have ventured to intimate in connexion with it, I hope and think we shall find that there are less of these fatal results than unfortunately have before obtained. The instruments employed in catheterism were formerly often too rigid, too large, or too carelessly passed, and have occasioned unhappy results. I trust that you will forgive me for having taken up so much of your time. Yet I am very conscious that I have failed to notice several points in the instructive and suggestive paper we have had the advantage of listening to this evening.

Mr. Berkeley Hill.—After the admirable remarks that have been made by Sir Henry Thompson on the excellent paper of Sir Andrew Clark, I shall not occupy the time of the meeting at any great length; but there are one or two points to which I think I may usefully advert. In the first place, as to the presence or absence of kidney disease. I certainly heard with considerable surprise Sir Andrew Clark tell us that in two cases where he had made a post-mortem examination he did not find disease of the kidneys. I have never met with a case of the third, insidious form of urinary fever, in which class I would place all of Sir Andrew Clark's cases (and which Sir Henry Thompson well distinguished from the other two), where kidney disease has not been present, and present—at all events, to my mind—in a satisfactory form to account for death. In the post-mortem details of one case which Sir Andrew Clark gave us, he told us that the bladder was thickened, that the walls were thick, and, if I recollect rightly, he said that the muscular coats were hypertrophied. What does that mean? Does it not mean that the disease which has been working in the urinary tract was not of short duration? There must have been an impediment to the outflow of urine somewhere for a long time to produce that change from the natural structure of the bladder. When the walls of the bladder are thickened, and the muscular bands hypertrophied, then comes difficulty in the escape of the urine from the ureter. From this impediment are produced dilatation of the ureters, and, conse-

quently, dilatation of the renal pelves (changes which we have heard were not present in the instance Sir Andrew Clark has related to us); thus strain upon the kidneys, which induces chronic interstitial nephritis. In the case which Sir Andrew first narrated, where he had not the opportunity of examining the kidneys after death, he described a condition of the urine which, I should say, was admirably indicative of chronic interstitial nephritis. He tells us that the patient passed too much urine; that it was of low specific gravity; and that, when the bladder came to be examined, a considerable quantity was retained, which Sir Benjamin Brodie drew off. It is true he does not say that the urine was turbid, which it usually is—I suppose, always is, perhaps only to a slight degree, but always in a case of chronic interstitial nephritis somewhat turbid—from an excessive secretion of mucus. Nor does he say that such was the case in the instance in which he was able to examine the urinary apparatus after death. Nevertheless, I must say that I have heard with extreme surprise of his having had a case of this slow insidious urinary or catheter fever, whichever term may be applied, without finding disease in the kidneys. I cannot think that the passage of the catheter had any specific influence in this long-delayed case of urinary fever. When that fever comes on within a few hours or days after the passing of an instrument, no doubt the instrument is the immediate cause of febrile disturbance, which, as Sir Henry Thompson has explained, has two forms. One, the sthenic form, is distinctly traceable to the influence of the catheter through the nervous connection existing between the kidneys and the urethra; this assertion is proved by the condition of the urine secreted during the first few hours after the passage of the instrument, a phenomenon clearly narrated by Mr. Marcus Beck in the paper to which Sir Andrew Clark has referred. But this change in the urine does not come on in the insidious form of urinary fever, the category to which the cases of Sir A. Clark belong. It is nothing more than an exaggeration of the condition which is present, I maintain, before the catheter is passed. In these cases, the fever was the product of a long neglect of the indications of nature, who changes the condition of the urine and calls forth symptoms which, had they been interpreted as Sir Henry Thompson has interpreted them to-night, would have led to the use of the catheter long before it was actually applied. I was not aware of the scope which Sir Andrew Clark's admirable paper was intended to take—

Sir Andrew Clark.—I purposely narrowed the scope of the paper to four propositions.

Mr. Berkeley Hill.—That being the case, I will only deal with one other proposition, which I think is included in these four, viz. the second. Sir Henry Thompson told us that, owing to his having no longer access to the hospital post-mortem rooms he was not able to get post-mortem examination of cases that came under his own observation as he would wish to do. Therefore I should like to state the result of an examination which I made yesterday of the 'Reports of the Surgical Registrar of University College Hospital' for eleven years; selecting only such cases as were admitted with enlarged prostate. I find that in that period there were fifty-nine cases admitted and treated in the hospital, and, of those, forty-four recovered, and were discharged, not cured, but having been well set going in their catheter-life; thirteen died, and of eleven it is recorded that chronic interstitial nephritis had been present in the kidneys, sometimes with acute nephritis superinduced; in two others, bronchitis is put down as the cause of death; but unfortunately no particular description is given in the Registrar's abstract of the state of the kidneys in those two cases. I think this is a tolerably strong proof that interstitial nephritis is the cause of death in these cases of *insidious* urinary fever which we have to discuss this evening.

Mr. Savory.—Allow me to express the pleasure I have experienced in hearing Sir Andrew Clark's paper, calling attention to a fact of great importance to all surgeons, and one well worn, like many others which are familiar to us, but likely to be overlooked in the multitude of new subjects that come before us day by day. Seeing how constantly catheters and all kinds of instruments are passed into the urethra, and how comparatively seldom it is that any serious consequences follow even when catheters and other instruments are somewhat roughly used, I think it follows that there must be something peculiar in those persons who suffer from the use of them, and of these persons I think we may say that one class consists of those who have mischief in the kidney. That a large number of persons who experience serious evil from the passage of the instrument, whether it be in the form of fever or something still worse—even to a fatal issue—I cannot doubt that in many of these cases the kidneys are seriously affected. I am also sure that in many of these cases the kidneys are not affected in the ordinary

way—that is to say, in ways corresponding to the descriptions which we get in works on certain diseases of the kidney. They are not characterised after death by very striking changes, but that they are affected I think is evident from certain facts which often come before us on careful inquiry during life. I have been accustomed for many years past previous to performing operations, as I suppose all surgeons would be careful to do, to examine into the state of the kidneys; and what I would look to, particularly in these cases, is not whether the kidneys yield a large quantity of albumen, or even a small quantity of albumen, or whether there be in the urine certain forms indicating advanced degeneration; but what I think is of more importance is the estimate of the quantity of urea which the kidneys are separating from the blood. In many cases I am convinced that the kidneys are in this respect weak, and of all disasters which can happen to the surgeon, probably the worst comes from those causes where this condition of the kidneys has been overlooked. In many respects the heart and lungs are organs more immediately vital than the kidneys; but with regard to serious operations, or in considerable shock to the patient, I would rather have thoroughly sound kidneys on my side than even a sound heart or lungs; and I think it is in this respect that the kidneys often fail. Therefore, I should take it that in some of these cases which were said to show no appearance of mischief after death, the kidneys have still been at fault. I was struck during the reading of Sir Andrew Clark's paper with the statement that the urine was low in density. The urine being low in density often suggests to surgeons that such mischief as this may lurk behind, therefore I exclude this class of cases by saying that they form a large proportion of those in which catheterism is likely to be attended with disastrous results. Then, where the kidneys may be perfectly sound, we come to a class which I should think is very rare, in which after the use of instruments we get undoubtedly blood-poisoning, septic inoculation. This subject of blood-poisoning is a very wide one; it presents various phases, and we all know that we can distinguish separate classes of cases of blood-poisoning in this regard. We now come to the cases to which I suppose Sir Andrew Clark more particularly alludes—those cases in which very severe fever (for I would accept the term "fever" without the objection which Sir Henry Thompson takes to it), or even a fatal issue occurs, and in which there is certainly neither renal degeneration nor any

form of blood-poisoning. I for one think that there are two distinct sources of fever, or as I would call it, to avoid objection, constitutional disturbance. Fifty years ago there was only one source of constitutional disturbance recognised, and that was the nervous system. Then, as our knowledge of blood-poisoning increased, the interest and importance of this so overshadowed the other that for some years past it seems completely to have shut it out of view, and no surgeon now talks about the nervous system as a source of constitutional disturbance consequent on local irritation. By the way, the term "local irritation" is an uncommonly good one, and I was glad to hear Sir Andrew Clark use it to-night. We all remember the classical work of Mr. Travers on 'Constitutional Irritation.' When he wrote that book one source only of constitutional irritation was understood, and that source was the nervous system; but so true and faithful are his descriptions of the cases which he gives that by the further light which we now possess it is quite easy for surgeons to read these cases through and separate them into their two distinct classes—to say that one class of these cases belongs to the nervous system and the other belongs to blood-poisoning. I take it that in the cases to which Sir Andrew Clark has more particularly alluded to-night we have a most conspicuous and very striking example of constitutional disturbance produced through the nervous system—in fact, what we venture to call, using the term in a broad and large sense, to which I see no objection, cases of shock, in which the effects of a shock either lingered on towards recovery, or in some cases terminated in death. And there is this remarkable fact, that of all parts of the body no part seems in so close and intimate association with certain nerve centres as is the urinary apparatus. There are many facts bearing upon this: every surgeon must be familiar with the fact, which is confirmed by physiology, that during the administration of anæsthetics after the power of reflex action has ceased in all other parts of the body, it can still be provoked by irritation about the urinary organs. If you decapitate a turtle and wait for some hours, the power of evoking reflex action dies out part by part, but the last part in which you can evoke it is the region of the cloaca. With regard to the urethra and the organs in connexion with it, all surgeons have for a long time past been quite familiar with the fact that certain "accidents," as the expression goes, occur when these parts are dealt with in various surgical ways. It is not merely the

passage of the instruments into an elderly person, or a person who has been for some time the subject of disease, but I suppose it must be in the experience of every surgeon that occasionally, even in the very young and healthy, a considerable amount of shock is caused by the introduction of the instrument. I suppose it must have occurred to all of you to see in some cases, even after sounding children for stone, that a decided shock is produced by the operation. I have seen death follow the amputation of the penis, and after death not a trace of injury has been discovered in any part of the body. I repeat, it is surely familiar knowledge to us all that of all parts of the body in this relation, the urinary organs are the most delicate tests of reflex action of what one must call a morbid kind. There is another fact which bears very strongly upon this, and that is the effect of catheterism to which Sir H. Thompson has alluded. We occasionally get faints, and perhaps more often rigors are produced. Now, this subject of rigors is in itself one of great importance. We all know that there are rigors and rigors. First of all, there are physiological and pathological rigors. There are rigors from mental emotion, rigors from cold, rigors from fear, rigors which are unattended by rise of temperature, and these we would call physiological. Then we come distinctly to rigors which are attended by a rise of temperature, and then we have what we may call pathological rigors. Now, although it is perfectly clear that in the great majority of cases severe and prolonged rigors, especially those which are attended or followed by sweating, indicate septic absorption, yet it must be clear also that we have rigors which indicate nothing of the kind; and if I were asked to defend this position I do not know that I could take any stronger case than those rigors which immediately or very soon afterwards follow the introduction of the instrument. Therefore, Sir, as the sum of all this, if I were called upon to attempt to answer the question which Sir Andrew Clark has raised to-night, I should say that those cases to which he has particularly alluded are cases in which constitutional disturbance or fever or pyrexia is produced by local irritation through the nervous system.

Mr. Reginald Harrison laid special stress on the antiseptic treatment of cases occuring in elderly people where the dilated bladder required catheterism. He was in the habit of substituting some antiseptic fluid for the urine which was drawn off, and thought

this procedure tended to diminish the liability to the occurrence of constitutional disturbance. He was inclined to accept the septic view of the nature of the fever.

Dr. Althaus spoke of different forms of retention of urine; some were of a spasmodic character.

Mr. Bennett May made a few remarks on the subject.

Dr. Maclagan regarded the view of Sir Andrew Clark and of Mr. Savory that the nervous system had much to say in some of these cases as of the greatest significance. His own experience entirely coincided with that opinion.

Sir Joseph Fayrer pointed out the greater liability to, and the greater severity of, the constitutional disturbance which occurred after catheterism or other surgical operations on the urinary organs when the patient had been, or was, the subject of malarious disease. His own experience in these matters in India had led him to views which agreed in many particulars with those which fell from the lips of Sir A. Clark, Sir H. Thompson, and Mr. Savory. He also specially alluded to the importance of a knowledge of the state of the kidneys in such cases.

Sir Andrew Clark, in reply, said: I beg to thank you, in the first place, for having so patiently listened to a physician who has been treading upon doubtful ground. To-night I will satisfy myself with referring to one or two of the principal objections that have been made. The first objection has been to the title of the paper. Before I answer that let me say that on a previous occasion I was misapprehended because I spoke without previous preparation, and I determined to-night to speak with previous preparation, and to speak as clearly as it was in my power to do, feeling assured that there could be no longer any misconception of my meaning. But I regret to say—as a body of Englishmen you will forgive me for saying it—I have been as much misunderstood as ever. With regard to the objection to the term catheter fever, I purposely said, and I repeat it, that I used the term for the moment, simply for provisional use, to indicate the variety of urinary fever, or whatever fever you like to call it, which I had in my mind. The second statement which I have to make is that the debate, interesting as it has been, has been in a great measure beside the paper. The paper was meant to call attention to a certain form of fever occurring after catheterisation without reference to the reason for catheterisation, and attention was fixed on a certain series of propositions

which I read as slowly and clearly as it was possible for me to do. Next it has been alleged—or if not alleged, clearly implied—that, after all, a great deal of fuss has been made about this; that catheterism is a very frequent affair, and that death from it cannot be so common, or we should hear more about it. One of the most careful observers that I know, who deliberately uses his words, estimates the percentage of death at 3 and 4 per cent. It cannot surely be that English surgeons have lost their respect for the value of human life. What does 3 or 4 per cent. mean? Surely a good deal if it involves the lives of heads of families; and I should be sorry to think—indeed I do not think, notwithstanding what has been said—that there is on the part of English surgeons any want of due reverence for the safety of human life. The next objection is one that has been made by Mr. Berkeley Hill, who is surprised that I should have examined cases—and I have examined more than I have stated—of fever following the use of the catheter, and found no evidence of disease, for he (except in two cases) has invariably found it, or it has been invariably found. Now, the first thing that I have to say in reply to Mr. Berkeley Hill is that I am not the only observer who has been unable to discover sensible lesions of the kidneys after death from catheter fever. Cases of this kind have been reported by several observers, and more especially by Velpeau, Marx, and Malherbe. And the latter, an advocate of the renal theory in death from urinary fever, confesses frankly that he knows several cases in which with the utmost care no structural lesion could be discovered. He gets out of the difficulty by saying, "Well you know, I am sure that there was some functional disorder." In answer to this it might be insinuated that the observers of the time were not competent; but the answer would be unjust; for when Malherbe spoke the study of the pathological histology of renal disease was the fashion of the hour. The second thing that I have to say is that Mr. Berkeley Hill's statement is obviously based on hospital experience, whilst mine is based on the experience of private practice, and that the two are widely different. What is the nature of hospital experience in such cases? Is it not true of the cases of this kind that come to the hospital that they come, not when the disease is beginning, but when it is advanced—when the patients are old, worn-out, and structurally unsound? The poor man with vesical disease has kept long at work, and has, although suffering much, never perhaps thought himself really ill until obliged

to desist from work and take to his bed, or get into the hospital. Now, Mr. Berkeley Hill finds as his experience that in all such feeble, worn-out cases there is renal disease. Beyond a certain stage I do not for a moment doubt the accuracy of this statement. Nevertheless I think I shall be able to show from an examination of the records of his own hospital, or from the researches of an equally distinguished colleague, that in many cases of death from urinary fever there is no notice of the existence of renal disease; and that in cases where renal disease was found it was declared to be inadequate to account for death. In twenty-six out of sixty-six cases of death from old-standing vesical and prostatic disease cited by Mr. Marcus Beck from the University College Hospital Records, no evidence of the existence of renal disease has been mentioned. Furthermore, Mr. Marcus Beck, speaking of the closing days of urinary fever, says, "At the same time"—that is at the time that he is asserting the existence of renal disease—"the secretion of the urine continues in fair quantity, and the patient therefore cannot be said properly to die of uræmia." And further on the same observer adds, "As before stated, in many cases the real cause of death is not the damage to the kidneys, for the secretion of the urine remains abundant to the last, but the absorption from the abscesses or from the putrid urine in the pelvis. On the other hand, the symptoms closely resemble, and as a matter of fact are often undistinguishable from those of septicæmia." I trust these statements will satisfy Mr. Berkeley Hill that catheter fever sometimes issues in death even when no visible structural change is found adequate to account for the fatal issue. Then there is another objection which has been made, indirectly indeed, but still with sufficient force, implying that I was objecting to the use of the catheter. On the contrary, I think the reason why such evils, not as I have been describing, but many other evils that I enumerate in cases following catheterisation, have existed, has been because the patient has been unwilling, owing perhaps to bad advice, to begin with catheter assistance at once. That is my feeling, and if it is supposed by any Fellows of the Society that I have any objection to the use of the catheter, I must beg to tell them that they are mistaken. On the contrary, the difficulties which I have seen as a physician being occasionally called into these cases have plainly arisen, as Sir Henry Thompson has said, from the catheterism not being early enough. Let me next refer to the very important observation—and all his observa-

tions are both interesting and important—of Mr. Savory. He mentions a point which, I think, if no other point had been brought out this evening, would remain a memorial of a useful meeting. It is that he himself attaches, and he believes that every wise surgeon will attach, more importance to the condition of the kidney than to almost any other organ of the body in approaching the performance of an operation. Sir, it is twenty-eight years since I said that; I am not thinking of priority; but I am only wishing to show that the subject has been in my mind. I do not pretend that I was the first to say it, but I said it at least twenty-eight years ago; and on various occasions since, under the term of renal inadequacy, I have pointed out the great peril which exists in all such cases in performing surgical operations. It is only another illustration that those who are faithful observers can come independently to the same conclusion. I do not think that at this late hour I should encroach further upon the time of this Society. I repeat humbly, but believingly, that the subject has not been thoroughly exhausted. There are most important points embodied in my propositions which have not been met in this discussion. I hope that some time—soon—we may be able under another, if not under this aspect, to reconsider this question, and keep closer than we have done to-night to the ground which has been laid open for discussion.

THE LETTSOMIAN LECTURES

Were delivered by R. BRUDENELL CARTER, F.R.C.S.

On January 7th, 21st, and February 4th.

SUBJECT—MODERN OPERATIONS FOR CATARACT.

January 14*th*, 1884.

ON AMPUTATION AT THE KNEE-JOINT BY LATERAL FLAPS.

By T. Pickering Pick, F.R.C.S.

In the present communication I do not propose to say anything on the advantages or disadvantages of amputation at the knee-joint in comparison with amputation in the lower part of the thigh. The subject has been so exhaustively treated by Mr. Pollock in a paper communicated to the Royal Medical and Chirurgical Society, that little or nothing can be added to the subject. The point to which I wish to draw the attention of the Fellows of this Society is the manner in which the operation is best performed.

The operation of amputation at the knee-joint is one of considerable antiquity, but it had not been performed, or at least recorded, more than a few times in the history of English surgery, until within the last fifty years. In the year 1830 Velpeau advocated its adoption, and some ten years later Syme performed it in some cases, but is stated by Fergusson to have subsequently abandoned it, imagining that greater danger was incurred by the larger surface of bone exposed. In a clinical lecture in 1854 Sir William Fergusson speaks very highly of this operation and strongly advocates its claims, both on account of its greater freedom from danger and on the utility of the stump after the operation.*

All these surgeons, however, insist upon the necessity of cutting through the condyles and removing the articular end of the femur and the structures covering it. And it must be in the recollection of many Fellows of the Society that a very few years ago amputation through this articulation was regarded as an unsound operation, unless the articular surface of the bone, together with the encrusting cartilage and investing synovial membrane, was removed. It was not, I believe, till 1857 that Mr. Samuel Lane first performed the operation which is now generally advocated by surgeons of amputating the leg at the knee-joint without removing the articular cartilages, and since that time the proceeding has been followed by

* 'Med. Times and Gazette,' July 8th, 1854.

numerous surgeons in a considerable number of cases. It must be borne in mind, however, that the cases in which this operation can be performed are limited, since it is evident that in a large proportion of patients for whom amputation is required this proceeding is not applicable. I allude, of course, to those cases in which the disease is in the knee-joint itself. Here the whole of the diseased structures would not be removed, and therefore this operation is only applicable to those cases where the limb requires removal for injury or disease *not* implicating the knee-joint.

The manner in which this operation is generally performed is by a long anterior, nearly rectangular flap and a short posterior one, and it is to this proceeding that I venture to take exception and to point out what I believe to be a far preferable mode of proceeding.

The following is Mr. Pollock's description of the operation:—"I feel for the interval between the edges of the condyle and head of tibia and commence my incision at that point and immediately behind the edge of the hamstring muscle as it crosses that space. I take especial care never to commence my incision higher than the margin of the condyle. The incision should be carried perpendicularly downwards on the side of the leg till nearly five inches below the lower edge of the patella, then gradually brought across the front of the leg, and when crossing the tibia should be quite five inches below the patella; then carried up the inner side to a point corresponding exactly to that from which the incision commenced. I usually make the posterior flap by cutting from without inwards; it should not be too short and should consist merely of integument. As soon as the flaps are completed, all the structures round the joint should be divided at right angles with the limb." Most surgeons advocate the same mode of proceeding. Thus Professor Lister says that amputation at the knee-joint "has been performed with good results, by raising a large anterior flap from the upper part of the leg, opening the joint and cutting a shorter posterior flap from within outwards, the patella being retained in the anterior flap."*

Mr. Erichsen, in a clinical lecture on a case of fibro-plastic tumour of the leg, states that he prefers a "long anterior flap, slightly rounded at the edges and leaving the patella."†

Mr. Holmes recommends that amputation at the knee-joint

* 'Holmes' System of Surgery,' 1st edition, vol. iii, p. 87.

† 'Lancet,' vol. ii, 1871, p. 460.

should be performed by a "long skin flap cut out of the front of the leg by an incision convex downwards, starting from the back of either condyle and reaching four or five inches down the leg."*

Mr. Bryant recommends that "in amputation at the knee a long anterior skin flap, and shorter, but not short posterior skin flap, should be made by cutting from without inwards, the muscles being divided by a circular sweep of the knife in disarticulating."†

The American surgeons adopt the same plan. Dr. Staples employs "a semilunar flap in front reaching an inch or an inch and a half below the tubercle of the tibia and a short posterior flap."‡

This operation has been a very favourite one at St. George's Hospital since Mr. Pollock first performed it there in 1864, and has been frequently resorted to by my colleagues and myself, but I have never been quite satisfied with it, because I have noticed the frequency with which a portion of the anterior flap has sloughed on account of its great length and deficient vitality, and this even when the operation has been carefully performed in the manner recommended by Mr. Pollock and with the precautions which he advises to prevent this untoward result.

That this sloughing does frequently occur is abundantly proved by the accompanying tables which I have extracted from the amputation records of St. George's Hospital, and which comprises, I believe, every instance of removal of the limb at the knee-joint which has taken place in the hospital since 1864. It includes thirty-three cases, but of these we must exclude two because the amputation was performed in another way. Of the remaining cases two died on or before the third day and must also be excluded, so that we have twenty-nine cases on which to base our inquiry. Of these twenty-nine cases it is definitely stated that in four there was no sloughing, and in eight other cases, as there is no note as to the occurrence of sloughing, it is fair to assume that there was none, as there is very little doubt that it would have been mentioned if it had taken place. One patient had phagedæna, and in the remaining cases, sixteen in number, more or less sloughing occurred. It is true that in some it was very slight; in one case, the note says: "A thin line of the anterior flap sloughed;" but still the fact remains that out of a total of twenty-nine cases of amputation at the knee-joint, in

* 'Principles and Practice of Surgery,' 3rd edition, p. 944.

† 'Practice of Surgery,' p. 1055.

‡ 'American Journ. of Med. Sciences,' January, 1872, p. 62.

no less a number than sixteen a portion of the anterior flap perished for want of sufficient blood-supply, and no further argument is wanted, I think, to condemn the operation.

In the year 1878 I admitted a woman with a large enchondromatous tumour, which was partially ossified, growing from and completely incorporated with the tibia, so that nothing short of amputation held out any prospect of removing the disease. In consequence, however, of the size of the tumour and its encroachment upon the front of the knee-joint, it was not possible to obtain sufficient tissue to form a long anterior flap and I therefore determined to amputate at the knee-joint by a short anterior and a long posterior flap. And this operation I performed with a very good result. There was no sloughing of the flap, and though the recovery was slow, the patient had a most excellent stump, upon which she could follow her employment, that of a cook, which necessitated a great amount of standing, and on which, as she informed me, she could walk five or six miles without fatigue or inconvenience. But upon thinking over this operation by the long posterior flap I did not feel altogether satisfied with it. In the first place it seemed to me that it prevented efficient drainage and did not allow of the ready discharge of matter from the stump, which would be apt to collect in the sort of bag formed by the posterior flap and thus materially interfere with union. Again, the constant dragging of the heavy posterior flap would keep up a considerable amount of tension on the sutures, tending to drag down the cicatrix over the ends of the bone and delay the union of the wound. Therefore I determined to abandon it.

On June 1st, 1881, a little boy, aged ten, was admitted into St. George's Hospital with a compound fracture of the leg. He had been playing in the road, when he was knocked down and run over by a tramcar, the wheel passing over his right leg. On admission there was a lacerated wound extending from about one to two inches below the tubercle of the tibia to about the same distance above the ankle-joint. The tibia was fractured near its centre, obliquely from without inwards and from above downwards, both ends projecting from the wound. The fibula was also fractured, and its ends buried in the neighbouring muscles. The soft parts of the limb were completely crushed. The boy was collapsed and did not appear to suffer much pain.

Ether having been administered, I proceeded to amputate through

the knee-joint by means of lateral flaps. An incision was commenced at the upper border of the patella and carried down the middle line of the limb as low as the tubercle of the tibia; it was then curved outwards over the outer side of the leg to the back and carried upwards along the middle line to a point corresponding to the commencement of the incision on the front of the leg. A similar incision was carried round the inner side of the leg, and thus two somewhat quadrilateral flaps with rounded corners, consisting only of skin and subcutaneous tissue, were mapped out. The lowest point of these flaps was about an inch and a half below the level of the tubercle of the tibia. They were dissected up as high as the articulation, the patella was removed, and the various structures around the joint divided by a circular sweep of the knife. A drainage-tube was inserted, the end being made to project through the posterior extremity of the wound, and the stump was dressed antiseptically. Very little blood was lost during the operation, but the patient was much collapsed, requiring brandy.

He went on well after the operation. On the seventh day the stump was dressed; the wound was quiet and healing with scarcely a trace of discharge. He seemed quite well and was free from pain, wanted to get up, and had been asking for a wooden leg. On the eleventh day the greater part of the wound was healed, most of the sutures were removed, and the drainage-tube shortened. On the fourteenth day the wound was healed, except the orifice through which the drainage-tube protruded. This was withdrawn, and the rest of the sutures removed. On the twenty-second day he was sent to the Convalescent Hospital at Wimbledon. He remained at Wimbledon till July 6th, when he was finally discharged with an admirable stump. The vertical cicatrix lay in the intercondyloid notch, and was not subjected to any pressure when the weight of the body was supported on the stump. There was a nice soft cushion over either condyle of the femur.

Since this date I have had two other opportunities of amputating at the knee-joint, and on both occasions have resorted to the plan of operating by lateral flaps. The first case was that of a feeble old man, sixty-two years of age, who was admitted into St. George's Hospital with a compound comminuted fracture of both tibia and fibula, with severe laceration of soft parts, for which there could be no doubt that amputation was the only alternative, though on account of the age of the patient and his extremely

collapsed condition the prospects of an operation were of a most gloomy nature. I performed the operation of amputation at the knee-joint by lateral flaps, as in the other case. The patient never rallied, had repeated attacks of syncope, and was unable to keep anything on his stomach, and in spite of the subcutaneous injection of ether and the free exhibition of stimulants he sank and died on the third day.

The other case was that of a man, aged thirty, who was admitted with a compound comminuted fracture of both bones of the leg on June 30th, 1881. On the day after his admission symptoms of delirium tremens developed themselves, and this was followed by diffuse cellular inflammation of the limb, necessitating free incision. On the fifteenth day after admission he developed symptoms of typhoid fever and was reduced to a state of extreme prostration, during which his life was despaired of. After a time he rallied; the diarrhœa, which had been profuse, ceased, and his general condition improved. The leg, however, was in a most unsatisfactory condition, and as it could clearly never be any use to him, and was evidently a drain on his strength, it was determined to remove it. Amputation was therefore performed by lateral flaps at the knee-joint. He appeared to rally for a day or two, and then began to sweat and have a high temperature. Some redness appeared about the stump, and diffuse cellular inflammation came on and spread up the thigh. The edges of the wound gaped and the external condyle became exposed. The man was again reduced to death's door, but owing I believe to the fact that he was able to take large quantities of nourishment and stimulants he rallied, and was eventually discharged with a healed stump on February 11th, 1882, after having been seven months and a half in the hospital. The cicatrix was partially adherent to the external condyle, but in spite of this the man when seen some six months later could bear firm pressure on the end of his stump, and could walk in his "bucket leg" with ease.

These two cases do not add much to our knowledge of the advantages or disadvantages of this plan of operating, and are only mentioned in order that I may put the Fellows of the Society in possession of the whole of my experience on this subject. In the first of the two cases the man did not live long enough to afford any data on which to base an opinion, and in the second case the complications which arose were so formidable that I think it is a matter of

congratulation that we were enabled to turn the man out of the hospital with a stump sufficiently useful to allow of his earning his daily bread.

When I performed these operations I was not aware that Dr. Stephen Smith, of New York, had, in the year 1870, advocated amputation at the knee-joint by lateral flaps. He, however, somewhat modifies the operation by commencing his incision an inch below the tubercle of the tibia, carrying it in a circular manner round the leg until it reaches its under surface, when it is curved upwards towards the median line. When this point is reached it is continued directly upwards to the centre of the articulation. A second incision begins at the same point as the first and pursues a similar direction upon the opposite side of the leg and meets it in the median line behind.* By these incisions Dr. Smith asserts that the cicatrix is not subjected to any pressure on the face of the stump, but that it sinks into the intercondyloid notch and offers no point of contact with the artificial appliance. There can be no doubt that this is so, but the same thing was the case in the amputation which I performed on the boy, and though I have no practical experience of Dr. Smith's operation, it seems to me that on account of the great length of the flaps, and the fact that the tissues on the front of the leg are not divided to a higher level than an inch below the tubercle of the tibia, there must be a great tendency to the bagging of matter, especially in the *cul-de-sac* or pouch of synovial membrane beneath the quadriceps extensor muscle.

The advantages claimed for this operation are (1) that by it we obtain as efficient, if not more efficient, drainage than by that of the long anterior flap; (2) that we have two moderately-sized and well-nourished flaps, which are not liable to slough, as is the case in the single, ill-nourished, long anterior flap; and (3) that the cicatrix is placed in the intercondyloid notch between the two prominent condyles of the femur, and is not therefore subjected to any direct pressure from the artificial limb.

* 'American Journ. of Med. Sciences,' January, 1870, p. 33.

Table of Cases of Amputation at the Knee-joint.

No.	Name.	Age.	Cause for which amputation was performed.	Nature of amputation.	Result.
1	James F.	51	Epithelioma of leg	Long anterior and short posterior flap	Slight portion of the margin of the anterior flap sloughed.
2	Mary G.	55	Elephantiasis of leg	Do.	Died, third day.
3	Hector M.	24	Anchylosis; wasted and useless limb	Do.	Phagedæna of stump; the anterior flap did not suffer much.
4	Maurice H.	66	Elephantiasis	Do.	A little sloughing of anterior flap.
5	James J.	13	Primary, for compound fracture	Do.	Anterior flap destroyed by sloughing.
6	Victor B.	33	Subastragaloid dislocation; sloughing and secondary hæmorrhage	Do.	A small portion of anterior flap sloughed.
7	E. L.	29	Primary, for compound fracture	Do.	Some portion of the anterior flap was lost by sloughing.
8	William S.	48	Phagedæna after compound fracture	Do.; a portion of the condyles of the femur were removed	No sloughing; a good stump.
9	Evan L.	29	Primary, for compound fracture	Long anterior and short posterior	Ant. flap sloughed.
10	Jemima J.	39	Elephantiasis; ulceration of leg and foot	Do.	A thin line of the anterior flap sloughed.
11	Julia C.	20	Myeloid tumour of head of tibia	Do.	No sloughing.
12	Mary L.	37	Enchondroma of tibia	Do.	Died, 30 days; no note as to sloughing.
13	Annie R.	17	Necrosis	Do.	Ant. flap sloughed.
14	Mary C.	42	Epithelioma	Do.	Died, 1 day.
15	George N.	25	Primary, after compound fracture	Do.	No note.
16	Henry M.	20	Compound fracture, primary	Do.	Died; no note; 24 days.
17	Benjamin B.	18	Do.	Do.	No note.
18	James D.	35	Do.	Do.	No note. Good stump.

No.	Name.	Age.	Cause for which amputation was performed.	Nature of amputation.	Result.
19	Ann N.	68	Compound comminuted fracture	Long anterior and short posterior	Died, 10 days.
20	Louisa A.	49	Elephantiasis	Do.; more than unusually long posterior flap	No sloughing.
21	Elizabeth C.	31	Elephantiasis	Long anterior and short posterior	Good deal of suppuration; great retraction of anterior flap.
22	Susan S.	22	Malignant tumour of leg	Do.	Secondary hæmorrhage. Died, 17 days; pyæmia.
23	Percy M.	19	Disease of tarsus	Do.	Ant. flap sloughed.
24	Anne P.	39	Epithelioma	Short anterior and long posterior flap	Good stump.
25	Fanny V.	27	Enchondroma	Do.	Good stump. Can follow her occupation as cook, and walk six miles on it.
26	William L.	29	Gangrene after ligature of femoral artery for aneurism	Long anterior and short posterior flap	Ant. flap sloughed.
27	Eliza Q.	30	Painful stump	Do.	Slight sloughing at edge of flap.
28	Thomas E.	40	Epithelioma of tibia	Do.	Secondary hæmorrhage; flap sloughed.
29	John L.	28	Primary for compound fracture	Do.; flaps somewhat irregular on account of bruising	Flap sloughed.
30	Ellen P.	25	Disease of tibia	Long anterior and short posterior flap	Flap sloughed.
31	Jane H.	24	Paralysis (congenital?)	Do.	No note.
32	Elizabeth G.	68	Old ulcer	Do.	Flap sloughed.
33	William S.	20	Primary, for compound fracture	Do.	Flap sloughed.

Abstract. No. of cases 33

Cases of sloughing 16

„ of no sloughing . . . 4

„ in which there is no note . . 8

„ phagedæna 1

„ died on or before third day . . 2

„ of long posterior flap . . . 2 = 33

Mr. Bryant thought the operation had not been recognised as it should have been. He did not think that sloughing and phagedæna were so common as Mr. Pick seemed to think. Mr. Bryant advocated amputation at the knee-joint. He had performed the operation several times, and regarded the operation as of the simplest and safest either for disease or compound fracture. He looked with much favour on the method brought forward by Dr. Stephen Smith. Mr. Pollock had advised the removal of the patella, but Mr. Bryant thought there was no necessity to remove it. The operation was one of life-saving value, and was not to be compared with any other operation in this respect.

Mr. Davy had performed the operation nine times; in one of the first five cases there was some sloughing, and out of the whole number there was but one death. The parts taken by the fascia lata and the unrestrained muscular action after the amputation were mentioned. In one case of the "coat-sleeve" operation there had been sloughing over the external condyle. He showed a piece of dried cartilage separated from the external condyle which had had much granulation tissue about it.

CASE OF PULMONARY STENOSIS IN A CHILD.

By William H. Day, M.D.

W. D—, aged six, a pallid, nervous boy, was admitted into the Samaritan Hospital under my care November 21st, 1883. He had previously been under the care of my colleague, Dr. Prickett, who was the first to recognise a peculiar murmur at the base of the heart. It was ascertained that he had been delicate and ailing from birth. Two years ago he had a fall and hurt his head; about this time he is said to have had two fits, in one of which he screamed and struggled, in the other he lay quite still. A week later he had a mild attack of measles, and since then he has lost flesh. He has never had rheumatism or scarlet fever. There is no history of phthisis in the family, and the parents and two other children have always experienced excellent health. Has never had a blow or injury to the chest.

On *examination* a loud and rough systolic murmur is heard over the third left costal cartilage and marked vibratory thrill below the clavicle at the second left interspace. The murmur diminishes in intensity towards the apex, but is loud over the conus; it is less loud and softer on the right side over the second costal cartilage, diminishing in intensity as the right subclavicular region is reached. This murmur I take as evidence of coexistent congenital malformation. It is not carried into the neck on either side. The mur-

mur is heard in both axillary regions, loudest on the left side and louder over the ensiform cartilage than at the apex. The murmur is also heard indistinctly at the angle of the left scapula. The chest is everywhere resonant on percussion, there is no cough, no œdema, no dyspnœa, and no laryngeal symptoms. He can run up and down stairs without being distressed. There is no cyanosis and no præcordial pain whatever; the pulse is regular, equal at both wrists, and averages 80 beats a minute. The fingers are not clubbed. The temperature both morning and evening has been normal since admission; the bowels are regular, the urine is non-albuminous.

I have thought this case of sufficient interest and rarity to bring before the notice of this Society, never before having met with a case precisely like it, and for some time it seemed difficult to make a satisfactory diagnosis of its true nature. It is fortunate that such cases are rare seeing that they are not amenable to treatment. My doubt as to its nature for some time was caused by the fact that if the mischief was located in the pulmonary artery, it was singular that the right side of the heart should not be dilated and some degree of cyanosis be present. Indeed, from all we know of these lesions in the pulmonary artery we must conclude that as time passes on the constriction in, or the pressure on the vessel will make itself more and more felt, and lead to gradually increasing congestion and dilatation of the right ventricle. There can be no doubt of the case being congenital. I presume such cases always are.

Dr. Sansom informs me that he has watched a similar case in a boy who lived to early manhood and then died of pulmonary phthisis. The post-mortem examination in Dr. Sansom's case revealed stenosis of pulmonary artery and no other lesion. Dr. Thorowgood informs me that he has known a similar systolic murmur to the left of the sternum caused by pressure on some portion of the aorta.

I was puzzled some years ago to ascertain the cause of a pulmonary murmur in a young woman the subject of an ovarian tumour. Three medical men who saw the case before me were equally puzzled. When the patient took a full inspiration the murmur disappeared, and reappeared on expiration, so that I came to the conclusion that it arose from retraction of the lung leaving the base of the heart uncovered.

The anæmic condition of this little boy may have something to

do with the production of the murmurs, but it is not sufficient to account for them. The case shows that a loud pulmonary murmur is not incompatible with a perfectly healthy heart.

ON FLAT-FOOT AND ITS CURE BY OPERATION.

By Alexander Ogston, C.M.

The improved results of wound treatment by Sir Joseph Lister's antiseptic method have placed in our hands modes of remedying surgical deformities that would formerly have been regarded as unjustifiable, but may now be practised with absolute confidence by skilled antisepticians, and with excellent results even by those less versed in the antiseptic methods.

The operations of osteotomy, though known and practised by a few before Listerism was introduced, have now become the property of every surgeon and are almost universally practised. But much remains to be done before we shall have turned our improved methods of wound treatment to the best possible account in the surgery of bones and joints.

In one part of this field I have been working for several years, and now venture to bring my results before my surgical brethren. The cure of flat-foot is the object I have aimed at.

Excepting those by apparatus, the modes of treating this disease at present employed are, so far as my experience goes, entirely unsatisfactory, and no instance of cure by any of them has come under my observation. It is true that by rest and time the pain that accompanies the deformity becomes ameliorated or disappears, so that the surgeon may if he pleases call the result a cure, but the deformity does not disappear or even become materially diminished. A cure, in the proper sense of the word, has not taken place In all the cases where I have employed the ordinary methods long and patiently there has resulted in the most favorable merely a cessation of the pain at the instep, but in the large majority the patients have wearied of treatment and withdrawn themselves from it. Want of time and money have hitherto prevented such patients carrying out a cure by orthopædic machines under my care.

Flat-foot, or pes valgus acquisitus, is, like scoliosis and knock-knee, almost invariably the result of a disproportion between the

strength of the foot and the work it has to accomplish. Hence it generally occurs in adolescents, is most frequently due to the rachitis adolescentium that produces so many of these static deformities, and is seldom found save in those who are overworked. In message-boys, young agricultural labourers, domestic servants, and even young people at school, we frequently see it slowly appear. If we examine into their general health we rarely miss the languid circulation, the cold extremities prone to perspire, and the rickety knottings of the bones, especially at the anterior ends of the ribs. We are told that there has been heavy labour, out of proportion to the strength, causing pain or dull aching about the instep, which passes off on rest but reappears on exertion and becomes more aggravated the longer the exertion is continued.

But the history of flat-foot varies much. I know of one case where a very marked double flat-foot, casts of which are on the table, occurred, without any pain being complained of, in a boy of seven years of age, who was very fat and in whom the weight of the large body seemed the only cause adequate to account for the onset of the deformity.

In flat-foot, as in all static deformities, it seems reasonable to believe that all the textures which normally contribute to maintain the correct posture of the foot become affected, though of course in different degrees, and the foot as a consequence becomes perverted in its form.

The alterations are worthy of some notice. The point on which they hinge is a yielding and flattening of the arch of the instep, all other changes being secondary to this and directly consequent on it. The flattening of the arch of the foot is the test of the existence of flat-foot, and the condition is better designated by the term flat-foot or pes planus, not unfrequently bestowed upon it, than by the name of pes valgus or everted foot that it often bears. The name pes valgus is misleading. The valgus or everted posture is not necessarily characteristic, as it includes another and different condition which is, I think, generally confounded with it. This condition is that of everted or valgus ankle.

Valgus ankle is usually seen in young girls passing out of childhood, and is sometimes temporary, sometimes permanent. The ankle, especially when seen from behind, is observed to have lost its straight form and to fall inwards, so that the malleoli approach the middle line and constitute a "knock-ankle." The rubbed con-

dition of the boot may show that the ankles do actually strike one another in walking. If the foot be inspected without its coverings the valgus ankle, which may be detectable only when the patient stands, is observed to be limited to the region of the ankle-joint, and seems due to a loss of form of the bones, the malleoli and astragalus, perhaps of the calcaneo-astragaloid articulation as well. But the foot below the ankle does not participate in the deformity save in so far as the valgus ankle entails on it an outward deviation. The arch of the instep remains unaltered.

In true flat-foot, on the contrary, the ankle-joint can hardly be said to participate. The arch of the foot suffers, and the bones, tendons, and ligaments that maintain the shape of the instep are so modified that the arch unfolds, its two extremities recede from one another, and its curve finally becomes a straight line, touching the ground along its whole length.

Examination of such a foot may reveal a slight laxity of all its articulations, but there are always great changes at the joint between the scaphoid and the head of the astragalus, forming the inner half of the mediotarsal or Chopart's articulation. Here the relaxation is very great, so that by acting on this joint alone we can, on the one hand, rectify the faulty position of the foot, and, on the other hand, move it into the worst possible degree of the deformity.

It is only when the disease is still recent that we can by manipulation cause the deformity to disappear. In recent cases it may even disappear if the patient sit down and lift up the foot for our inspection. The superincumbent weight being removed the flattening of the arch disappears, sometimes quickly, sometimes more slowly, so that the foot may be normal in outline, and our attention be called to what really exists only by the patient complaining of a dull aching pain at the instep, sometimes aggravated by firm pressure about the scaphoid bone. In such instances mistakes in diagnosis are frequent. When the patient stands, however, the deformity reappears, and if he again sit down and lift up the foot it resumes once more its normal outline.

When we try manually to enlarge the arch which the instep forms, the deformity can be made as evident as when the patient stands, and we can remedy in like fashion the distortion we have produced. When the foot is so manipulated, it is only needful to move Chopart's joint, one hand fixing the astragalus and the other

grasping the scaphoid. Then the maximum degree of the disease can be caused to appear or disappear, while similar movement at any other joint shows that its share in producing the deformity is very slight. So long as the hands maintain Chopart's joint in its proper position the flat-foot cannot be rendered evident.

In process of time the deformity becomes permanent, and can no longer be made to disappear. It is in such cases that the details of the deformity can best be studied. We discover that the tarsal bones along the inner side of the foot are ranged in a line parallel with the ground and everywhere in contact with it. The first metatarsal and the internal cuneiform are horizontal in direction, the scaphoid and head of the astragalus behind them form a marked prominence in the sole and on the inner side of the foot, a callosity covering them on the sole, and a bunion-like patch of skin on the inner side of the foot. The articulation between them, so mobile in the earlier stage, is now fixed and rigid. On the application of great force it can sometimes be caused to yield so that Chopart's joint may be restored to position, the deformity disappearing, but often the joint remains rigid and refuses to move upwards under any force. This is due to an alteration in the shape of the bones, detectable by palpation, and also visible when the joint is opened.

The manner of production of this alteration is as follows:— When the arch is flattened the ligaments beneath become elongated, and on the under surface of the joint the bones tend to become separated from one another, while on the upper side the pressure between them is increased. Hence growth is checked, or absorption even takes place, at the upper halves of the articulations of the scaphoid and astragalus, while the lower halves, where the mutual pressure is lessened, show a tendency to separation and increased growth of the separated surfaces. The increased growth takes place mainly in the astragalus, so that the joint is not found gaping below, but the caput tali is changed in shape, becomes somewhat square in form, and presents an abnormal ridge or projecting angle dividing its articular facet into two portions, one articulating with the scaphoid, the other with the inferior calcaneo-scaphoid ligament. When this disposition of bone becomes very prominent, the flat foot enters on its permanent stage, and the more marked the projecting angle becomes the greater is the resistance offered to the reduction of the deformity. The angle ultimately projects so much

that it locks on the scaphoid and no reduction is possible until it be removed.

The relaxation and subsequent alteration of shape in the bones of Chopart's joint are the key to the disease and its successful operative treatment.

There are two secondary changes in the foot, however, that deserve notice as completing the picture of flat-foot, first, the everted or valgus position, and second, the attitude of the great toe. It naturally follows that, after the descent of the arch of the inner side of the instep, the changed relations of the tarsal bones produce an appearance of the foot resting unnaturally on its inner edge, or in other words, becoming everted. In most cases of flat-foot this phenomenon is very apparent. It seems due to the falling down of the inner arch, and is generally unconnected with valgus ankle. When the arch is raised into its proper place eversion vanishes.

By the increased separation of the extremities of the arch the structures of the sole of the foot are made tense, and the muscles that flex the great toe, being put on the strain, and atrophied by the pressure against the ground, flex the great toe at its ball, and it consequently ceases to form an angle with the metatarsus and comes to be in a straight line with it. In this way the metatarso-phalangeal joint appears very prominent above, and from its abnormal exposure to pressure is tender or forms a bunion, while the ball of the great toe, a feature of the normal sole, is diminished in size. The position of the toe is at first remediable, and a restoration of the arch of the instep produced at Chopart's joint by the hand remedies at the same time, without anything further being done, the malposition of the toe, but it reappears in proportion with the reappearance of the flattened arch. In long-standing cases the toe becomes permanently deformed and may ultimately give rise to much suffering.

In the very worst cases, which are rare, the yielding of the foot at Chopart's joint goes even further than to allow the head of the astragalus to touch the ground. The sole divides into two parts, that before and that behind Chopart's joint, and each obeys a different tendency. The part anterior to the joint remains as the walking sole of the foot and continues in contact with the ground, while the other portion, consisting of the os calcis and astragalus, has its posterior end drawn up so that the tuberosity of the calcaneum is elevated a finger's breath or thereabout from the ground, and the

portion of the sole corresponding with the posterior fourth of the foot does not come into contact with the ground. The bones are rigidly fixed in this perverse posture and the feet are shaped not like arches, but like canoes.

The difference between a flat-foot where the deformity is still reducible and one where it has become rigid depends upon a change of form of the bones, already partly described, that can to some degree be observed by palpation from without, but which is very evident when the joint is opened.

In the extreme dorsal flexion of the astragalo-scaphoid joint present in flat-foot, the caput tali is no longer so much covered below by the scaphoid as it normally is, but escapes from it downwards and inwards, so that it finally forms on the inner side of the sole a prominence greatly exceeding in size that of the tuberosity of the scaphoid. The articular surface of the scaphoid is altered in direction so that it looks more downwards, and tends to forsake its contact with the caput tali. There would be an actual gap between the bones below did not the astragalus accommodate itself to the void and assume an angular form with two facets nearly at right angles to each other, one looking forwards and articulating with the scaphoid, and the other looking downwards to the ground, parallel to it, and resting on the inferior calcaneo-scaphoid ligament.

So soon as the altered shape of the astragalus head becomes pronounced the deformity ceases to be easily reducible, and it shortly comes to pass that it offers an insuperable obstacle to the reduction; the foot is henceforth fixed, because the prominent angle cannot be made to ascend behind the scaphoid since it locks upon it with every attempt at plantar flexion.

I have given much attention to these cases, and have tried many plans of treatment. Boots with the inner margin of the soles raised, arched steel supports under the inner side of the sole, well-moulded pads of cork and other materials, or hollow cushions of caoutchouc, have not produced a cure even in mild cases, while they are of course inapplicable in the severe forms. Lateral supports to counteract the valgus position have been equally unsuccessful. Prolonged rest, with or without stiff bandages, has always relieved the pain for the time being, but has never cured a single case in my hands. Neither have I had any success with Langenbeck's method of forcibly reducing the deformity with the hands,

and then treating in plaster-of-Paris bandages, for it is inapplicable in the reducible cases and impossible in the more advanced forms. To give the experience of a good many years in one sentence—none of the plans of treatment I have tried have had in my hands any effect whatever in the cure of the disease.

When observing the disease and my failures in its treatment, I could not help being struck with the prominent part played in its production by Chopart's joint, and became convinced that, could any method be devised of restoring the joint to its normal position and rigidity, or even of causing bony ankylosis there, it would almost surely result in a cure. From observing that so long as the manipulating hand held Chopart's joint firmly reduced so long the deformity was apparently cured, the inference was natural that any method of imitating from within the effect of this support from without would have a good prospect of radically curing the disease. Ankylosis between the astragalus and the scaphoid could do no great harm, for there are so many other points at which the tarsus is moveable that probably the rigidity of this one joint would entail no inconvenience.

The first attempts at reducing this idea to practice were made in the year 1877, when a series of cases of flat-foot were treated by reducing the deformity as perfectly as possible, fixing them in the improved position by plaster-of-Paris bandages, and maintaining them immoveable for periods of three months. Some of them had a fenestra cut in the bandage and frequent injections of carbolic acid lotion made into the neighbourhood of the joint in the hope of causing rigidity by the prolonged rest and the irritation of the injections. Although some seem to have benefited by the treatment, the improvement was not permanent, and in none was a satisfactory cure obtained.

It was next decided to open the joint and remove the articular cartilage from a small portion of either bone at corresponding points under the idea that ankylosis would result from it. Bidder's experiments, performed in 1877,* had shown that new bone is not formed when an ivory peg is driven through the articular surface of a bone, and hence it was inferred that nailing the bones together by pegs, without removal of the articular cartilage, would fail in producing ankylosis, while removal of a portion of the articular

* 'Langenbeck's Archiv,' vol. xxii, Heft 1, "Regeneration des Knockengewebes."

cartilage would probably result in bony union of the denuded parts.

Accordingly in the next case that was submitted to treatment an incision was made along the inner side of the foot over the joint, and while the foot was held in the best possible position a notch was made by a small saw in the two bones, the saw being made to cut a linear track into the head of the astragalus and through the joint for some distance into the scaphoid. It was hoped that bony union between the two clefts would result, and to favour this the foot, still held in the rectified position, was put up in plaster of Paris. After three months' rest, this patient was dismissed much improved, although I am unable to state that her cure was permanent, as careful inquiry failed to trace her after she had left. Before she left I believed I could make out slight movement in Chopart's joint, and as this would have jeopardised the cure it was decided to treat the next cases by more extensive denudation.

In the year 1878, two patients were subjected to the following operation. An incision was made along the inner border of the foot, down to the joint, and a small wedge of bone, three quarters of an inch deep and of a like breadth, was chiselled out of each of the bones, leaving notches at points corresponding with each other, the foot being held in the position of most complete rectification while this was being done. In both cases the patients were dismissed in two months seemingly cured; in one of them the cure was permanent a year after the operation. In the second case the result was not satisfactory as she was still complaining nearly two years after the operation.

Yet it seemed probable that a more certain means of producing bony ankylosis would yield better results, and after much consideration the following method was devised, and has since been carried out with excellent results in seventeen cases.

The object of the proceeding is to denude as much of the cartilaginous surfaces of the astragalo-scaphoid joint as can conveniently be reached, to place the foot in proper position and secure its immobility by uniting the two bones by ivory pegs. It is applicable both to the milder cases which are reducible, and to the severer which are not so. I have never had an opportunity of using it in the very aggravated forms where the sole becomes boat-shaped and the heel leaves the ground.

On purpose to secure the utmost benefits of the antiseptic

method, the feet are, as a preliminary step, washed daily in a 1·20 carbolic lotion and done up in a large Lister's dressing while the patient is confined to bed, and this is carried out for four or five days, so as to secure the greatest possible purity of the thick layers of epidermis that exist on the sole and over Chopart's joint. All loose epidermis is peeled or rubbed off with pumice-stone, and when a state of purity has been reached the patient is put under chloroform or ether, both feet, if the disease be double, being operated at one sitting.

The dressings are removed, the elastic tourniquet is placed round the leg below the knee, and the leg, ankle, and foot are washed once more with carbolic lotion and finally with oil of turpentine. The foot is then laid on a disinfected piece of macintosh, and the carbolic spray is turned on it. It is convenient to stand on the left side of the patient, while an assistant stands opposite holding the foot by the ankle and metatarsus.

In a normal foot Chopart's joint lies about an inch in front of the internal malleolus, and the most prominent bony point on the inner side of the foot is the scaphoid tuberosity just anterior to it. But in the flat-footed the astragalus head is so greatly displaced from the scaphoid and so prominent that it forms the large projection seen and felt on the inner side of the foot about an inch in front of the tibia, and the scaphoid is comparatively indistinct, while the line of Chopart's joint is half an inch further from the tibia than usual. By moving the metatarsus this can generally be felt to be the case. Hence an incision to open the joint has to be made further from the ankle than in a normal foot.

The foot, lying in the assistant's grasp without any attempt being made to reduce the deformity, is placed with its outer side resting on the operation table, and an incision, an inch and a quarter long and parallel to the sole, is made along its inner side over the joint, dividing all the structures down to the bones. If this incision commence about an inch from the tibia its centre will be over the articulation. No important structure is divided save some small branches of the internal saphena vein or the small vein itself, and these may be tied with catgut or left unligatured.*

* A longitudinal incision gives best access to the joint. It may, in aggravated cases, advantageously be slightly curved with its convexity downwards if it seem desirable. An incision across the border of the foot, parallel to the line of the articulation, is attended with more risk of missing the joint and

If the incision be carried down to the bones by the first movement of the knife, the head of the astragalus, partly covered with cartilage, will be observed through a button-hole in the capsular ligament in the depth of the wound if its edges be retracted by aneurism needles. If not, a second cut with the knife completes the division of the soft parts. After the caput tali has become visible, free access to the joint has to be obtained by separating the attachments of the ligamentous capsule to the edge of the scaphoid for a distance of half an inch on each side of the wound. The ligament is seized by a dissection forceps, elevated and detached from its insertion into the scaphoid, its connections with the periosteum and fibrous structures over the scaphoid being maintained as far as possible by cutting with the edge of the scalpel directed towards the toes, the blade lying parallel with the bone.

In this manner a somewhat T-shaped opening is made into the joint, and sufficient access is gained for the next step of the operation, the denudation of the bones.

A stout chisel, half an inch broad, bevelled on one side and provided with a wooden handle, is held in the right hand with the bevelled side away from the caput tali, while by its means the articular cartilage is shaved away from the whole of the exposed surface of the bone over as great an extent as is possible, a thin layer of the subcartilaginous bone being also removed so that the cancellous structure is well laid bare. The chisel is next applied to the scaphoid, the bevelled side being held towards it, as the surface to be here denuded is concave, and by repeated shavings the denudation is carried as far as possible between the bones. In this manner each bone is bared of its cartilage, and if the arch be now restored to its normal position by the assistant the two surfaces are found to correspond, the head of the astragalus retracting into its normal position behind the scaphoid.

If the deformity be of old standing and the bones have adapted themselves to their altered position, it is not possible to restore the arch until, by means of the chisel, the prominent angle that has formed on the lower surface of the astragalus head has been shaved off, and the rounded form of the head restored; but when this is accomplished the arch is easily put into its proper shape.*

wounding important structures, while it gives less ready access to the head of the astragalus than the incision parallel to the sole.

* The denudation can be accomplished by means of a stout scalpel, a Volk-

The next step of the operation is to nail the bones together by ivory pegs. The joint is washed out with 1 in 20 carbolic lotion and the arch of the foot is restored to its normal shape by the assistant depressing the metatarsus. The scaphoid after this movement covers the head of the astragalus, and the denuded surfaces lie opposite one another, separated by an interval of about a quarter of an inch, caused by the removal of so much bone. In the milder cases the arch is perfectly restored to its position; even in the severer cases it is much improved. In its restored shape it is held fast by the assistant, while the operator drills two holes through the scaphoid into the caput tali. The anterior angle of the wound is drawn apart by retractors so as to expose the scaphoid. The point of the drill* is placed on the upper and inner side of the scaphoid, the drill is pointed towards the centre of the caput tali, and when its direction has been well determined it is set in motion, piercing a hole an inch and a quarter in depth through the two bones. On withdrawing the drill,† an ivory peg‡ is held ready to be put in its place. When it is withdrawn one of the pegs is placed in the hole and driven home by a series of light taps with a small mallet. After it has entered for rather more than

mann's sharp spoon or curette, or a mallet and chisel, but the most convenient plan has seemed to me that which I have described, by the chisel held in the hand. The soft bones are easily denuded, and the shavings are carefully lifted out by a dissection forceps. A little gluey synovia often escapes during the process.

* The best drill, to my thinking, is an archimedean drill, such as is used by carpenters, fitted with what are sold as "broaches" (No. 40), by the wholesale watchmakers, and which form good drill points. They should be somewhat less in size than the ivory pegs to be employed. The drills cost 3s., the broaches 3½d. each.

† The best method of disinfecting the drill and its point is, I believe, to wash it well with oil of turpentine.

‡ The pegs are prepared from the finest ivory knitting needles, sold in the Berlin wool shops as ivory knitting pins, No. 13, Wynn's bell gauge, and are about eight inches long, and of the size of a No. 7 or 8 catheter, French scale, or a No. 2 English scale. They should be cut with a fine saw into pieces three inches in length, which are sharpened at one end by a file, and boiled in 1·20 carbolic water for ten or fifteen minutes until they lose their whiteness and become impregnated with the disinfectant lotion, after which they are preserved in 1·8 carbolic oil; or they may be prepared by Neuber's method of prolonged soaking in German oil of juniper and afterwards preserved in absolute alcohol. I have not yet used them as prepared by the latter method.

half its length, its projecting end is cut off level with the scaphoid by a bone forceps. A second perforation is then made parallel to the first and nearly half an inch distant from it through both bones, and a second peg is driven home in it. One of the perforations may be made beneath one margin of the skin and the other beneath the opposite margin.

The bones are so firmly fixed together by the pegs, which can be seen crossing the gap that separates them, that when the assistant removes his hand the foot remains in the improved position.

The wound is well cleaned, the lips brought together by a series of catgut sutures an eighth of an inch apart. I do not usually loosen the elastic tourniquet until after this has been done and the Lister's dressings applied, but if there be any dread of hæmorrhage the elastic cord can be removed before the wound is closed. If this be done the bleeding is usually free and comes from the bones. But as no arteries of any magnitude have been divided the compression of a well-applied dressing is the best means of preventing the escape of any quantity of blood. A Lister's dressing is therefore carefully put on, enclosing the whole foot from a hand's breath above the ankle to beyond the toes. The spray is then turned off. A few turns of plaster-of-Paris bandage outside the dressing steady the foot, and after they have been applied the tourniquet is removed.

The patient suffers sharp pain for twenty-four hours, and after that the convalescence is painless. The dressings may be removed on the fourth day, or left on for weeks. On their removal the wound has always been found healed, or existing merely as a superficial sore. The patients are kept in bed for two or three months, and are then permitted to rise. In a week or two afterwards they are able to walk freely.

This operation I have performed seventeen times in ten patients, and in one case, operated on 8th of December last, I pegged on the left foot, the joint between the scaphoid and internal cuneiform as well as Chopart's joint, because its movement seemed unusually free.

My patients have all, without exception, remained free from fever, the thermometer not rising above 100° Fahr. save when two of them, while still under treatment, were attacked by sorethroat, lasting for a day or two. The wounds remained aseptic, and the dressings were renewed only a very few times. In some of the

patients a slight tenderness and pain on trying to walk at the end of six or eight weeks indicated that the joint was still unankylosed, doubtless from the slow repair due to defective circulation. They were often permitted to walk a little at the end of two months, and to use the feet freely at the end of eight months, but I think that any use of the feet should, as a rule, not be permitted until three months have elapsed, as bony union is slow in such individuals.

In all my patients, to the best of my belief, great benefit resulted from the operation, and in most of them bony ankylosis and a painless arch was obtained. They mostly resumed their laborious occupations, and were able fully to bear all the demands made upon them. One patient subsequently died of heart disease.

In one patient an ivory peg was spontaneously and painlessly extruded by a small cutaneous opening five months after the operation, but in every other instance they remained, probably to undergo slow absorption or vascularisation.*

Another patient, while under treatment, complained that one of his operated feet ached a little, and was never quite satisfied with the result on that foot. Yet he underwent a season of heavy harvest work after leaving the infirmary. He returned again last November, and a little fulness was then detected about the calcaneo-cuboid joint of the right foot, the one which had ached.

Both were put up in plaster of Paris for two months, and when these were removed at the beginning of this month the feet looked perfect. The restoration of the arch of the instep was absolutely perfect in this case.

In a few of the other patients the plantar arch was restored to perfection. But this was not generally the case. In all it was, however, much improved, and added to the patient's lightness of step even when to a surgical eye traces of the deformity remained. One of my cases is still under treatment.

Knowing how much the prolonged rest following the operation would of itself tend to improve such cases, and alive to the temptation to be sanguine and over-confident in judging of the results of one's own work, I have endeavoured to free myself from any bias, and to form a true estimate of what was due to the operation itself in the improvement that resulted. Such of the patients as were accessible were seen or written to at considerable periods after the

* Riedinger, 'Verhand. d. deutschen Gesell. f. Chir.,' 1881, x cong., p. 167. Trendelenburg, the same, p. 136.

operation, and were warned that the information they gave was required not to gratify me, but to determine whether the same operation should be tried upon others. They all, save the patient mentioned above, maintained that the operation had cured them, and that they would willingly undergo it again on account of the benefit they had derived from it.

My own impression is that this mode of treatment is likely to be of use in suitable cases, uncomplicated by other diseases, and where none of our other methods can be relied upon. I therefore think it desirable that it should be tested by others, should they deem it worthy of a trial, and receive its verdict according to the results it may yield in their hands.

Mr. BRYANT thanked Dr. Ogston for bringing forward so good an operation. Mr. Golding-Bird had carried out a very similar procedure in such cases at Guy's Hospital for some time past. Mr. Bryant did not see the necessity for pegging the bones together. Moreover, the ivory pegs often remained unaltered; he doubted whether vascularisation of them could take place.

Mr. BAKER thought the contracted tendons ought to be divided in cases of flat-foot.

Mr. DAVY said although he was rather favorable to the resections of the tarsal arch, he was not convinced of the utility of the procedure. He would have liked to have seen the patients themselves, so that he might judge of the "cure" which had been effected. Mr. Hilton had said that the inner three fifths of the foot represented the elastic element, the outer two fifths the sustentative element. By interfering with the inner part the rigidity of the foot was increased, which could not be regarded as an advantage. Mr. Davy had performed twenty-four resections of the tarsus with only one unfavorable issue, and these without anything more than mere "cleanliness" as contrasted with Listerism.

Sir JOSEPH FAYRER spoke of flat-foot in soldiers and sailors, and inquired what ought to be done for these as well as for children with flat-foot where as yet no structural alterations had been set up?

Dr. OGSTON, in reply, said that he had not had to deal with contracted tendons. He had no experience of those very severe cases where the tendo Achillis was much contracted and the os calcis raised. He did not pretend to go into the pathology of what happened to the ivory pegs; but he quite sympathised with Mr. Davy's scepticism.

Table of Cases of Flat-foot, treated by Excision and Pegging of the Astragalo-Scaphoid Articulation.

No.	Sex.	Age.	Occupation.	Right or left foot.	Date of operation.	Course of cure.	Date of dismissal.	Final result.
1	F.	18	Farm servant	Right and left	Jan. 4th, 1880	Afebrile recovery, save for intercurrent sorethroat	June 5th, 1880	Apparently cured at dismissal. Writes on Dec. 10th, 1883, that she is "quite well, much better of the operation," and her feet "stronger than they were before."
2	F.	14	Domestic servant	Right	Sept. 30th, 1880	Afebrile recovery	Nov. 22nd, 1880	Sent to convalescent hospital, and subsequently dismissed cured. On April 1st, 1881, returned to report herself. Had been in service for a month, and was nearly well. On Sept. 8th, 1881, she returned to report, and was seen by Mr. William Adams, London. Quite cured.
3	F.	17	Farm servant	Right and left	Sept. 30th, 1880	Do.	?	Returned to report herself on Sept. 29th, 1881. Feet almost perfect in shape; valgus position gone. Is satisfied that she is cured.
4	F.	13	Mill-worker	Do.	March 5th, 1881	On 24th May, 1881, pain on walking; again kept at rest	July 7th, 1881	Apparently cured at dismissal. She died, I was afterwards informed, of heart disease and general dropsy, in June, 1882.
5	F.	15	Crofter's daughter	Left	Jan. 25th, 1882	Afebrile recovery	March 11th, 1882	Apparently cured at dismissal. Writes on Dec. 8th, 1883, that the operation improved her foot very much, though it is "weak at times yet."
6	F.	17	Domestic servant	Left	Jan. 25th, 1882	Do.	March 13th, 1882	Returned to report herself Oct. 9th, 1882. Chopart's joint seems ankylosed at inner side. Patient is satisfied she is cured.
7	M.	18	Farm servant	Right and left	Dec. 30th, 1882	Do.	?	Complained of a little pain in right foot on dismissal. Supposed to be malingering. Worked through harvest of 1883, and returned on Nov. 1st, 1883. Outer part of right Chopart's joint a little swelled. Left foot perfect. Put up in plaster bandages until Jan. 5th, 1884. The feet were then perfect in shape and seemed soundly cured.
8	F.	18	Domestic servant	Do.	March 1st, 1883	Do.	?	Returned Oct. 22nd, 1883, and reported herself cured. Both feet perfect. A month previously an ivory peg painlessly appeared at a small opening, and was drawn out.
9	M.	14	School-boy	Do.	Oct. 9th, 1883	Do.	Nov. 27th, 1883	Sent home with every appearance of being perfectly cured.
10	F.	12	Farm servant	Do.	Dec. 8th, 1883	Do.	Still under treatment; giving every promise of a good result.	

January 28th, 1884.

ON THE NATURE OF SNAKE-POISON, ITS EFFECTS ON LIVING CREATURES AND THE PRESENT ASPECT OF "TREATMENT OF THE POISONED."

By Sir Joseph Fayrer, K.C.S.I., M.D., F.R.S.

The communication which I have the honour of making to you this evening is in fulfilment of a promise made last April, after the discussion of a paper on a similar subject by Dr. Badaloni, of Nocera, which excited considerable interest and some criticism with regard to the purely scientific aspect of snake-poisoning, its treatment, and its relation to the vital statistics of countries in which venomous snakes are more numerous than in our own (in this respect) more highly favoured one.

I purpose to describe the nature and the mode of action of snake-poison on living creatures; and, being most familiar with the Ophidia of India, I shall select some illustrations from that source, especially as it affords typical examples of snakes which are endowed with this terrible power of destroying life.

Let me ask your attention to some points in the structure of the apparatus which is concerned with the elaboration and inoculation of the poison which it is the purpose of this paper to describe.

The order Ophidia has three subdivisions:

1. *Ophidii columbriformes* (innocent).
2. *Ophidii columbriformes venenosi.*
3. *Ophidii viperiformes.*

The two latter are all poisonous—they are the Thanatophidia, and well merit this name in India, where they destroy, probably, 20,000 human beings annually.

The general anatomical structure and distinctive characters of a snake are well known, but I will ask you to notice certain differences between:

1. An innocuous and a poisonous snake.
2. Between a poisonous colubrine and a viperine snake.

The crania, drawings and dissections before you illustrate these differences. Snakes are provided with sharp recurved teeth, which

are firmly fixed in the maxillary, palatine and pterygoid bones; by the form and arrangement of these teeth, poisonous may be distinguished from innocent snakes.

The harmless snake has two complete rows of ungrooved small teeth, one outer or maxillary, and one inner or palatine row; in the majority there are from twenty to twenty-five teeth in the outer row.

In the venomous snakes, the outer row is represented by one or more large tubular fangs, firmly anchylosed to the maxillary bone, which is moveable, and by its movement causes the erection or reclination of the fang which is so marked in Viperidæ. In the innocent snake, the maxillary bone is elongated, and gives insertion to a row of teeth; in the poisonous colubrine it is much shorter, giving insertion to only one or more teeth, the anterior and largest of which is the poison-fang.

In the viperine snakes, the maxillary bone is reduced to a mere wedge, giving insertion to a long curved and tubular fang, which is a much more formidable weapon than the fang of the cobra, or other colubrine snake.

These fangs, when reclined, are covered by a sheath of mucous membrane, in which lie also several loose reserve fangs, in different stages of growth. When the working fang is lost by accident, or is shed, one of the reserve fangs takes its place, becoming fixed to the maxillary bone, and placed in communication with the duct of the poison-gland.

The teeth vary considerably in the different subdivisions of the order. They are described as being perforated. Though this is apparently the case, it is not really so. They are dense and compact, enclosing the usual pulp-cavity; but being folded on themselves form either an open groove, as in the Hydrophidæ; a complete canal, as in cobra; or a more complete tube still, as in Viperidæ.

During development the laminated tooth folds like a leaf on itself, and so forms the channel along which the virus is conveyed; and thus the tooth makes a most complete hypodermic syringe.

The poison is secreted by a conglobate racemose gland situated in the temporal region behind the eye. It is of considerable size—about that of an almond in the cobra—and is furnished with a duct which opens into the capsule of mucous membrane enveloping the base of the fang; the venom thence flows into the dental canal,

and is injected into the wound when the tooth penetrates the bitten object. At the orifice of the duct it seems probable that there may be a sphincteric arrangement of muscular fibres which would enable the snake to control the ejaculation of the virus.

I have not been able to make out such a sphincter in the Elapidæ; but Dr. Weir Mitchell says it exists in crotalus. I may have overlooked it, and think it probable that further examination may detect it in other poisonous snakes. I may here just refer to the remarkable mechanism by which the ectopterygoid bone being pushed forwards, the maxillary is made to rotate, and to erect the fang in the viperine snakes; and to the action of the temporal and masseter muscles, which, whilst they close the jaw in the act of biting, at the same time compress the gland, and force the poison through the duct. Time does not admit of anatomical details; but they are fully described in the 'Thanatophidia' (pp. 1 to 5), and are represented in the sketches and specimens before you.

Before I pass on to consider the poison, let me say a few words about the poisonous snakes themselves. Here I may remind you that the only poisonous snake in Great Britain, and, indeed, in a great part of Europe, is the adder—*Pelias berus*—a viper (or some variety of it); and that, in comparison with the cobras and vipers of India and the Tropics, it is feeble in its venomous power.

The venomous colubrine snakes of India are: of Elapidæ, the *Naja tripudians* or cobra, *Ophiophagus elaps* or hamadryad, *Bungarus ceruleus* or krait, *Bungarus fasciatus* or raj-samp, sankni; of Xenurelaps, *X. bungaroides*, and the various species of callophis; Hydrophidæ, a very numerous family of sea-snakes, all are very poisonous, but, being confined entirely to a marine or estuarial life, are not so dangerous to human life as others.

The viperine snakes are represented by *Daboia Russelii* (or tic polonga, or chain-viper); *Echis carinata* (or kupur, or phoorsa-snake); these are true vipers; while the Crotalidæ, or pit-vipers, are only feebly represented by the *Trimeresuri peltopelor*, halys, and hypnale; these are much less poisonous than their American congeners, crotalus, lachesis, cruspedocephalus, and others.

The Najadæ are the most virulent of the columbrine snakes; none are more deadly than the cobra or hamadryad. Of Viperidæ the daboia and echis are probably as deadly as any of the African forms.*

* Of American Elapidæ, elaps corallinus, and lemniscatus. Of American

In 1868, I resumed an investigation, begun in 1854, on the subject of poisonous snakes and the nature and effects of their venom. During that inquiry, which continued till 1871, I ascertained from official sources that out of a population of 120,972,263 (Dr. Hunter), 11,416 persons died of snake-bite in the year 1869. Subsequent returns show that the mortality continues at very much the same rate. The Sanitary Commissioner, in his report on the North-West Provinces and Oude for 1882, tells us that 6515 persons were killed in that year by snakes and wild beasts, out of a population of 44,107,869. In 1881, in all India, there were 22,377 deaths from the same cause.

In destructiveness the snakes stand in about the following order: cobra, krait, echis, daboia.

The *Ophiophagus elaps*, *Bungarus fasciatus*, and Hydrophidæ are deadly but less numerous, and therefore less destructive to life.

The returns cited represent only a portion of India, and there is good reason to believe that the total annual mortality of the whole peninsula is not much, if at all, under 20,000 persons, or roughly about one in every 10,000.

The subject is of much general interest, and it is as important to humanity as to science to ascertain the nature and properties of the poison, and to discover what may best counteract it.

Snake-poison is secreted by glands which represent the parotids in other creatures (a small gland is connected with the duct of the poison-gland in daboia, and was figured in a drawing by me made in 1869. Dr. Wall suggests that its secretion may in some way modify the action of the poison, perhaps giving it the peculiarity in which it differs from the cobra-venom) and is probably a modification of the saliva, though different in its action from that innocent and indispensable secretion. The analogy is more probable if, as suggested by some physiologists, Mr. Busk and others, there be an active principle in it, closely allied to the ptyaline of saliva.

The virus is a transparent, slightly viscid fluid, faintly acid in reaction, of varying specific gravity, 1·058 being the average (according to Wall) of a mixture of virus taken from several cobras. It has a bitter taste in the cobra, but not bitter in daboia.

Crotalidæ, crotalus (rattlesnake), laches-mutus; Craspedocephalus (West Indian). Of African Elapidæ, naja-haje, naja-hæmachates: of Viperidæ, cerastes, and four or five others are very dangerous.

It is of a faintly straw-coloured hue in cobra; in the ophiophagus of a golden yellow. When dried it loses from 50 to 75 per cent. of water (Wall) and forms a semi-crystalline substance like gum arabic. It is secreted in considerable quantities, and if a fresh and vigorous cobra be made to bite through a leaf stretched across a spoon or shell, several drops can be thus obtained. Examined under the microscope it is structureless, but a few cell-forms and micrococci may be detected. The mucus of the mouth may be the origin of these organisms, and it is probable that there is nothing characteristic in them (Wall), for the most active venom is free from them. The poison is exhausted when the snake has bitten frequently, and it is then comparatively harmless; but it rapidly becomes dangerous again.

"If the virus be kept in the liquid state it first becomes neutral, then alkaline, and a few feathery cubic crystals form; if preserved in a loosely corked test-tube, it will become cloudy, smell offensively, and swarm with bacteria, but still it is poisonous.

"The alkalinity now lessens, and the reaction becomes again acid; the fluid then coagulates into a firm whitish opaque substance, somewhat like the coagulated white of an egg, but of a lemon colour.

"If a small quantity of fluid be left uncoagulated it is poisonous, and the washings of the coagulum are also poisonous" (Wall).

Heating cobra-poison to boiling point (Wall says) does not destroy its physiological action, though less local inflammation is caused by it when so treated.

Snake-poison has been examined by chemists, but a complete or exhaustive analysis has not yet been given.

Fontana, in 1781, and Prince L. Bonaparte, in 1843, made an analysis of the virus of the adder (*Pelias berus*), and came to the conclusion that it contained an active principle, to which he gave the name of echidnine or viperine, which he succeeded in separating. The paper in which he describes the process was read before the Union degli Scienziate Italiani at Lucca in 1843, and is in our library; so far as I know but little has been added since Prince Louis Bonaparte's investigations; further analysis will probably confirm or modify his views, and perhaps add to our information. The Prince laboured under the disadvantage of having only adder-poison to analyse. With a better supply of cobra, daboia, or crotalus virus, which might now be obtained, there are good

grounds for hoping that the chemistry of snake-poison will be exhaustively worked out. This is now being done in America by Drs. Weir Mitchell and J. E. Reichardt, who have published some results of their work.

In 1873, cobra-poison from Bengal was submitted to Dr. Armstrong, F.R.S., for analysis, and he obtained the following results:

Crude poison.	Alcohol precip.	Alcohol extract.	Albumen for comparison.	
				(Ralfe)
Carbon, 43·56	45·76	43·04	53·5	53·5
Nitrogen, 40·30	14·30	12·45	15·7	15·5
Hydrogen	6·60	7·0	7·1	7·0
Sulphur	2·5	...	...	1·6
Oxygen	...	...	...	22·0
Phosphorus	...	...	...	0·4

This is an incomplete analysis, but it is to be hoped that the same eminent chemist may be disposed to continue the investigation when supplied with more virus.

The following is an epitome of Weir Mitchell and Reichardt's investigations, which relate chiefly to crotaline poison, but include a partial analysis of some dried (colubrine) poison from India. They find that the venom of the crotaline snakes can be subjected to the action of the temperature of boiling water, without completely losing its poisonous powers. The activity of the venom, however, of *Crotalus adamanteus* seems to be destroyed by a temperature below 176° Fahr. Mitchell, some years ago, showed that the venom of *Crotalus durissus* is not destroyed by boiling, and the curious fact is noted that the venom of *Crotalus adamanteus* should thus differ from the venom of other snakes.

The symptoms caused by the venom of the different snakes with which they have operated do not, they say, differ radically, save in degree, but there are symptoms which suggest that further investigation may enable them to point out certain differences by which it will be possible to discriminate one form of poisoning from the other. This is partly in accordance with what has already been observed in India, and notably by Dr. Wall.

The investigations, so far, lead them to conclude that the poison of the cobra is the most active, next the copper-head, then the

mocassin, and lastly, the rattle-snake; but their reseaches on this head are not yet complete.

They are unable to confirm the statement of Gautier, of Paris, that an alkaloid, resembling a ptomaine, exists in cobra poison. Professor Wolcott Gibbs, they say, was unable to find an alkaloid in the poison of crotalus, but they have satisfied themselves that the venom contains three distinct proteid bodies, two of which are soluble in distilled water, one of which is not soluble. These bodies have certain properties and reactions, which are detailed in their monograph on the subject.

Hitherto, observers have regarded the venom of different snakes as each representing a single poison; but it appears that, of the three proteids before mentioned, one is analogous to peptones, and is a putrefacient poison; another is allied to globulin, and is a most fatal poison, probably attacking the respiratory centres, and destroying the power of blood to clot, while the third resembles albumen, and is probably innocuous. The separation of the poisons necessitates a long and elaborate series of researches, the results of which will be subsequently reported. They have also ascertained that the poison of the rattle-snake (*Crotalus adamanteus*), copper-head (*Trigonocephalus contortrix*), and mocassin (*Toxicophis pisci-vorus*), are destroyed by bromine, iodine, hydrobromic acid (33 per cent.), sodium hydrate and potassium permanganate.

It appears that the activity of the venom differs not only in character and intensity in different genera and species, but also in the same individual under varying conditions of temperature, climate, health, and state of vigour or exhaustion at the time. It is a most virulent poison, and it takes effect when absorbed into the circulation, either by inoculation, or, as I demonstrated in India (quite against all former and universal belief), when applied to a mucous or serous membrane, proving that it may neither be sucked from a bite nor swallowed with impunity.

It acts most rapidly on warm-blooded creatures, sometimes with lightning-like rapidity, when it enters a vein; it is deadly also to cold-blooded creatures, and to the lowest forms of invertebrate life. Strange to say—and this, to me, is one of the greatest of its mysteries—a snake cannot poison itself, or one of its own species, scarcely its own congeners, and only slightly any other genus of venomous snake, but it kills innocent snakes quickly. It has been ascertained that a vigorous cobra can kill several dogs or from a

dozen to twenty fowls before its bite becomes impotent, and then the immunity is of brief duration, for the virus is rapidly reformed.

In 1868 and 1869 I observed that, whilst the general characters of the effects of snake-poison are alike, yet viperine differs from colubrine poison. The poison of naja kills without destroying coagulability of the blood, whilst that of the daboia (viper) produces complete permanent fluidity ('Thanatophidia,' p. 4), and in connection with this, "the blood of an animal killed by snake-poison is itself poisonous, and, if injected into an animal, rapidly produces its poisonous effects. I have transmitted the venom through a series of three animals with fatal results."

In 1868 I described the difference of the action of cobra and daboia-venom in the case of two horses bitten by these snakes ('Thanatophidia,' p. 79). At pp. 72-73, op. cit., I also pointed out the peculiar action of daboia-venom in causing early convulsions. In some the early convulsions are more marked, and, in others, death is preceded by a more decided state of lethargy. In the bite of the echis the local symptoms are peculiarly severe, so ('Thanatophidia,' p. 631) Dr. Wall gives a more complete exposition of the varying effects, and shows them to be greater than I supposed.

Snake-poison is a narcotic and kills by extinguishing in some way (some molecular change) the source of nerve-energy. It is also a blood-poison and an irritant, if applied to mucous and serous surfaces it causes inflammation; absorption then takes place, and the symptoms of general poisoning are induced. It causes great local disturbance as well as blood-change; for, if the bitten creature survive long enough, the areolar tissue may inflame, suppurate, and slough. If it enter by a large vein, life may be destroyed in a few seconds. It was supposed the more active poisons acted by shock through the nervous system, but the rapidity with which a poison can be distributed through the circulation would account for the most rapid death from snake-bite. The chief effect is on the respiratory apparatus, and death occurs by asphyxia; but the whole voluntary muscular system is also affected, and general paralysis results; whilst the long continuation of cardiac pulsation after apparent death, proves that it is not due to failure of circulation.

The action of snake-poison is discussed at full length in the 'Proceedings of the Royal Society' by Dr. T. L. Brunton and myself (1873-74-75-78). These researches led us to conclude that

the action of the poison is: (1) on the cerebral and spinal centres, especially the medulla, inducing general paralysis, especially of respiration; (2) in some cases, where the poison has been conveyed through a large vein directly to the heart by tetanic arrest of cardiac action, probably owing to action in the cardiac ganglia; (3) by a combination of these causes; (4) by blood-poisoning of a secondary character.

The phenomena vary according to the nature of the snake and the individual peculiarities of the creature injured, the chief difference being observed in viperine as contrasted with colubrine poison. The latter is a nerve-poison of great deadliness; but as a blood-poison it is not of much power. Viperine poison, on the other hand, is a more potent blood-poison. Dr. Wall summarises the difference in the action of daboia (viperine) or cobra (colubrine) poison as follows: "Cobra-poison, when introduced slowly into the circulation, produces gradual general paralysis, but, at the same time, shows a preference for certain nerve centres; paralysis of the tongue, lips, and larynx being very marked symptoms, and respiration is very quickly extinguished after the paralysis shows itself. Death is often attended with convulsions which are clearly due to carbonic acid poisoning. Introduced with a fair amount of rapidity, these symptoms are rapidly developed, the paralysis being preceded by gentle stimulation which causes slight muscular twitchings. Injected in a large quantity into the circulation, the stimulation is so violent as to cause general convulsions of which, however, the respiratory muscles have the chief share, and which are immediately followed by paralysis and death.

"Daboia-poison, though not injected directly into the circulation, causes the most violent convulsions, which are in no way necessarily followed by paralysis and death, but may be, for the time, completely recovered from. They do not depend on carbonic acid poisoning. The paralysis that succeeds is general, and lasts a very considerable time before respiration is extinguished. There is no evidence of the tongue, lips, and larynx being especially paralysed; they probably also suffer in the same degree as other parts. Cobra-poison very quickly destroys the respiratory functions—after slight acceleration the respiration becomes slower, and the excursus is lessened. Daboia-poison at first quickens the respiration very much more than cobra-poison does, and the lessening of the excursus and the slowing of the breathing does

not occur so soon. The respiration generally in daboia-poisoning has a peculiarly irregular character. This function certainly exists longer under the influence of daboia-poison than under that of cobra-poison. The effect of cobra-poison on the pupil is so slight as to be a matter of doubt. Daboia-poison nearly always causes wide dilatation in the earlier stages of the poisoning. Salivation is a constant symptom of cobra-poisoning; it is exceedingly rare in daboia-poisoning.

"The effect of cobra-poison in the blood is not very great. Sanious discharges are rare, albuminuria has not been seen, and recovery is striking and complete when it takes place. In daboia-poisoning, on the other hand, sanious discharges are the rule. Albuminuria is usual should the victim live any time; and, after the nerve symptoms have passed away, the subject has to go through a period of blood-poisoning little, if at all, less dangerous than the primary symptoms; we have, in addition, the greater local mischief caused by daboia-poisoning, and the greater power it has of destroying the coagulation of the blood.

"The physiological properties of daboia-poison undergo great change by its being heated to 100° C. in solution, losing the power of producing primary convulsions, whereas cobra-poison remains unaltered. Daboia-poison kills birds at once in convulsions, whereas, with cobra-poison, unless the poison has been directly injected into the circulation, death occurs only after paralysis.

"Lastly, amphibia recover from an amount of daboia-poison that would be necessarily fatal in the case of cobra-poison."

Without unreservedly accepting Dr. Wall's conclusions, I regard them as an able summary of the action of different kinds of snake-poisons, and they confirm the deadly nature of Indian as compared with European snake-poison.

The local effects of the poison are partial paralysis of the bitten part, pain, infiltration, swelling, inflammation, and ecchymosis round the spot where the poison has been introduced, and sometimes in other and distant parts, and if the animal survive for some hours, infiltration and incipient decomposition of the tissues and hæmorrhagic discharges. The general symptoms are depression, faintness, cold sweats, nausea, vomiting, exhaustion, lethargy, unconsciousness.

Dogs vomit, and are profusely salivated. They present an appearance as if the hair were "staring." As the poisoning pro-

ceeds, paralysis appears in the limbs, commencing generally in the hinder parts with a tendency to creep over the whole body, involving the muscles of deglutition, and loss of co-ordinating power of muscles of locomotion. Albuminuria (especially in viperine-poisoning), hæmorrhagic dischages, relaxation of sphincters; exhaustion, lethargy, and convulsions precede death.

In fowls the appearance is that of great drowsiness. The head falls forwards, rests on the point of the beak, and gradually the fowl, no longer able to support itself, rolls over on its side. There are frequent startings, as if of sudden awaking from the drowsy state, then convulsions and death.

In cases where the quantity of poison injected is large, and it is at the same time very active (as in cobra), the bitten animal small and weak, or if it have entered a vein, death is almost instantaneous, as from shock. In such case, the cardiac ganglia are probably paralysed; at all events, the heart suddenly ceases to beat.

The effects of snake-poison on man are much of the same character, and may be studied in the details of sixty-five cases recorded in the 'Thanatophidia,' which also give an idea of the duration of life. Dr. Wall has summarised them as follows: "The average length of time of the sixty-five cases is 15·17 hours; but the average is raised by the exceptionally long duration of a few cases of viperine poisoning, so that a better estimate of the probable duration of time will be obtained by dividing the period in spaces of one hour each, and determining what percentage of deaths occur in each.

	Percentage.		Percentage.
One hour and under ...	... 10·76	Between 7 and 8 hours	... 4·61
Between 1 and 2 hours	... 12·3	„ 8 „ 9 „	... 3·07
„ 2 „ 3 „	... 13·84	„ 9 „ 10 „	... 7·69
„ 3 „ 4 „	... 7·61	„ 10 „ 12 „	... 4·61
„ 4 „ 5 „	... 1·54	„ 12 „ 24 „	... 9·36
„ 5 „ 6 „	... 1·54	Over 24 hours	... 20·00
„ 6 „ 7 „	... 3·07		

"The most fatal periods appear to be between two and three hours, and more than 25 per cent. of the total deaths take place between one and three hours after the infliction of the bite."

It appears also from the above report, in which in fifty-four cases the exact spot is described, that 94·54 per cent. are wounded in the extremities.

Place of bite.	Percentage of cases.	Place of bite.	Percentage of cases.
Fingers and wrist	31·48	Leg	3·70
Forearm	1·85	Thigh	1·85
Elbow	5·56	Breast	1·85
Shoulder	1·85	Ear	1·85
Feet, toes, ankle	48·15	Perinæum	1·85

This is a matter of some interest, as the hope of success lies in preventing access to the circulation, and in the facility of removing the part injured, and with it the inoculated venom.

The greater proportion of deaths recorded result from the direct effects of the poison; chronic cases, in which death or recovery resulted after protracted periods, are less frequently referred to.

Snake-poisoning in this country is of the viperine character, and though happily the activity of our viper is feeble compared with that of the tropical viperidæ, and except in the case of very weak or young creatures its immediate effects as a nerve-poison are feeble; yet the effects on the blood, and, locally, on the tissues, may be productive of severe and even dangerous symptoms.

The result of my experience is that, so far, no physiological antidote to snake-virus is known, and that when the full effect on the respiratory centres is produced, remedies are of little, if any, avail; albeit, when the poison has entered in smaller quantities, treatment may be of service on general principles.

Viewing the apparent analogy between curare and snake poisoning, death in both being caused by paralysis of the respiratory apparatus, Dr. L. Brunton and I hoped that, by keeping up artificial respiration and supporting the body temperature, we might keep an animal poisoned by snake-virus alive until elimination had taken place; and the result of some experiments justified the anticipation to some extent, for animals were kept alive for many hours, but succumbed at length when the artificial respiration was withdrawn. Mr. V. Richards, who repeated our experiments in India, succeeded in thus keeping an animal alive for days, though it too succumbed finally. In the case of curare, artificial respiration is completely successful, though not so in snake-poisoning. This seems to show that the damage done by the snake-poison is of a more serious and permanent nature than that by curare, as indeed I am inclined to believe, though I do not say that a physiological antidote is impossible; all I assert is, that it has not yet been found. I would encourage efforts to devise a

method of treating snake-poisoning in whatever degree it presents tself, for some means of neutralising the poison or of restoring the damaged nervous system and blood may still be found; at any rate, it is with snake-virus as with other deadly poisons: there must be a quantity, however small, which, though dangerous, is not of necessity fatal, in such cases we may influence the result by treatment, and save life in some. But after long and repeated observation in India, and subsequently in England, I am forced to the conclusion that all the remedies hitherto regarded as antidotes are absolutely without any specific effect on the condition produced by the poison, and that such aid as we can give must depend on preventive and local treatment.

I will now briefly describe the measures to be adopted in the treatment of snake-poisoning, and especially refer to the permanganate of potash as a remedy, and shall read a letter from Dr. M. de Laçerda, of Rio de Janiero, written by that distinguished physician as a commentary on the discussion which took place here last April on Dr. Badaloni's paper.

The first and most important indication is to prevent the poison entering into the circulation; to this all else is subsidiary. The rapidity with which this takes place depends a good deal on the part of the body bitten, and on its vascularity. When the poison enters a vein, if the bite be inflicted by a vigorous snake, the result is generally rapidly fatal. Experiments on animals show that bites inflicted on parts, even where large veins are not implicated, may produce their effects so rapidly that only immediate severance of the part or complete constriction prevents absorption.

It is necessary, therefore, as quickly as possible after a bite has been inflicted to apply a ligature above it, and so tighten it as to completely arrest the circulation. As it happens in 94 per cent. of cases that the bite is inflicted on an extremity, this may frequently be accomplished; but, in parts where no ligature or elastic bandage or cord can be applied, proceed at once to excise the bitten part; this, indeed, should be done in all cases, ligatured or not; then make an incision through the bite, and reflect the skin; expose the tissue wherever this is altered in colour, dissect it out, and be careful to remove every part of it; then apply cautery, some escharotic, or the permanganate solution, taking care that it reaches, as much as possible, in every direction where the poison may have infiltrated. After this is done, the ligature may be

relaxed; for, if the virus be destroyed, the danger of its entering the circulation is past. Should it have already entered, as is only too probable, all that can be done is to give stimulants, keep the patient warm, at rest, and, when the respiration begins to fail, use artificial respiration, and endeavour to keep the patient alive till the poison be eliminated.

In 1869 I gave instructions for the treatment of snake-bite, and, excepting that I would substitute Esmarch's bandage for the ligature, as recommended by Dr. Wall,—that permanganate, 5 per cent. solution, should be applied to the wound when the venom has been carefully dissected out, or injected when it has not been cut out, I have nothing to alter in these suggestions.

Suction, being unlikely to be of much avail, is practically useless to the patient and dangerous to the operator, and should neither be encouraged nor relied on. Insist on the importance of quiet and perfect rest; the temperature should be kept up; the respiration, if it begin to fail, supported by artificial methods. Where the poison has happily been limited to the seat of inoculation, and in cases where no great quantity of virus has been absorbed, we may hope to do good; but where the poison has entered the circulation in larger quantities, and the physiological symptoms are developed, the prognosis is exceedingly unsatisfactory.

As soon as possible after a person is bitten by a snake apply a ligature made of a piece of cord or elastic bandage round the limb or part, at about two or three inches above the bite. Introduce a piece of stick or other lever between the cord and the part, and, by twisting, tighten the ligature to the utmost. After the ligature has been applied, cut the punctures, to the depth of a quarter of an inch, with a penknife or other similar cutting instrument; let the wounds bleed freely, or, better still, excise the punctured part and all the infiltrated areolar tissue subjacent to it. Apply either a hot iron or a live coal to the bottom of these wounds as quickly as possible, or inject into the subcutaneous cellular tissue a solution of permanganate of potash, 5 per cent., or some carbolic or nitric acid. If the bite be where a ligature cannot be applied, with a sharp penknife cut out the bitten part and all the infiltrated cellular tissue to the depth of a quarter or half of an inch; then apply a hot coal or hot iron to the very bottom of the wound, or, better, the permanganate of potash. Give fifteen drops of liquor ammoniæ diluted with an ounce of water immediately, and repeat

it every quarter of an hour for three or four doses, or longer if symptoms of poisoning appear; or give hot brandy, or rum, or whisky, or spirits with equal parts of water, about an ounce of each (for an adult), at the same intervals. Suction of the wounds is not very likely to be beneficial, and as it may be dangerous to the operator it cannot be recommended.

If symptoms of poisoning set in and increase, if the patient become faint or depressed, unconscious, nauseated, or sick, and respiration begin to fail, with symptoms of paralysis of tongue and fauces, apply mustard poultices, or liq. ammoniæ on a cloth, on the stomach and heart; continue the stimulants, and keep the patient warm; but do not shut him up in a hot stifling room, or a small native hut; rather leave him in the fresh air than do this.

Chronic, *i.e.*, milder cases, must be treated on the same and general principles. Do not make the patient walk about; if depressed, rouse him with stimulants, mustard poultices, or ammonia, but let him rest.

If the person be brought, as he or she probably will be, some time after the bite has been inflicted, and symptoms of poisoning are present, the same measures are to be resorted to. They are less likely to be successful, but nothing else can be done.

In many cases the prostration is due to fear; the bite may have been that of a harmless or exhausted snake, and such will rapidly recover if so treated and encouraged. If poisoned, but, as frequently is the case, not fatally, these measures are still the most expedient.

A plain summary or translation of these suggestions might be hung up in public places. The people should be warned against incantations, popular antidotes, and delay in seeking for aid. Every police inspector, of whatever grade, might be taught the application of the simple measures I have described, and should be enjoined to make them known as widely as possible among the police and the people.

There can be little doubt that recoveries from Indian snake-poisoning occur chiefly in cases where the snake has been exhausted or harmless, or has bitten imperfectly, and in a few cases where prompt interference has prevented the entry of the poison into the circulation.

Let me now make some remarks on the remedial value of permanganate of potash. During my investigation of the value of

remedies for snake-poisoning, permanganate of potash was not omitted, and I made the following experiments :—

June 12th, 1869.—First, a fowl was bitten by a cobra in the thigh at 3 p.m.; at 3.1 fifteen drops of liq. potass. permanganate were injected into the spot; dead in seven minutes, 3.35. Second, forty drops of liq. pot. permanganate injected into the external jugular of a dog. This produced no apparent effect on the animal. At 3.48, bitten by a cobra (which had bitten before and was not fresh) in the thigh; the fang punctures were at once washed with the strong solution of permanganate, which was well rubbed in; 3.52, sixty more drops injected into the vein; 3.54, two drachms injected into the bowel, all the symptoms of cobra-poisoning advancing rapidly; 4.12, forty more drops injected into jugular vein; 4.25, dead in thirty-seven minutes.

In 1878 Dr. Brunton and I made the following experiments which confirm the power of the permanganate to neutralise the poison before it has entered the circulation, but show its inefficiency when it follows it.

Experiment 1.—Five milligrammes of poison were dissolved in one cubic centimètre of water, and mixed with one cubic centimètre of liq. potassæ permanganatis, and injected under the skin of a guinea-pig. No symptoms were produced, and the animal remained quite unaffected.

Experiment 2.—Two rabbits of the same litter, each weighing exactly two pounds, were taken. Five centigrammes of cobra-poison were dissolved in one cubic centimètre of liquor potassæ permanganitis, and allowed to stand for about eight minutes. The mixture was then injected under the skin of the flank of one rabbit. No symptoms whatever were produced, and the animal, though kept under observation for some weeks, remained quite unaffected by the poison. Five milligrammes of cobra-poison, dissolved in two cubic centimètres of water, were injected into the other rabbit at the same time. During the injection a little of the poison was lost, so that the animal did not receive the full dose, yet it died in thirty minutes.

Experiment 3.—April 4th, 1878. Guinea-pig, weighing $1\frac{1}{2}$ lbs.: injected four centigrammes of cobra-poison into leg. 4.1 p.m., ligature applied immediately; permanganate of potash applied immediately. 4.5 p.m., twitching; 4.10 p.m., dying; 4.13 p.m., convulsion; 4.14 p.m., dead.

Experiment 4.—April 4th, 1878. Guinea-pig weighing 1 lb. 3.45′ 20″ p.m. Injected ¾ grain (= 4 centigrammes) of cobra poison, under skin of leg. A ligature was applied round the leg in one minute, and in five minutes permanganate of potash was rubbed into an incision made over the site of injection. 3.52 p.m. ligature cut; 3·53, twitching violently, leg paralysed; 3.57 p.m., dying; 3.58 p.m., dead—less than thirteen minutes.

Dr. Wall, who has carefully investigated the subject, makes the following pertinent remarks ('Indian Snake-Poisons,' p. 129): "As it was found that potassium permanganate does destroy the poison, steps were taken to see if it would be of any practical use in the treatment of animals suffering from snake-bite. It was found, by experiment, that a considerable quantity of potassium permanganate, dissolved in a weak saline solution, could be injected into the circulation of an animal without producing any immediate effect (I found the same with a strong solution). A dog, suffering from cobra-poisoning, had a cannula placed in its saphena vein; a solution of potash was injected, but though a large quantity was cautiously and gradually introduced into the circulation, and though at the same time life was prolonged by artificial respiration, in no way was the least benefit to be perceived from the remedy. The reason is obvious. It is quite true that potassium permanganate destroys the active agent of cobra-poison by oxidising it; but, when introduced into the blood, it of course commences oxidising indifferently all the organic matter with which it comes in contact; but it has no power of selecting one organic substance for oxidation rather than another. The oxidising power of the permanganate is, therefore, exerted on the constituents of the blood generally, instead of being reserved for the cobra-poison in it alone; so, if cobra-poison is dissolved in an organic solution, and the permanganate is added before injection, the poison suffers little, if any, diminution in strength, for oxidation has taken place chiefly at the expense of the other organic matter. Thus, it would be necessary to destroy all the constituents of the blood by oxidation before all the poison in it could be destroyed too. If a substance should be found having the power of oxidation, with a special affinity of exercising it on snake-poison, the problem of the treatment of snake-bite would be solved, but potassium permanganate has not the special power."

It has been pointed out that there are other substances which

greatly diminish or destroy the action of snake-poison when mixed with it out of the body. Of all such agents, permanganate of potash is probably the best; still it seems to be of little practical use.

Wall further remarks: "It may be asked why, if metallic salts, tannic acid, hydrate of potash, and permanganate of potash destroy snake-poison, should not these substances be used in preference to excision? The reply is obvious. If we could know the exact position of the poison, and if there were only one deposit, we might probably succeed in destroying it by injection. But to remove the poison deposited by the bite of a snake requires a more intelligent observation, guided by eyesight and judgment, but an injection of a chemical agent must be, to a great extent, made by guesswork, and the solution, instead of following the poison, takes the line of least resistance in the tissues, often leading it far from the poison."

In a pamphlet ('Experiments on Permanganate of Potash and its Use in Snake-poisoning') dated 1882, Richards says: "A solution of 5 per cent. of permanganate of potash is able to neutralise the poison;" and recommends that this should be injected into the bitten part after a ligature has been applied; it is less likely to cause sloughing of the tissues than any other agent which could neutralise the venom. In his letter dated July 22nd, 1882, he says: "It is, in my experience, the best local application we possess. It is not a physiological antidote, but is a chemical one, and is utterly powerless to effect any influence on the lethal action of snake-poisoning." (He means the constitutional action.) He is of opinion "that whenever opportunity offers, the injection of permanganate of potash should be resorted to, assuming that a ligature has been efficiently applied (where it can be applied at all) within five minutes from the bite. In the average run of cases, the permanganate will certainly destroy the poison lying beyond the ligatured part," if it come in contact with it; but, as Wall pointed out, the difficulty of insuring its contact with the poison is so great as to render it practically unreliable. I agree with Richards that, so far as it goes, it is a good local application, and as such it ought to be used, or, in its absence, tannic acid or liquor potassæ might be resorted to with the same object; but as a constitutional remedy, as a physiological antidote, it is powerless, like all others that have been tried and failed to do good. Dr. de

Laçerda himself, although he attributes the highest value to it as a chemical antidote, both as a powerful oxidising agent, and by the action of the potash says : "As to the idea of finding a physiological antidote for snake-poisoning, I entirely agree with you that it is a Utopia."

Dr. de Laçerda's letter is most interesting and instructive. He says that he has been led to write it by reading the report of a discussion at this Society on April 16th, 1883. With some preliminary observations, he continues : "I beg leave to protest against an opinion attributed to me by some of your colleagues, but which I have never sustained. I refer to the opinion that attributes to bacteria the effects of the poison. I have weighty reasons for considering such an hypothesis as entirely false. I recognised, indeed, by means of repeated and careful observations, that the venom contains micrococci in great numbers, and I made a communication on this subject some three years ago to the Academy of Sciences of Paris. These corpuscles, however, exist in the venom in an accidental manner, as also in the human saliva, and play no important part in the effects of the poison. This last acts as a chemical agent producing a rapid alteration in the molecular composition of the albumina which enters into the formation of almost all animal tissues. On the blood, given certain conditions, its effects are very rapid, almost instantaneous; the same happens with the nervous and other elements whose functions are disturbed immediately that the venom comes in contact with them. Now, such immediate action can never be attributed to bacteria. You see, therefore, that this unsustainable theory cannot be invoked in endeavouring to explain the neutralising effects of permanganate of potash.

"Having made this protest, I will proceed to indicate the points on which I cannot agree with certain of your colleagues and with yourself, in regard to certain questions relative to snake-poisons. In the first place, I do not consider it exact to say that this venom, inoculated in the tissues of an animal, invades rapidly the organism.* On the contrary, numerous experiments made during

* Dr. de Laçerda may possibly have operated only with the crotaline snakes, and if so, he has not had the opportunity of witnessing the different action excited by colubrine poison. In my experience, frequent experiments showed that direct general contamination follows the bite even when no large vein has been wounded.

three years have proved to me that the venom is slowly absorbed by fractions, acting first locally on the tissues in which it has been inoculated, the elements of which imbibe the venom little by little and fix it. This destructive local action is at times, of itself alone, sufficient to produce, a short time after the inoculation has been effected, general disorders of a reflex character which are not unfrequently confounded with the disorders due to the generalisation of the venom, which require a greater time for their manifestation.

"In those cases in which the effects of a generalisation of the venom were produced within a short time after the inoculation, some vessel had been opened by the inoculating instrument, giving the venom free entrance into the circulation.

"Another point in regard to which I cannot agree with some of your colleagues is that there are species of snakes whose venom acts principally upon the blood, while others act specially upon the nervous centres. For the Brazilian species, at least, I can affirm that this opinion is erroneous, and it does not appear to me probable that the species inhabiting India furnish an exception to the rule of unity of action of the venom that I have verified for Brazil. With the venom from a single species, I may even say, of a single individual, an animal may be made to succumb by causing profound perturbations in the central nervous system, without apparent alteration in the blood; or *vice versâ*, with slightly pronounced disorders of the nerve-centres and profound alteration of the blood. Everything depends on the conditions in which the experiment is made.

"Passing now to the essential point of the discussion that took place in the Medical Society, I will give in a few words how I comprehend and how I judge that the efficacious effect of permanganate of potash should be comprehended. You yourself, by experiments made in 1869, recognised that permanganate of potash mixed with the venom took from it its noxious properties. Certain conditions of the experiments led you, however, to deny the efficacy of this chemical agent in the cases in which the venom had been inoculated in the tissues. As you know, however, I have demonstrated by numerous experiments and innumerable clinical facts that the neutralisation takes place even in the midst of the tissues, which makes this substance a chemical antidote of great value.

"The permanganate of potash acts upon the venom, destroying

it in two ways: first, as a powerful oxidising agent; second, by the potash that forms the base of the salt. Passing a current of nascent oxygen through a concentrated solution of the venom, this loses entirely its noxious properties. This experiment, which I have repeated many times, gave me always the same result. Let us suppose now that an individual is bitten. If injections are made in the place of the bite from five to ten minutes after the inoculation of the venom, this is promptly neutralised *in situ*, and the individual runs no further danger. A great number of facts like this have been observed in Brazil. If aid is given late, hours after the bite, when the tumefaction of the wounded part is very pronounced, and the phenomena that indicate the entrance of the venom into the circulation have already declared themselves, injections repeated in various parts of the wounded member, parting from the wounds made by the fangs of the reptile, still give very good results. Nor is it difficult to explain the good results in this case. The venom, as I have said, acts first locally, and only enters the general circulation after the lapse of a certain time, and by portions. The permanganate of potash, meeting in the tissues with the venom which is little by little diffusing itself, neutralises it in the various points where it has been diffused and thus stops the source of supply. The entrance of new and successive portions of the venom into the general circulation being thus impeded, the organism takes charge of the elimination of what has already been introduced, and which was insufficient to compromise the life of the individual.

"We will now suppose a case of greater gravity, in which a vein is wounded, and there is a rapid penetration of a large quantity of venom into the circulation. Even here an injection of a solution of one hundredth of permanganate of potash may be practised in the vein, since we have recognised that no bad effects are produced in dogs by a dose of from two to three cubic centimètres. In this case the good results are problematic, in view of the rapid diffusion of the venom in the organism; but then, if permanganate of potash does no good, no other substance could be useful. These cases, fortunately uncommon, are beyond all help.

"As to the idea of finding a physiological antidote for snake-poison, I entirely agree with you that it is a Utopia."

After careful consideration, fully admitting that in permanganate of potash we have an agent which can chemically neutralise snake-

poison (as, indeed, was shown by Dr. Brunton and myself in 1878), I do not see that more has been done than to draw attention to a local remedy already known as a chemical antidote, the value of which depends on its efficient application to the contaminated part, which, as Dr. Wall has pointed out, is too uncertain to be reliable. We are still then as far off an antidote as ever; and the remarks made by me in 1868 are as applicable now as they were then. They were as follows:

"To conceive of an antidote, as that term is usually understood, we must imagine a substance so subtle as to follow, overtake, and neutralise the venom in the blood, and that shall have the power of counteracting or neutralising the poisonous and deadly influence it has exerted on the vital force. Such a substance has still to be found, nor does our present experience of drugs give hopeful anticipation that we shall find it. But I repeat that where the poisonous effects are produced in a minor degree, or where secondary consequences are to be dealt with, we may do much to aid the natural powers in bringing about recovery."

In conclusion, fully acknowledging the value of recent researches, I would express a hope that the subject may receive further vigorous investigation, and that efforts may be prosecuted, especially in the direction of search for some method of increasing elimination of the poison, of ascertaining the exact nature of the lesion of the nervous system and blood, and how far they are removable; that, as to local measures, with the view of preventing entry of the virus into the circulation, and of neutralising it *in situ*, improvements on present methods may be sought for. As to advance in the investigation of the physiological and chemical aspects of the question, much may still be done, as also in respect of the chemistry and microscopical character of the virus itself, and the blood and tissues of the poisoned. But these inquiries, of such importance to the human race, can, I fear, make but little progress whilst the present restriction on all physiological research continues to be maintained.

A review of the subject of snake-poisoning would be quite incomplete without acknowledgment of the valuable labours of such Indian observers as Dr. Short, Dr. Nicholson, and Dr. Stradling; Drs. Stuart, Ewart, Richards, and Wall, who have added materially to our knowledge, as also have Dr. Halford, in Australia, Dr. de Laçerda, in Rio de Janiero, Dr. Lauder Brunton, F.R.S., in

London, and Drs. Weir Mitchell and Reichardt, in America, who are now engaged in the most important and much-needed investigations into the chemistry of the poison, and the condition of the blood and tissues of the poisoned.

I am indebted to the Director-General of the Army Medical Department for the following interesting case, which will appear in the next 'Army Medical Department Report.'

Colonel M., while serving in Zululand, near the lower Tugela river, was bitten in the leg just below the knee, and, after the lapse of a few seconds, became sensible of extreme shock, and at once felt certain that a snake had bitten him. He rode back to camp, and, when first seen, ten minutes after the infliction of the injury, was in the following condition:—There was pain, ecchymosis, swelling, and partial paralysis of the bitten part. He was so exhausted that he had nearly fallen off his horse. The forehead and hands were bathed in cold perspiration; the extremities were cold and pale; there was great nervous depression, with sense of impending death; respiration was hurried. Quickly following this, bilious vomiting set in, with loss of co-ordinating power; numbness of extremities and lips, and dragging sensation of the face; intense pain in neck, troublesome cough, with thick viscid expectoration. The pulse was, from the first, weak and rapid, rising from 120 to 150; restlessness and anxiety became very distressing. Vomiting ceased at 9 p.m., but soon afterwards still graver symptoms developed; vision rapidly failed; the eyelids drooped, the speech became thick and nasal; there was paralysis of the tongue and soft palate, with dysphagia. There were also clonic convulsions of the upper extremities, and of the muscles of the chest; the breathing was stertorous with low muttering. At 12.45 he spoke for the last time, and then lapsed into a semi-comatose condition, and died at 2 a.m., ten hours after the bite.

Tight compression was made above the seat of injury, between the bite and the heart; the wound was enlarged, and an attempt was made to remove all the blood and poison from it. Nitrate of silver and ammonia were applied freely to the surface of the wound. Ammonia and diffusible stimulants were administered by mouth. To relieve the distressing vomiting, sinapisms were applied to region of stomach, and brandy with soda-water given; the restlessness was combated with hypodermic injections of morphia (half a grain for a dose); the morphia gave great relief, which, however, was

only transitory. Hot-water bottles were applied to the feet, and stimulants were given with an unsparing hand, but were not always retained. Ammonia was also injected subcutaneously.

Post-mortem examination made nine hours after death: Body well nourished. Cadaveric rigidity well marked. Hypostatic congestion. Great discolouration of scrotum and finger-nails. Situation of bite on left leg at upper and inner side of calf, about three inches below internal condyle of femur, and immediately over internal saphena vein. Appearance that of a small pin-puncture; lower part of leg rather swollen. On removing the skin from the region of the wound there was found great sero-sanguineous extravasation into the surrounding tissues, and the muscles were soft and infiltrated with blood; the internal saphena vein was punctured. The venous system on the left side much congested. The glands in left groin, in long axis of limb, enormously enlarged and congested. Glands in right groin normal; pericardium normal. Heart: right cavities full of fluid blood; left cavities empty; valves healthy; no clots. Lungs normal. Liver congested, and slightly enlarged. Gall-bladder fully distended. Spleen somewhat enlarged, otherwise normal. Stomach slightly congested, rugæ well marked; contents, a small quantity of glairy mucus. Kidneys normal. Omentum contained much adipose tissue. Intestines normal. Bladder normal, contained a small quantity of urine. Brain somewhat congested, otherwise normal. Blood in a fluid state.

The snake which inflicted the fatal wound was not seen; in all probability Colonel M. trod on one asleep, which then struck at him. The systemic shock was at once apparent after receipt of the injury, which is accounted for by the puncture of the internal saphena vein, and the introduction of the poison direct into the general circulation. The clothing traversed by the fang of the snake was, first, cloth garter; second, khakee riding-breeches; third, drawers of light material. From the high situation of the puncture the opinion of competent judges was that the snake which inflicted the wound was a "black mamba," one of the large African vipers, species not determined.

The following were exhibited:
Crania of innocent snakes.
Crania of venomous colubrine snakes.

Crania of viperine snakes.

Dissections of muscular apparatus for erecting fangs.

Dissections of poison gland and duct.

Models of fangs.

Drawings of the above.

Also coloured figures of venomous snakes of India.

The following snakes were exhibited:

Naja tripudians, bungarus ceruleus, bungarus fasciatus, callophis, elaps corallinus, daboia Russellii, echis carinata, vipera rhinoceros, vipera cerastes, lachesis mutus, crotalus durissus, pelias berus, and several non-venomous snakes.

Dr. Waring asked whether Sir Joseph Fayrer had himself known any serious effect to have followed the sucking of a poisoned wound.

Dr. Thorowgood related a case which had come under his notice, in which death had resulted from asphyxia. Some alarming symptoms produced in the person of Frank Buckland, the naturalist, were also mentioned as having resulted from the use of a penknife to clean the nails, which penknife had previously been employed to lay open a rat stung to death by the bite of a cobra. The symptoms were relieved by sal volatile.

Dr. Sansom had made experiments long ago which proved that bacteria and fungi could live in strong solutions of permanganate of potash; these would show that the snake-venom could hardly be of a living nature, and was probably alkaloidal.

Mr. Walter Pye spoke of the differences between viperine and colubrine poison. He suggested that the poison in the one might be alkaloidal and in the other of a living nature.

Dr. Norman Chevers thought it possible that the juices of the snake might be antidotal to its poison, since the bite of a snake is innocuous on itself or on its own species. He alluded to the curious sero-sanguinolent exudation met with in most of the cases described, including the African one narrated by the President. The infiltration had the appearance of thin red-currant jelly.

Dr. Mair had seen a fatal case of the very short duration of forty-five minutes. The valuelessness of permanganate of potash or ammonia, when once the poison was absorbed, was also believed in by him. He understood that the cobra poison was not harmful when taken by the stomach. He spoke of the alleged immunity of the mungoose.

Dr. Paul related a severe case of poisoning which terminated in recovery.

The President, in reply, said that he most certainly believed the poison could be absorbed by a mucous membrane; he had seen the effects of such absorption over and over again in dogs, birds, frogs, &c. He had mentioned in his paper many facts which he had not the time to read. The blood of a person poisoned from a colubrine snake generally clotted, whilst that from a viperine poisoning remained fluid. We had very much to learn in all directions of this most interesting subject. He did not believe in the alleged immunity of the mungoose, for the reason that he had seen this active animal die after the infliction of a fair bite from a cobra.

February 11*th*, 1884.

ON TREATMENT OF IRREDUCIBLE HERNIA, AND AN IMPROVED METHOD OF ADAPTING A TRUSS IN ALL FORMS OF HERNIA.

By Thomas Bryant, F.R.C.S.

I would draw your attention this evening to the subject of irreducible hernia, and more particularly with reference to its treatment, since I have reason to think that surgeons, as a body, are too ready to regard this condition of hernia as irremediable, except by an operation, as well as too prone to resign its palliative treatment into the hands of mechanists.

I need hardly add that neither the opinion given, nor practice mentioned, meets with my support; for I am convinced by experience, and I trust I can show you, that many cases of supposed irreducible hernia can be made reducible by treatment, and that the majority of cases of truly irreducible hernia, including umbilical, femoral, and inguinal, can be fitted with a truss which supplies the three desiderata of such an instrument and gives protection to the hernia, guards against its increase, and is comfortable to the wearer, and, what is more, tends greatly to make the hernia reducible.

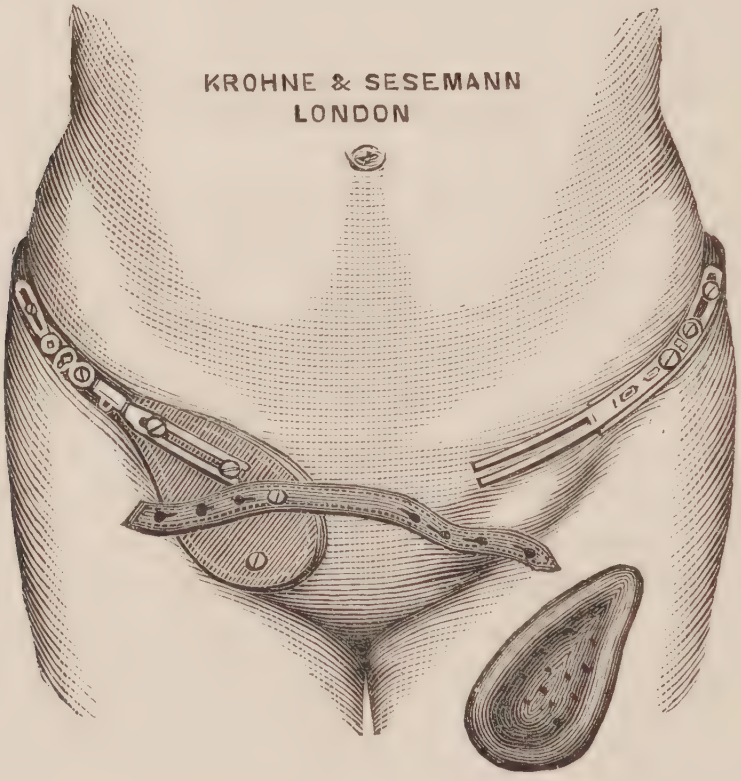
And, first of all, with respect to the reduction of a supposed irreducible hernia, under what circumstances is such a result possible, and, when possible, by what means is its reduction to be brought about?

With reference to the possibility of reduction, I take it, we may fairly assume that every case of hernia is capable of reduction if no adhesion exist between the sac and its contents, and if the contents themselves be not matted together by old inflammatory effusions; or in other words, that all herniæ can be rendered reducible, so long as they have not been the subject of some antecedent strangulation, incarceration, or obstruction.

With these exceptions, I think I am right in saying that all herniæ recently descended, and many that have been down weeks, or even months, can be rendered reducible by treatment.

The means adopted to bring about this result are not complicated,

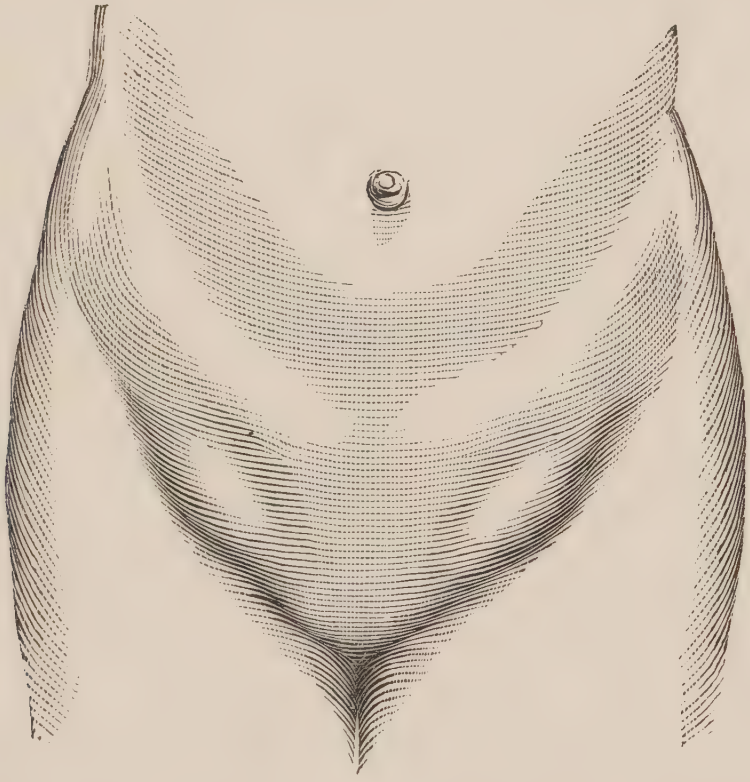
They are the recumbent position, associated with limited diet, milk-diet being the best, and the administration of small and repeated doses of saline purgatives, together with the local application of cold to the hernial protrusion by means of an ice-bag, or one of Leiter's metallic coils. By these means the bowels are emptied of their contents, and the blood-vessels in their walls are rendered less turgid. The fat that may be in the omentum is likewise absorbed. Altogether, the hernial contents are diminished. This treatment, to be effectual, should be persisted in for days, and in some cases for weeks, and under it a good result may, with some confidence, be anticipated.



I have had many cases in which success followed the rigid adoption of these means, and some failures; but where the latter occurred I have always felt it was from a want of confidence in the result, and, as a consequence, a carelessness or half-heartedness in the application of the practice. Success has, however, often come to me by persistency and attention, soon after despair of obtaining it has almost induced me to abandon the attempt. I have, by the steady persistency of these means, continued from a few days up to nine weeks, brought about the reduction of herniæ which had been down

from five up to twelve weeks; and I think it will be admitted that, where success followed the practice, it was worth the trouble; for it is to be remembered that it was brought about by means that in no way endangered life, and at the worst were only wearisome.

The point, however, to which I want more particularly to draw your attention is the second, which refers to the truss which is required for an irreducible hernia; for I feel confident that I shall obtain the support of all my medical and surgical brethren, when I say that if our present trusses for reducible hernia are not all that we should wish, we have not at present a single truss for an irreducible hernia which can be praised, or even passed as efficient.



How far the one I am now about to introduce to your notice will meet with your support I know not; but I may say that I have used it in a sufficient number of cases—two dozen, at least—to test its value, and that in all it has proved a success; indeed, I am induced to think, from the principle upon which the truss is formed being sound, and at the same time simple, that it is the only one upon which an efficient truss for an irreducible hernia can be expected to succeed; and I believe it to be of equal benefit for a reducible.

The practice is like in kind to that which has been laid down for rendering an irreducible hernia reducible, but it differs in degree; for the object I have in view in these truly irreducible herniæ is to render them as small as it is possible to get them by rest, diet, and the local application of cold.

Having attained this point, I take a plaster-of-Paris "mould" of the hernia, and the parts around its neck, and from this mould make a "cast." Upon the "cast" I have a metal plate of tin or copper moulded, and covered with wash-leather, and when this has been efficiently made, I have a pad which accurately fits the hernia —whether umbilical, femoral, or inguinal—and this pad has only to be fixed to a spring or belt to make the instrument complete.

The pad, being a mould of the hernia, forms a most efficient protection to it; and, touching as it does every point of the protrusion, it fairly guarantees no increase in its size; for a like reason, the truss keeps its place, and is really comfortable to the wearer, since it presses equally.

To illustrate these points more clearly, I have induced Mr. Krohne, of 8, Duke Street, Manchester Square, to have two of the casts of the groins of two of my patients with irreducible hernia grafted on to a cast of the pelvis, &c., of a female figure (*vide* Plate), on the right side of which the hollow part made in the way described is fitted; on the left it has been removed from the spring, and so placed that you can see and examine it. In both the fit is perfect. Indeed, I could give you abundant testimony of the value of these trusses, but one will suffice. It was sent me in a letter, dated September 6th, 1883, from a medical friend who has had a personal experience of the instrument.

"Mrs. A— has had an irreducible umbilical hernia for many years, about the size of a closed hand. She has worn umbilical trusses, belts, and air-pads, of all kinds, but none of them gave her the necessary support, and the hernia constantly slipped out under the pad, causing much pain, and preventing her from walking. Acting on your suggestion, I kept her in bed for a few days, and when the hernia had gone back as much as it would, I took a mould of the tumour, and sent it to Krohne, who made from it a metal case, lined with wash-leather, which fits the hernia. This is attached to an ordinary umbilical belt. Mrs. A— has never before been so comfortable, is able to walk well, and she says the hernia is smaller.

"In another case," writes my medical friend, "the benefit was even greater. Mrs. F—, a large flabby woman, had also an umbilical hernia. It was the largest I had ever seen, and could not be covered by both hands spread over it. The pressure of the belt had caused abrasion

between the over-lapping hernia and the skin. She was confined to her room, and had not been downstairs for weeks. I kept her in bed, both to diminish the bulk of the tumour and to allow the skin to heal, and then took a mould in plaster of Paris, and sent it to Krohne's, as in the other case, and a moulded metal pad was returned to her. I saw her a few days ago walking. She says she is quite comfortable, and that the tumour is smaller.

"There is a little knack in taking this cast. I snip off all the hairs likely to be in the way, so that they should not adhere to the plaster, and oil the tumour. I send to a dentist for the plaster, as the commoner kind cracks and makes a mess.

"Yours truly,
"JOHN WILTON."

"Sutton, Surrey."

The late Mr. Millikin, of St. Thomas's Street, was the first maker who carried out these suggestions for me about twenty years ago, and it was for a man who had a hernia with an undescended testicle, in which protection was required as much as support. The result was so satisfactory that I have followed the practice ever since. As a mode of adapting a truss for a reducible hernia, I have good reason to believe that a like practice is of equal service. I have employed it in a dozen cases with marked success, and I have done so in patients who had found it impossible to be fitted otherwise with comfort. And can this be a matter of surprise when we know that every groin has its own special shape, and that a pad which is well adapted to one is not suitable to another?

I maintain, therefore, that in all cases of irreducible hernia, this method of adapting a truss should be followed where it can be, and that, in all cases of reducible umbilical, femoral, and inguinal hernia, the only perfect truss is one in which the pad has been moulded on a cast of the seat of hernia of the individual sufferer.

For the public generally, this perfect instrument cannot probably be made on account of expense, but for those who want comfort with efficiency it is strongly to be advised.

In London and the large towns there will never be any difficulty in obtaining a cast of the affected part. In country practice, our medical friends must learn to make the mould, if not the cast, themselves, when any good instrument maker can readily make the metal moulded truss-pad, and fix it to the spring which is to hold the pad *in sitû*.

When plaster is not at hand for the mould, a piece of gutta percha, dipped into hot water and softened, may be used for the purpose; and it is possible that this method may prove to be the

most useful, since a hard mould is secured by this method which is not likely to break.

I need hardly add that the practice advocated in these papers is applicable to other cases than hernia. I have already used it in examples of spina bifida, and for the protection of outgrowths; but I believe its general applicability to surgical cases will be at once acknowledged.

Mr. Pearce Gould acknowledged that many herniæ were difficult and troublesome to manage, and here a pad made from an exact mould was most comfortable and efficient. He suggested that the mould might be so made that the pad should take the place of the weakened pillars at the neck of the hernia.

Mr. Francis Mason asked whether complete success attended the use of the truss.

Sir Joseph Fayrer thought the truss would prove of great service. He felt sure that such additional means for treating large old incarcerated herniæ were very welcome. He had often employed, and still recommended, an incision in the tense pillar of the neck of such herniæ so as to allow of their complete reduction; and some form of plug to the inguinal canal might be of much value.

Mr. Bryant, in reply, said he contrived first to get the hernia as small as possible before taking the mould. The employment of this principle had hitherto not been done systematically.

NOTES ON LUPUS.

By Jonathan Hutchinson, F.R.S.

My design in the present paper is to somewhat enlarge the meaning given to the term lupus, and to show that we ought to include under it a group of diseases some of which differ considerably in external features from others. Hitherto we have been content for the most part to recognise only lupus vulgaris and lupus erythematosus, and grave doubts have been expressed by some good authorities as to the propriety of continuing to associate them under one name. My argument will be that they ought to be so associated, that they possess natural and close affinities, and that certain other more rare affections ought also to be placed in their company. In order that we may be in a position to judge rightly on this matter and to avoid mistakes in classification it is desirable to preface a few words as to our definition of the word lupus and our conception of the pathological processes to which it is applicable. Originally

it included the malignant affection which we now know as rodent ulcer, and the term lupus exedens was perhaps generally applied to this disease. It is not very long ago that an excellent woodcut showing the ravages of rodent cancer did duty in Drewitt's 'Manual of Surgery,' as a portrait of lupus exedens. It is now well recognised that lupus is a disease of skin and mucous membrane only and that it never tends to eat deeply. The term lupus exedens has passed out of use, and I believe no one now attempts to make any division of lupus vulgaris into two groups in reference to the depth and extent of the ulceration.

We know, however, that the lupus process shows very different tendencies in different cases in this respect and that it is not uncommon to observe lupus growth, whether in single or multiple patches, persist for many years and spread extensively without any tendency whatever to ulcerate. The histology of lupus vulgaris as an infective cell-growth in the corium has long been established, and recent observations have shown that there is commonly in association with it a bacillus resembling that of tubercle. I shall not in what I have to say this evening lay any great stress upon the nature of the final products which attend the process, since it is probable in the highest degree, so far as I can understand the matter, that they are products and not causes. That they are of great importance as characteristic of the fully-established lupus process no one can doubt, but to assert that they are present in the very onset of the disease and in all forms and stages of it is a very different matter. The definition of lupus should, I think, be a clinical and not a histological one, and should be based upon the recognition of causes and modes of extension rather than of results. Whoever investigates the beginnings of lupus will I think be forced to conclude that it starts as a common inflammation and gets its peculiarities from the diathesis of the patient. Its exciting causes are in many instances very definitely those which originate in inflammation. Exposure to cold, such as causes chilblains, or exposure to sun, the irritation of prolonged catarrh, the stings of insects or slight wounds or scratches are among the influences which sometimes definitely excite it. In no cases do we recognise any probability that it is the result of contagion. Its earliest stage is always an area of inflammatory action which slowly takes on the peculiarities of the disease. When the lupus process is established we have an infective new growth which spreads by continuity with

the adjacent tissues, and which, although it never affects the lymphatic glands or the internal organs, seems capable of travelling considerable distances away from the original patch. I do not know how otherwise to interpret the multiplicity which lupus so frequently assumes than by supposing that it is the consequence of infection through the lymphatic spaces of the skin. This multiplicity is almost always observed in proximity to the original patch, at any rate in the first instance, for we must admit that in some cases later on the diffusion becomes general and that secondary developments may take place at the greatest possible distances from the first.

I by no means wish to deny that the multiplicity may be in some cases primary. In lupus erythematosus it is probably usually so, but in lupus vulgaris these cases of primary and independent multiplicity, or what might be called constitutional multiplicity and tendency to symmetry, are very rare. So inveterate is the infective power of the lupus growth that it is a very rare event to see the disease die out of itself. When it does so it is almost always after a duration of many years and in connection with the advent of something like senility in the individual.

In this infected region it far exceeds the processes of inflammation which we recognise as strumous, and which although themselves very chronic almost always tend in the end to spontaneous cessation.

Side by side with its infecting vigour we observe in the lupus growth a distinct tendency to retrogressive change. Its formations after a certain duration, often very considerable, wither and atrophy, leaving the part which they had affected in the condition of scar.

The production of a scar may, I suppose, be taken as an invariable result of any process which can claim the name of lupus, but it is to be clearly understood that the character of the scar will depend upon the extent of the previous growth or infiltration. Where the lupus growth has been inconsiderable a correspondingly slightly marked scar will be left, and in many cases it may be quite impossible to the unaided eye, or even without making microscopic sections, to prove that the integrity of the skin has been in any way damaged. Any hesitation that is felt in accepting this assertion may easily be removed by the examination of the condition left by lupus in different patches on the same patient. It will often be seen that whilst the scar is very conspicuous on one patch it is impossible to

detect it on another. This, however, is only a difficulty in appreciation, and we unreservedly accept it as part of the definition of lupus, that to some extent it disorganises the part which it affects and leaves a scar when it departs. *A slowly creeping and infective form of inflammatory new growth in the skin and mucous membranes which invariably leaves a scar,* such I think might be a fair working definition of lupus. It would include scarcely anything else excepting rodent ulcer and certain syphilitic affections. Respecting these latter we may admit at once that it would be quite impossible to frame any definition which should exclude them, for the simple reason that they are exact imitations of the lupus process in all its varieties and differ from it only in that they acknowledge a syphilitic cause. The rodent ulcer may be distinguished by its tendency to ulcerate deeply, to affect other parts than the skin, and in most cases to show but little tendency to the formation of scar at all.

We will now discuss the features of resemblance and difference between lupus erythematosus and lupus vulgaris. In well-marked cases of these two diseases the differences are very marked indeed. By far the most important of them, since it probably implies essential difference in the order of origin, is the almost invariable symmetry of the one and the almost invariable non-symmetry of the other. We know exactly where to expect the patches of lupus erythematosus. It will begin on the nose and spread in bats' wing areas on the cheeks, and next, without continuity, it will affect both ears. If the patient be young and the disease severe we may next observe it on the two hands. In some instances it begins in the first instance with symmetrical patches on the two cheeks. This definite tendency to symmetry without continuity of extension proves that the disease depends upon inborn peculiarity of structure involving susceptibility to very slight exciting causes. Lupus vulgaris, on the other hand, although when it begins on the nose it may spread equally on both sides of the face, does so only by direct continuity, and nothing is more common than to see it begin as a single patch on one side of the face or limb and although it may become multiple it may remain conspicuously not symmetrical throughout. This fact would appear to imply that the disease depends comparatively little upon such peculiarities of structure as are bilateral, and that it is difficult to originate, although when once started remarkably persistent and infective. It would suggest

an alliance with cancer in the case of common lupus, and with such diseases as chilblains and psoriasis in the case of lupus erythematosus. But although we may thus draw strong lines of distinction between the two diseases in their well-characterised forms, it is necessary in the next breath to admit that there are a host of ill-characterised forms which constitute connecting links between them. I have omitted to state, and perhaps it was almost unnecessary to do so, that the erythematous lupus is sometimes little more than an erythema, a mere congestion with a little roughness of surface and an abruptly margined border, but with no evidence to the naked eye, or scarcely any to the microscope, of new growth or cellular infiltration. In common lupus, on the contrary, the yellow apple jelly-like layer of new growth may be a third of an inch in thickness and there may be no congestion whatever.

There is, however, another feature of erythematous lupus which is held to be very characteristic. I allude to the formation of little discs depressed in the centre and elevated at the edge. Now, these discs are often attended by so much of thickening that it is absolutely impossible to draw any line of distinction between them and the growths of common lupus. Again, we meet with cases in which erythematous lupus is by no means accurately symmetrical, but is attended by the formation of patches placed irregularly, and apparently not the result of independent proclivity of tissue, but of infection from the parent patch. In a mixed case of this kind which I have at present under observation the disease is unquestionably erythematous on the nose and cheeks, and the two ears are also characteristically affected, but discs and patches are scattered irregularly over the face and lips. A few occur on the body and a single one on one thigh. The soft palate is affected and is in a condition which could not be distinguished from common lupus, and there is, to crown the complexity, that the present disc, which is still present and not bigger than a sixpence, began on the chin and had existed for two or three years before the others showed themselves.

I could easily instance a host of cases in which the features of lupus erythematosus and lupus vulgaris were inextricably mixed in the same patient.

Whatever is true of lupus erythematosus in this respect is true also of lupus sebaceus, which is a variety of it. Asserting then that whilst it is exceedingly easy to discriminate between lupus erythematosus and common lupus in their well-marked forms and that it

is very essential they should be distinguished one from the other, I yet find good reason for placing them in the same family group and for believing that they depend upon similar though not the same causal influences. I must hasten to the discussion of other and less generally recognised forms.

Permit me first to say a few words as to the peculiar features which common lupus assumes when it occurs on the extremities. I never in my life saw a quiet patch of non-ulcerated lupoid growth on either the hands or the feet. Nor is it common on these situations to be able to recognise the apple jelly-like growth at all. The disease is here always a mere inflammation, very chronic in its processes, attended with much swelling, and with the formation of papillary granulation masses. The cellular tissue is always much more involved than in other parts, and the resulting cicatrix is much deeper and may even cause considerable deformity. What I am describing is wholly distinct from the erythematous lupus of the hands and is never, like it, symmetrical. The peculiarities which I have mentioned have been recognised by several authors.

Under the name of disseminating follicular lupus simulating acne, Dr. Tilbury Fox ably described in a clinical lecture,* the disease which I had called acne lupus. He recognised under that term precisely the same features which I do. At the time of Dr. Fox's paper I had written nothing respecting the disease, but I had long well recognised it and had exhibited in the Annual Museum of the British Medical Association, as acne lupus, the portraits which I have shown this evening. As Dr. Fox points out, the terms acneform lupus and lupus acneique, in use long previously in Paris, had been applied to the sebaceous forms of erythematous lupus, and were, therefore, not available for this totally different disease.

My impression is that the junction of the two substantives gives a clearer, shorter, and more explicit designation than that which Dr. Fox proposed. The disease does not, in any rate not in all cases, merely resemble acne. It is acne and lupus in association. The spots, originally common acne, take on lupus growth.

There is a form of disease in which the features of acne are mixed with those of lupus. It is usually seen in conjunction with the common form of ulcerating lupus on the nose. Scattered over the cheeks are a number of acne pustules many of which, but not all, become the seat of lupus growth. Instead of receding as an

* See 'Lancet,' July 13, 1878.

acne pustule will usually do after it has been emptied of its contents, these spots grow into little lupus nodules, and unless promptly treated will advance and coalesce.

I have seen this lupus acne attack only one cheek, although both were affected by common acne. In this case the patient, a delicate lad of a tubercular family, suffered also from lupus of his palate, and his sister had a most peculiar affection of her hands to which I should ask to be allowed to apply the name of lupus eczema.

In explanation of the phenomena of acne lupus I suppose that we may adopt the hypothesis that inflamed acne spots in close proximity to a patch of lupus attract to themselves through the lymph channels infective material. Each one in turn becomes a source of infection to adjacent glands. I have never seen acne lupus excepting as a concomitant and sequel of common lupus.

The disease to which I would give the name of eczema lupus is a very peculiar one. The conditions presented are such that equally good observers might differ in opinion as to what the disease ought to be named. The patch weeps like an eczema, and it may be red, abraded, and cracked, but unlike eczema it is absolutely incurable, remains for years without any alteration excepting slow extension of its edge, and when it undergoes resolution a thin scar is left. It is not attended, as a rule, by any tendency to the development of eczema elsewhere. It is certainly essentially a lupus, but its features are so peculiar that it is almost invariably wrongly named. I have not seen more than six or eight well-marked examples of this affection, and it may be well that I should describe in a little detail two or three of them.

In close association with what I have ventured to call eczema lupus is a form of disease which attacks the skin of the extremities, usually I think in very early life, and which has a very remarkable result in producing contraction of the digits and in arresting their growth. There is never any exfoliation of bone, yet the digits become shortened and reduced to mere stumps. Some interstitial absorption must probably take place to explain this, but in large part it is due to arrest of growth of the bones, consequent on the occurrence of severe disease all round them at an early period of life. In severe cases the deformity produced is such that the term lupus mutilans might be appropriate. It is, however, only a very severe form of eczema lupus, or of the common ulcerated lupus of the hands and feet.

I have seen two examples of the disease exactly like the portrait which I exhibit, and three or four others which resembled it, but less closely.

In the case which the portrait illustrates it will be seen that the whole of the extremity nearly to the elbow has been reduced to the condition of a thickened glossy scar. All the fingers were shortened to mere stumps, the thumb, index, and middle finger being none of them more than about an inch in length, and the ring and little finger still shorter. Their phalanges were displaced by the contraction of the scar. It will be seen that there are no abrupt margins to the patch, no ulcers and no crusts; everywhere the scar is red, shiny, abraded, and eczematous. It is clear that the cellular tissue as well as the skin itself has been involved in the process, and there is a good deal of general thickening about the wrist-joint.

A case exactly resembling this in all features, excepting that it extended higher up on the arm, was under my observation when I was house surgeon at the York County Hospital. In it also the patient was a young woman.

In some examples of lupus mutilans, however, the disease departs much less definitely from the type common to lupus and attacks the hands and feet, and the degree of mutilation produced appears to depend very much upon the early age at which the disease begins. Of this a remarkable example has several times recently been under observation at the examinations at the College of Surgeons, the shortening of the fingers being almost as great as that in the portrait which I show. In this patient, although the palm of the hand shows lupus in the exzematous condition, the back has an abruptly margined patch, swollen, with ulcerated edges, the man has a similar patch on one foot.

There is another very peculiar malady to which some years ago I ventured to give the name of lupus marginatus, but which might perhaps quite as well be known as lichen lupus, for it is attended by the formation of little hard lichen nodules which constitute the margins of the patches. When I first described this disease it was claimed by a distinguished authority to be lupus erythematosus, but it is not only unattended by erythema, but it is never developed symmetrically. These features conclusively mark it as having a nearer affinity to common lupus than to the erythematous variety. It is a very peculiar disease, but as it is distinctly infective, slowly

serpigenous, and leaves scars, I claim it as one of the lupus group. I have seen only three examples of it.

We have next the malady which I have tried to name lupus lymphaticus.

The last subject which I will submit for your consideration is the question whether we ought not to regard the very remarkable disease which Hebra and Kaposi were the first to observe, and which may be well designated Kaposi's disease, as a family form of lupus. By family form I mean a form which, owing to some intensification of hereditary transmission, shows itself at a very early age and in several members of the same family of children. Probably, most forms of constitutional taint and tendency to disease are prone occasionally to assume the family form, and in doing so, frequently present peculiar modifications. What is inherited is not a disease in its entirety, but a peculiar modification of tissue giving proclivity to disease under special but it may be very slight exciting influences. Now, the facts as regards Kaposi's disease appear to be these:

In vol. xxx of the 'Transactions' of the Pathological Society, the late Dr. Tilbury Fox and Dr. Colcott Fox give a detailed report of their case, which was in some features similar to what I have described under the name of lupus lymphaticus. Their case was designated one of lymphangietodes. It was brought before the Pathological Society on October 15th, 1878, and in connection with it I mentioned two of my cases of lupus lymphaticus, neither of which I had at that time published. Dr. Fox's patient, a young man of twenty-one, had at birth two large port wine stains on the left thigh. At the age of six months the veins of the left calf were noticed to be enlarged, but it was not till two years old that the lymphatic complications were noticed. Then little "warty" growths appeared near to, but quite distinct from, the nævi. He had repeated attacks at intervals of a few years of a sort of low fever, during which these patches became somewhat inflamed and tender. It should be added that he had been born in the Mauritius.

I believe I may say that the condition of the lymph warts was exactly the same as those seen in my patients. I did not think so at the time I saw Dr. Fox's patient, but Dr. Colcott Fox, who has since had opportunities of seeing mine, assures me that it was. Dr. Fox gives a detailed description of the changes present as demon-

strated by the microscope, in proof that the lympathics were affected and in justification of the title given. It is to be observed that his case differed from mine, first, in the congenital presence of nævi, and, secondly, in a very considerable implication of the venous system as the disease progressed. Only in one of my three cases was any tendency observed to inflammation of the patches, and in it on a single occasion a rather sharp attack happened, very similar probably to those which occurred repeatedly in Dr. Fox's patient. It will be seen that I am especially interested in adducing in support of the name which I have given to the disease the evidence so ably brought forward by Dr. Fox, in support of the opinion that the lymphatics are the seat of the morbid process.

The case of Miss N—.—I think that no one who will compare the portrait which I next show with that of the arm of the boy whose case I have just been describing will hesitate to accept them as illustrating the same disease. In each instance the patches are arranged in a long streak which passes down the outer side of the upper arm, and, crossing obliquely the back of the forearm, passes down to the side of the little finger. On the wrist-joint and ulnar side of the hand there is a large patch much larger than any of those higher up. The patch over the olecranon it would be difficult to distinguish from one of a rough papillary form of psoriasis. Those on the upper arm consist of aggregations of small lichen spots, very hard and rough. Those on the lower arm show minute lichen spots at the edges of abruptly margined long patches which are cicatricial in the middle. It is a little doubtful how far this cicatricial condition is to be explained as the result of former treatment. The patch on the hand is again rough, thickened, elevated, and papillary. It shows no tendency either to ulcerate or to produce scar. In this case, as in the boy, the disease began in very early life, but not actually in infancy. The patient is now a young lady of fifteen. There is no question that the disease has made very considerable progress of late years.

I shall have to note, in speaking of the lupus lymphaticus which is attended by little tufts of dilated capillaries, that it occurs sometimes in apparent connection with congenital nævoid conditions. When it does so, however, there is always very definite and progressive extension afterwards. The arrangement of the patches in long streaks in two cases which I have just described will, I feel sure, induce the suspicion in many minds that the disease may be in

part at least connected with conditions which are present at birth. Granting the plausibility of this hypothesis we still have, both in this instance and in that of lupus lymphaticus, to account for the fact of subsequent aggression. This subsequent aggression is often so out of all proportion to the original disease that the former may have been almost forgotten by the patient's parents. The opinion which I wish to advocate is that even admitting that in some cases a congenital peculiarity of skin, something of the nature of nævus or mole was the starting-point. The progressive disease developed in it is of a lupus nature.

The portrait which I shall presently show is of extreme interest in this connection. For in it the state of the patient's skin has been most anxiously watched through her whole life. An insignificant port wine stain was all that was observed when the child was born, and it underwent no change until the age of five years. At present a disease characterised by delicately-ringed patches is slowly extending down the forearm very much in the same direction as in the two cases the portraits of which are before you.

I recorded in 1879 (see 'Medical Times and Gazette,' vol. i, page 3) the case of a little girl aged two years and a half, in which a congenital nævus on the face became serpigenous and spread very widely, leaving a scar in the middle. It continued to spread; at the age of four years it had destroyed part of the columna and septum of the nose and produced very extensive scars. Although I have said that there was congenital nævus, it should be added that the child's mother denied this, and said nothing was present at birth, but that some little red spots began to appear within a few weeks afterwards, affecting the nose, ears, and hands.

Sixthly, cases in which a sort of erythematous psoriasis spreads rapidly over large areas of the limbs and body.

In deciding as to whether any form of lupus is more nearly allied to the erythematous type or the vulgaris type, the tendency to symmetry is I feel sure the most important feature to be noticed. This is the one which takes us deepest as to to the real nature of the process, connecting the disease with inborn peculiarities of tissue tendency and vascular supply and putting it at a distance from infective new growths. The more definitely the disease is limited to the part first attacked or to those adjacent to it and therefore presumably infected by it, the more closely does the process approach to that of the new growths and the more definitely

does it belong to lupus vulgaris. Tried by this test we shall find that the lupus lymphaticus, lupus marginatus, and a peculiar variety of lupus which occurs in connection with nævoid structures, although conspicuously attended by the enlargement of blood-vessels, belong really to lupus vulgaris, and not to the erythematous form. It is further to be noted respecting these also that they appear to have no connection whatever with proneness to chilblains, a feature which very definitely characterises all the best-marked examples of lupus erythematosus.

Of lupus erythematosus, taking as its most essential feature the tendency to symmetry, we have the following forms:

Firstly, one in which the patches are simply erythematous and slightly scaly, without any formation of discs and without any conspicuous implication of the sebaceous glands. Even in this form, however, there are almost always little sebaceous patches in the concha of each ear.

Secondly, the very rare form in which in association with simple erythema the hands as well as the face are affected and there is a hæmorrhagic tendency.

Thirdly, cases in which discs develop, lupus erythematosus discoides, usually limited to the face.

Fourthly, lupus sebaceus, in which dry, orange-peel like patches form symmetrically on different parts of the face. This was the form originally described by Hebra, and is often but little erythematous.

Fifthly, a form so closely allied to chilblains that it is scarcely possible to distinguish the two. Usually amongst the varieties of common lupus I think we may mention the following:

First, lupus occurring in single patches slowly aggressive at their borders, with a tendency to cicatrise in the centre, but with little or no tendency to ulcerate or inflame or to cause any infection of adjacent parts. These single patches may exist as such through half the patient's life.

Second, many-patched lupus. In these cases the secondary patches always begin near to the first, and there is never any exact symmetry; ulceration may be present or wholly absent.

Third, lupus with ulceration. This, it must be admitted, is a very variable factor. Any variety of lupus may ulcerate, and the tendency to do so varies somewhat with the precise part affected.

Fourth, lupus acne, the lupus follicularis disseminatus of Dr. Tilbury Fox.

Fifth, lupus eczema, a form of disease which begins as an eczematous process and ends as a lupus one and which looks like an eczema throughout, but which leaves scars.

Sixth, the papillary form of lupus, the lupus verrucosus of McAll Anderson.

Seventh, a condition of things which is a mixture of the lupus eczema and the papillary form and which produces an extraordinary mutilation of the digits, may be conveniently known as lupus mutilans.

Eighth, a lichen lupus, or lupus marginatus.

Ninth, lupus lymphaticus, and

Tenth, nævus lupus, a form of lupus originating in parts which were affected by congenital nævi.

Dr. Radcliffe Crocker said that he could not altogether agree with Mr. Hutchinson's definition of lupus as it would include syphilis. We must admit that different processes could produce like results. So that degeneration and pressure-atrophy might result from other than lupus processes. He recognised the truth of the clinical picture drawn so faithfully by Mr. Hutchinson, but considered that we ought to wait for more cases before coming to a definite conclusion. The multiplication of compound names led to confusion, and ought not at present to be persisted in. Lupus might be found to attack certain constituents of the skin. In acne lupus, so called, the sebaceous glands were found not to be specially involved.

Mr. Noble Smith showed a case of meningocele in a child aged seven weeks. The tumour was three quarters of an inch from above down and nearly an inch wide, globular in form. It was situated over the centre of the occipital bone. Its appearance was very like a congenital cyst, but it could be emptied by very firm pressure, and the margins of the opening could then be felt. The skin was natural, except that in a good light the central part was thinner than the sides. There was no pulsation. It was not appreciably altered when the child cried. The tumour was translucent. A gutta-percha splint moulded to a cast of the tumour was being worn. The child being apparently in good health Mr. Noble Smith proposed dealing with it by pressure, reserving operative interference in the event of the patient getting worse.

Dr. Godson exhibited a mechanical nurse or "couveuse."

Mr. Pearce Gould exhibited a pathological specimen of Meningo-Encephalocele.

February 18th and 25th, 1884.

ON THE COLD-BATH TREATMENT OF ENTERIC FEVER.

By Sidney Coupland, M.D., F.R.C.P.

In the remarks I propose to make upon what is called the "cold-bath treatment" of enteric fever I do not intend to discuss at any length the *rationale* of the treatment. This has been done by many —by no one more clearly or scientifically than by my friend and senior colleague, Dr. Cayley,* whom I should have much preferred to have opened this question to-night, his knowledge and experience being so much greater and more thorough than mine; and I feel bound to express my personal obligations to him for example and precept on this very important method of treatment, which seems to be founded on sound pathological data. My own experience is based upon cases of enteric fever admitted under my care at the Middlesex Hospital, in which I have endeavoured, imperfectly, to act upon the principles to which I refer; and my choice of this subject has been mainly influenced by the desire to formulate the results of this experience, which I will endeavour to do with impartiality.†

In dealing with a case of enteric, or indeed any specific fever, three distinct lines of treatment are open to us, which may be termed the specific, the expectant, and the antipyretic. Of the first of these I can say nothing, for, in spite of much advocacy on the part of some, it is plain that we have not yet attained to it. It means the employment of measures directed against the special poison or germ of the fever—an antidotal or germicidal method, according to the view held as to the pathogeny of the disease. In

* 'Croonian Lectures on the Etiology, Pathology, and Treatment of Typhoid Fever.' London: Churchill, 1880.

† The reports of the medical registrars of the Middlesex Hospital for several years past contain detailed analyses of the cases of enteric fever treated in that institution. I have utilized information obtained from these reports in this paper; and I must take this opportunity of thanking my friend Dr. J. W. Browne for allowing me the use of the proof sheets and manuscripts of his reports for the years 1881 and 1882.

enteric fever, such remedies as calomel, iodine, carbolic acid, salicylic acid, sulphurous acid, &c., have been variously prescribed with this intent; but the fact that none of them have become universally adopted is enough to show that their influence has been overrated. Even with such notable examples as syphilis and malaria in view, it behoves us to be most sceptical about the value of "specifics" in any disease, although no one can assert that such may not some day be found. For my own part, I am inclined to think that the increasing evidence in support of the germ theory of these diseases, instead of giving hope of such a consummation, points rather the other way, for the introduction into the human body of an agent sufficiently powerful to neutralise or destroy the morbific virus would be likely to produce dangers as great as or even greater than those it is intended to avert.

The expectant treatment is that which is most generally adopted. It recognises the futility (at present) of seeking for specifics, and reserves itself for emergencies as they arise. It is not pure empiricism, for it acts upon the knowledge of the specific lesions of the fever, and is directed to mitigate the effects of these lesions, to control excessive diarrhœa, check hæmorrhage, give rest to the inflamed and ulcerated bowel by careful regulation of diet, to relieve the bronchitic and pulmonary symptoms, and to check excessive rise in temperature. In so far as antipyretic methods are resorted to, only when the fever attains dangerous heights do they fall under the head of "expectancy," and as such they have been practised from all time. The fever has been treated symptomatically. This treatment also guards against and deals with complications of the fever, whether these be due to the local lesion, as peritonitis, or to the pyrexia, as in the administration of stimulants to maintain the flagging heart; and, finally, it has to deal with such sequelæ as may arise out of the changes induced by the long-continued fever.

Now, the third method, that upon which I have undertaken to dwell, is based on the recognition of the continued pyrexia being of itself an element of danger, not only from actual excess of body-heat, hyperpyrexia, but from the secondary effects of continued fever, the excessive tissue metamorphosis and degenerations, particularly as seen on the circulatory and nervous systems, with the consequent tendency to fatal hypostatic congestion of the lungs. Those who advocate the antipyretic treatment to be systematically adopted,

believe that its adoption would result in a greatly diminished mortality, and in a very marked difference in the condition of the individual patient as regards physical and mental vigour, during convalescence. Dr. Brand, of Stettin,* who for more than twenty years has been the consistent advocate of systematic cold bathing in fever, goes further than this, and avers that his experience justifies the assertion that in controlling the pyrexia from the first, the specific lesions of the fever do not run through all those stages which we mostly think to be inevitable—in other words, that the swollen and infiltrated follicular glands do not pass on to ulceration, so that diarrhœas, hæmorrhages, and perforations are unknown, and typhoid is robbed of its terrors. If this be true—and I am not in a position to either confirm or confute it (for I have not found it possible to carry out the Brand method in its entirety)—there is barely any room for expectant treatment, and anxiety as to the issue would depend mainly upon the previous condition of the individual. Brand in his own practice states that of 381 hospital cases fifteen died, a mortality of 3·9 per cent., and of 257 cases in private practice all recovered. No wonder that his faith in the Wasserbehandlung is great, and his belief (shared by his pupil Glenard) firm that could every case of typhoid fever be subjected to such treatment from the first it would cease to be a fatal disease. I may add that he explains this influence not only upon the pyrexia, but upon the specific lesions, by the ingenious assumption that the development of the typhoid germ, or rather the typhoid process, is arrested by refrigeration as fermentation is by cold. I do not ask your acceptance of this hypothesis, but desire to deal with the antipyretic treatment on the sole ground that it controls pyrexia.

The means at our disposal for carrying out this treatment are (1) drugs, and (2) the external application of cold.

The drugs on which most reliance can be placed are quinine in large doses, salicin or salicylates, and kairin, the newly-introduced agent; but digitalis, veratria, and other powerful remedies have been occasionally employed. In my own cases I have been content to prescribe quinine in ten- or twenty-grain doses, generally as an adjuvant to the bath or cold sponging; in but a few cases have I given salicylate of soda, and in only two kairin. It is no part of my present purpose to speak of the respective merits and demerits

* 'Die Wasserbehandlung der Typhösen Fieber,' von Dr. E. Brand. 2te Aufl. Tübingen, 1887. His first monograph on the subject appeared in 1861.

of these remedies, but I should like to state my conviction that it would not be wise to adopt either one or the other on the same systematic plan as is done in the treatment by cold, especially as we cannot speak with such confidence upon the mode of action of a drug as we can of the action of cold. There are some who have observed deleterious effects arise from the use of quinine, and it is not always tolerated by the stomach. As to kairin, a very powerful antipyretic but of very transient action, I submit that although it may be safely given in some cases it is not without danger in others. I cannot speak from any experience on this point, for it was only given for a short time in the two cases in which I employed it; but Mr. Fardon, the resident medical officer of the Middlesex Hospital, tells me that in two cases which were under this treatment, the subsultus, tremors, delirium, &c., remained during the depression of temperature as in high fever, these symptoms disappearing after the bath had been substituted for the drug.

The means of reducing excess of body-temperature by cold include, amongst others, affusion, sponging, the compress or ice-bag, and the cold bath. Of affusion (save in connexion with the bath) I have no experience, although I believe that Currie attributed much of his success to the stimulant action of this measure on the torpid nerve-centres in fever. But I will say a few words upon each of the other methods, and their application.

1. *Sponging.*—The face, trunk, and upper limbs are sponged over for a period of ten or fifteen minutes with either tepid or ice-cold water (it is best to commence the treatment with the former) whenever the temperature of the body reaches 102° F. The practice is generally, but not invariably, followed by a fall in temperature, which is not, as a rule, of more than 2° or 3°—a fall the extent of which depends more upon the stage of the fever than anything else. For in the later weeks of an attack the temperature is more labile; the effect produced by sponging is more marked than in the earlier weeks, or when the fever is at its height. And it requires very frequent repetition to be at all effectual in attaining the object of preventing the pyrexia becoming severe. In consequence of this, sponging alone is open to grave objection; for if it be carried out on the strict principle, it involves so frequent a disturbance of the patient that he is deprived of continuous sleep. Moreover, I have met with cases in which, for this reason and for others, the bath has been preferred by the patient. Both methods may be combined

in severe cases, the sponging being employed to obviate too frequent recourse to bathing. It has the advantage of not requiring the removal of the patient from his bed, and may be used when bathing is contra-indicated (Plate I).

2. *The compress.*—This consists in the application to the surface of the body or of a limb of cloths wrung out in ice-cold water, and it is had recourse to at the same temperature as the sponging, and on much the same grounds. It is also useful in cases of moderate pyrexia; but, like sponging, it does not reduce the temperature to the extent that the bath does. The compress should be changed very frequently, and its application continued so long as the temperature remains above 99°, so that it requires very constant attention on the part of the nurse. Here, again, the disturbance of the patient interferes somewhat with sleep. I have employed it in a few cases as the sole antipyretic agent, and am convinced of its efficacy. It is especially useful as a substitute for the bath in the later stage of the disease, when there is much abdominal distension. I should add that in all cases of treatment by the application of cold the patient is kept only covered by a sheet and thin coverlet (Plate II).

3. *The bath.*—This is undoubtedly the most powerful antipyretic agent we possess, and almost the only measure that has succeeded in saving life threatened by hyperpyrexia. Even those who mistrust its routine employment in typhoid fever recognise its power, and no one would dispute its efficacy or value as a last resort in such urgent cases. But it is the employment of the bath to control the whole course of the fever that I am considering. Brand's rules are explicit enough; he urges the early commencement of the treatment, and the repetition of the bath every three hours if the temperature rises to 102·2° F. (39° C.) in the axilla, the temperature of the bath not being above 68° F. (20° C.). I have myself so far departed from these rules as to prescribe the bath at a temperature of 103° F., and in several cases it has been necessary to repeat it in three hours, when the fever is at its height. The usual temperature of the bath was 75° F. or 70° F., and its duration ten or fifteen minutes, but both these conditions are varied according to the way in which the patient bears the immersion. In some, especially spare people, the graduated bath—*i.e.*, commencing at 90° F., and cooled to 75° F., with longer immersion, or one at 85° F., is borne better than the colder bath of shorter duration. It often happens that the tempe-

rature of the body has risen above 104° F., in spite of hourly observations between the baths, when the next bath is given. Again, in very severe cases, to one of which I shall allude in greater detail presently, hardly any reduction in temperature is effected, or the actual reduction and duration of after-fall is so slight that within a very short time the fever has risen to its previous height.* Dr. Ord has recommended the graduated bath, and says that it is most efficient and most safely applied early in enteric fever, but he does not think it necessary to repeat it at shorter intervals than twelve hours, "an apparent revival of the temperature often subsiding after such a period."† It may be that the cooler bath acts as a more powerful stimulant on the nerve-centres, more resembles Currie's affusion than the warmer, but in either plan the difference between the temperature of the body and that of the water is great enough to give rise to a considerable amount of "shock." Shivering is of course an invariable result of the immersion, and sometimes this is so severe as to necessitate the curtailment of the process. I have rarely seen any serious collapse occur, but often consider it advisable to administer a little stimulant. Much of course depends on the mental attitude adopted by the patient; it is not as in hyperpyrexia, where we have to deal with one perhaps semi-comatose; but at a temperature of 103° F. the patient is fully conscious, only wakeful, restless, and uncomfortable. No wonder then that he does not at first like being plunged into water of the temperature of 70° F., and occasionally the dread of the bath is so great as to render it unadvisable to pursue it, and then one has to rely upon other antipyretic measures. Those who do submit to the temporary discomfort soon, however, come to appreciate its advantages, and even to look forward to the repetition of the bath, for with hardly an exception the patient afterwards sinks into a calm sleep, and for a few hours at least is in a state of unwonted comfort.

Apart from special contra-indications, the bath has the disadvantage of being difficult of application under all circumstances, an objection which will apply with special force to private practice; but I do not think its employment should be abandoned without an

* An analysis of the very varying effects upon the febrile temperature, wholly unrelated apparently to the initial temperature or duration of the bath, will be found in the "Report of the Committee on Rheumatic Hyperpyrexia," 'Clin. Soc. Trans.,' vol. xv.

† St. Thomas's Hospital 'Reports,' 1879.

effort, seeing how much it ministers to the well-being of the patient. Again, in hospitals with many fever cases under treatment it would hardly be possible to carry out the routine treatment with the ordinary complement of nurses and attendants, unless some special means were devised to relieve them of the strain and labour involved in lifting adults in and out of a bath.* The bath should be large enough to allow of the patient being completely immersed, except the head and shoulders, which may be douched and sponged. After the immersion and removal to bed the body is very lightly dried, and only a light covering placed over the patient, unless there be much shivering, when his feet are wrapped in a blanket. It is not necessary to take the temperature of the body (if so it must of course be a rectal observation) when in the bath or immediately on coming out. It suffices to make the observation half an hour after removal, and repeated at intervals of one or two hours subsequently.

The comparative superiority of the bath to other methods, where these latter have been adopted in preference or as alternatives, is shown in charts (Pl. III). It may be expressed in the statement that the daily mean of the temperature is much lower under bath

* Thanks to Mr. Fardon, who planned the idea of a mechanical arrangement to relieve nurses from this task, and to Mr. Hawksley, who constructed the apparatus, we have had for the past four years at the Middlesex Hospital the apparatus shown in the adjoining room. It is not very sightly, but is very serviceable, and consists of a large wooden frame on wheels, about five feet and a half between the uprights, the crossbar above being about seven feet from the ground. This is large enough to stand across the bed and the bath, which is drawn by the side of the bed; and by means of an endless chain and pulley a hammock can be slung parallel with the bed, the uppermost pulley being attached to rollers which run on a horizontal metal bar beneath the crossbeam. The apparatus being in position, and the patient prepared, the canvas webbing of the hammock is passed under his body, the hammock poles adjusted and attached by ropes to the lower pulley; the hammock is now raised by means of the pulleys, and when it has cleared the level of the bed it is run along until it comes over the bath, when the action of the chain is reversed and he is gradually immersed. The bath is a large one—about six feet and a half long at its upper part and five feet and a half at its base, and a foot and a half deep. Its width is twenty-eight inches at the head and eighteen inches at the foot. It runs on wheels furnished with rubber tires, and is supplied with a tap for withdrawing superfluous water. A square and a circular water-pillow are placed in the bath, the former supported on an inclined plane at the head, so that the patient's shoulders may be a little raised.

treatment than under compresses or sponging; for although the maxima may be higher in the one case, the minima are lower.

Once more let me point out that the object of systematic antipyretic treatment is not to meet the emergency of a hyperpyretic storm, but to safeguard the organism from the effects of continued fever. The mode of action of the methods employed for this purpose may not be the same; it is unlikely, to say the least, that quinine or kairin acts in the same way as sponging or the bath; but it is highly probable that the powerful effects produced by the external application of cold are due as much to the rousing into action of the heat-controlling centres, which are paralysed by the specific virus, as to the mere abstraction of heat. Moreover, the whole effect of the bath may not be merely because it lessens heat expenditure or heat production; it may not be a mere question of diminished body heat; it may also operate upon the central nervous system generally, and be beneficial for other reasons than those of subduing fever only. Certain it is that patients submitted to this treatment pass through their fever more easily and have fewer of the more serious sequelæ which are liable to occur in cases where no such treatment is adopted.

In estimating the value of any therapeutic measure, we naturally inquire, firstly, what influence it has upon the mortality of the disease, and, secondly, what is its effect upon the course of the disease. The first requires an appeal to statistics. These are open to the objection that it is assumed that in the disease that is analysed the conditions and the rate of mortality are constant, or nearly so. Now, this is obviously not the case with the specific fevers, the mortality varying considerably with the type of the epidemic, the hygienic surroundings, and the individual himself. I have placed before you the number of cases of enteric fever admitted into the Middlesex Hospital in every year from 1867 to 1883 (inclusive), with the annual rate of mortality, which has ranged from 28·8 per cent. in 1876 (forty-five cases) to 2·5 per cent. in 1880 (forty cases). The mean mortality for the whole period of seventeen years is 14·8 per cent. of 823 cases admitted, or

	Cases.	Deaths.	Rate.
In 5 years ending 1871 ...	151	20	13·2 per cent.
4 „ 1875 ...	165	28	16·9 „
4 „ 1879 ...	213	34	15·9 „
4 „ 1883 ...	294	40	13·6 „

From figures given by Dr. Cayley in his lectures, as well as from others I have gathered from hospital reports, we may take it that the mean mortality from this disease in the London hospitals is about 15 to 18 per cent, The rates of mortality in hospitals are no doubt higher than in private practice, among the upper and middle classes, where the care of the patient begins as a rule earlier than in hospitals; for no matter whether the treatment be "expectant" or "antipyretic," the earlier the case is cared for the more hopeful is the outlook. But it is maintained by Brand and his followers that the rigid adoption of the bath treatment will diminish the mortality to a far greater extent than does treatment upon the expectant plan. I have already mentioned Brand's general statistics, and will content myself with drawing your attention to these figures published by various authorities abroad, where the adoption of Brand's system is well-nigh universal, and where its results have been so favorably regarded that the Imperial Board of Health issued instructions directing it to be carried out in the army.

Enteric Fever Mortality Statistics.

		Expectant Treatment.		Bath Treatment.	
		Cases.	Per cent. mortality.	Cases.	Per cent. mortality.
Brand (from all sources)		8296	21·7	8141	7·4
Jürgensen (Kiel) ...	1850–61	330	15·4	—	—
	1863–66	—	—	160	3·1
Liebermeister (Basle)	1843–64	1718	27·3	—	—
	1865–66	746	21·3	—	—
	1866–74	—	—	1163	11·2
Liebermeister (Tübingen)	1860–71	61	23·0	—	—
	1871–80	—	—	110	5·5
Mosler (Greifswald)		—	—	29	3·5
V. Ziemssen and Immermann (Erlangen)		63	30·2	32	9·4
Goltdammer (Berlin)		—	18·1	—	13·2
Glenard (Lyons)		—	26·0	—	9·0
Mayet (Lyons)		—	14·48	—	10·74

This matter of mortality may, however, be scrutinised more deeply. We may inquire whether the mode of death, or, rather, the immediate cause of death, has undergone any change since the adoption of antipyretic measures. I think it has, and that the greater regard paid to the evil influence of continued pyrexia, even where Brand's method has been only partially followed, has resulted in a remarkable alteration in these respects as well as in the period

at which the fatal result occurs. I have compared the fatal cases which occurred in 1873 at the Middlesex Hospital, when I was medical registrar, with the series of eighty-nine cases which form the basis of my present paper. In that year (1873) seventy-five cases were admitted, of whom nine died. One severe case of hyperpyrexia was treated by the cold bath, and recovered; but systematic antipyretic treatment was not in vogue. Of these fatal cases four were admitted in the *first* week of their illness, three of whom died within a fortnight from pulmonary congestion and high fever, and one, a severe case marked by high fever, at the end of the fourth week from perforation of the bowel. Two of the cases were treated from the *second* week, one dying from pulmonary congestion in the fourth week, the other after two months' stay from a rare sequela—suppurating mediastinal glands. Two cases of *relapse* proved fatal from perforation; and one case (date of illness not known) from meningitis during convalescence. In my series of eighty-nine cases I find that the seven fatal ones may be thus distributed: one case of perforation was in a man who unfortunately contracted the disease during his convalescence from acute rheumatism; not one of the twenty-seven cases admitted in the *first* week were fatal; of the forty-one cases admitted in the *second* week, one died of perforation;* of the eleven in the *third* week, one died from perforation; the other deaths were in cases of *relapse* (one from pulmonary congestion), except one who died from cardiac disease after the fever had passed away. If this comparison be a fair one, it points to this fact, that early submission to treatment on the expectant plan did not prevent cases dying from pulmonary congestion—*i.e.* the direct effect of the pyrexia; whilst the only case of death from that cause in the antipyretic series was in a case of relapse.

Employing the same mode of comparison on a larger scale, I have examined the records of the 94 fatal cases of enteric fever that occurred in the hospital from 1873 to 1883 inclusive, and have drawn them up in a tabular form, as well as illustrated the point diagrammatically (Pl. IV). Contrasting the two periods (one of six years—viz. 1873–78, and one of five years, 1879–83), I have endeavoured to distinguish those cases which proved fatal (*a*) from the direct effects of the pyrexia, (*b*) from the local intestinal lesions, (*c*) from intercurrent affections complicating the fever, and (*d*) from sequelæ:

1873–78. 278 cases; 45 deaths: (*a*) Hyperpyrexia and pulmonary

* This patient was not treated by the bath.

congestion, 22; pneumonia, 2—total 24. (*b*) Intestinal perforation, 11; hæmorrhage, 2; pyæmia following extreme intestinal ulceration, 1—total 14. (*c*) Pleuro-pneumonia, 1; septicæmia (early), 1—total 2. (*d*) Peritonitis (late), 1; suppurating mediastinal glands, 1; meningitis, 1; diphtheria, 1; asthenia and suppurating broncho-pneumonia, 1—total 5.

1879–83. 365 cases; 49 deaths: (*a*) Pulmonary congestion, &c., 10; syncope, 1—total 11. (*b*) Perforation, 24; hæmorrhage 7; severe diarrhœa and collapse, 2—total 33. (*c*) Acute nephritis, 2. (*d*) Asthenia (bedsores), 1; phagedæna of the face, 1; thrombosis (mitral disease), 1—total 3.

Thus the rate of mortality on the whole number of cases admitted was in the first period 16·2 per cent., the rates under each of the above heads being—(*a*) 8·7 per cent.; (*b*) 5·0 per cent.; (*c* and *d*) 2·5 per cent. In the second period the rate was 13·4 per cent., and was thus made up—(*a*) 3·0 per cent.; (*b*) 9·0 per cent.; (*c* and *d*) 1·4 per cent.

It may be a mere assumption on my part to argue that the singular diminution in mortality of late years from what I may call pyrexial causes is due to a more extended pursuance of antipyretic methods, especially as such treatment was in vogue, although I believe less generally, before 1879; but when taken in conjunction with a similar amelioration in the clinical course of the fever, the assumption may be of some value. Unfortunately the deaths from intestinal lesion have *pari passu* increased, but this is a point upon which I shall remark again, as it opens up ground for serious consideration.

I pass now to consider more particularly the eighty-nine cases consecutively under my care in the Middlesex Hospital from 1879 to 1883, which include patients of all ages, from three to forty years, fifty-one being males. As may be expected, the cases were of all degrees of severity, and antipyretic treatment of some kind was adopted in the majority of them. I have drawn up a summary of these cases, arranged in a tabular form according to the day of the attack (reckoned as nearly as possible) upon which the patient was admitted. (See Tables A and B.) The tables were drawn up with a view of showing the period of adoption and the nature of the antipyretic measures; and a tolerably fair notion of the relative severity of the cases, *quoad* pyrexia, may be gathered from the frequency with which the treatment was had recourse to. The

difference in the severity of the cases in which baths were had recourse to, and those in which no baths were given, may also be indicated by noting the duration of the stay in hospital of those which recovered.

Amongst the bathed cases.

Cases.	Admitted in the	Average stay.	Relapsed.
18	1st week	50·4 days	2 cases.
27	2nd „	58·4 „	6 „

Amongst the cases not bathed.

Cases.	Admitted in the	Average stay.	Relapsed.
9	1st week	40·0 days	1 case.
13	2nd „	42·5 „	0 „

In fifteen cases no antipyretic measures were taken, and in none of them was the fever high; in some extremely mild. In eighteen cases the measures were limited to occasional cold sponging or compresses, or the administration of quinine or salicylate of soda. Some of these cases were severe, and in a few the bath was only withheld because of the condition of the patient. Again, in thirteen cases, in addition to other measures, only one bath was given; and in thirteen others from two to four baths. In eleven cases the number of baths was five, but less than ten; in sixteen cases it was ten, but less than twenty; and in only three was it deemed necessary to give more than twenty baths. It is plain, therefore, that so far as the bath treatment is concerned it has been employed only as one means to the general end; and the table shows how often other measures were employed to aid in the control of the fever. Therefore I cannot claim to be a strict follower of Brand's instructions, any more than I am as regards the temperature at which bathing was had recourse to. And in the nature of things it was seldom possible to commence bathing very early in the disease. But the attempt was made in every case to get the full effect of antipyretics in one way or another; and now that I am enabled to pass the whole series of cases in review, I cannot feel dissatisfied with the result. In some cases the occurrence of hæmorrhage, or great aversion on the patient's part, compelled the disuse of the bath, the latter reason, also, in some cases preventing its adoption at all.

I shall not attempt to present a complete analysis of all these cases, but may refer briefly to the leading symptoms present in the nineteen which were bathed from ten to forty-eight times, these being perhaps the most severe of the whole series; for in all, without exception, the temperature attained 104° at least once, in spite of the controlling measures, and in twelve of them the pulse rate during the height of the fever was 120, while in a few it reached 140. Diarrhœa was marked in ten of the cases; in the rest constipation was more the rule. In one case there was profuse hæmorrhage leading to the abandonment of bathing after twenty-four baths had been given; and this patient, a woman thirty-three years old, recovered. There was slight hæmorrhage noted in three other cases, in one shortly after admission and before any baths were had recourse to. In most of the cases there were bronchitic signs; but the bronchitis could only be said to be severe in three, in which there were also signs of hypostatic congestion, and, except in one case, in which the fever could not be thoroughly controlled, the pulmonary condition did not notably increase under the treatment. Occasional transient and mild delirium occurred in seven of the cases; it was severe, and accompanied by insomnia and tremors in the case just referred to as resisting the antipyretic effect of the bath. In one case there was slight albuminuria. Four cases relapsed, but in most the duration of the fever was prolonged, the average stay in hospital of the seventeen cases that recurred being eight weeks. Yet sequelæ were few—viz., periostitis in one, axillary abscess in another, and bedsores in a third.

I can best illustrate the practice by briefly relating the particulars of two cases. I select these cases, because in the one the bath treatment was actively employed from the first, and in the other it was used in a relapse.

The subject of the first case was a thin, delicate lad, aged twelve, admitted on Nov. 8th, 1880. He was liable to attacks of bronchitis, and his illness which commenced with headache, muscular pain, debility, cough, and later diarrhœa, had lasted about a week before his admission. His aspect was depressed, skin dry, but no rose-spots could be seen; the temperature was 102°. The belly was tympanitic, and the "tâche" was readily elicited. Marked bronchitic rhonchi, but no dulness at bases of lungs; respiration 30; pulse weak (120); heart sounds feeble, and cardiac impulse very readily visible through the thin parietes. No increase in the normal area of splenic and hepatic dulness. At 7 p.m. on the day of admission the temperature rose to 104·4°, and he was placed in a bath of 70° for ten minutes, which reduced the temperature

to 99·6°. The bath was repeated at 10 p.m., the temperature having risen to 103·8°, and a dose of quinine (ten grains) was given afterwards. The next day, the ninth of his illness, the temperature only once rose above 103°—viz., at 7 p.m., when a third bath was given, but with less effect, for in eight hours the temperature was again 103·8°, and a fourth bath was given. During this day, the tenth, the temperature rose very rapidly, and no fewer than seven baths were given, the effect of each being to reduce the temperature from nearly 104° to 100° or 101°. A dose of quinine was prescribed after the last bath given at midnight. During the eleventh day he had four baths given at varying intervals, only one being required in the forenoon, showing apparently the value of the quinine in controlling the pyrexia; the maximum temperature on this day was 103·4°. On the twelfth day he had one bath in the forenoon, and three from 3 p.m. to 9 p.m., the temperature having risen rapidly on each occasion to 104·4°, 104·8°, and 104·2° respectively. On the thirteenth day six baths were given at about regular intervals, the temperature only occasionally mounting up to 104° by the time the bath was prepared. On the fourteenth day the fever was at its height, for in spite of watchfulness it attained 104·8° and 105·2° on two occasions; seven baths were given on this day. On the fifteenth day, four baths were given, all afternoon, the temperature not reaching above 103·6°. On the sixteenth day six baths were given until 3.30 p.m., when, owing to the motions being tinged with blood, this treatment was suspended, and cold sponging was practised at intervals during this day and the following. The signs of hæmorrhage not being repeated, and the sponging proving ineffectual to control the pyrexia to any marked extent, three baths were given on the eighteenth day after 6 p.m., and the last, or forty-eighth bath, was given at 3 p.m. on the nineteenth day, from which time the temperature never rose again to 103°. For some days there were considerable fluctuations in temperature—as much as three or four degrees between the morning and evening records; but a week later—namely, on the twenty-fourth day—a normal temperature both morning and evening indicated that defervescence had occurred. A week later there was a slight and transient recurrence of pyrexia, but no true relapse. The case was a severe one; for although the diarrhœa was not marked, only about one or two loose motions daily, and on one occasion only any sign of hæmorrhage, yet there was considerable tympanites, and very marked bronchitis, which was not influenced either for good or ill by the immersions. The main anxiety lay rather in the indications of the pulse, which was throughout feeble, and its rate on the thirteenth day as high as 156; so that alcohol was not sparingly given. But a notable feature was the entire absence of delirium, and the fact that he slept well in the intervals of the bathing. He took nutriment well, but convalescence was rather slow, the emaciation and debility being very marked. His stay in hospital, about seven weeks, was not, however, so prolonged as in many cases. (Plate V.)

The other case I have selected is that of a bricklayer, aged twenty-six, admitted on July 12th, 1881, in the second week of his illness, his previous health having been good and his habits temperate. He was a well-built muscular man, but very prostrate on admission. Abundant rose-rash; no distension of belly; no evidence of splenic enlargement. Temperature 102°, pulse 120, respiration 24. He had a short cough, with muco-purulent expectoration; and physical examination revealed abundant

rhonchi over the chest; weak breathing at the bases of lungs. At 6 p.m., the temperature being 103°, sponging was prescribed, and was repeated whenever the temperature rose to 103°, which it did repeatedly during the night. On the next day (about the thirteenth of his illness) the temperature was 104·2° at noon, and he passed blood in the motions. Quinine was given—twenty grains in two doses—but it excited vomiting, and the hæmorrhage recurred, but at no time after this. From the fifteenth to the twentieth day the pyrexia was mild; maximum temperature about 106°, with normal temperatures in the morning. The bronchitis, however, persisted, and the patient's debility and loss of flesh increased. On the twenty-fifth day a recrudescence (if not a true relapse) of the fever occurred, the temperature rising to 102°, and on the twenty-sixth day to 103·6°, when quinine was given. He complained of abdominal pain, but there was no diarrhœa. From the twenty-sixth day to the thirtieth day the temperature was frequently above 102°, quinine being given in ten-grain doses at intervals without much appreciable effect. At 3 p.m. on the thirtieth day, the temperature being 103·8°, he was placed in a bath of 78° for fifteen minutes; this reduced the temperature to 99° (half an hour after removal from the bath), but it rose again to 103° in six hours, and the bath was repeated. On the thirty-first day he had two baths, on the thirty-second five baths, on the thirty-third two baths, on the thirty-fourth four, on the thirty-fifth two, and on the thirty-sixth one, the temperature often being 104°, and once 105°—*i.e.* eighteen baths in seven days. This relapse, if it may be so called, was also characterised by an abundant crop of rose-spots, by slight diarrhœa, by the spleen becoming palpable, by great depression, by the tongue being dry and brown, and by the persistence of marked bronchitic signs. The pulse rate rose from 100 to 144 on the thirty-fifth day, and the cardiac sounds became very feeble. On August 9th—*i.e.* about the fortieth day—the temperatures were normal in morning and evening, and the bronchitis had nearly disappeared. He had only just entered upon convalescence and was still very feeble, when he insisted on leaving the hospital on August 18th.

These cases will suffice to indicate the line of practice—viz. to employ the bath as an antipyretic agent, and to continue its use without intermission, unless in the presence of some contra-indication as the occurrence of intestinal hæmorrhage, so long as the temperature rose above 103°, severe bronchitis notwithstanding. It will be seen that the temperature of the body often exceeded this limit when a bath was given, but this merely means that in the hour or two hours that elapsed between one thermometric observation and another, the temperature had passed from below 103° to above it; and I do not think it would have been advisable in any case to give more baths within the twenty-four hours than was given on some of the days in the first case. Nor were these cases treated with the rigour prescribed by Brand, not only as regards the temperature at which they were bathed, but as regards the temperature of the bath; and in the second case it is quite possible that

the severity of the relapse owed something to the lack of rigid antipyretic measures in the primary attack. Nor was the feeble circulation allowed to interfere with the adoption of the treatment, although necessarily forming an anxious element in the cases. Lastly, the value of the antipyretic measures in preventing cerebral symptoms was well shown.*

Whilst refraining from inflicting upon you details of any other of the cases, which terminated in recovery, I think it right and necessary to indicate the leading features of the seven *fatal* cases that are to be found in this series.

Case 1.—A man, aged thirty, admitted in his fourth attack of rheumatic fever on Feb. 15th, 1883. He had a mitral bruit, but his rheumatism yielded rapidly to salicylate treatment. He was in a bed adjoining a severe and prolonged case of enteric fever; but, when a week after he had been allowed to get up he again showed febrile symptoms, the idea that he had contracted this disease was not at first entertained, especially as the pyrexia was accompanied by a slight return of arthritis. For nearly three weeks he had irregular pyrexia without any abdominal signs, and then came a period of apyrexia of about a week, to be followed with what proved to be a severe and fatal enteric relapse. Diarrhœa set in, crops of rose spots appeared, and the fever became high, so that on the fourth and fifth days of the relapse he had in all four baths at 78° for fifteen minutes, having been taking ammonia and salicylate of soda regularly since the pyrexia first set in. On the seventh day of the relapse there was intestinal hæmorrhage, and on the eighth perforation, followed by acute peritonitis, and rapid collapse. The post-mortem examination showed recently inflamed and swollen follicular glands in the intestine, with sloughing and partially cicatrised ulcers. There was recent as well as chronic mitral endocarditis. This case was an instance of a not infrequent event in the history of typhoid fever—viz. a mild primary attack followed by a severe relapse, and it is specially instructive in the light it throws on such cases in the fact that, owing to its nature being unsuspected, the diet during the primary attack was not rigidly restricted.

Case 2.—A fishmonger, aged nineteen, admitted on Feb. 2nd, 1882, towards the close of the third week of his illness, with high fever, much tympanites and diarrhœa. During the first six days ten baths were given, with occasional sponging in the intervals. About thirty hours after the last bath he was attacked with severe abdominal pain and vomiting, followed by evidence of acute peritonitis; he survived five days, a second attack of severe pain occurring twelve hours before death. The post-mortem revealed twelve denuded and deep ulcers in the ileum, three of which had perforated.

Case 3.—A married woman, aged twenty-one, was admitted on Nov. 21st, 1883, in an enfeebled, emaciated state, with high fever (temperature 105°) and distended abdomen. She had been ill for six weeks,

* I have records of all the cases included in this review, some of which will be found in the Appendix, p. 222.

having apparently passed through a primary attack of the fever three weeks before. One bath was given, perhaps without due caution; although signs of perforation did not appear till the following day. The lesions were limited to the lowermost ten inches of the ileum, where deep ulcers, with partially detached sloughs, occurred; in one of these the necrotic process had extended through the serous coat.

Case 4.—A labouring man, aged twenty-four, thin and worn, was admitted on Nov. 28th, 1881, towards the close of the second week of illness. There was considerable bronchitis and pulmonary congestion, a feeble, rapid pulse, much tympanites, diarrhœa, and abdominal pain. No bath was given, but sponging was practised. He died from the effects of perforation eight days after admission, most of the intestinal ulcers being still covered with adherent sloughs.

Case 5.—This case, the other death from intestinal lesion, was that of a domestic nurse, aged thirty-four, who was admitted on Oct. 2nd, 1883. She had contracted the fever in Scotland four weeks before, and the disease must have been specially malignant, since two out of four members of the family who fell ill at the same time also died. For the first week from her admission there was constipation and but mild pyrexia, then diarrhœa set in and the temperature rose. From Oct. 13th to 17th nine baths were given. On the 18th some blood appeared in the motions, and the antipyretic treatment was limited to the application of compresses, followed later by kairin, which was only given for two days. On Oct. 23rd, or three weeks after admission, the hæmorrhage recurred and became uncontrollable, death occurring on the 27th. There was most extensive and severe ulceration, involving not only the ileum, but also the large intestine in its whole extent, especially in the cæcum (the source of the bleeding), ascending and transverse colon. In the ileum were two large necrotic areas involving all the coats, so that perforation was inevitable had life been prolonged a few hours.

Case 6.—I need only allude to this case, that of a young woman, aged twenty-one, admitted Oct. 10th, 1883, in which, owing to the existence of mitral stenosis and cardiac dilatation, aided doubtless by the effect of the fever, death occurred from cardiac thrombosis some weeks after the fever had passed away. The case was not diagnosed as, nor treated for, enteric fever, presenting none of the features of the disease.

Case 7.—I have reserved to the last mention of the only case of the series in which death could be directly attributed to the intensity of the febrile process itself. A young carpenter, aged seventeen, of good physique, and whose health had always been good, was admitted on Oct. 26th, 1882. He stated that he had been "feverish" four weeks before, but that he continued at his employment in spite of headache, anorexia, some abdominal pain, and diarrhœa. On the 25th he vomited, and was attacked with shivering. He was admitted next day. Temperature 104·8°, pulse 116, respiration 44; and for twenty-four hours the fever, being uncontrolled, attained great heights (hyperpyrexia, 105·8°). At 1 p.m. on the 27th, the temperature being 105·8°, he was given a bath at 70° for fifteen minutes. In half an hour after the temperature was 104·2°, and at 4 p.m. 103·6°. The bath was repeated at 5 p.m., and again at 7 p.m., the third being followed by quinine (twenty grains). The later baths were prescribed

of a higher temperature (viz. 85°), and of a longer duration (thirty minutes), than the earlier, as the colder ones produced so much shivering that it was feared the reactive rise in temperature might be partially due to this. Throughout the case there was a copious rose-rash, and in the later days diarrhœa, some delirium, very rapid pulse, signs of pulmonary engorgement, and shortly before death profuse epistaxis (see p. 233).

I remember at the time considering that in this case we had lost valuable time in not commencing antipyretic treatment from the very first, and in not more determinedly pushing it; but it never occurred to me then, nor, on further consideration, does it seem to me now, that the treatment pursued intensified the febrile process. Hyperpyrexia does not always yield to treatment; indeed, until the value of cold was demonstrated in dealing with it, it used to be invariably fatal. But there are many cases in which this treatment has been thoroughly pursued in rheumatic hyperpyrexia without avail. I consider this lad was the victim of this remarkable and uncontrollable febrile disturbance, and think it not unlikely that, had more care been taken of him in the primary, though mild, attack to which this was the relapse, the hyperpyrexial condition might never have been developed. The post-mortem showed fluidity of blood, blood-stained serum in the serous sacs, pulmonary engorgement with apoplexies, greatly swollen spleen (20½ in.) and liver, swelling and infiltration of the intestinal follicular glands, and a few denuded ulcers.

I have thought it right to dwell more particularly upon these fatal cases in order to be sure whether the line of treatment had any influence in inducing the fatal termination, and especially whether the employment of the bath could be charged with this. I have just expressed my view as to the solitary case of death from pulmonary congestion, with hyperpyrexia; but I imagine the chief point of attack upon the treatment will be that it may favour intestinal hæmorrhage,* and perhaps induce perforation of the bowel. Undoubtedly in a severe case, which comes under treatment late in the disease, the risk of perforation from the slightest disturbance is a very pressing one; and in one of my cases, which was admitted in a relapse in the sixth week, the placing her in a bath may have determined the fatal rent. But neither in this case nor in any of the others did the signs of perforation occur until

* Upon this question I agree with Dr. Cayley, who has pointed out that intestinal hæmorrhage does not occur more frequently under bathing; and that it is a common practice to use cold to the surface to check internal hæmorrhage.

some hours had elapsed after the bath; so that we have no positive evidence that either the disturbance caused by the moving of the patient or the possible excitation of undue peristalsis from the application of cold to the surface (if such be possible) was really the cause of the mischief. Again, is it not a fact that perforation is due to the extension of the necrotic process through the serous coat? And when perforation is induced by undigested substances, is it not because of the direct injury inflicted on an ulcer whose base is only formed by the serous coat? In spite, then, of the striking increase in the last few years of mortality from intestinal lesions at the Middlesex Hospital, I see no reason to attribute it to the treatment. Some of these cases I find on inquiry were admitted after perforation had occurred; in others it took place very soon after admission; but although many of the remainder had baths in their course, the striking and obvious signs of perforation in hardly one instance coincided with the time of taking the bath. At the same time, it is a wise precaution not to run the risk of hastening what is, I fear, in all these cases, an inevitable sequence of the deeply ulcerated bowel; and therefore it may be both prudent and wise to withhold bathing from patients admitted with marked tympanites and other signs of severe ulceration, and rest content with the effects of the application of compresses and the administration of opiates. Only within the last fortnight a young girl has died in my ward from this untoward circumstance, and for these very reasons I did not resort to the bath in her case at all; and a few days later a child was admitted moribund from the same cause. One other remark on this point. It is only in the last three years, and more especially in 1883, that the mortality from intestinal lesions has been so high in the Middlesex Hospital; and, in face of this, it is justifiable to assume that the type of the fever recently has been marked by severe and deep ulceration of the intestine.

In the course of this paper I have said very little of the opinions of others on the subject of the cold-bath treatment, but before I conclude I should like to advert to the very decided opposition manifested to it by some of the leading physicians in Paris twelve months ago in the course of a debate upon typhoid fever at the Academy of Medicine,* which commenced on October 24th, 1882, and was continued every week until its close on March 20th, 1883.

* 'Bulletin de l'Académie de Médecine,' Paris, 1882 and 1883.

The debate ranged over the whole subject of typhoid fever, its etiology, prophylaxis, mortality, and therapeutics. So far as the last-named topic is concerned the matter was raised by M. Herard advocating large doses of quinine, and also referring to salicylic and carbolic acids, and to cold baths as being sometimes of service. M. Dujardin Beaumetz declared himself a supporter of "armed expectation," and said that he had out of eighty-seven cases only lost four. The cold-bath treatment had, he said, been abandoned in France because it exposed the patient to the dangers of pulmonary congestion. He averred that whereas baths only lowered temperature, sponging acted on the nerve centres. M. Hardy, the President, followed on the same side, and condemned quinine and salicylic acid equally with the bath. "Hyperthermia" was, he said, the *bête noire* of some, and he ridiculed the energy put forth to combat it. The thermometer had usurped the pulse, whose indications were of far greater significance. On January 9th, 1883, the debate was kindled anew by the reading of a paper by M. Glenard, of Lyons, upholding Brand's method, contrasting the French and German army mortality statistics of typhoid, and declaring that no one had a right to judge of the alleged efficacy of the treatment as practised by Brand who did not faithfully act up to its principles. M. Glenard's paper was accompanied by a statement signed by twenty-two of the twenty-four hospital physicians of Lyons, concurring in his views—surely a remarkable testimony. The Academy referred this paper to a committee, who reported mainly on the subject of mortality in the army. M. Germain Sée in a long address reviewed the whole subject of the treatment of fever. He was opposed to the cold bath; said that its advocates could not agree amongst themselves; asserted that fever was rather increased than lessened by the refrigeration of the surface, which drives the blood from the periphery, and maintained that increased oxidation and disintegration were produced by it; statements which it may be observed are directly contrary to the recent very laborious research of Sassetzky,* as well as those of other observers. Moreover, M. Sée declared that this treatment induced acute pneumonia, that it greatly increased the liability to intestinal hæmorrhage, that it enfeebled the heart, and, in short, that it was dangerous, and not to be compared in efficacy with the effects of cold affusion and sponging. M. Bouley, who was the only speaker in favour of the

* 'Virchow's Archiv,' vol. 94, December, 1883.

method, staunchly supported Glenard's contention. M. Jaccoud deprecated the tendency to regard the pyrexia as the whole disease, and supported expectant methods. Of the two Lyons physicians who did not sign the attestation, one, M. Teissier, sent a letter to the effect that he reserved cold bathing for hyperpyrexia, believing that the systematic method of Brand was answerable for pleurisies, pneumonias, pericarditis, and intestinal hæmorrhage; the other, M. Bondet, extending the catalogue of disasters by the addition of syncope, apoplexy, rheumatic fever and periostitis. To this M. Bouley rejoined by producing further testimony from Lyons, notably in letters by MM. Renaut and Chauveau. By far the most determined attack came from M. Peter, who had six years before written powerfully against the method. According to him this "vulgar, blind, empirical, and detestable practice of giving cold baths, *coup sur coup*," was responsible for fatal cases of pulmonary congestion, for hæmorrhages, including one form hitherto foreign to typhoid fever—viz., hæmoptysis, and for pulmonary inflammation. It was a "pompier's" duty this of quenching fever by the cold bath; nor was it based on Currie's doctrine, that of stimulating the nerve-centres; it was simply attempting to reduce fever heat. Why, he asked, if it were so useful, had not the English, who are a practical and a patriotic people, followed the example set them nearly a century ago by their countryman Currie? M. Peter cited cases known to him of death from pulmonary congestion, and concluded his remarks by enumerating many "systems" of treatment which had enjoyed a brief popularity amongst the simple, the rash, and the incompetent ("*naïfs, imprudents, et incompétents*"). To this vigorous counterblast M. Bouley replied, pointing out that M. Claude Bernard, then, must be classed among the "simple" ones; but M. Peter had the last word, and the Academy clearly sympathised with him.

I do not suppose it would be possible to find in medical literature more discordant opinions upon a question of the value of a particular mode of treatment of a disease than we have here. On the one side an unbounded faith in its efficacy, on the other a most decided opinion that it is dangerous, and responsible for increasing the mortality from the disease in which it is adopted. I have already indicated the conclusion to which my own experience has led me, which is in total opposition to the views held by these latter authorities whose opinions one cannot but respect. Pulmonary congestion,

instead of being favoured by the treatment, is, in my experience, notably diminished; pulmonary hæmorrhage I have never seen; pneumonia is less frequent, and has never appeared to be provoked by it; intestinal hæmorrhage has not occurred more frequently than on the expectant plan; but naturally it is met with in the more severe cases, which are precisely those in which more frequent recourse is had to the bath. How can I, then, acquiesce in such a condemnation, and to what other conclusion can I come than that the experience of the objectors is based on imperfect data, and that they have mistaken the natural course of the fever for the ill-effects of a therapeutic measure, perhaps adopted in a way wholly different from its original promoter's intention? Convictions like those of M. Glenard, and of his *confrères* at Lyons, cannot be shaken by *ex cathedrâ* statements of authorities however high, nor by any amount of sarcasm or ridicule; nor is it just to say that the antipyretic treatment is pursued in disregard of other indications, notably the pulse. It might as well be urged that the cold bath had replaced the dietetic rules which govern the whole management of a case of enteric fever. But the thermometer is the main index of the pyrexia; and the pyrexia, if long-continued, is so grave an element of danger, directly and indirectly, that it must be combated. It can only be successfully controlled by the unceasing application of antipyretic measures, and the earlier resort is had to them the greater the hope of success.

What, finally, are the good effects that may be expected from the steady pursuit of these measures? I think they are mainly these:—If commenced early enough, and carried out systematically, it may curtail the disease by rendering it abortive; but that as it is practically impossible either to be certain of the nature of a pyrexia at its inception, and therefore to commence antipyretic measures so soon as might be desired, it may be conceded that if it do not exert any control over the progress of the intestinal lesions it does appreciably diminish the dangers to life due to pyrexia itself, and place the patient in a better condition. For under antipyretic treatment nervous symptoms are allayed, sleep favoured, delirium banished or much reduced. The prostration is less, the so-called "typhoid" state is mostly conspicuous by its absence. The pulse gains in force, and the tendency to pulmonary congestion and collapse of lung, which endangers life in the early weeks of every severe case of enteric fever, is obviated. Further, the digestive and assimilative

functions are improved, so that the difficulty is to avoid yielding to the patient's desire for a solid diet before defervescence has occurred; and, at the close, the patient's mental and physical vigour is far more speedily restored, and such sequelæ as mania and other conditions indicative of profound nutritional disorder of the nervous system rarely occur. It does not appear to increase the liability to intestinal hæmorrhage or to perforation, which depend on the severity of the local lesions. And it reduces the mortality from the disease.

As to the rules, so far as it is possible to formulate any, of the treatment, they may be summed up as follows:—The patient should be as lightly overed as possible, and whenever the temperature reaches 102° sponging should be practised; but if, as mostly happens, the pyrexia cannot be adequately controlled by this measure, then the bath-treatment may at once be commenced. The temperature of the bath may be about 75°, and the time of immersion ten minutes; but this may be varied, as already explained. If possible the bodily temperature should not be suffered to rise above 103° without having recourse to the bath; and if this entail the repetition of the bath more than every three hours sponging may be practised in the intervals, or quinine given after a bath. The treatment must not be abandoned in the face of pulmonary complications, as bronchitis, congestion, or pneumonia; but when necessity compels a resort to it in the later stages of the fever, and symptoms of grave intestinal lesion are present, caution should be practised in bathing, for which it is well often to substitute the application of compresses or cold sponging. If there be intestinal hæmorrhage or signs of peritonitis, the bath must not be given. Although by aid of stimulants bathing may safely be practised when the pulse is rapid and feeble I should regard extreme debility, especially in old subjects, or the presence of organic heart disease, as distinct contra-indications for its employment. It is not prudent to relax rigid dietetic rules, although the patient, under the influence of the treatment, may have his digestive functions so improved as to create a craving for solids before actual defervescence sets in.

To pursue the antipyretic treatment of enteric fever so thoroughly as to attain the desired ends requires much attention and close watchfulness on the part of those in charge of the case; and although in many cases it may seem afterwards as if the resort to such measures had been unnecessary, so far as the recovery of the patient is concerned, the gain derived from increase in comfort, and

deliverance from the dangers, direct or remote, of continued pyrexia, is surely worth all the labour and care that may have been expended.

Appendix of Cases.

Case 1. *Enteric fever; high range of pyrexia and marked cardiac feebleness. Antipyretic measures—forty-eight baths; quinine.*—George H—, aged twelve, schoolboy. Admitted November 8th, 1880, on eighth day of attack—the leading features of which are given in the text of the paper (p. 211), and need not here be reproduced.

Case 2. *Enteric fever, severe; repeated attacks of intestinal hæmorrhage; marked asthenia; vomiting; albuminuria, twenty-nine baths, and quinine.*—Kate G—, aged thirty-three, married. Admitted September 22nd, 1880, on tenth day of attack; a healthy-looking, well-nourished woman, who had hitherto enjoyed excellent health. She stated that ten days ago she began to suffer from drowsiness, headache, and loss of appetite. For past five days has been deaf. Diarrhœa for last two days.

On admission.—T. 103°, pulse 100, resp. 28. Skin hot and dry. Numerous rose-spots; well-defined "tâche" elicited. Abdomen full, not distended; splenic area increased. Scattered rhonchi audible over chest; no basic dulness. First sound of heart feeble.

Prescribed dilute hydrochloric acid, and brandy ʒiij daily.

2.30 p.m.—T. 104°. First bath at 65° F. for ten minutes.

3.30 p.m.—T. 102°.

5.30 p.m.—T. 104·8°. Second bath at 65° F. for ten minutes.

6.30 p.m.—T. 102·2°.

8.30 p.m.—T. 103·4°. Third bath at 65° F. for ten minutes.

9.30 p.m.—T. 102°, pulse 108. Bowels opened once slightly since admission.

11.30 p.m.—T. 104·2°. Fourth bath at 65° F. for ten minutes, followed by Quin. sulph., gr. xxx.

September 23rd, 12.30 p.m.—T. 97°.

2.30 a.m.—T. 102°.

4.30 a.m.—T. 103·8°. Fifth bath, 65° for fifteen minutes.

5.30 a.m.—T. 102·6°. 7.30 a.m.—T. 100·8°. 8.30 a.m.—T. 101·2°.

10.30 a.m.—T. 104·2°. Sixth bath, 65° for fifteen minutes.

11.30 a.m.—T. 98·6°, pulse 96.

Several fresh rose-spots on chest and abdomen. Aspect less depressed; cheeks flushed; eyes suffused. Tongue thickly coated. Bowels twice open loosely. Pulse soft, full, and compressible.

2.30 p.m.—T. 102·4°.

4·30 p.m.—T. 103·8°. Seventh bath, 65° for twenty minutes.

6 p.m.—T. 102·4°.

8 p.m.—T. 103·8°. Eighth bath, 65° for fifteen minutes.

9 p.m.—T. 98·4°, pulse 90, resp. 30. Bowels once moved during the day.

11 p.m.—T. 102·2°.

September 24th, 1.45 a.m.—T. 103·6°. Ninth bath, 65° for twenty minutes, followed by Quin. sulph., gr. xxx.

4 a.m.—T. 103°.

4.30 a.m.—T. 103·4°. Tenth bath, fifteen minutes.

5 a.m.—T. 100°.

9.30 a.m.—T. 100·8°, pulse 96. Abdomen tympanitic. Bowels twice open loosely. Fine crepitation at bases of lungs. Deafness increased.

11.30 a.m.—T. 100·2°. 1.30 p.m.—T. 101·2°. 3.30 p.m.—T. 102·2°.

5.30 p.m.—T. 103·6°. Eleventh bath, fifteen minutes.

6.30 p.m.—T. 99°.

8.30 p.m.—T. 104°. Twelfth bath, fifteen minutes.

9.30 p.m.—T. 103·2°, pulse 108. Thirteenth bath, fifteen minutes.

10.30 p.m.—T. 100·2°.

September 25th, 1.30 a.m.—T. 104·4°. Fourteenth bath, 65° for twenty minutes, followed by Quin. sulph., gr. xxx.

5 a.m.—T. 104·3°. Fifteenth bath, fifteen minutes.

6 a.m.—T. 99·8°.

10 a.m.—T. 104·2°. Sixteenth bath, fifteen minutes.

11 a.m.—T. 99°, pulse 108. Tongue clean and moist. Bowels twice open loosely. Slight albuminuria.

1.30 p.m.—T. 103·2°. Seventeenth bath, fifteen minutes.

2.30 p.m.—T. 99·4°.

6.30 p.m.—T. 104·2°. Eighteenth bath, twenty minutes.

7.30 p.m.—T. 102·6°.

9.30 p.m.—T. 104·4°. Nineteenth bath, fifteen minutes.

10 p.m.—T. 101·4°.

September 26th, 12 midnight.—T. 103·4°. Twentieth bath, twenty minutes.

1.30 a.m.—T. 99·8°.

5.30 a.m.—T. 104·4°. Twenty-first bath, fifteen minutes.

8.15 a.m.—T. 101°. Bowels twice open. Abdomen again distended and tender. Crepitation at posterior bases of lungs, with impaired resonance.

11 a.m.—T. 103·8°. Twenty-second bath, fifteen minutes.

12 a.m.—T. 99·4°.

2 p.m.—T. 104·2°. Twenty-third bath, twenty minutes.

3 p.m.—T. 98·4°.

5 p.m.—T. 104°. Twenty-fourth bath, twenty minutes.

6 p.m.—T. 100°.

8.30 p.m.—T. 104·6°. Twenty-fifth bath, 65° for twenty minutes.

10 p.m.—T. 102°, pulse 96. Bowels open three times; motions loose and watery.

11 p.m.—T. 103·6°. Twenty-sixth bath, 65° for twenty minutes.

12.30 p.m.—T. 98·2°.

September 27th, 2.30 a.m.—T. 104·6°. Twenty-seventh bath, fifteen minutes.

4 a.m.—T. 101·4°.

6 a.m.—T. 103·2°. Twenty-eighth bath, fifteen minutes.

7 a.m.—T. 101°.

9 a.m.—T. 103°; pulse 108, small and weak. Considerable prostration; increased abdominal distension. About 8 a.m. passed a scanty tarry motion.

Brandy increased to ʒviij for twenty-four hours, and turpentine mixture prescribed; the bath being suspended.

From 11 a.m. to 7 p.m. the temperature kept above 104°, reaching 104·8° at 5 p.m. At 6 p.m. an ice bag was applied to the abdomen.

The bowels were moved four times in the day; motions of a dark coffee colour.

8 p.m.—T. 102·6°. 12 midnight.—T. 101·2°.
September 28th, 2 a.m.—T. 102°. 4 a.m.—T. 102·6°.
6 a.m.—T. 104·4°. Twenty-ninth bath, 65° for fifteen minutes.
7 a.m.—T. 98·4°. 10 a.m.—T. 102°.

As the motions still contained blood, and there was considerable abdominal tenderness, it was deemed prudent to discontinue the bath treatment. Hypodermic injections of ergotine and ergotine with morphia were given on two occasions on this day. At 6 p.m. the temperature rose to 104·4°, and cold sponging was practised for fifteen minutes. Pulse smaller. Bowels open seven times during day; motions fluid and blood-stained.

September 29th.—The temperature now fell rather rapidly, reaching as low as 95·6° at 10 a.m., and 94·6° at 12. The patient had vomited frequently, but the hæmorrhage was less. The brandy was reduced to five ounces, and champagne ordered. Ergotine and morphia injections repeated. At 10 p.m., T. 101°, pulse 96. Bowels not open during day, but sickness continued. Urine had to be withdrawn by catheter.

September 30th, 2 a.m.—T. 101°. 6 a.m.—T. 98·6°.
10 a.m.—T. 102·4°, pulse 120. Two motions in night, one "tarry." Tongue rather dry. Vomiting continues, but less, and less distension. Urine free from albumen.
12.—T. 103·4°. 4 p.m.—T. 102·6°. 6 p.m.—T. 103·2°. Sponged.
7 p.m.—T. 103·6°. Sponged. 9 p.m.—T. 103·6°.
10 p.m.—T. 101·8°, pulse 124. Bowels twice moved; fluid, black motion.
October 1st, 2 a.m.—T. 101·4°. 10 a.m.—T. 103·2°, pulse 120. First heart sound short and feeble. Bowels not open. Control over bladder regained. Quin. sulph., gr. xx.
2 p.m.—T. 101·8°. 4 p.m.—T. 99·9°. 5 p.m.—T. 98·6°.
8 p.m.—T. 97°. Bowels twice open; pale, yellow motions.
October 2nd, 10 a.m.—T. 100·6°, pulse 102. Face flushed. Aspect depressed. Sleeps well. During the day temperature ranged about 102° (Plate VI).

It is needless to pursue this prolonged case in all its details; suffice it to say that for some days she remained in a very prostrate condition, the hæmorrhage recurring on October 5th, 12th, and 15th, together with vomiting. The pyrexia remained about 101° to 102°, and sponging was occasionally had recourse to. Defervesence did not actually occur till October 30th, previous to which she had become mentally enfeebled and frequently had delirium.

She was discharged on December 31st.

Case 3. *Enteric fever, severe, prolonged; marked cardiac weakness; periostitis. Twenty-six baths; sponging; compresses; quinine.*—Jane H—, aged twenty-four, single, housemaid. Admitted August 27th, 1883, in second week of fever, which commenced rather abruptly with headache, giddiness, and sickness, followed by attacks of shivering. She remained at work for a week, and then went away "for a change," but not improving was brought to the hospital. Her previous health had been good.

On admission.—T. 104·4°, pulse 112. A well-nourished young-woman, suffering from headache and malaise; cheeks flushed; lips dry. Several rose-spots on surface of abdomen, which is slightly distended. Slight tenderness in right iliac region. Splenic area obscured by tympanites. Tongue coated and rather dry; anorexia; bowels loosely

open. Chest resonant; a few rhonchi over the back. Pulse full and soft.

7.30 p m.—T. 104°. Sponged. 8.30 p.m.—T. 103·2°.

10 p.m.—T. 104·4°. Bath given at 75° for fifteen minutes. Temp. after bath 104·4°.

August 28th, 2 a.m.—T. 102·6°. 6 a.m.—T. 104°. Second bath.

7 a.m.—T. 100°. 10 a.m.—T. 103·2°.

11 a.m.—T. 98·4°. Bowels not open. Tongue thinly coated and moist; lips covered with sordes. Marked tenderness in left iliac fossa. Urine acid, sp. gr. 1015, trace of albumen.

2 p.m.—T. 103°. Third bath. 3.15 p.m.—T. 100·8°.

6 p.m.—T. 103°. Fourth bath, followed by Quin. sulph., gr. x.

7 p.m.—T. 100°. 10 p.m.—T. 102·6°.

August 29th, 6 a.m.—T. 101·8°.

10 a.m.—T. 101·2°. Slept fairly well. Vomited slightly once. Bowels confined.

2 p.m.—T. 102·6°. 6 p.m.—T. 104°. Fifth bath.

7 p.m.—T. 100°. 10 p.m.—102·8°.

August 30th, 2 a.m.—T. 104°. Sixth bath. 3 a.m.—T. 99·2°.

6 a.m.—T. 102·6°.

10 a.m.—T. 104°. Seventh bath. After bath temp. 101°.

Did not sleep well. Bowels not open. Tongue moist. Abdomen rather distended; tenderness in left iliac fossa. Numerous rose-spots. Takes her food well. Urine sp. gr. 1017, acid, no albumen.

2 p.m.—T. 103·6°. 6 p.m.—T. 104°. 10 p.m.—T. 103·8°.

August 31st, 2 a.m.—T. 102°.

6 a.m.—T. 102·6°. Bowels twice moved; a little blood with one motion. Tongue moist. Abdomen distended. Slept fairly well. Pulse 112, small.

10 a.m.—T. 102·2°.

2 p.m.—T. 103°. Ordered Quin. sulph., gr. xx and Tinct. Opii, gr. x.

6 p.m.—T. 103·4°. 7 p.m.—T. 102·6°. 10 p.m.—.T 101 4°.

September 1st, 2 a.m.—T. 100·2°. 6 a.m.—T. 97·2°. 10 a.m.—T. 98·6°. Vomited once during night; bowels not open. Tongue less furred. Abdomen softer; more tender on left than on right side. Pulse 104, dicrotic.

2 p.m.—T. 100·8°. 6 p.m.—T. 102·6°.

10 p.m.—T. 103°. Eighth bath. 11.15 p.m.—T. 100·8°.

September 2nd, 2 a.m.—T. 102·2°. 6 a.m.—T. 102·4°.

10 a.m.—T. 102·8. Ninth bath. After bath temp. 99·2°.

2 p.m.—T. 103·4°. Tenth bath, twenty-five minutes.

6 p.m.—T. 102·6°.

10 p.m.—T. 103·4°. Eleventh bath, fifteen minutes.

11 p.m.—T. 101°.

September 3rd, 2 a.m.—T. 102·4°. 6 a.m.—T. 103·2°. Twelfth bath.

7 a.m.—T. 99·8°. 10 a.m.—T. 102°, pulse 112. Bowels once moved; no blood; less abdominal tenderness.

2 p.m.—T. 103°. Thirteenth bath. 3.30 p.m.—T. 100·8°.

6 p.m.—T. 102·4°. 10 p.m.—T. 103·2°. Fourteenth bath.

11 p.m.—T. 98·4°.

September 4th, 2 a.m.—T. 102·8°. 6 a.m.—T. 102·2°.

10 a.m.—T. 103°, pulse 120. Fifteenth bath. Bowels not open. Shivering slightly.

11.30 a.m.—T. 100°. 2 p.m.—T. 102·4°. 6 p.m.—T. 103·4°.

About 5.45 she had an attack of shivering, lasting more than a hour.

7 p.m.—T. 102·8°. 10 p.m.—T. 102°.

September 5th, 2 a.m.—T. 102·4. 6 a.m.—T. 102·8°.

10 a.m.—T. 102·4°, pulse 130, full. Passed a quiet night. More headache and cough.

2 p.m.—T. 100·8°. 6 p.m.—T. 102°. 10 p.m.—T. 102°. Headache worse.

September 6th, 2 a.m.—T. 102·4°. 6 a.m.—T. 102°.

10 a.m.—T. 102·8°, pulse 120. Did not sleep much. Headache less. Is apathetic, lying low in bed with knees raised. Fresh spots have appeared. Small trace of albumen in urine. No dulness at base of lungs. Scanty rhonchi.

2 p.m.—T. 102·8°. 6 p.m.—T. 103·6°. A compress applied to abdomen.

10 p.m.—T. 101·8°, pulse 120. Bowels twice moved; scanty, semi-solid motions.

September 7th, 2 a.m.—T. 103·6°. Compress now employed, and repeated during the next two days at frequent intervals. The temperature throughout the 7th and 8th was thus kept about 102°, but the pulse-rate rose.

September 9th, 2 a.m.—T. 104°. Compress. 6 a.m.—T. 99·8.

10 a.m.—T. 102·2°, pulse 140. Compresses re-applied and continued till 6 a.m. next day, being changed every ten minutes.

10 p.m.—T. 104·6°; so Quin. sulph., gr. x, given; which repeated at 3 a.m. on 10th.

September 10th, 3 a.m.—T. 102·8°. 6 a.m.—T. 100°.

10 a.m.—T. 99·8°, pulse 134, less dicrotic. Night disturbed owing to changing of compresses. Is cheerful; and tongue is cleaner.

2 p.m.—T. 101·4°.

6 p.m.—T. 103·8°. Sixteenth bath, 75°, for ten minutes.

7.30 p.m.—T. 99·4°. 10 p.m.—T. 103·6°, pulse 124. Quin. sulph., gr. x.

11.15.—T. 101·2°.

September 11th, 2 a.m.—T. 103·4°. Seventeenth bath.

6 a.m.—T. 101·4. 10 a.m.—T. 103·2. Has been shivering at times for the last two hours. Bowels open twice in the night, stools very loose and pale.

11 a.m.—T. 103·4°. Eighteenth bath. After bath, T. 101·6°. Urine sp. gr. 1015, acid, turbid, containing pus.

1.30 p.m.—Pulse 148, very compressible. Aspect depressed. Faint flush on cheeks and neck; and a marked erythema over the arms and upper part of each forearm and back. Lips covered with sordes. Tongue dry. First heart-sound short. Abdomen tumid. Täche well marked. Spleen easily felt.

2 p.m.—T. 103·4°. Nineteenth bath. After bath, T. 101·4°.

6 p.m.—T. 103·2°. 10 p.m.—T. 102°. Less depressed. Sleeps at intervals.

September 12th, 2 a.m.—T. 104°. Twentieth bath.

3.20 a.m.—T. 102°. 6 a.m.—T. 102·8°. Quin. sulph., gr. x.

10 a.m.—T. 101·4°, pulse 160, wavy; resp. 40. Aspect depressed. Abdomen more distended. No fresh spots. Dozed at intervals during night; no continuous sleep; no delirium. Bowels once open; stools loose. Ordered mixture containing digitalis.

3 p.m.—T. 102·4°. 6 p.m.—T. 103·4°, pulse 160.

7 p.m.—Twenty-first bath. 9 p.m.—T. 103·4°. Given Quin. sulph., gr. x, but vomited at once. At 10 p.m. given Liq. Opii Sed., ℥ xv, which produced sleep.

September 13th, 2 a.m.—T. 102·4°. 6 a.m.—T. 101°.

9 a.m.—T. 101°, pulse 144. Bowels opened twice in last twenty-four hours. Lips and tongue very dry and brown. Heart-sounds short but distinct. Abdomen distended. Champagne given instead of brandy.

3 p.m.—T. 103·6°. Twenty-second bath. 3·30 p.m.—T. 99°.

6 p.m.—T. 104·2°. Twenty-third bath. 6.30 p.m.—T. 101·8°.

9 p.m.—T. 102·6°, pulse 136. Feels easy. One large motion, loose and pale. At 10 p.m. opiate repeated.

September 14th, 3 a.m.—T. 101·6. 6 a.m.—T. 104°. Twenty-fourth bath.

7.15 a.m.—T. 98·6°. 9 a.m.—T. 101°. Sleeping quietly.

12.—T. 103·2°. Cold sponging.

3 p.m.—T. 103·4°. Twenty-fifth bath. 3.30 p.m.—T. 100·8°.

6 p.m.—T. 102·8°. 9 p.m.—T. 102·2°, pulse 156. Bowels open, stools pale and formed. Opiate given but rejected.

September 15th, 3 a.m.—T. 102·4°. 6 a.m.—T. 100·8°.

9 a.m.—T. 104°. 10 a.m.—Twenty-sixth bath. 10.30.—T. 99°.

11 a.m.—Drowsy. Lips very dry and covered with sordes. Pulse 140, resp. 36. Abdomen more distended. Abundant crop of rose-spots.

3 p.m.—T. 103°. Compresses now again applied to abdomen and maintained more or less during rest of pyrexial period. Bowels twice moved; stools semi-formed. Urine acid, sp. gr. 1022, contains more albumen.

6 p.m.—T. 102·4°, pulse 160. 9 p.m.—T. 102·6°.

September 16th, 6 a.m.—T. 102·4°. 10 a.m.—T. 100·6, pulse 160, running and very dicrotic, resp. 36.

To-day a fresh and copious eruption of rose-spots appeared. The bronchitic râles were more abundant; the cardiac sounds weaker, so that it was deemed advisable to considerably increase the stimulant. The tongue was very dry, brown, and cracked. Abdomen distended and tender. Bowels opened in the morning, motion formed, and twice in the evening, the last being a liquid, blood-stained motion. Compresses still continued.

September 17th.—Slept fairly well. Bowels open twice during the night; one motion slightly blood stained. Pulse counted at heart, 168. The digitalis mixture omitted. 11 a.m.—Pulse 140.

September 18th and 19th.—Her condition had now become very critical, owing mainly to the very marked feebleness of the heart and signs of increasing pulmonary congestion; dulness and moist râles appearing at the bases. The temperature only once reached above 103°; mostly between 101° and 102°. She still continued to pass blood in the motions, but not in large quantity. On the 19th there was muttering delirium (for the first time), the pulse very rapid, 160; respiration 40, and shallow. Stimulant was increased, and her position altered.

September 20th to 24th.—She now began slowly to improve; the pulse falling to 140 and 128; the pyrexia also took a lower range, and the chest-signs to clear up. On the 24th stimulant was diminished. She slept better, and on the 25th the temperature at 9 a.m. was 98·6°. Rose-spots continued to appear until the 29th. Defervescence did not really

take place till September 29th, *i.e.* in the seventh week of the fever, and after that a mild relapse occurred from October 6th to October 12th. She sat up for the first time on October 27th, having taken solid food since the 22nd (Plate VII).

Convalescence was retarded by a limited periostitis over the left tibia, signs of which appeared on November 5th. A tender reddened swelling indicated this. Much relief was given by a subcutaneous incision by Mr. Gould on November 9th, and the part was painted with iodine. On the 19th a drop of pus was obtained by a hypodermic syringe, but a free incision was never required, and she left the hospital, fairly strong and well, December 11th, for Eastbourne.

Case 4. *Enteric fever, with intercurrent attack of "rötheln;" marked asthenia. Nineteen baths; quinine.*—Charlotte S—, aged thirty-one, parlour-maid; admitted October 25th, 1880, on eighth day, but had been indisposed for some weeks. On the 17th she was sick, shivered, and took to her bed; then had much anorexia, and thirst, severe headaches, insomnia; some cough. Had never been ill before.

On admission.—Temp. 104·6°, pulse 132, resp. 40. Dark hair, flushed cheeks, dry lips, depressed aspect; headache, thirst, anorexia, abdominal pain, and slight cough. Scanty bronchitic râles. Soft systolic bruit at apex of heart. Abdomen distended; tender. Skin hot and dry; a few rose-spots. Spleen not to be felt. Pulse soft and compressible.

6 p.m.—T. 104·8°. Bath 70° F., given for ten minutes.

6.30 p.m.—T. 101·8°.

9 p.m.—T. 103·4°. Second bath. 10 p.m.—T. 101·4°; pulse 102.

12 midnight.—T. 103·4°. Third bath, followed by Quin. sulph., gr. xx.

October 26th, 1 a.m.—T. 102°.

3 a.m.—T. 103·2°. Fourth bath. 4 a.m.—T. 100·4°. Tongue more moist. Temperature remained about 101° thoughout the day rising to 102·6° at 10 p.m.

October 27th, 4 a.m.—T. 103·2°. Fifth bath. 70°, for fifteen minutes. Retention of urine, requiring catheterism. Bowels opened once; motion pale brown and liquid. Temperature during day about 102°.

11 p.m.—T. 104·2°. Sixth bath, 70° F., for fifteen minutes.

12.15.—T. 98·8°.

October 28th, 4 a.m.—T. 103·8°. Seventh bath. 5.15 a.m.—T. 99·6°.

9 a.m.—T. 102·4°, pulse 112. Much prostration. First cardiac sound feeble. Bowels once moved. Scanty rose rash. Cheeks flushed.

11 a.m.—T. 101·8°. 3 p.m.—T. 102·4°. 7 p.m.—T. 102·6°.

9 p.m.—T. 104°. Eighth bath. 9·30.—T. 98·8°. Bowels open.

October 29th, 12 midnight.—T. 103°. Ninth bath.

1.30 a.m.—T. 101°.

7 a.m.—T. 103·4°. Tenth bath. 8 a.m.—T. 99.

10 a.m.—T. 99·8°, pulse 104, very small. Bowels twice moved. Has expectorated scanty blood-stained mucus. Is drowsy.

2 p.m.—T. 103·4°. Eleventh bath. 3 p.m.—T. 99°.

7 p.m.—T. 105°. Twelfth bath. 8 p.m.—T. 98·8°.

October 30th, 12 midnight.—T. 103·4°. Thirteenth bath.

1 a.m.—T. 99·8°.

5 a.m.—T. 102·6°. 7 a.m.—T. 102·2°. 9 a.m.—T. 100·8°.

11 a.m.—T. 101·8°, pulse 114. First heart-sound almost inaudible. Bowels once open, motion liquid. Occasional crackling râles in front

of chest; no dulness; scanty reddish-brown sputum. Fresh crop of spots.

5 p.m.—T. 103°. Fourteenth bath. 6 p.m.—T. 99·8°.

October 31st, 12 midnight.—T. 103·4. Fifteenth bath. 1 a.m.—T. 97·8°.

5 a.m.—T. 102·2°. 9 a.m.—T. 101·8°.

11 a.m.—T. 102·4°, pulse 100. Bowels twice opened. Increased apathy. Purplish circumscribed flesh on cheeks and nose. A measly rash has appeared on chest. Tongue moister. Pulse very feeble.

1 p.m.—T. 101·2°. 5 p.m.—T. 102°. 9 p.m.—T. 102·6°.

11 p.m.—T. 103°. Sixteenth bath. 12 midnight.—T. 97·6°.

November 1st, 4 a.m.—T. 102°. 6 a.m.—T. 102·6°. 8 a.m.—T. 102°. Patient lies in apathetic state. Bowels are slightly opened. Seen by Dr. Cayley, who pronounced rash to be that of rötheln; it has now extended to legs, and a few papules are seen in pharynx.

1 p.m.—T. 101·8°. 3 p.m.—T. 102°. 5 p.m.—T. 102·6°.

7 p.m.—T. 103·6°. Seventeenth bath. 8 p.m.—T. 99°.

9 p.m.—T. 98·4, pulse 108. Bowels opened twice.

November 2nd, 2 a.m.—T. 102·2°. 5 a.m.—T. 102°. 8 a.m.—102·4°.

10 a.m.—T. 101·4°, pulse 120. Rash fading. Soft systolic murmur at apex of heart. Tongue dry and fissured. Face still flushed. Feels better.

12 noon.—T. 102·6°. 4 p.m.—T. 102·4°. 8 p.m.—T. 102°, pulse 126.

10 p.m.—T. 103·4°. Eighteenth bath. 11 p.m.—T. 98·2°.

November 3rd, 1 a.m.—T. 100·8°. 3 a.m.—T. 102·2°.

4.30 a.m.—T. 103·2. Nineteenth bath. 6 a.m.—T. 97°.

7 a.m.—T. 99°. 10 a.m.—T. 101°, pulse 120. Bowels not open since the 1st.

November 4th.—She now began to decidedly improve. Pulse and heart gained in force and former in volume. Prostration less. Pyrexia took a lower range, between 100° and 101°.

November 5th.—Continued improvement; maximum temperature 102·4° at 10 p.m. An enema given owing to constipation and pain in belly.

November 6th.—At 4 a.m., temp. 103°, but after this it ranged about 101°. The bowels had been well opened by the enema, but abdomen remained much distended and she suffered from cramp in her legs.

November 7th.—Rambled slightly in her sleep. Temperature between 101° and 102°.

November 8th to 10th.—Continued improvement in strength; tongue clean and moist; but more bronchitis. Skin desquamating. Constipation requiring enemata. Temperature several times 99°.

Convalescence now set in; temperature falling to normal on the 11th. She slowly regained strength; the apex murmur did not disappear, and she left for Eastbourne on December 6th.

Case 5. *Enteric fever; much bronchitis; some intestinal hæmorrhage; great debility. Eighteen baths; sponging; quinine.*—William B—, aged twenty-six, married, bricklayer; admitted July 12th, 1881, in the second week of fever. Previous health good, and habits temperate. His illness began about a fortnight before, with pain in back, loss of appetite, thirst, vomiting, cough, loss of strength, and latterly relaxed bowels.

On admission.—T. 102°, pulse 120, resp. 24. Dark-haired, muscular,

but prostrate; tongue thickly coated; abdomen not distended; no increase of splenic dulness; numerous acne spots. Numerous bronchitic râles; weak breathing at bases. Short cough with muco-purulent expectoration.

6 p.m.—T. 103°. Sponged with tepid water, which repeated whenever temperature rose to 103°.

9 p.m.—T. 103·5°. 12 midnight.—T. 103·4°.

July 13th, 3 a.m.—T. 103°. 6 a.m.—T. 101·2°. 9 a.m.—T. 102°.

12 noon.—T. 104·2°, pulse 108. Bowels twice opened, motions dark and containing altered blood. Much bronchitis.

2 p.m.—T. 103·6°. Given Quin. sulph., gr. xx, in two doses. Vomited in afternoon. Recurrence of hæmorrhage from bowel in evening. No pain or tenderness.

July 14th, 3 a.m.—T. 100°. 9 a.m.—T. 98°. Bowels not open. Appearance of two suspicious rose-spots. Bronchitis increased. Maximum temperature 101° at 9 p.m., pulse 108, resp. 48.

July 15th to 20th.—There was no recurrence after hæmorrhage, but the abdomen became more distended and the tongue dry. The bronchitis diminished and the pyrexia was mild, ranging between 98·2° and 101°.

July 21st.—Maximum temperature 99·8° at 9 p.m. Cough less; physical sign mainly prolonged expiratic and sibilant rhonchi. Tongue moister, and patient complaining of languor. Bowels confined.

July 25th to 26th.—A relapse set in. Patient had become very thin. Temperature 99° at 9 a.m., rose to 102° at noon, and for many days remained above this figure. At noon on 26th, T. 103°; 9. p.m., 103·6°, pulse 120, and complained of pain in belly. Given Quin. sulph., gr. x.

July 27th, 3 a.m.—T. 100·6°. 6 a.m.—T. 102°. 9 a.m.—T. 102·6°. Abdominal pain continued. Bowels once open. Face flushed. Urine, sp. gr. 1015, acid; no albumen.

9 p.m.—T. 103·6°. Quin. sulph., gr. x.

July 28th, 3 a.m.—T. 103·2°. Quinine repeated.

9 a.m.—T. 101·4°, pulse 100. Lips covered with sordes. Tongue dry. Still abdominal pain and some sickness. Emaciation progressing.

12 noon.—T. 102·4°. 2 p.m.—T. 103·2°.

6 p.m.—T. 103·6, pulse 96°.

July 29th.—No change. Temperature between 102° and 103°, and at midnight 104°.

July 30th, 3 a.m.—T. 102·6°. 6 a.m.—T. 103°.

9 a.m.—T. 102·6°, pulse 100. Quinine had been continued at intervals without much effect.

3 p.m.—T. 103·8°. Bath, 78°, for fifteen minutes. T. 99° half hour after.

9 p.m.—T. 103°. Second bath. 9.30.—T. 100·6°.

July 31st, 3 a.m.—T. 104°. Third bath. 2.30.—T. 100°.

6 a.m.—T. 102·6°. 8 a.m.—T. 102·8°.

10 a.m.—T. 102·6°, pulse 120. Tongue thickly coated; bowels open, motions pale and loose. More bronchitic râle and rhonchus.

4 p.m.—T. 101·2°.

8 p.m.—T. 103·8°. Fourth bath. 8.30.—T. 100·4°.

August 1st, 12 midnight.—T. 104°. Fifth bath. 2.30.—T. 102·4°.

4 a.m.—T. 105°, pulse 136. Sixth bath, for twenty minutes.

4.30 a.m.—T. 98·6°. 6 a.m.—T. 101·2°. 8 a.m.—T. 102·4°.

10 a.m —T. 103·4°. Seventh bath.

10.30 a.m.—T. 100°, pulse 108, resp. 30. Spleen felt below ribs. Tongue thickly coated. Has a depressed aspect. Some tenderness in right iliac region. Cough rather troublesome; râles more abundant.

4 p.m.—T. 103·4°. Eighth bath. 4.30.—T. 100·4°.

6 p.m.—T. 102°. 8 p.m.—T. 102·6°.

10 p.m.—T. 104·6°. Ninth bath. 10.30.—T. 100.

August 2nd, 6 a.m.—T. 103·4°, pulse 132. Tenth bath. 6.30.—T. 99·2°.

10 a.m.—T. 100°, pulse 100, resp. 20. Tongue red at tip and edges, dry and brown in centre. Cough worse; bronchitic signs more marked. Several typical rose-spots have appeared on abdomen.

2 p.m.—T. 101·6°. 4 p.m.—T. 102·6°.

6 p.m.—T. 104°. Eleventh bath. 6.30.—T. 99·8°.

August 3rd, 12 midnight.—T. 104°. Twelfth bath. 12.30.—T. 98·4°.

10 a.m.—T. 103·2°. Thirteenth bath.

10.30—T. 98·6°, pulse 114, resp. 28. Spots fading. Abdomen not distended. Motions loose, light brown in colour.

12 noon.—T. 101·6°. 6 p.m.—T. 103·4°. Fourteenth bath.

6.30 p.m.—T. 101°, pulse 102, dicrotic. 10 p.m.—T. 102·6°.

12 midnight.—T. 103·6°. Fifteenth bath. 12.30 a.m.—T. 98°.

August 4th, 2 a.m.—T. 101°. 4 a.m.—T. 101·4°. 6 a.m.—T. 102°.

10 a.m.—T. 102·6°, pulse 144. Tongue very dry and brown. Still much bronchitis. First cardiac sound very weak.

12 noon.—T. 102·8°.

2 p.m.—T. 103·6°. Sixteenth bath. 2.30.—T. 97·6°.

7 p.m.—T. 103·6°. Seventeenth bath. 7.30.—T. 98·2°.

August 5th.—The temperature during greater part of day maintained a lower level, but it was 103·4° at 11 a.m., and at 8 p.m., T. 103·6°. Eighteenth bath. 8.30 p.m.—T. 99·6°. Midnight.—T. 102°.

August 6th.—Bronchitis still marked; and tongue brown and dry, but pyrexia less. At 2.30 p.m.—T. 98·8°; 10 p.m.—T. 102·4°, pulse 120.

August 7th.—Morning temperatures—100° to 101°; evening, reaching to 102·6°.

August 8th to 10th.—Although the bronchitis persisted the pyrexia was abating much; temperature falling to 97·6° on the 9th. On the 10th eggs were added to diet.

From August 11th the fever passed away, and temperature became subnormal. Only a few dry rhonchi could occasionally be heard in chest. He insisted on leaving on August 18th, although he was too feeble to walk.

Case 6. *Enteric fever; relapse in fifth week; cardiac asthenia; high pyrexia; slow convalescence. Seventeen baths; sponging; quinine.*—Catherine M—, aged twenty-one, married, housewife. Admitted January 24th, 1882, on eighth day. She had always enjoyed good health. Had been confined eight weeks before. For past two weeks her appetite had fallen off, and eight days ago she felt cold and vomited. No pain or diarrhœa.

On admission.—Temp. 103°. Depressed aspect; face dusky; pulse rapid and compressible. Rose-spots on abdomen, which is distended. Breath-sounds feeble; no râles.

9 p.m.—T. 103·8°. Bath given. 10.30.—T. 102·8°.

January 25th, 12.30 a.m.—T. 105·3°. Second bath. 1 a.m.—T. 101·2°.

4 a.m.—T. 104. Quin. sulph., gr. x. Sponging. 5 a.m.—T. 102°.

7 a.m.—T. 103·6°. Sponged. 8 a.m.—T. 100°. Delirious during the night.

10 a.m.—T. 102·2°, pulse 100, resp. 50. Pulse compressible; cheeks flushed; no lung signs.

6.30 p.m.—T. 103·6°. Third bath for seven minutes.

8 p.m.—T. 102·6°.

10 p.m.—T. 104°. Fourth bath for seven minutes.

11 p.m.—T. 101°.

January 26th, 1 a.m.—T. 104·6°. Sponged. 2 a.m.—T. 101·8°.

3 a.m.—T. 103°. 6 a.m.—T. 101·8°.

10 a.m.—T. 101·8°. Slept at intervals in night. Bowels opened; motion loose and pale.

3 p.m.—T. 104·2°. Fifth bath for twenty minutes.

4 p.m.—T. 101·4°.

10 p.m.—T. 103·2°. Quin. sulph., gr. x. 12 midnight.—T. 101·8°.

January 27th, 3 a.m.—T. 102·8°. Quin. sulph., gr. x.

10 a.m.—T. 102·4°, pulse 96. Bowels once open. Looks brighter.

2 p.m.—T. 102·4°. 6 p.m.—T. 102·8°.

10 p.m.—T. 103·8°. Sixth bath, 80°, for twenty minutes.

11.30 p.m.—T. 102·2°.

January 28th, 2 a.m.—T. 104·2°. Seventh bath. 4 a.m.—T. 101°.

10 a.m.—T. 100·2°, pulse 100, regular, somewhat dicrotic. Tongue dry and brown. Abdomen not distended. Bowels are open.

2 p.m.—T. 102·8°. 10 p.m.—T. 102°.

January 29th, 4 a.m.—T. 102·6°. 10 a.m.—T. 101·8°.

2 p.m.—T. 100°.

6 p.m.—T. 104·4°. Eighth bath. After bath temp. 103·4°.

10 p.m.—T. 103·8°. Ninth bath. After bath temp. 101·8°.

January 30th, 6 a.m.—T. 103°. Tenth bath. After bath temp. 101°. Tongue dry and brown. Abdomen tympanitic. Fresh eruption.

2 p.m.—T. 102·8°. 6 p.m.—T. 102°.

January 31st.—Maximum temp. 102·2°, pulse 108.

February 1st, 6 a.m.—T. 102·8°. 10 a.m.—T. 104°; pulse 108. Eleventh bath. 11.45.—T. 100·2°.

2 p.m.—T. 103·8°. Twelfth bath. 6 p.m.—T. 102·4°.

February 2nd.—Temperature ranging between 101° and 102·6°. Bowels, which had been confined for four days, were opened involuntarily twice. Given brandy ʒiv daily.

February 3rd, 10 a.m.—T. 104°. Thirteenth bath. After bath temp. 99·4°.

February 4th to 11th.—The pulse became weaker and stimulant was increased. The fever now took a lower range, falling from 101° to 99° on the 9th. A bedsore formed on the right buttock on the 11th.

February 12th to 19th.—A week of apyrexia; improved appetite; allowed rusks in diet. A relapse now set in. T. 102° on 20th.

February 21st, 10 a.m.—T. 102·6°. 2 p.m.—T. 101·8°.

6 p.m.—T. 103·6°. Sponged. The bedsore was healing well. Bowels confined, and an enema prescribed.

2 p.m.—T. 103·4°.

February 22nd, 10 a.m.—T. 103°. 2 p.m.—T. 105·4° Fourteenth bath. After 103°.

6 p.m.—T. 105°. Fifteenth bath. After bath 103·2°.

10 p.m.—T. 102·8°.

February 23rd, 2 a.m.—T. 104·2°. Sixteenth bath. After temp. 100·6°.

10 a.m.—T. 104°. Seventeenth bath.
6 p.m.—T. 104°. Quin. sulph., gr. x.
7.30 p.m.—T. 103°. Quin. sulph. repeated.
10 p.m.—T. 102·6°.
February 24th.—Tongue moist; temp. not above 102·2°.
February 25th, 2 a.m.—T. 102·4°. 6 a.m.—T. 101·6°.
10 a.m.—T. 103·6°, pulse 108.
2 p.m.—T. 104·8°. Quin. sulph., gr. x.
3 p.m.—T. 104°. Repeat Quin. sulph.
6 p.m.—T. 103°. 10 p.m.—T. 102·4°.
February 26th, 27th.—On each of these days the temperature rose to 104·4° at 2 p.m., when quinine was given. On the 28th it fell to 100°.
March 1st.—Pyrexia again increased; 104° at 2 p.m. Quinine, 20 gr., given, and for some days after, during which pulse became very feeble and rapid (132) and with cough and bronchitic râles in chest gave some anxiety. A decided improvement took place on March 7th, and although convalescence was rather slow it now took place uninterruptedly. Oysters were given on the 7th, and a chop on the 10th, but she did not leave the hospital until April 20th.

Case 7. *Enteric fever; relapse of very severe type; hyperpyrexia; pulmonary congestion; death on tenth (?) day of relapse. Seventeen baths; sponging; compresses; quinine.*—John S—, aged seventeen, a carpenter, admitted October 26th, 1882, in the fourth week. His previous health had been good. The attack commenced four weeks before with headache and abdominal pain. He saw a chemist, who told him he was "feverish," and gave him some physic. But he continued to work at his employment until the 25th, when he consulted a medical man, who told him that he had typhoid fever, and advised his admission. The headache had persisted more or less continuously throughout this period, and he had also several repeated attacks of abdominal pain. The appetite continued fair until the 22nd, when he began to lose it. Since the 20th there has been diarrhœa, and on the 25th he was sick. He had an attack of shivering the same evening. Has slept badly. No cough.
On admission.—T. 104·8°, pulse 116, resp. 44. A stout, well-nourished, dark-haired lad, with deep red flush on cheeks. Skin very hot and dry; no eruption. Tongue dry and coated in centre with brown fur. Abdomen tumid, painful below epigastrium. Splenic dulness not increased. Chest resonant; a few scattered sonorous rhonchi occasionally heard. Heart-sounds normal.
Ordered milk and beef tea. Acetate of ammonia.
7 p.m.—T. 105°. 9 p.m.—T. 105·8°. 11 p.m.—T. 105·2°.
October 27th, 1 a.m.—T. 105°. 3 a.m.—T. 105·2°.
5 a.m.—T. 104·6°. Bowels open slightly; motion dark.
7 a.m.—T. 105·2°. 9 a.m.—T. 105·2°.
11 a.m.—T. 105·2°, pulse 112, resp. 28. Appearance of two rose spots on abdomen. To-day decided increase in area of splenic dulness, which measures five inches vertically. Urine, sp. gr. 1030, acid, no albumen.
1 p.m.—T. 105·8°. Placed in a bath at temp. 70° for fifteen minutes. T. 104·2° thirty minutes after removal from bath.
4 p.m.—T. 103·6°.

5 p.m.—T. 104·8°. Second bath, 70°, for fifteen minutes. T. 103·6° thirty minutes after bath.

7 p.m.—T. 104·4°. Third bath, 70°, for fifteen minutes. T. 103·6° thirty minutes after bath.

8.30 p.m.—T. 103·6°. Quin. sulphatis, gr. xx, in two doses.

9.30 p.m.—T. 103·8°.

10 p.m.—T. 104, pulse 114, full, bounding. He shivered violently after the bath and for half an hour, but face remained flushed. The flush is most on the left cheek, and is of a deeper colour. He is wakeful, but not delirious. Has a great aversion to the baths. Tongue dry and brown in centre, creamy at sides. Lips dry and covered with sordes. Heart's action tumultuous, forcible, sounds loud and rough, especially the first in region of apex. Abdomen more tumid; bowels not open.

10.30 p.m.—T. 104·2°. Fourth bath, 70°, for fifteen minutes. Complained of cramp in legs; shivered much in the bath and for half an hour afterwards.

11 p.m.—Quiniæ sulph., gr. xx.

12 midnight.—T. 103·8°.

October 28th, 1.30 a.m.—T. 103·4°. 2.30 a.m.—T. 103·8°.

3.30 a.m.—T. 103·8°. 4.30 a.m.—T. 103·2°. 5.30 a.m.—T. 102·6°.

6.30 a.m.—T. 102·2°. 7.30 a.m.—T. 101·2°.

8.30 a.m.—T. 101·6°, pulse 100, resp. 28. Slept for about one hour after the last bath. No delirium. Slight deafness and tinnitus. Complains of pain in umbilical region; no tenderness, and distension less marked. Face still intensely flushed. Lips and tongue very dry. Pupils large. Heart's impulse less forcible, more undulatory; sounds less loud, and pulse is softer. Bowels opened once in night; motion semi-solid and drab coloured.

12.30 p.m.—T. 103°, pulse 104. At 1 p.m. a compress wrung out of ice-water applied to left arm, and constantly renewed until

3.30 p.m.—T. 104°. Fifth bath.

4.30 p.m.—T. 103·2°. 6.30.—T. 103·4°. 8.30.—T. 104·4°.

9.30 p.m.—T. 103°. Compress reapplied to left arm.

10.30 p.m.—T. 104·6°. 11 p.m.—Quin. sulph., gr. xx.

12.30 p.m.—T. 103·6°.

October 29th, 1.30 a.m.—T. 102·8°. 2.30 a.m.—T. 103·6°.

4.30 a.m.—T. 103°. 6.30 a.m.—T. 102·6°. 7.30 a.m.—T. 103·8°.

8.30 a.m.—T. 103°.

10.30 a.m.—T. 103°. The iced compresses have now been constantly applied and renewed for twelve and a half hours continuously, the arm and hand being quite cold, the latter livid. Slept at short intervals, about two hours in all. Bowels opened once; motion semi-solid, pale drab colour. Flush on cheeks still marked, but more dusky red. Lips covered with sordes. Tongue dry and cracked. Intellect clear; no increase of deafness. A few fresh rose-spots have appeared on abdomen. Pulse 100, dicrotic, resp. 28. Slight working of alæ nasi. Weak breathing at bases of lungs; no dulness.

10.30 a.m.—T. 103°. Sixth bath.

1.30 p.m.—T. 103·4°. Bowels opened; motion loose, paler.

5.30 p.m.—T. 104·2°.

6 p.m.—T. 104°. Seventh bath.

Has borne the last two baths better than the preceding ones; much less shivering.

6.30 p.m.—T. 100·6°.
7.30 p.m.—T. 103·4°.
9.30 p.m.—T. 104·2°. Eighth bath. After this bath he slept for two hours.
October 30th, 2.30 a.m.—T. 103·8°. Involuntary passages.
3.30 a.m.—T. 104°. Ninth bath.
5 a.m.—T. 103·4°. Slept for nearly an hour.
7 a.m.—T. 103·8°.
9 a.m.—T. 104·4°. Tenth bath, followed by a short sleep.
12 noon.—T. 103·8°, pulse 120, weaker. Ordered brandy ℥ss every six hours. Takes beef tea and milk well. Fresh crop of rose spots on chest. Tongue and lips dry. No delirium. Urine, sp. gr. 1032, acid, no albumen.
2.30 p.m.—T. 104·2°. Eleventh bath.
4 p.m.—T. 103·1°. 5 p.m.—T. 102·8°. 6 p.m.—T. 103·8°.
7 p.m.—T. 105°. Twelfth bath, followed by Quin. sulph., gr. xx.
8.30 p.m.—T. 104·2°. 9 p.m.—T. 103·6°.
10.30 p.m.—T. 104·8°. Thirteenth bath. Pulse 124, sharper, dicrotic; resp. 48. Percussion note dull over lower one third of each back, where the breath-sound is weak; the resonance is also short and high pitched in each lower axilla. Brandy ℥ss every two hours.
11.30 p.m.—T. 102·6°.
October 31st, 1 a.m.—T. 104·5°. Fourteenth bath, temp. 85°, for thirty minutes.
2.30 a.m.—T. 102·4°. Slept for four hours.
6.30 a.m.—T. 102·8°.
7.30 a.m.—T. 103°. Sponged, which also produced slight shivering.
8.30 a.m.—T. 103·2°. Sponged.
9.30. a.m.—T. 101·8°.
11 a.m.—T. 104·1°, pulse 124, dicrotic. Bowels not open. Has snatches of sleep. Tongue very dry. No delirium.
12 noon.—T. 104·1°. Fifteenth bath, 85°, for thirty minutes.
2 p.m.—T. 103·7°.
3 p.m.—T. 104°. Sponged.
4 p.m.—T. 104·2°. Sponged.
5 p.m.—T. 104·4°. Sponged.
Slept for twenty minutes at 6 p.m.
6.30 p.m.—T. 104·7°. Sixteenth bath, 85°, for thirty minutes.
8.30 p.m.—T. 103·8°. Slept for an hour.
9.30 p.m.—T. 104·5°.
10.30 p.m.—T. 104·4°. Bowels moved involuntarily; motions very offensive, pale.
11.30 p.m.—T. 104·4°.
November 1st, 12.30 a.m.—T. 104·8°. Seventeenth bath, 85°, for thirty minutes. Bath better borne for first twenty minutes.
1.30 a.m.—Quin. sulph., gr. xx, in two doses at fifteen minutes' interval. Bowels again open; stools as before. Ordered Supposit. Opii, gr. j.
3 a.m.—T. 104°. Slight delirium, followed at 4 a.m. by a four hours' sleep.
8 a.m.—T. 103°.
10 a.m.—T. 103·4°. Slept for one hour.
11.30 a.m.—T. 104°. 12.30.—T. 103·6°. Sponged.
1.30 p.m.—T. 102·8°. 2.30 p.m.—T. 104°, pulse 132, resp. 24. Pulse sharp and short. Dulness still marked in lower axilla and over

lower one third of both lungs, with weak breathing; no râles. Heart-sounds fairly distinct, the first being murmurish. Marked rose rash on chest. Tongue remains dry and glazed, and lips and teeth covered with sordes. No delirium since 4 a.m.; is more talkative and brighter.

3 p.m.—Sponged. Bowels open.

Slept for two hours from 4.30 p.m.

6.30 p.m..—T. 104°. Sponged.

For next five hours temp. about 103°, and bowels frequently opened involuntarily. Slept at intervals.

November 2nd, 5 a.m.—T. 102·5°. Bowels opened voluntarily. Suppository repeated. Slept from 5 to 7.30 a.m. T. 102·4°.

8.30 a.m.—T. 102·4°, and again slept for two hours.

1 p.m.—T. 103·2°.

2 p.m.—T. 103·8°, pulse 148, small, quick; resp. 20, deep, regular. Lies in a drowsy state, but can easily be roused. Pupils small. Lips still very dry and cracked. Tongue moist at margins, brown and cracked in centre. Complains of nausea, and unable to take beef tea. Abdomen tympanitic. Bowels not open since suppository. There has been slight epistaxis. Given Brand's essence of beef. Dilute mineral acids. Brandy ʒss every ninety minutes.

3 p.m.—T. 103·4°. 4 p.m.—T. 104·2°. Sponged.

8 p.m.—T. 103·6°. 9 p.m.—T. 103·4°.

10 p.m.—T. 102·8°. Slept till 12.

November 3rd.—Slept for nearly two hours till 2 a.m. T. 103°.

4 a.m.—T. 102·6°.

5 a.m.—T. 102·8°. Has vomited coffee-ground material (? blood swallowed from epistaxis). Is continually picking his nose and the bed-clothes, but is not delirious.

5.30 a.m.—Bowels open involuntarily.

7.30 a.m.—T. 101·8°. 8.30.—T. 102·6°. 10.30.—T. 103·1°.

12.30.—T. 102·6°, pulse 150, very small. No recurrence of vomiting. No delirium. Hearing is acute. Continues to pick bed-clothes, &c. Tongue very dry and glazed. Resp. 28, tranquil.

He died the following day, November 4th (Plate VIII).

Post-mortem examination.—Body fairly nourished. Commencing decomposition. Muscles dark. About an ounce of dark brown serum in peritoneal cavity. No peritonitis. About six ounces of very deeply blood-stained fluid in left pleural cavity, three ounces in the right; no adhesions. About three ounces of dark brown serum in pericardium. Parietal layer of right pleura extremely vascular, with numerous points of ecchymosis; also in left side. Cardiac cavities contained mostly fluid blood, and endocardium was blood stained. Muscular tissue firm. Valves normal. Bronchi full of dark-brown, blood-stained fluid slightly mixed with air.

Case 8. *Enteric fever in phthisical subject, otherwise uncomplicated; pyrexia high. Seventeen baths; sponging; compresses; quinine; kairin.* —Sarah B—, aged eleven, living at home, admitted October 24th, 1883, in second week. She was always delicate, and six years before was in hospital for inflammation of the lungs; since then had shown phthisical symptoms. About a fortnight before her admission she began to suffer from headaches, then from anorexia, vomiting, and for past three days diarrhœa with abdominal pain.

On admission.—T. 103·8°, pulse 120, resp. 24. Small, thin, fair,

pale, with dry lips, coated tongue, anorexia, thirst. General bronchitis; impaired resonance with crepitation at left anterior apex. Cardiac sounds week.

October 25th.—Bowels open once; stools pale and liquid. Temperature ranged about 102·5° before noon, but afterwards rose above 103°, for the reduction of which kairin in five-grain doses was given at 5, 6, and 7 p.m., and again at 11 p.m., and 1 and 2 a.m. She became delirious, and it was therefore discontinued.

October 26th, 2 a.m.—T. 100·6°. Fifth dose of kairin given.

3 a.m.—T. 99·6°. 6 a.m.—T. 99·4°.

10 a.m.—T. 103·6°. Compress applied to abdomen. Pulse 120, resp. 30. Bowels open thrice; stools yellow, liquid. Rambled much in night. Abdomen distended.

2 p.m.—T. 103·4°. Bath given, 75°, for ten minutes.

3.30 p.m.—T. 101·4.

6 p.m.—T. 104·2°. Second bath.

8.30 p.m.—T. 101°. Bowels twice moved. Pulse 116, resp. 32. Mild delirium throughout the day, easily raised.

10 p.m.—T. 104·2°. Third bath, 75°, fifteen minutes.

11.30 p.m.—T. 98°.

October 27th, 2 a.m.—T. 103·2°. Compress applied.

10 a.m.—T. 103·2°. Fourth bath at 12.30.

Passed a restless night. Bowels twice open. Much thirst. Tongue dry. 1 p.m.—T. 99·4°.

6 p.m.—T. 103·8°. Fifth bath. 8 p.m.—T. 101·6°.

10 p.m.—T. 103·6°. Slept a little after bath, and delirium became less. Complains of headache.

October 28th, 2 a.m.—T. 103·6°. Compresses renewed and continued.

10 a.m.—She was now very depressed and prostrate. Pulse 108, small and compressible, resp. 30. Occasional cough; sibilant rhonchi all over chest. Heart-sounds very weak. Numerous rose-spots. Abdomen distended. Tongue glazed. Brandy ʒiv daily.

2 p.m.—T. 103°. Sixth bath. 3.30 p.m.—T. 101.

6 p.m.—T. 104·8°. Seventh bath. 7.30 p.m.—T. 97·8°.

9.30 p.m.—T. 103°.

October 29th.—Eighth, ninth, tenth, eleventh baths, each 75°, for ten minutes, given at 2.30 a.m. (T. 103°), 9.30 a.m. (T. 103·2°), 5 p.m. (T. 103°), 8.30 p.m. (T. 103°), and occasional sponging whenever temperature rose above 102° between the baths. She slept at intervals during the day.

October 30th.—Twelfth, thirteenth, and fourteenth baths, given at 1 a.m. (T. 103·2°), 6 a.m. (T. 103·6°), and 10 p.m. (T. 103·2°), and sponging several times, with rising temperature. Diarrhœa. No fresh eruption. Pulse 120, resp. 176. Less depressed.

October 31st.—Temperature between 100° and 101°. No baths given.

November 1st, 1 to 10 a.m.—T. 101·6° mostly. Diarrhœa has ceased, and flush no longer on cheeks. Pulse 125, resp. 24.

1 p.m.—T. 103·8°. Fifteenth bath, 75°, for fifteen minutes. Half hour later 98·9°.

7 p.m.—T. 104·2°. Sixteenth bath. Half hour later 98·6°.

November 2nd, 1 a.m.—T. 103·6°. Seventeenth bath. Half hour after 99°.

No more baths were given, the temperature remaining mostly between 101° and 102°, and when rising to latter level reduced by

sponging. Defervescence took place on the 9th. The child had become very emaciated, and the persistence of pulmonary signs raised the fear that her condition might pass into ostitis. Convalescence was further hampered by otitis, but she took food well, and slowly regained strength. She did not leave her bed till December 12th, and was not sent to a convalescent home until December 31st.

Case 9. *Enteric fever following pneumonia; much cardiac depression. Fourteen baths; sponging.*—William R—, aged fifteen, schoolboy. Admitted February 20th, 1880, about the tenth day of the fever. He had been an in-patient with left pleuro-pneumonia from December 29th, 1879, to January 30th, so that unless an unusually prolonged incubation period be admitted in his case it seems hardly possible that he contracted the disease in the hospital. The fever began very definitely on February 11th, *i.e.* twelve days after leaving the hospital.

On admission.—T. 103·2°, pulse 120, resp. 32. A fairly well-nourished lad; weak; pain in left side of chest; anorexia; thirst; anxious. Coarse rhonchi audible on breathing; impaired resonance at left posterior base (probably from late attack of pneumonia); cardiac sounds feeble. Abdomen tense, tympanitic; some resistance in right iliac fossa, where gurgling is to be felt. A few rose-spots. Tongue dry and coated.

4 p.m.—T. 104·5°. 4.30.—Bath given, 70°, for ten minutes.

5 p.m.—T. 99·8°.

7 p.m.—T. 104·2°. Second bath. 7.30.—T. 99·6°.

9 p.m.—T. 103·6°, pulse 112. Rambling at times. Lies on back with knees drawn up.

11 p.m.—T. 104·2°. Third bath, 60°, for ten minutes.

11.40.—T. 99°.

February 21st, 3 a.m.—T. 103·4°. Fourth bath, 60°, for ten minutes.

4 a.m.—T. 99·5°.

10 a.m.—T. 103·2°, pulse 132. Slept fairly well, but rambled a good deal. Tongue still coated; abdomen distended.

12 noon.—T. 103·8°. 1.30 p.m.—Fifth bath, 70°, for ten minutes.

2 p.m.—T. 100·2°.

6 p.m.—T. 103·6°. 6.30 p.m.—Sixth bath, 60°, for ten minutes.

7 p.m.—T. 100·8°.

11 p.m.—T. 104°. 11.30 p.m.—Seventh bath, 60°, for ten minutes.

12.15.—T. 98·6°.

February 22nd, 4 a.m.—T. 103·2°. Eighth bath, 60°, for ten minutes.

4.45.—T. 98°.

10 a.m.—T. 102·4°, pulse 120, resp. 36. Abdomen still tense and tympanitic.

4 p.m.—T. 103·8°. Ninth bath, 60°, for ten minutes.

5.30.—T. 99·2°.

8 p.m.—T. 103·8°. Tenth bath, 60°, for ten minutes.

9 p.m.—T. 104·2°.

February 23rd, 3 a.m.—T. 103·4°. Eleventh bath, 60°, for ten minutes.

4 a.m.—T. 99·4°.

12 noon.—T. 103·2°, pulse 132, regular, small, dicrotic. Tongue moist, coated on dorsum. Numerous rose-spots. Abdomen still much distended. Urine, sp. gr. 1022, acid. 2 p.m.—T. 101·8°.

6 p.m.—T. 103·6°. Sponged for twenty-five minutes.

6.35 p.m.—T. 103·4°.

7.30 p.m.—Twelfth bath. 8 p.m.—T. 99·6°.

12 midnight.—T. 102·8°. Thirteenth bath. 1 a.m.—T. 99·6°.

February 24th, 3 a.m.—T. 102°. 5 a.m.—T. 102·8°. 7 a.m.—T. 102·6°.

9 a.m.—T. 101·8°, pulse 132, regular, compressible. Bowels opened once; motion loose and pale. For next twelve hours temperature over 103°.

9 p.m.—T. 104·2°. 10.30.—Fourteenth bath for ten minutes, 60°.

11 p.m.—T. 99·2°.

February 25th.—Temperature reached 103° at 3 a.m., 2 p.m., 6 p.m., and 10 p.m., and at other times it was mostly above 102·8°, but no baths were given. There was a slight improvement in general condition.

February 26th.—Temperature never reached 103°. It was for the most part about 102·4° to 102·8°, only twice below 102°.

February 27th.—Temperature only once reached 102°, at 8 a.m., but never fell below 101°.

February 28th.—Temperature only thrice above 100°, and only once as low as 99°.

March 1st.—Defervescence. Temperature reaching 97·8° at noon, rising to 100·2° at 10 p.m.

March 2nd.—Temperature for the most part subnormal, only reaching 99° at 8 p.m. There is little to add. Convalescence was uninterrupted. Although the case was uncomplicated the degree of wasting was considerable. It must not be forgotten that he was attacked with the fever whilst convalescing from an acute disease, and perhaps this explains the severe effect produced by the pyrexia.

He was discharged on April 2nd.

Case 10. *Enteric fever; mild primary, with severe relapse, marked by diarrhœa; great cardiac weakness. Fourteen baths; sponging.* Thomas W—, aged fifteen, a cardboard-maker, admitted January 22nd, 1883, on fourth day of illness, which commenced with headache and pain in chest; loss of appetite; no vomiting or diarrhœa.

On admission.—T. 99·8°, pulse 112, resp. 25. Well nourished, fair, pale; frontal headache, pain in back, anorexia; depressed aspect; pulse feeble. Tongue thickly coated. No rash. No abdominal distension. No increase of splenic dulness. Breathing vesicular. First cardiac sound feeble. Even. temp. 101·6°.

January 23rd.—T. 101·4, pulse 116, resp. 20. Tongue coated. Bowels not open.

7 p.m.—T. 103°. Sponged. 8 p.m.—T. 102·8°.

12.—T. 103·2°. Bath given, 77°, ten minutes. 1.30 a.m.—T. 100·4°.

January 24th.—Signs still negative. Maximum temperature 102·2° at 5 a.m.; minimum 99·6° at 1 p.m.

January 25th.—Bowels still confined; urine, sp. gr. 1035, acid, copious deposit of lithates. Maximum temp. 102·6° (7 p.m.), minimum 99·8° (5 p.m.).

January 26th.—Bowels opened twice, motions forced. Maximum temperature 101·4°, minimum 98·6°; pulse 100, short and weak; resp. 28.

For the next fortnight (till February 11th) there was very mild pyrexia, the temperature occasionally falling to normal, and never reaching to 102°. On February 5th and 6th it was mostly between 97° and 99°, but he remained feeble with slow, weak pulse, and languid circulation. Rusks and custard pudding were allowed him on the 5th,

but owing to constipation an enema was ordered on the 11th; the motion was solid. Perhaps owing to this enema the temperature, which on the 8th and 11th had twice been above 102°, again took a more continued higher range. On the 12th, evening it was above 102°, and at 5 and 9 p.m. 102·8°. But there was no abdominal pain and the tongue was clean.

February 13th.—Bowels open loosely, pulse 90, resp. 26. Bread omitted from diet.

1 p.m.—T. 101·4°. 9 p.m.—T. 103·4°. Sponged.

February 14th.—Pulse 80, resp. 30. Temperature between 98·8° and 101·4°. A severe relapse now set in.

February 15th, 9 a.m.—T. 98·4°. Bowels open; motions liquid and scanty. Tongue dry and glazed. Lips dry. Extreme prostration. One or two doubtful rose-spots. No abdominal distension. Splenic dulness 3½ inches. Heart-sounds weak. Pulse 100, resp. 36. Ordered brandy, ℥iv daily. A slight trace of albumen in urine.

1 p.m.—T. 103·2°. Sponged. 5 p.m.—T. 103·6°. Sponged.

6 p.m.—T. 101·6°. 9 p.m.—T. 103·6°. Sponged. 10 p.m.—T. 101·8°.

February 16th, 1 a.m.—T. 102°, pulse 110, resp. 25.

5 a.m.—T. 101°. Pulse very weak and dicrotic. Numerous rose-spots. Brandy increased and champagne ordered.

1 p.m.—T. 102·2°. Sponged. 3 p.m.—T. 102·6°. 4 p.m.—T. 103°.

5 p.m.—T. 102·4. Second bath, 85°, fifteen minutes. 6.40.—T. 98·8°.

9 p.m.—T. 104°. Third bath, 85°, fifteen minutes.

11 p.m.—T. 97·4°.

February 17th, 1 a.m.—T. 102·8°. 5 a.m.—T. 101·4°.

9 a.m.—T. 100·4°. Is less depressed; pulse 96, extremely small and weak, resp. 25.

1 p.m.—T. 100·6°. 5 p.m.—T. 102·6°.

7 p.m.—T. 103·2°. Fourth bath, 85°, ten minutes.

8.40 p.m.—T. 99·6°.

February 18th, 3 a.m.—T. 102·6°. 7 a.m.—T. 100·6°.

11 a.m.—T. 100·6°. Bowels opened three times, motions loose.

1 p.m.—T. 102·2°. 3 p.m.—T. 101°. 5 p.m.—T. 103·4°.

6.15 p.m.—T. 103°. Fifth bath, 85°, fifteen minutes.

7.15 p.m.—T. 99·8°.

February 19th.—Diarrhœa was now pronounced, about four loose motions daily for two days. Patient is very listless. Temperature from 100° to 102·2° in morning.

3 p.m.—T. 103°. Sponged. Pulse 108, resp. 32. 4 p.m.—T. 101·6°.

5 p.m.—T. 103·2°. Sixth bath. 7 p.m.—T. 102·8°.

11 p.m.—T. 104·2°. Seventh bath. 12.15.—T. 100·6°.

February 20th, 2 a.m.—T. 102·6°. 4 a.m.—T. 102·8. 9 a.m.—T. 99°.

11 a.m.—T. 101°, pulse 110. Looks pinched; lies with mouth half open; very listless. Sordes on teeth. Skin dry and harsh. Pulse very small; heart-sounds barely audible.

1 p.m.—T. 101°. 3 p.m.—T. 103°.

5 p.m.—T. 103·6°. Eighth bath, 85°, for fifteen minutes.

7.15.—T. 101·6°.

11 p.m.—T. 103·2°. Ninth bath, 85°, for fifteen minutes.

12.15.—T. 99·8°.

February 21st, 2 a.m.—T. 101·6°. 6 a.m.—T. 101°.

10 a.m.—T. 99·8°, pulse 100, resp. 25. Looks and feels better. Tongue moister. Pulse stronger.

2 p.m.—T. 99·2°. 8 p.m.—T. 101·6°.

February 22nd, 2 a.m.—T. 100·6°. 8 a.m.—T. 101°.

10 a.m.—T. 99·4°, pulse 96, resp. 24. Pinched features; depressed aspect. Tongue dry and glazed. Heart-sounds tender, pulse firmer. Numerous rose-spots. Breathing weak at base of lungs. Urine acid, sp. gr. 1025, trace of albumen.

1 p.m.—T. 101·2°. 5 p.m.—T. 102°. 9 p.m.—T. 103°.

10 p.m.—T. 103°. Tenth bath, 85°, for fifteen minutes.

11 p.m.—T. 100°.

February 23rd, 5 a.m.—T. 101°. 9 a.m.—T. 98°.

11 a.m.—T. 99·6°. Bowels once moved, stool loose. Tongue very dry and glazed. Pulse 100.

1 p.m.—T. 99·4°. 5 p.m.—T. 101·8°. 9 p.m.—T. 102·8°.

February 24th, 5 a.m.—T. 100·6°.

9 a.m.—T. 99·4°, pulse 98, resp. 32. Bowels open three times in last twenty-four hours. Is very apathetic and still very pinched looking. Pulse slightly improved. Urine contains a trace of albumen.

1 p.m.—T. 100·2°.

5 p.m.—T. 103·6°. Eleventh bath, 85°, for fifteen minutes.

6.30 p.m..—T. 100·8°.

February 25th, 1 a.m.—T. 100·4°. 5 a.m.—T. 99·8°. 9 a.m.—T. 98°.

5 p.m.—T. 103·8°. Twelfth bath, 85°, for fifteen minutes.

6.30 p.m.—T. 101·4°. 9 p.m.—T. 102·6°.

February 26th, 1 a.m.—T. 101·2°. 7 a.m.—T. 99°.

11 a.m.—T. 96·8°; pulse 96, very dicrotic; but heart-sounds better pronounced. Less apathy. Tongue still dry and caked.

1 p.m.—T. 97·8°. 3 p.m.—T. 98·6°. 5 p.m.—T. 102·6°.

7 p.m.—T. 104·6°. Thirteenth bath, 85°, for fifteen minutes.

Half an hour after.—T. 103·4°.

9 p.m.—T. 104·6°. Fourteenth bath, 85°, for fifteen minutes.

11 p.m.—T. 99·4°.

February 27th.—After this outburst, the patient became very collapsed, temperature falling to 98° at 5 a.m.; 97·6° at 7 a.m.; and to 95° at 9 a.m., where it remained for some hours; even at 1 p.m. it only registered 95·3° in rectum. Extremities were cold and pulse imperceptible; yet cardiac sounds fairly pronounced. Urine contained a trace of albumen. Carbonate of ammonia was given and temperature rose: 5 p.m.—T. 96·4°; 7 p.m.—T. 97·4°; 9 p.m.—T. 98·2°; 11 p.m.—T. 98·4°.

February 28th, 1 a.m.—T. 99·4°. 3 a.m.—T. 102·6°. 4 a.m.—T. 104°. 6 a.m.—T. 102·6°. 8 a.m.—T. 100·4°. 10 a.m.—T. 99·6°. Still lies in a very apathetic, dull state, with half-open eyes and mouth. Tongue dry and glazed. Radial pulses very feeble. At left posterior base dulness, bronchophony, and fine crepitation. Pulse 100, resp. 28. For next twelve hours temperature again subnormal, from 97·6° to 95°.

March 1st.—Temperature rose from 97·2° at 2 a.m. to 101·8° at 10 a.m.; remained about 100° and 101° till 6 p.m., and fell to 97 6° at midnight. Pulse 120, resp. 34. Extreme asthenia. Signs of consolidation well marked at base of left lung; bronchitic signs on right side. Urine passed involuntarily. Bowels open once voluntarily.

March 2nd.—As yesterday; temperature subnormal in early morning, rising to 100·6° at noon, 102° at 2 p.m., and remaining about 101° till 10 p.m., when it was 100·4°.

March 3rd.—Fretful and hungry. Pulse still very weak. Some bronchial breathing and fine crepitation still audible at left base. No

albumen in urine. Temperature 99° to 100° before morning; from 98·8° to 97° at 9 p.m. Fish allowed.

From this date the course was apyrexial.

The signs of pulmonary consolidation cleared up, and were quite absent on the 8th. He took food well. The tongue cleaned and became moist. His mental condition improved. Constipation requiring enemata. An abscess formed over the left great trochanter, discharging on the 11th. He gained flesh, but convalescence was tedious.

Discharged April 7th.

Case 11. *Enteric fever; at first mild, then severe; marked by constipation; otorrhœa. Thirteen baths.*—Joseph C—, aged seventeen, cigar-maker, admitted September 22nd, 1880, on the fifth day. His previous health had been good; except for scarlet fever in childhood. The attack commenced four days before with headache and earache; next day he was weak and feverish, but continued at work till the following day. He then began to have slight diarrhœa, for which he took some "liver pills."

On admission.—T. 102°, pulse 108, resp. 18; well nourished; complaining of frontal headache, thirst, malaise. Face flushed; lips dry; tongue thickly coated and tremulous. Abdomen distended, tympanitic. No rose rash. Abdominal "tâche" well marked. No increase of splenic dulness. Pulmonary signs normal. First sound of heart short, second sound reduplicated at third left cartilage.

7.30 p.m.—T. 103°. Bath given, 65°, for ten minutes. 8 p.m.—T. 100°.

12 p.m.—T. 102·6°, pulse 84; asleep.

September 23rd, 12 midnight.—T. 103·2°. Second bath.

1.30 a.m.—T. 99·4°. 3 a.m.—T. 101°. 7 a.m.—T. 100·6°. 9 a.m.—T. 101·4°.

11 a.m.—T. 102·2°; pulse 108, good volume. Much headache. Bowels not open. Urine, sp. gr. 1020, no albumen. 1 p.m.—T. 101·6°.

5 p.m.—T. 102·2°. 9 p.m.—T. 101·2°, pulse 96.

September 24th.—Throughout the day temperature remained between 100° and 101°; pulse 76. Belly still distended, and bowels opened at 9 p.m slightly; a formed clay-coloured motion; tongue being thickly furred.

September 25th.—At 9 a.m. temperature fell to 98·4°, rising at 9 p.m. to 101·2°; pulse from 72 to 90. No eruption has appeared, but there is purulent otorrhœa. Bowels once moved; stool as before.

September 26th.—Temperature from 98·4° to 101°. Still headache.

September 27th.—Temperature from 99·6° to 101°.

September 28th.—Temperature from 98·6° at 9 a.m. to 102·4° (5 p.m). Abdomen not so distended, no tenderness. Tongue cleaner. Face less flushed.

September 29th, 1 a.m..—T. 102·6° 5 a.m.—T. 99·4°.

10 a.m.—T. 103·6°, pulse 120. Skin dry. Complains much of headache. Tongue thickly coated.

11 a.m.—T. 103·8°. Third bath, 65°, for ten minutes.

12.30 p.m.—T. 101·6°. 4.30 p.m.—T. 105. Fourth bath, 65°, for fifteen minutes.

6 p.m.—T. 102. 8 p.m.—T. 103·6°. Fifth bath, 65°, for ten minutes.

9 p.m.—T. 99°. Restlessness since last bath.

September 30th, 12 midnight.—T. 103°. Sixth bath. 1 a.m.—T. 100·4°.

6 a.m.—T. 103·6°. Seventh bath. 7 a.m.—T. 102·8°.

10 a.m.—T. 101°, pulse very rapid. Headache better. Bright flush on cheeks. Tongue still coated. 11 a.m.—T. 99·4°. 2 p.m.—T. 102·4°.
4 p.m.—T. 100°. 8 p.m.—T. 104·6°. Eighth bath. 9 p.m.—T. 103·6°.
10 p.m.—T. 103·6°. Ninth bath. Pulse 144, small.
October 1st.—During the morning temperature ranged between 101° and 102°; Pulse fell to 84. Tongue thickly coated and tremulous. Abdomen distended and tender; bowels once open, motion pale, but semi-solid. A few rose-spots on trunk.
3 p.m.—T. 103·6°. Tenth bath. 4 p.m.—T. 100·2°.
9 p.m.—T. 102·6°, and then it fell; pulse 120.
October 2nd, 1 a.m.—T. 100°. 3 a.m.—T. 102°. 5 a.m.—T. 100·2°.
9 a.m.—T. 100·2°, pulse 72. Spleen can be felt. Tongue coated and tremulous. Some enlarged and painful glands on right side of neck.
11 a.m.—T. 99·4°. 5 p.m.—T. 103·2°. Eleventh bath.
6 p.m.—T. 99·4°. 9 p.m.—T. 101°, pulse 84. 11 p.m.—T. 102°.
October 3rd, 1 a.m.—T. 102·6°. 8.30 a.m.—T. 98·6°. Passed a restless night. 1.30 p.m.—T. 102·4°.
4.30 p.m.—T. 104·6°. Twelfth bath. 5 p.m.—T. 99·8°.
October 4th, 1 a.m.—T. 101·6°. 7 a.m.—T. 99·6°.
11 a.m.—T. 101·4°, pulse 102. 2 p.m.—T. 101·2°. 6 p.m.—T. 101·2°.
12 midnight.—T. 100·8°.
October 5th.—Morning temperatures below 100° mostly; afternoon rose between 101° and 102°, and at 10 p.m. 102·4°; pulse 123. Bowels confined.
October 6th.—Morning temperatures below 100°. Bowels opened, semi-solid clay-coloured motion. 2 p.m.—T. 100·6°. 4 p.m.—T. 102·4°.
6 p.m.—T. 104°. Thirteenth bath. 7 p.m.—T. 100·8°.
October 7th, 2 a.m.—T. 100·2°. 8 a.m.—T. 102°.
10 a.m.—T. 101·6°, pulse 90. Tongue clear and moist. Bowels once moved.
4 p.m.—T. 102·6°. 8 p.m.—T. 102·6°. 12 midnight.—T. 100·6°.
October 8th, 2 a.m.—T. 102°. 6 a.m.—T. 99°.
10 a.m.—T. 99·6°; pulse 72. 2 p.m.—T. 102·2°. 6 p.m.—T. 101·2°.
10 p.m.—T. 100°.
October 9th.—Defervescence; temperature only once reaching 99°. Abdomen not distended. A small abscess on dorsum of left foot.
He was given custard pudding on the 11th, fish on 14th, and meat on 19th, and got up on 23rd. He rapidly gained flesh, and left the hospital on November 12th.

Case 12. *Enteric fever; relapse; much debility. Thirteen baths.*—James B—, aged nineteen, a brassworker. Admitted January 8th, 1881, in fourth week of his illness, which began with loss of appetite, vomiting, and increased debility.
On admission.—T. 102·2°, pulse 96, resp. 20. Fair haired; well nourished, marked depression, pale; feeling "weak and low." Tongue very tremulous, dry brown, red at tip and edges. Slight tremor of hands. Cardiac signs natural. Breath-sounds clear. Abdomen full, not tender; a few rose-spots on surface; hepatic area normal; splenic increased, measuring four inches. No headache. Anorexia. Urine, sp. gr. 1010, lithatic deposit.
3.30 p.m.—T. 103·8°. Bath given, 75°, for ten minutes.
5 p.m.—T. 103·2°. Second bath. 7 p.m.—T. 102·2°.
9 p.m.—T. 103·4°. Third bath. Pulse 88, small. Brandy ʒiij.

January 9th, 1 a.m.—T. 102·2°. 3 a.m.—T. 102·6°. 7 a.m.—T. 102·2°.
9 a.m.—T. 103°. Fourth bath.
11 a.m.—T. 101·6°; pulse 90, dicrotic. Is drowsy. No fresh eruption.
3 p.m.—T. 103°. Fifth bath, 68°, for fifteen minutes.
5 p.m.—T. 101·8°.
9 p.m.—T. 103·2°. Sixth bath, 65°, for fifteen minutes.
11 p.m.—T. 101·2°.

January 11th.—Temperature kept about 102° during morning hours; pulse 90, dicrotic. Bowels not opened. Tongue as before and tremulous. 1 p.m.—T. 102·8°.
3 p.m.—T. 103·4°. Seventh bath. 5 p.m.—T. 102°.
9 p.m.—T. 104°. Eighth bath. Bowels once open; motion semi-solid. Takes food well. 11 p.m.—T. 100·8°.

January 11th, 1 a.m.—T. 102·4°. 5 a.m.—T. 102·4°.
7 a.m.—T. 103·4°. Ninth bath. 9 a.m.—T. 100·4°.
11 a.m.—T. 101·6°; pulse 90, soft, dicrotic. Splenic area larger than on admission. A few fresh spots. 1 p.m.—T. 102·6°.
5 p.m.—T. 103·6°. Tenth bath, 65°, for fifteen minutes.
7 p.m.—T. 100·6°.

January 12th, 12.30 a.m.—T. 103·4°. Eleventh bath.
2 a.m.—T. 100·6°.
11 a.m.—T. 102·6°. Skin harsh and dry. Tongue dry.
1 p.m.—T. 102·8°.
2 p.m.—T. 103·2°. Twelfth bath. 3.15 p.m.—T. 100·2°.
8 p.m.—T. 102·6°.

January 13th, 1 a.m.—T. 102°. 5 a.m.—T. 101·8°.
11 a.m.—T. 102·4°; pulse 84. Abdomen tympanitic; tender in right iliac fossa and in splenic region. No rose-spots now visible.
1.30 p.m.—T. 102·6°.
2 p.m.—T. 103·4°. Thirteenth bath.
3.30 p.m.—T. 98·6°. The pulse now began to flag very markedly, but under increased stimulation it improved.

Although during the 14th and 15th the evening temperature was above 102°, it never rose to 103°, and no more baths were given. The bowels were open once daily; motions formed. On the 16th temperature ranged between 101° and 102°, on the 17th between 100° and 102°, on the 18th 100° and 101·4°, on the 19th 99° and 101°, on the 20th 100° and 101°, on the 21st 98·4° and 99·8°, and from that date it was normal and subnormal (Plate IX). Convalescence was retarded by an axillary abscess which appeared on the 28th, and he did not leave the hospital till March 2nd.

Case 13. *Enteric fever; advanced stage; much bronchitis and pulmonary congestion. Twelve baths; quinine.*—Andrew S—, aged sixteen, a carman. Admitted January 16th, 1881, in fifth week. Never had a day's illness before. A month ago drains connected with his house were altered; he had a "fever," marked by headache, diarrhœa, thirst, sleeplessness, for three weeks, and had been only out of bed four days, resuming work, when taken ill again on the 11th with a return of fever and diarrhœa.

On admission.—T. 103°, pulse 108, resp. 30. Thin, depressed, feeling very weak. Dry tongue. Lips covered with sordes. Distended belly; spleen not to be felt. Impaired resonance at bases of lungs, where subcrepitant râles were audible in addition to sonoro-sibilant

rhonchus, which is universal. First sound at apex of heart is weak; second pulmonary sound accentuated. Pulse small, compressible, dicrotic. Numerous rose-spots on chest, back, and abdomen. Skin hot and dry; a well-marked "tâche" obtainable. Insomnia; no headache; dilated pupils.

6 p.m.—T. 103°. Bath given, 75°, for ten minutes.

6.45 p.m.—T. 102·2°.

9 p.m.—T. 103·8°. Second bath, 72°, for ten minutes, followed by Quin. sulph., gr. xx. Rambled a little.

10.15 p.m.—T. 101°, pulse 108.

January 17th, 12.30 a.m.—T. 103°. Third bath, 75°, for ten minutes.

1.30 a.m.—T. 100°. 3.30 a.m.—T. 102°. 5.30 a.m.—T. 101°.

9.30 a.m.—T. 101·2°, pulse 102. Has been restless; not delirious. Pulse improved.

1.30 p.m.—T. 100·4°. 5.30 p.m.—T. 100·6°.

9.30 p.m.—T. 101·8°, pulse 108. Delirious all evening.

January 18th, 2.30 a.m.—T. 103·2°. Fourth bath, 75°, for fifteen minutes, followed by Quin. sulph., gr. xx. Cheeks flushed. Tongue and lips very dry. Bowels moved once; stool pale and flaky. Pulse 126, small. Dulness at posterior bases of both lungs, with moist râles, and sonoro-sibilant rhonchi general over chest. 11 a.m.—T. 101·8°.

6 p.m.—T. 103·2°. Fifth bath. 7.45 p.m.—T. 100°. Mild delirium.

11 p.m.—T. 103°. Sixth bath. 12 midnight.—T. 100°.

January 19th, 2 a.m.—T. 103°. Seventh bath, 70°, for ten minutes.

3 a.m.—T. 99·8°.

5.30 a.m.—T. 103·4°. Eighth bath, 70°, for ten minutes.

6.30 a.m.—T. 100°.

10.30 a.m.—T. 101·4°; pulse 132, small, compressible. Face dusky, occasionally pale. Cheeks flushed. Lips dry. Tongue dry. Muttering delirium all night. No change in pulmonary condition.

2.30 p.m.—T. 103·2°. Ninth bath. 3.30 p.m.—T. 100°.

5.30 p.m.—T. 102·4°. 9.30 p.m.—T. 102·8°; pulse 120.

10.30 p.m.—T. 103·4°. Tenth bath, 70°, for fifteen minutes.

11.30 p.m.—T. 99·6°.

January 20th, 2 a.m.—T. 102·8°. Eleventh bath, 70°, for fifteen minutes. 3.30 a.m.—T. 98·6°.

10 a.m.—T. 101·8°, pulse 120. Slept for last two hours, lying on side. Face still flushed and dusky. Muttering delirium. Takes food well. 3.30 p.m.—T. 100·8°. 5 p.m.—T. 102°.

Slept well during the day.

11.30 p.m.—T. 103°. Twelfth bath, 70°, for fifteen minutes, followed by Quin. sulph., gr. xx. 12.40 a.m.—T. 99·2°.

January 21st.—During this day temperature only 102° at 4 a.m., and for rest of day between 101° and 101·8°. Fresh rose-spots appeared. Signs of bronchitis and pulmonary congestion diminished. No longer delirious, and sleeps well, lying on side. Pulse 120.

January 22nd, 2.30 a.m.—T. 102°. 10.30 p.m.—T. 102·6°. At other times mostly between 101° and 102°. Takes food well. Sleeps lying on side. Tongue moist and cleaning. Dulness cleared away from bases of lungs; rhonchus less.

January 23rd.—Temperature above 101° in early morning, and in evening; at other times about 100°, and once, viz. 5 p.m., 98·2°. As bowels confined since 18th an enema given; pale semi-solid motion passed. Pulse 90.

January 24th.—Temperature fell from 101·4° at 1 a.m. to 98·2° at 3 p.m., and remained at lower level till 9 p.m., when it rose to 101·2°; pulse 96. Very few râles in chest. Tongue only coated in centre.

Defervescence was now established. On the 26th salicylate of soda given with the idea of preventing relapse. There was considerable weakness and tendency to constipation, requiring enemata. Allowed fish on February 8th, and got up on 19th.

Discharged March 9th, 1881, for Eastbourne.

Case 14. *Enteric fever; moderately severe, but ill-marked. Twelve baths. Sponging.*—Jane W—, aged eleven, schoolgirl, admitted February 11th, 1882. Three months before had been under treatment for chorea; and about a month or more ago began to suffer from painful swellings over the legs.

On admission.—A well-nourished blonde, with marked erythema nodosum over both shins, and suffering from general malaise. Moist and coated tongue. At this time no suspicion of enteric fever. T. 100·6° to 102·4°, pulse 108.

February 12th.—T. 99·6° to 102·6°, pulse 96.

February 13th.—T. 99·2° to 101·4° Erythema faded. Face flushed.

February 14th.—T. 99·6° to 103·4°. Bowels opened twice. Tongue coated.

February 15th.—T. 102·2°, pulse 132. Takes food well and sleeps well. Bowels not open. No sorethroat. No pulmonary signs. Brown mottling over shins at seat of previous erythema.

6 p.m.—T. 102·4°.

February 16th, 6 a.m.—T. 100°, pulse 108. Bowels twice open; motions pale and loose. 6 p.m.—T. 103·6°.

February 17th, 6 a.m.—T. 100·4°, pulse 96. Tongue moist and coated. Feels well, and has good appetite. Bowels again moved; motion loose, less pale. Belly not distended. 1 p.m.—T. 102°.

6.30 p.m.—T. 104°. Bath given. After bath T. 99°. 10 p.m.—T. 102·8°.

February 18th, 2 to 10 a.m.—T. 102·2°; pulse 108. Splenic dulness increased. Face pale. Bowels open; motions formed.

2 p.m.—T. 103·6°. Second bath. After bath T. 101°.

6 p.m.—T. 102·6°. 10 p.m.—T. 102·4°.

February 19th, from 2 a.m. to 2 p.m.—T. 101° to 102°. Bowels confined. Tongue thickly coated.

6.30 p.m.—T. 104·2°. Third bath. After bath T. 101°.

February 20th, from 2 a.m. to 10 a.m.—T. 101° to 102°, pulse 96.

2 p.m.—T. 104°. Fourth bath. After bath T. 101·8°.

6 p.m.—T. 104·8°. Fifth bath. After bath T. 100·8°.

February 21st, from 2 a.m. to 10 a.m.—T. 102° to 102·4°, pulse 96. Abdomen slightly distended. Bowels not open. No eruption.

2 p.m.—T. 104·6°. Sixth bath. After bath T. 101°.

10 p.m.—T. 103°. Sponged. After sponging T. 103·4°.

12 midnight.—T. 104·2°. Seventh bath. After bath T. 100·2°.

February 22nd, 6 a.m.—T. 102·6°. 10 a.m.—T. 103°, pulse 108, resp. 30. Coarse râles at bases of lungs.

6 p.m.—T. 104°. Eighth bath. After bath T. 98·2°.

February 23rd, 2 a.m.—T. 102·2°. 10 a.m.—T. 101·6°, pulse 114.

2 p.m.—T. 102·6°. Ninth bath. After bath T. 100°.

6 p.m.—T. 103·2°. Tenth bath. After bath T. 100·2°.

February 24th, 2 a.m.—T. 102°. 10 a.m.—T. 102·6°, pulse 108, full, bounding.

6 p.m.—T. 104·2°. Eleventh bath. After bath T. 100°.
February 25th, 2 a.m.—T. 101·8°. 6 a.m.—T. 102·4°.
10 a.m.—T. 101·2°, pulse 96°. Formed motion.
6 p.m.—T. 104·2°. Twelfth bath. After bath T. 98·4°.
February 26th.—Temperature between 101° and 102°.
February 27th.—Temperature between 99° and 101°.
February 28th.—Temperature between 99° and 100°.
March 1st.—Temperature between 98° and 99°, and apyrexial afterwards. The diagnosis was never quite certain, owing to absence of definite signs. Discharged March 28th, 1882.

Case 15. *Enteric fever; moderately severe; constipation; insomnia and headache. Eleven baths. Sponging. Quinine.*—Louisa G—, aged twenty-three, hospital nurse, admitted December 22nd, 1883, at end of first week, or early in second week; had been ailing a fortnight, with loss of appetite; headache past five days. Bowels regular till three days ago. On 21st shivered and felt hot.

On admission.—T. 103°, pulse 100, resp. 30; well-nourished brunette. Anorexia, thirst, malaise. Lips dry. Tongue coated. Constipation. No eruption. Given calomel, followed by magnesia, which produced three loose evacuations. 6 p.m.—T. 104°. 9.30 p.m.—T. 104°.

December 23rd, 2 a.m.—T. 104·4°. 6 a.m.—T. 102·6°.
10 a.m.—T. 103·6°, pulse 100. Headache. Tongue dry. Abdomen more tumid.
2 p.m.—T. 103·4°. Sponged. 3.30 p.m.—T. 101°.
5.30 p.m.—T. 102·4°. Sponged. 7 p.m.—T. 99·4°. 9 p.m.—T. 102·6°.
11 p.m.—T. 102·2°. Sponged. Slept well.
December 24th, 2 a.m.—T. 101·4°. 4 a.m.—T. 102·8°. Sponged. Headache still marked. Constipation. Abdomen tumid, tender. Cough troublesome. Temperature remained about 101° by sponging. When done at 11 a.m.—T. 102·8°.
4 p.m.—T. 103·4°. Bath given, 75°, for ten minutes, followed by Quin. sulph., gr. x. 6 p.m.—T. 102·8°. 8 p.m.—T. 103·2°.
11.40 p.m.—T. 105°. Second bath, 75°—70°, twenty minutes, pulse 120. Half hour after T. 103·2°. Bore the bath well, but shivered in it.
December 25th, 2.30 a.m.—T. 103·2°. Sponged. Half hour after 101·6°.
7 a.m. to 9 a.m.—T. 100·8°, pulse 84, resp. 24. Cough troublesome. Slept at intervals. More spots. More distension.
12 noon.—T. 101·6°. 2 p.m.—T. 102·6°. Sponged.
3 p.m. to 8 p.m.—T. 102·4°.
10 p.m.—T. 103°. Third bath, 75°, twenty minutes. After bath T. 102°.
December 26th, 1 a.m.—T. 101·2°. 5 a.m.—T. 102·8°. Sponged. After sponging T. 101°.
8 a.m.—T. 101°. 10 a.m.—T. 102·6°, pulse 104. Constipation. Slept fairly well. Headache less. Abdomen full.
12 noon.—T. 102·2°. 2 p.m.—T. 103·2°. Sponged. 3 p.m.—T. 103·2°.
7 p.m.—T. 104·5°. Fourth bath, 75°—70°, twenty-five minutes. Half hour after T. 101°.
10 p.m.—T. 103·6°, pulse 96, soft, compressible. No abdominal tenderness; some muscular pain from coughing. Cheeks flushed. Takes food well.
10.45 p.m.—T. 102·6°. Fifth bath, 75°, twenty minutes; followed by Quin. sulph., gr. xx. 12.30 a.m.—T. 101·2°.
December 27th, 2.30 a.m.—T. 101·2°. 4 a.m.—T. 102°.

6.30 a.m.—T.100·4°. 8.30 a.m.—T. 101°. 10.30 a.m.—T. 101·4°, pulse 96°, resp. 30. Slept fairly well; but disturbed by cough. More rose-spots.

12.30 p.m.—T. 101°. 2.30 p.m.—T. 102·2°. 3 p.m.—T. 102·4°. Sponged.

4 p.m.—T. 100°. 6 p.m.—T. 102·2°. Sponged.

7 p.m.—T. 101·4°. 9 p.m.—T. 102°, pulse 96. Lying on back; cheeks flushed. 10.45 p.m.—Quin. sulph., gr. x. 11.15 p.m.—T. 101·8°

December 28th, 1 a.m.—T. 101·4°. 3 a.m. to 7 a.m.—T. 101.°

9 a.m.—T. 102·2°. Quin. sulph., gr. x. Pulse 96. Tongue cleaner. Cough less troublesome.

11 a.m. to 1 p.m.—T. 101·6° 3 p.m.—T. 102·4°. Sponged.

4.30 p.m.—T. 101°. 6.30 p.m.—T. 103°. 7.30.—Sixth bath, 73°, twenty minutes; followed by Quin., gr. x. 8 p.m.—T. 101°. 10 p.m.—T. 102·4°.

12 midnight.—T. 103·4°. Seventh bath, 70°, thirty minutes.

December 29th, 1 a.m.—T. 100·8°. 3 to 9 a.m.—T. 101°.

11 a.m.—T. 102°, pulse 96, resp. 20. 1 p.m.—T. 100°.

3 p.m.—T. 102°. 5 p.m.—T. 102·8°.

5.30 p.m.—T. 103·2°. Eighth bath, 75°, followed by Quin. sulph., gr. xx.

6.30 p.m.—T. 101·6°. 8.30 p.m.—T. 102·4°. 9.30 p.m.—T. 102°. Given inj. morph. and atrop. for sleep. 12 midnight.—T. 101·8°.

December 30th, 2 a.m.—T. 101·8°. 6 a.m.—T. 101°.

10 a.m.—T. 100·8°. Several rose-spots have appeared. Bowels once slightly open.

12 noon.—T. 102·4°. Sponged. 2 p.m.—T. 102·6°.

6 p.m.—T. 103·4°. Ninth bath. 8 p.m.—T. 101·6°.

11 p.m.—T. 103·4°. Tenth bath, at 70° F.; followed by Quin. sulph., gr. x.

December 31st, 2 a.m.—T. 101·4°. 7 a.m.—T. 101·8°

9 a.m—T. 102·2°. Slept well. Bowels not open. More rose-spots. Less cough.

1 p.m.—T. 102·4°. Sponged. 2.30 p.m.—T. 100·8°.

4.30 p.m.—T. 103·6°. Eleventh bath, 70°, fifteen minutes.

6.30 p.m.—T. 98·8°. 9 p.m.—T. 100·2°, pulse 84; retention of urine; catheterism.

January 1st, 1884.—Has hypodermic injections of morphia every night to procure sleep. Is less depressed, but has not regained control over bladder. Urine 1020, contains a trace of albumen.

During this day temperature ranged between 101° and 102°; at 6 p.m. it was 102·4°, and sponging was practised.

January 2nd.—Condition improving; and she feels hungry. Urine still has to be drawn off. Temperature only once reached 102°, viz. at 9 p.m., when sponging practised. Bowels open; formed dark motion.

January 3rd.—Temperature between 100° and 102°. She now continued to improve, the pyrexia ceasing on the 9th. The urine contained traces of albumen until the 26th.

Discharged on January 31st.

Case 16. *Enteric fever; probably a relapse; marked "typhoid" symptoms; diarrhœa; bronchitis. Ten baths.*—Emma C—, aged thirty-five, a clothworker. Admitted December 27th, 1881, having been ill for two months with a "low fever," in which she had lost much flesh and become very weak.

On admission.—Emaciated; depressed; anorexia; thirst; headache. Doubtful rose-spots on abdomen, which is tympanitic. Splenic dulness three and a half inches. Tongue dry and brown. Bowels

open; motion formed. Sonoro-sibilant rhonchus audible over lungs. T. 102·6°, pulse 108.

December 28th.—T. 100·6, °pulse 108. E. T. 101°.

December 29th.—Tongue dry and brown. Motions formed. T. 100° to 101°.

December 30th, 6 a.m.—T. 101·6°, pulse 108. Numerous rose-spots. Cough troublesome.

2 p.m.—T. 103·6°. Bath given. After bath T. 98·2°.

6 p.m.—T. 103·6°. 7 p.m.—T. 104·6°. Second bath. After bath T. 98·6°

11 p.m.—T. 103·4° Third bath. After bath T. 98·4°.

December 31st, 6 a.m.—T. 102·8°, pulse 108. Face dusky. Tongue dry and brown. Bronchitis. Motions formed.

10 a.m.—T. 103·6°. Fourth bath. After bath T. 100·4°.

6 p.m.—T. 103·6°. Fifth bath. After bath T. 101·4°.

January 1st, 1882, 6 a.m.—T. 102·4°, pulse 96. Bowels open four times; motions typhoidal.

6 p.m.—T. 104·3°. Sixth bath. After bath T. 100°.

January 2nd, 2 a.m.—T. 100°.

10 a.m.—T. 99·4°, pulse 108. Much depressed; lies low in bed; dry and brown tongue; sordes on lips. Bronchitic signs have nearly disappeared.

6 p.m.—T. 104·6°. Seventh bath. After bath T. 98·8°.

January 3rd, 10 a.m.—T. 101·2°, pulse 120. Bowels twice moved.

8.30 p.m.—T. 103·6°. Eighth bath.

January 4th, 6 a.m.—T. 100·6°, pulse 120, dicrotic. Abdomen distended. Spots fading. Cough again troublesome, and bronchitic signs more abundant.

10 a.m.—T. 103·8°. Ninth bath. After bath T. 98·2°.

3 p.m.—T. 103·6°. Tenth bath. After bath T. 97°.

January 5th, 2 a.m.—T. 101°.

10 a.m.—T. 99°, pulse 84. Sweating profusely.

3 p.m.—T. 101°. 6 p.m.—T. 98·4°.

January 6th.—Defervescence occurred here by crisis, for on this day the temperature was subnormal, falling to 96·6°. On the 7th it rose to 100° in evening, but from that time onward was normal or subnormal. The pulse remained feeble for some days. Fish was given on the 18th, and bread allowed on the 29th.

Discharged February 11th, 1882.

Case 17. *Enteric fever; late stage; perforation of bowel; peritonitis; death. Ten baths; sponging.*— William K—, aged nineteen, fishmonger. Admitted February 2nd, 1882, in the third week, his illness commencing with a sorethroat, followed by headache and feverishness, but he remained at work until February 1st. No diarrhœa, and has complained chiefly of general aching pain.

On admission.—T. 102·6°, pulse 112, resp. 30. Well nourished; cheeks flushed; skin dry. Tongue coated with creamy fur. Abdomen full, tympanitic. Splenic dulness begins at eighth rib in axilla. A few rose-spots on back; none elsewhere. Breathing normal. Heart-sounds normal.

3 p.m.—T. 104·6°. Given bath. After bath T. 102·6°.

9 p.m.—T. 102·6° Sponged. Bowels acted.

February 3rd, 1 a.m.—T. 104·2°. Second bath. After bath T. 101·8°.

3 a.m.—T. 101·6°. 10 a.m.—T. 101·4°, pulse 96, soft. Aspect depressed. Face dusky. Bowels acted loosely.

1 p.m.—T. 103°. Third bath. 2.30 p.m.—T. 100·8°.

9 p.m.—T. 103°. Fourth bath. 10.15 p.m.—T. 101·4°.

February 4th, 1 a.m.—T. 101·6°. 5 a.m.—T. 101·8°.

9 a.m.—T. 103°. Fifth bath.

10.15 a.m.—T. 102·2°, pulse 96. Abdomen much distended. Tongue moist. Bowels open; loose.

1 p.m.—T. 102·2°. Sponged. 5 p.m.—T. 101·8°.

9 p.m.—T. 103°. Sixth bath. 10 p.m.—T. 101·2°.

February 5th, 1 a.m.—T. 101·8°. 5 a.m.—T. 102·6°. Sponged.

9 a.m.—T. 102·2°. Sponged. 1 p.m.—T. 101·8°.

5 p.m.—T. 102·6°. Sponged. 9 p.m.—T. 102·4°. Sponged.

February 6th, 1 a.m.—T. 101·8°. 5 a.m.—T. 102·8°.

9 a.m.—T. 101·6°, pulse 84, rather compressible. Numerous spots on chest and belly. Bowels open twice. Abdomen very distended. Coughing produces pain. 1 p.m.—T. 101·8°.

5 p.m.—T. 103·4°. Seventh bath. After bath T. 101·2°.

9 p.m.—T. 101·8°. Bowels slightly moved.

February 7th, 1 a.m.—T. 103·2°. Eighth bath. After bath T. 101·4°.

9 a.m.—T. 101·4°, pulse 96, dicrotic. Depressed. Face dusky. No râles in chest. Abdomen as before. Bowels open twice in night.

1 p.m.—T. 102°.

9 p.m.—T. 103·4°. Ninth bath. 10.15 p.m.—T. 100·4°.

February 8th.—T. 101·8°. 5 a.m.—T. 101·6°.

9 a.m.—T. 100·2°. Diarrhœa; bowels moved six times in night and five times during day. Belly very distended. 1 p.m.—T. 101·2°.

5 p.m.—T. 103°. Tenth bath. After bath T. 100·4°.

9 p.m.—T. 100·6°.

February 9th, 1 a.m.—T. 101·8°. 5 a.m.—T. 101·6°.

9 a.m.—T. 100·2°, pulse 96. Bowels moved three times in night. Has some pain in right hypochondriac region.

1 p.m.—T. 101·4°. 5 p.m.—T. 100·6°. 9 p.m.—T. 100°.

February 10th.—At 1.30 a.m. was suddenly seized with severe pain in left side of abdomen, with vomiting. Injected morphia, gr. $\frac{1}{6}$.

5 a.m.—T. 100·6°.

9 a.m.—T. 100·8°. Abdomen very distended. Bowels open three times during night. Ordered Extr. Opii, gr. $\frac{1}{2}$ in pill every four hours.

1 p.m.—T. 100·6°. 5 p.m.—T. 93·6°. 9 p.m.—T. 98°.

February 11th, 1 a.m.—T. 98·2°. 5 a.m.—T. 99·4°.

9 a.m.—T. 98°, pulse 112. Face dusky; expression anxious. Vomiting continues occasionally. Pain less. Sleeps fitfully. Tympanites marked, the resonance obliterating hepatic dulness. It was evident that perforation had occurred on the 10th. The temperature fell below normal during rest of day, and in early morning after 12th, nor did it rise to 100° except once at 5 p.m. on the latter day. The abdominal distension became extreme, and although the pain was controlled the vomiting continued unabated till his death. His face became pinched. No motions passed after the 11th. The pulse rose to 126 and 144. Breathing was throughout quiet, but heart's apex was displaced upwards (to 4th rib just below nipple). At 8 a.m. on the 15th he had another severe attack of pain on the left side, followed two hours after by attacks of shivering, and he died at 10 p.m.

Post-mortem examination (abridged from Dr. Fowler's notes).—Ema-

ciation. Considerable post-mortem congestion. Much gas escaped from peritoneal cavity on incising it; omentum adherent over right iliac fossa to ileum, which was intensely reddened. On detaching it it tore away a slough of the bowel the size of a sixpenny piece. Intense peritonitis with exudation and agglutination of coils of ileum, especially at its lower part, where coats of bowel blackened. Three perforations found, but fœcal extravasation very limited by the lymph. Mesenteric glands inflamed and swollen. Twelve ulcers were found in the ileum, all in the lower seven inches; five were large and seven were small. From all the sloughs had separated. Of the three which had perforated two were of the size of a shilling and one of a florin. One of these was an inch and a half from valve, the second one higher, and the third four and a half inches higher still. In thorax there were a few old pleuritic adhesions on both sides. The lungs though congested were crepitant throughout. The liver was separated from diaphragm; it was fatty looking. Spleen 9¼ oz., dark, soft, almost diffluent. Kidneys rather swollen and much congested.

Case 18. *Enteric fever; prolonged pyrexia; much bronchitis. Ten baths; quinine.*—Emma S—, aged fifteen, domestic servant. Admitted March 24th, 1882, on the tenth day of her illness, which began with pains in the head and back, thirst, and loss of appetite. Kept her bed since the 16th. Diarrhœa began on the 22nd. Four years ago was ill for three weeks with rheumatic fever.

On admission.—T. 100·4°. Well-nourished brunette of depressed aspect; flushed cheeks; sordes on lips; tongue thickly coated. Pulse 120, rather small and sharp. Abdomen full, not tender; splenic dulness measures vertically three inches. Heart-sounds normal. Normal breathing; no râles. Rose-spots.

3 p.m.—T. 103·4°. Bath given, 80°, for fifteen minutes.

4 p.m.—T. 102°.

10 p.m.—T. 104·2°. Second bath, 72°, for fifteen minutes.

12 midnight.—T. 101·6°.

March 25th, 2 a.m.—T. 102·4°. Quin. sulph., gr. x.

6 a.m.—T. 101·4°. Repeat Quin. sulph.

10 a.m.—T. 100·2°. Repeat Quin. sulph., gr. v. 2 p.m.—T. 101·6°.

6 p.m.—T. 101°. 10 p.m.—T. 101°. Repeat Quin. sulph., gr. v. Bowels loosely open three times.

March 26th, 6 a.m.—T. 100°. Quin. sulph., gr. v.

10 a.m.—T. 100·4°, pulse 88. Abdomen more distended. Fresh rose-spots. 6 p.m.—T. 102·4°.

10 p.m.—T. 102·2°. Repeat Quin. sulph., gr. v.

March 27th, 10 a.m.—T. 102·4°, pulse 108. Bowels acted once.

2 p.m.—T. 103°.

5 p.m.—T. 103·2°. Third bath. Half hour later T. 100·4°.

March 28th, 6 a.m.—T. 102·4°.

10 a.m.—T. 102·4°. Bowels open three times. Tongue thickly coated with brown fur. Pulse 102, soft, compressible. Abdomen more distended. Fresh rose-spots. 2 p.m.—T. 101·8°.

9 p.m.—T. 103·6°. Fourth bath. Half hour after T. 100·4°.

March 29th, 2 a.m. to 2 p.m.—T. 102·4° to 102°. Feels better.

4 p.m.—T. 103·4°. Fifth bath. 6 p.m.—T. 102·6°.

10 p.m.—T. 103·4°. Quin. sulph., gr. x. Slight sickness followed.

11.30 p.m.—T. 101·1°.

March 30th, 2 a.m.—T. 100·8°. 6 a.m.—T. 101·8°.

10 a.m.—T. 101·2°. Bowels opon twice. Pulse 120. Sonoro-sibilant rhonchi at bases of lungs. Abdomen tender.

2 p.m.—T. 100·4°. 4 p.m.—T. 100·4°. 6 p.m.—T. 103·6°.

8 p.m.—T. 103·4°. Sixth bath. 10.30 p.m.—T. 101·4°.

March 31st, 2 a.m.—T. 104°. Quin. sulph., gr. x.

3 a.m.—T. 103°. 6 a.m.—T. 101·6°.

10 a.m.—T. 102·8°, pulse 120, small. Tongue dry and brown. Sordes on lips. More rose-spots. More distension. More rhonchi. Slight tremor of tongue. Bowels moved once in night.

2 p.m.—T. 103°. Quin. sulph., gr. v.

5 p.m.—T. 101·4°. 7 p.m.—T. 103·8°. 9 p.m.—T. 103·4°.

10 p.m.—T. 103°. During day bowels acted seven times.

April 1st, 2 a.m.—T. 104·6°. Quin. sulph., gr. v.

6 a.m.—T. 103·6°. 9 a.m.—T. 103°. Bowels open three times in night. Looks brighter. Tongue coated, but moist. Pulse 132, compressible.

11 a.m.—T. 103°. Seventh bath.

6 p.m.—T. 101°. Bowels moved five times during day.

10 p.m.—T. 101·8°.

April 2nd, 2 a.m.—T. 101·8°. 6 a.m.—T. 102·8°.

10 a.m.—T. 102·4°. Bowels open three times; motion typhoidal. Pulse 120, small, dicrotic. A few first spots, but sound of heart feeble. Rhonchi as before.

2 p.m.—T. 103°. 6 p.m.—T. 101·6°. 10 p.m.—T. 102·6°.

April 3rd, 6 a.m.—T. 103°. Bowels open twice in night as before.

2 p.m.—T. 103°. 10 p.m.—T. 103·2°.

April 4th, 2 a.m.—T. 102·2°. 10 a.m.—T. 103·8°. 12 noon.—T. 103°.

2 p.m.—T. 103·4°. 6 p.m.—T. 103·4°.

10 p.m.—T. 102·4°, pulse 120. Delirious in night. Bowels open once.

April 5th, 2 a.m.—T. 102°. 10 a.m.—T. 102·6°. 2 p.m.—T. 103°.

6 p.m.—T. 103°. Tongue very dry and brown. Abundant rhonchus. Sputa bloodstained. Bowels acted once.

10 p.m.—T. 103·2°. Quin. sulph., gr. x. 11.30 p.m.—T. 101·5°.

April 6th, 2 a.m.—T. 102°. Less delirious. Hands tremulous. Tongue dry and tremulous. Pulse 120, dicrotic. 2 p.m.—T. 103°.

6 p.m.—T. 104·2°. Eighth bath. Half hour later T. 99·2°.

April 7th, 2 a.m. to 10 a.m.—T. 101° to 101·4°, pulse 114. Bowels open four times. Bronchitis still very marked. Tongue as before.

2 p.m.—T. 104·4°. Ninth bath. Half hour later T. 101·6°.

6 p.m.—T. 104·6°. Tenth bath, and Quin. sulph., gr. x. Half hour later T. 101·4°.

April 8th.—T. 101° to 100° to 102°, pulse 100, small, weak, dicrotic. A few spots still. Tongue still dry and brown, and sordes on lips. Bowels open twice.

April 9th, 2 a.m.—T. 101°. 10 a.m.—T. 102·4°. Bowels once open. Less distension. Fresh eruption of rose-spots. Pulse 120. Tongue and lips as before.

2 p.m.—T. 102·8°. 6 p.m.—T. 103°. 10 p.m.—T. 101·8°.

April 10th, 2 a.m.—T. 102°. 6 a.m.—T. 103°.

10 a.m.—T. 102·6°, pulse 120, small. Heart-sounds feeble. More spots. Abdomen not distended. Bowels open twice.

2 p.m.—T. 102·6°. 6 p.m.—T. 102·5°. 10 p.m.—T. 102·4°.

April 11th.—Throughout day temperature about 102°; maximum 102·6°. Bowels open once.

April 12th to 16th.—Pyrexia more irregular. Temperature between 100° and 102°; once (on 14th at 6 a.m.) 99°. Pulse fell from 120° to 96°. Slept better. Tongue became moist and clear. Bowels open once in two days.

April 17th to 21st.—Temperature oscillating, 100° to 102° or slightly above. Bowels confined, and enema on 19th. Defervescence set in on 22nd.

Discharged May 28th.

Case 19. *Enteric fever; moderately severe; marked by constipation. Ten baths; sponging.*—Cecilia T—, aged twenty-two, charwoman; admittted November 13th, 1883, in second week. Her previous health had always been good, beyond her being subject to "rheumatism." A fortnight ago she was attacked with headache and giddiness, and since then had felt weak and ill.

On admission.—Aspect depressed; weakness; malaise; pains in limbs, shiverings. Bowels confined. Tongue furred. A few rose-spots on abdomen, which is not distended. Splenic dulness, three inches vertically. First sound of heart reduplicate. Beyond a few moist râles over left back chest was healthy. Urine high-coloured, sp. gr. 1025, acid. Pulse 138, full; resp. 20. 7 p.m.—T. 104·4°. Sponged.

8.45 p.m.—T. 103°. 11 p.m.—T. 103·4°.

November 14th, 4 a.m.—T. 103·8°, Sponged. 4.45 a.m.—T. 102·2°.

7 a.m.—T. 103°. 11 a.m.—T. 102·8°. Several fresh spots. Pulse 124, resp. 20. 1 p.m.—T. 103·4°. Sponged. 2 p.m.—T. 103·4°.

6 p.m.—T. 103°.

8 p.m.—T. 104°. Sponged. 8.15 p.m.—T. 102°.

10.15 p.m.—T. 103°.

November 15th, 12.15 a.m.—T. 103·4.° Sponged. 1 a.m.—T. 102°.

5 a.m.—T. 103°. 10 a.m.—T. 102·6°. Very depressed. Bowels confined. Pulse 128, resp. 24. Temperature between 102·4° and 103·8°; sponging over 103° reducing it a degree.

November 16.—Temperature from 102·6° to 104°. Sponging as before.

November 17.—Same course of temperature. Sponging at 103°. Splenic area, four inches. Pulse 120. No bronchitis.

8.30 p.m.—T. 103·4°. Bath given, 80°, for twenty minutes.

11 p.m.—T. 100°. Enema given at 6 p.m; after which bowels acted freely—motion semi-solid and pale yellow. Slept continuously for two hours after bath, and more or less throughout the night.

November 18th, 1 a.m. to 8 a.m.—T. 102·4° to 102·8°. Pulse very weak and dicroted 136; resp. 32. Sonorous rhonchi over chest, weak breathing, but no dulness at bases. Tongue thickly coated. Lips covered with sores.

10 a.m.—T. 103·8°. Second bath, 85°, twenty minutes.

1 p.m.—T. 102°.

5 p.m.—T. 103·8°. Third bath, 85°, twenty minutes.

6.30 p.m.—T. 101·8°.

10 p.m.—T. 103·8°. Fourth bath, 85°, twenty minutes.

11·30 p.m.—T. 101·2°.

November 19th, 5.30 a.m.—T. 103·6°. Fifth bath, 85°, twenty minutes.

7 a.m.—T. 101·6°; pulse 138, thrilling, hyperdicrotic; resp. 20. Fresh crops of rose-spots. 11 a.m.—T. 102°.

5 p.m.—T. 103·4°. Sixth bath, 85°, twenty minutes.

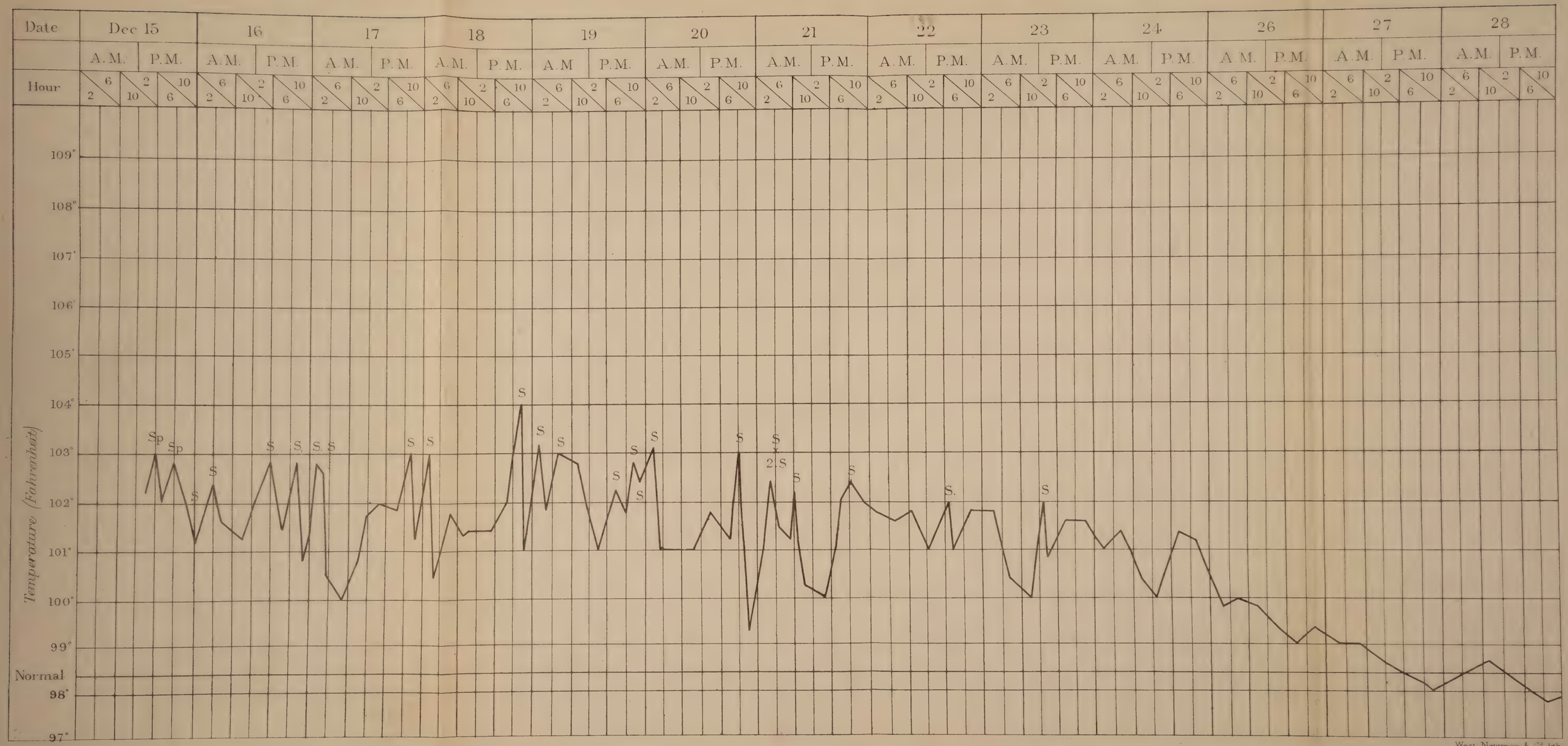
6.30 p.m.—T. 101·6°.

8.30 p.m.—T. 103·4°. Seventh bath, 85°, twenty minutes.
11 p.m.—T. 100·4°.
November 20th, 1 a.m. to 10 a.m.—T. 100·4° to 102·6°, pulse 138, resp. 32. Slept but little. Prefers tepid bath to sponging (iced water). No delirium. Splenic dulness, 4½ inch. Breath-sound fair at bases of lungs, where rhonchus. Rose-spots abundant.
1 p.m.—T. 102·4°. 3 p.m.—T. 102·2°.
5 p.m.—T. 104·2°. Eighth bath, 85°, twenty minutes.
6.30 p.m.—T. 101·8°.
8.30 p.m.—T. 103·2°. Ninth bath, 85°, twenty minutes.
10 p.m.—T. 100·2°.
November 21st, 2 a.m.—T. 102·8°. 6 a.m.—T. 103°.
11 a.m.—T. 102·4°, pulse 126. Slept better.
1 p.m.—T. 102·6°. 9 p.m.— T. 103·2°.
November 22nd, 2 a.m.—T. 102·2°. 6 a.m.—T. 102°.
10 a.m.—T. 102·2°, pulse 132, resp. 24.
2 p.m.—T. 103·6° 6 p.m.—T. 103·2°. 9 p.m.—T. 103°.
10 p.m.—T. 103·2°. Tenth bath, 85°, twenty minutes.
11.30 p.m.—T. 99·8°.
November 23rd.—Temperature during day about 102°, slightly above and slightly below; pulse 132, improved in volume and less dicrotic, resp. 28. Tongue moist, slightly furred in centre. Scanty rhonchus at bases of lungs, but air enters freely. Rose-spots still numerous on chest and back; sudamina. Sleeps fairly well (Plate III).
November 24th.—Temperature varying between 102° and 100·4°. Pulse 132.
November 25th.—Temperature between 101·6° and 100·4°. Pulse 120, improving in form.
November 26th.—Temperature 100° to 99°, pulse 108.
November 27th.—Temperature 99·4° to 98°, pulse 114, resp. 24. Bowels open for first time since 18th. Motions solid. The constipation caused some irritation, which was relieved by enema on the 29th. On December 1st, pulse-rate was 72.
Constipation was the chief symptom after this date, and on December 12th a relapse in temperature took place attributable to this. For twenty-four hours the temperature varied between 103° and 101°. Enemata followed by solid evacuation. Enemata were also given later more than once. Sole was given on the 23rd. She got up on the 27th, and was discharged on January 11th, 1883.
The foregoing include all the cases in which baths to the number of ten and upwards were given. They will suffice to demonstrate the treatment, which is further analysed below.

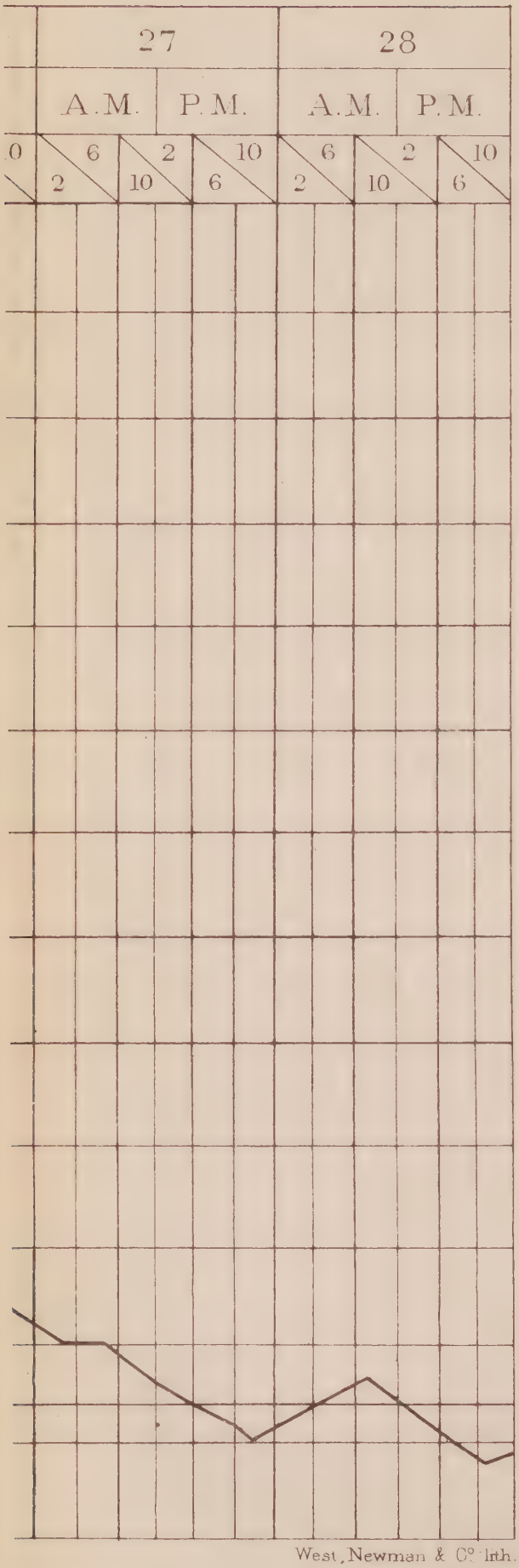
Analysis of Antipyretic Treatment of Enteric Fever.

The accompanying Tables comprise all the cases (eighty-nine in number) upon which the paper is based.
The cases were in the Middlesex Hospital under the author's care from 1879 to 1883 inclusive.
Table A includes every case in which the cold (or tepid) bath was employed at some time in its course—in all fifty-six cases.
Table B includes the remaining thirty-three cases in which no baths were given. In about one half of these cases no antipyretic treatment at all was adopted.

Plate I. See page 203. Enteric Fever. *Temperature Range of Case in which Cold Sponging was employed. No 88.*



West, Newman & Co lith.

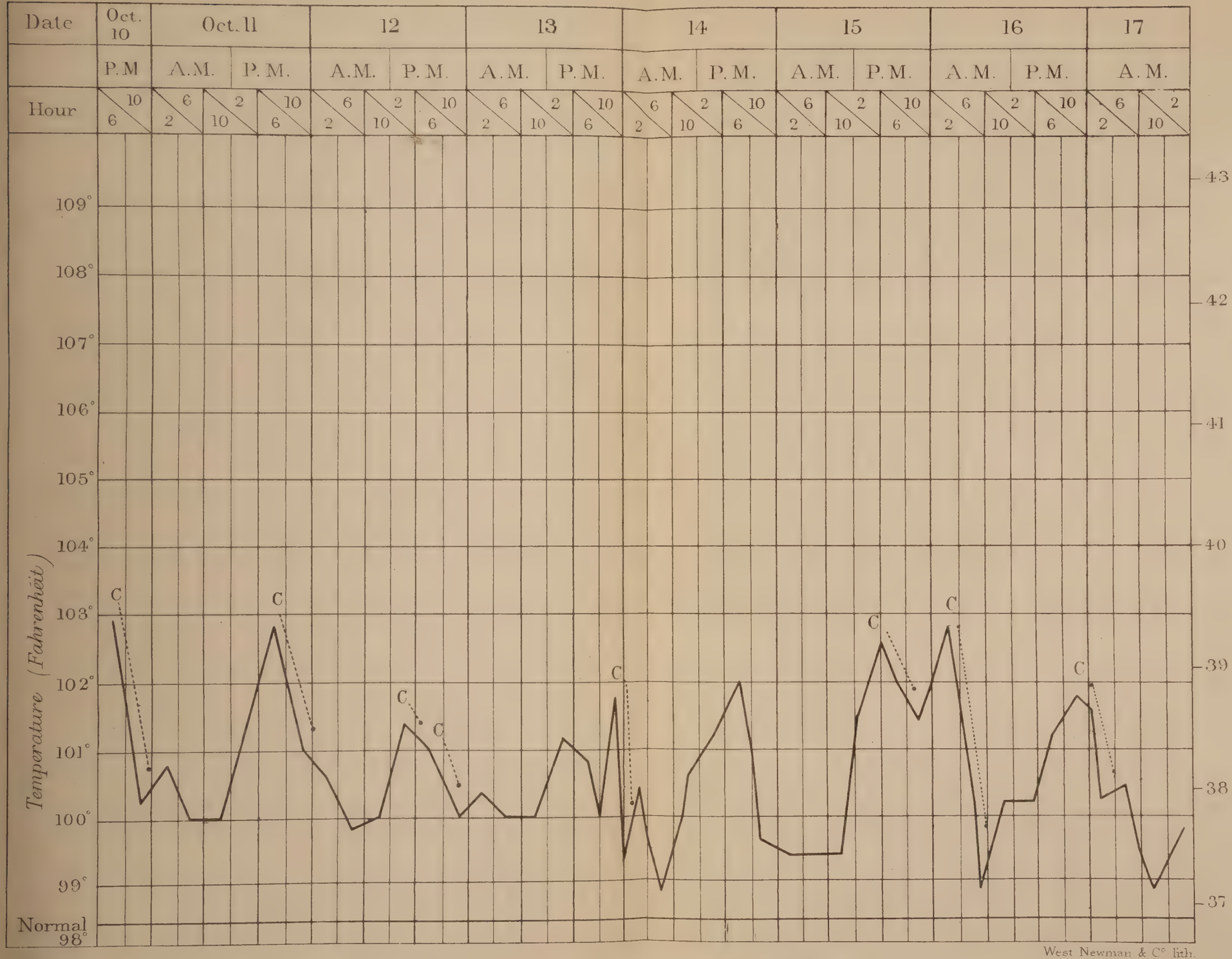


West, Newman & Co. lith.

Plate II. See page 203.

Enteric Fever. Temperature Range under Treatment by application of Cold Compresses to Abdomen. N° 83.

I.

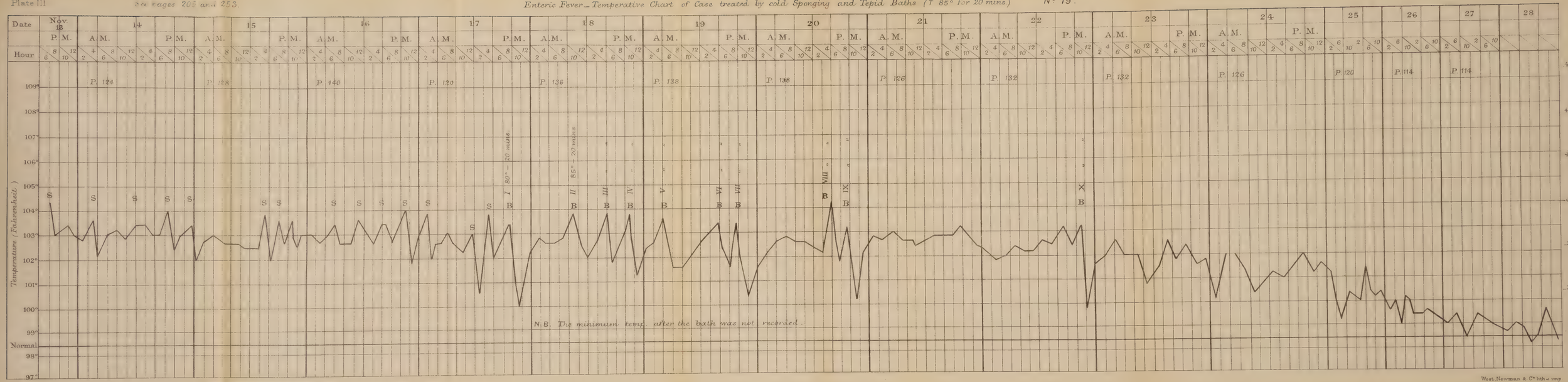


Compress = C....., shewing duration of application.

Plate III

See pages 205 and 253

Enteric Fever – Temperative Chart of Case treated by cold Sponging and Tepid Baths (T 85° for 20 mins.) N° 19.



West, Newman & C.o lith.rs imp

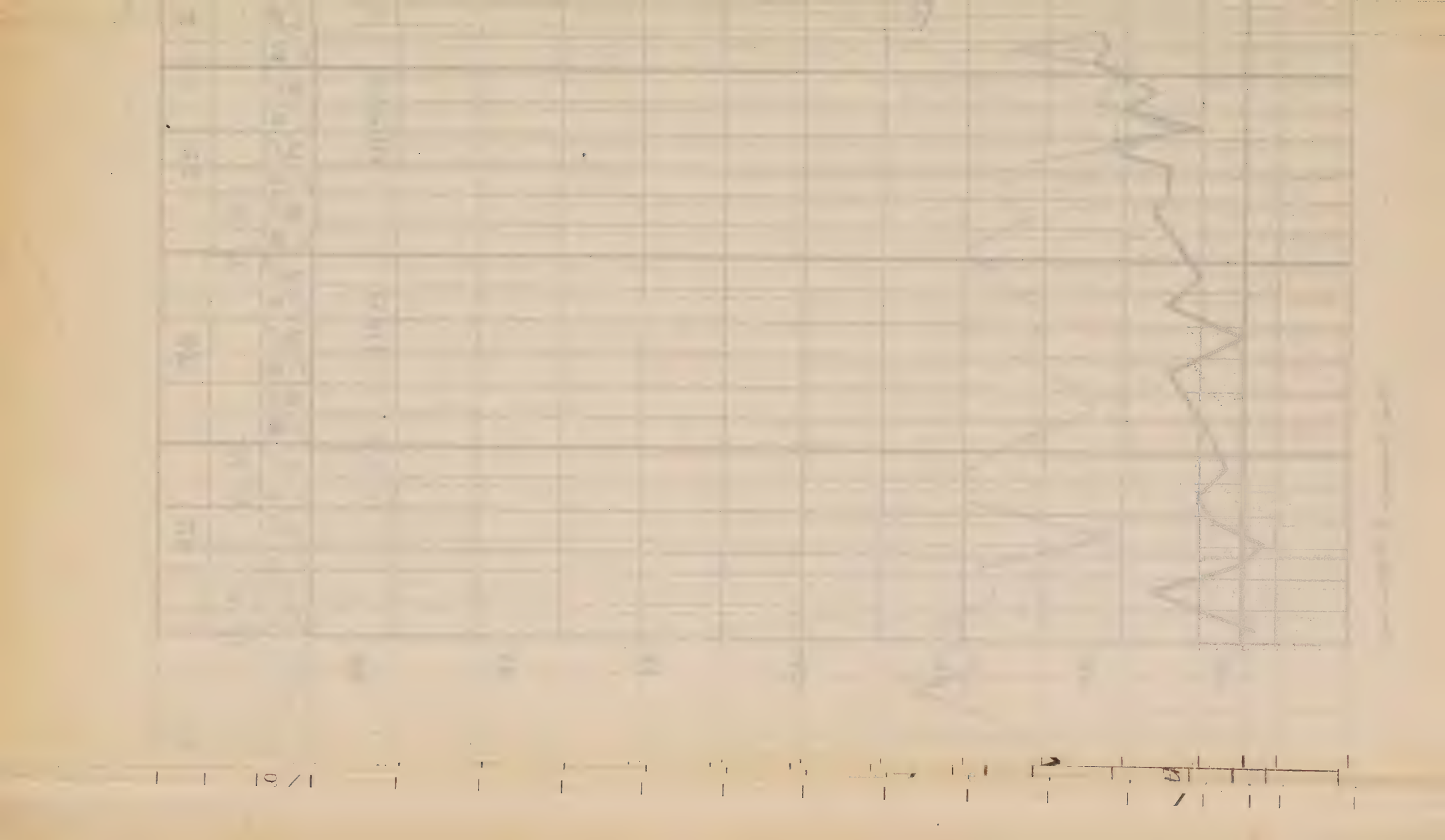


Plate IV.

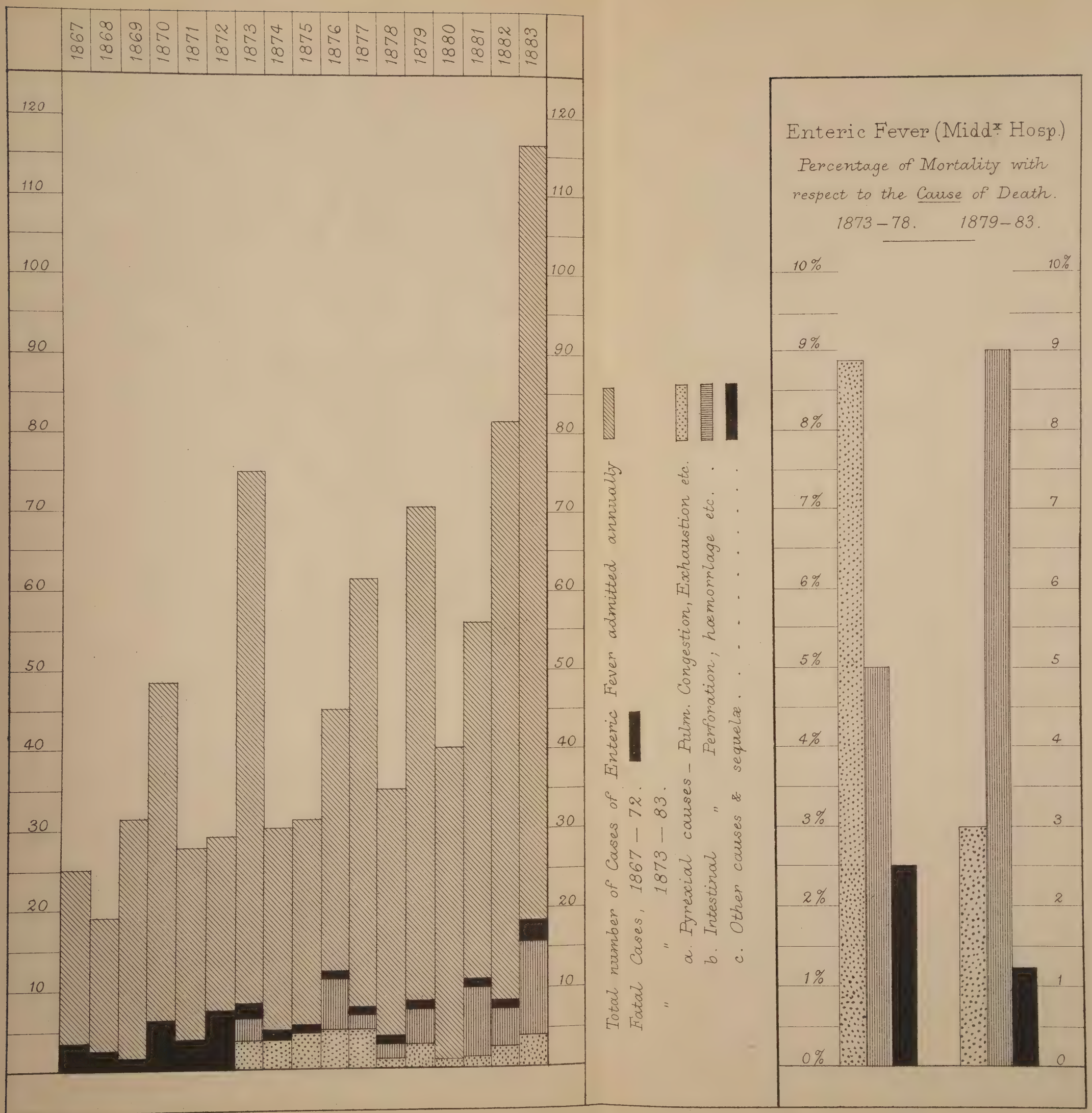


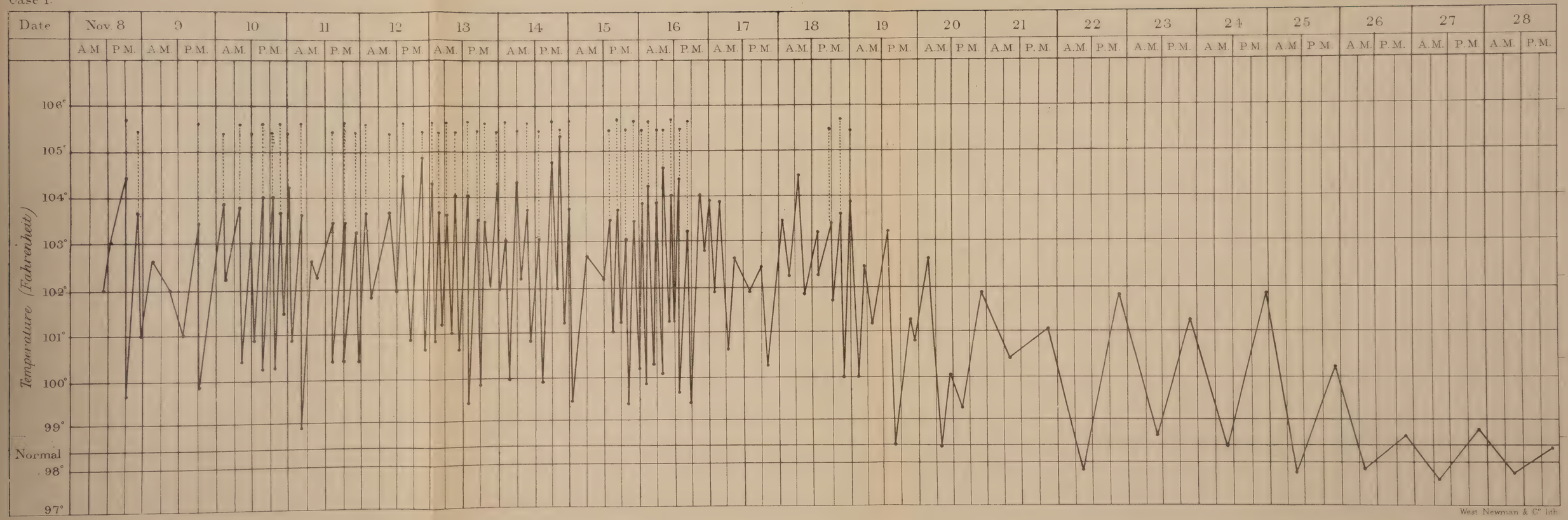
Chart showing Mortality in Enteric Fever at Middlesex Hospital, and (from 1873 – 83) the relative frequency of the Causes of Death.

West, Newman & C° sc.

Plate V.

See page 212.

Case 1.



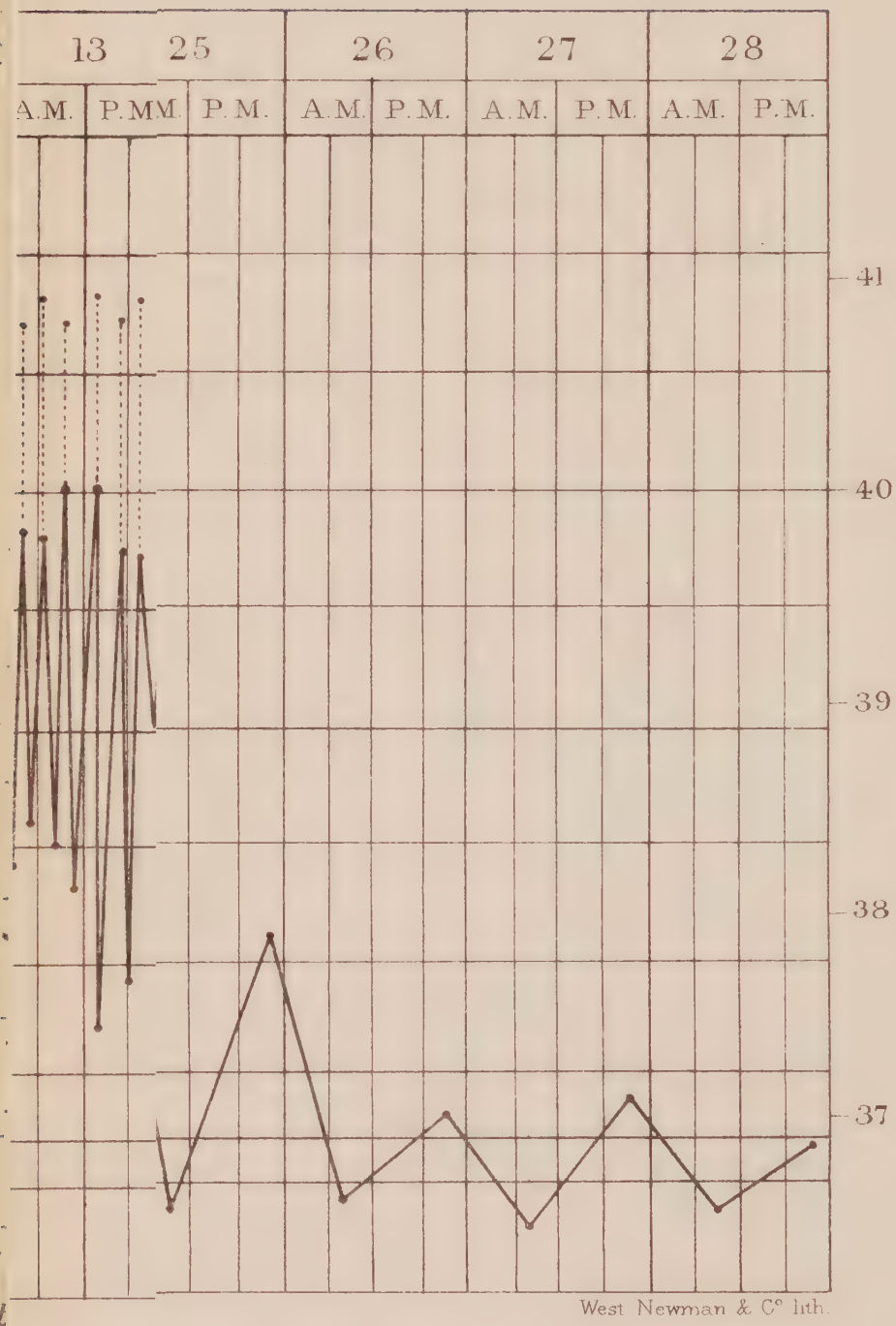
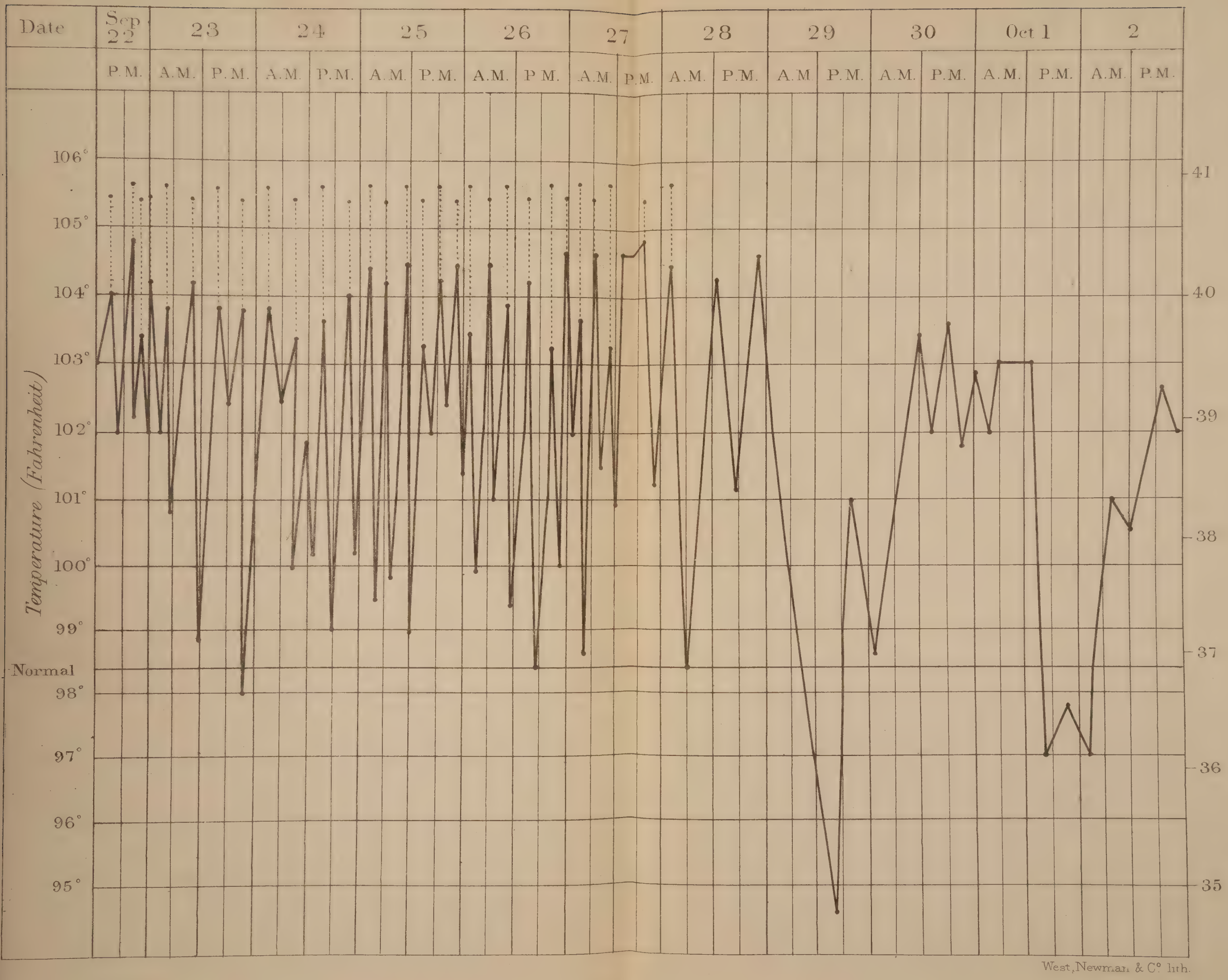

13
25
26
27
28
A.M.
P.M.
A.M.
P.M.
A.M.
P.M.
A.M.
P.M.
A.M.
P.M.
41
40
39
38
37
West Newman & C° lith.

Plate VI.

See page 224.

Case 2.

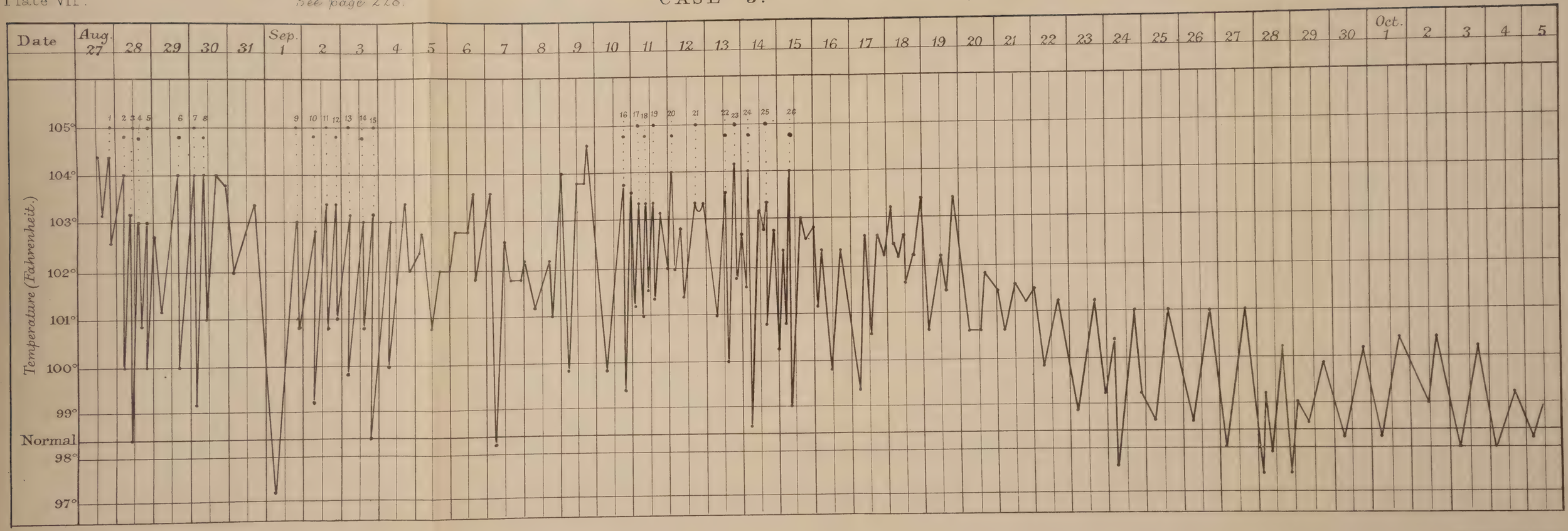


West, Newman & C° lith.

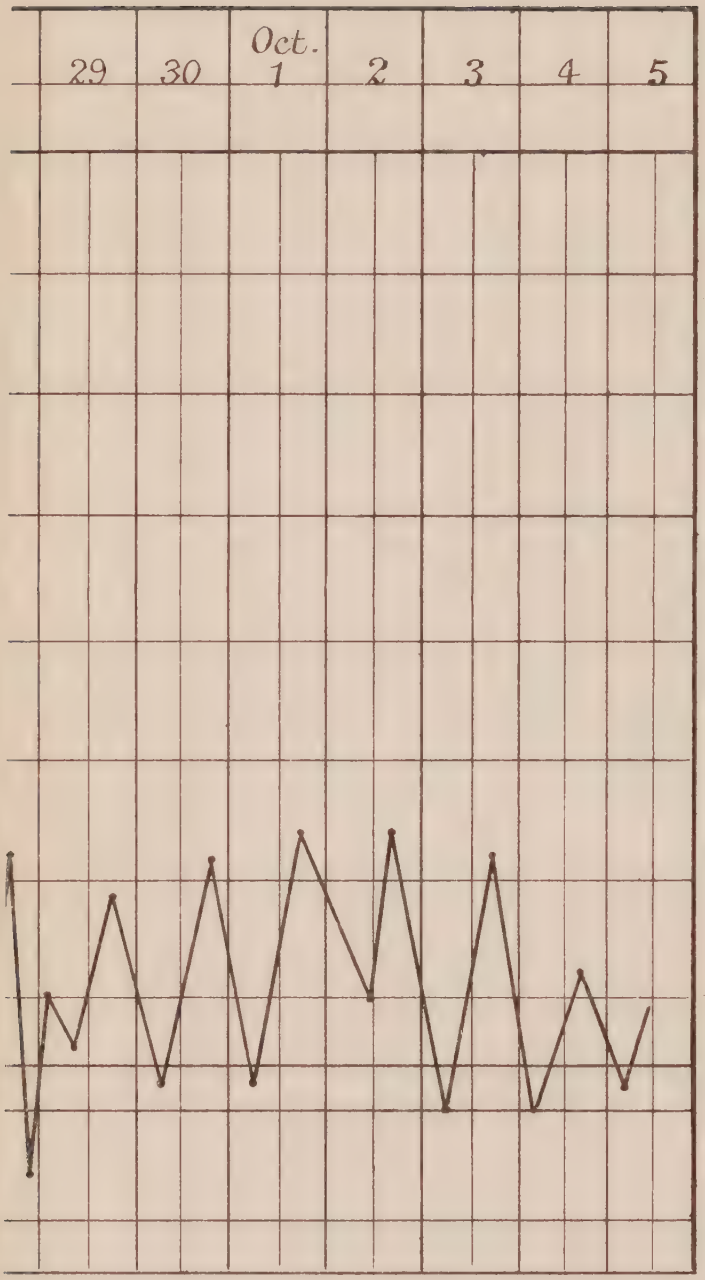
Plate VII.

See page 228.

CASE 3.



West, Newman & Co. lith.



West, Newman & Co. lith.

Plate VIII. *See page 236.* CASE 7.

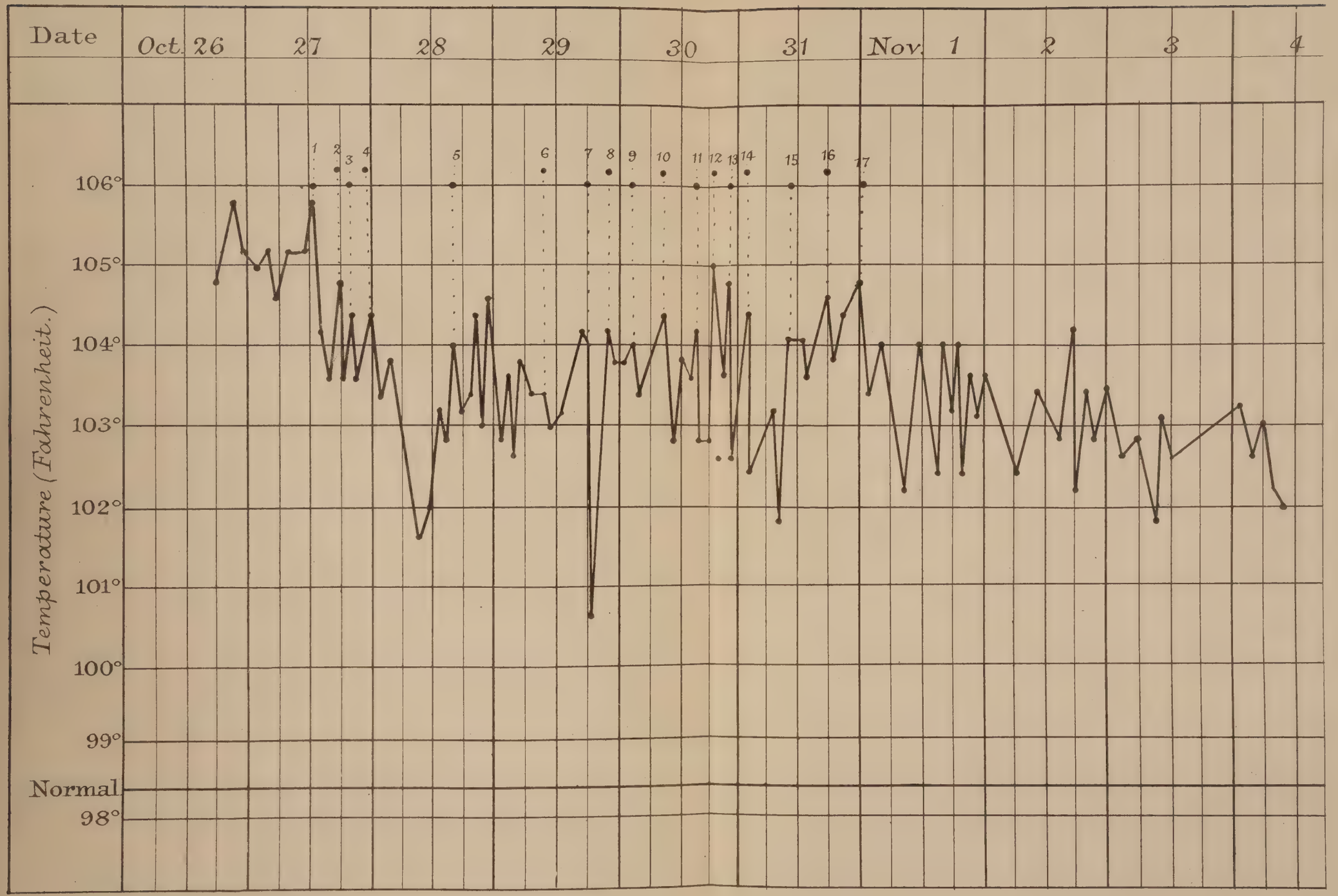
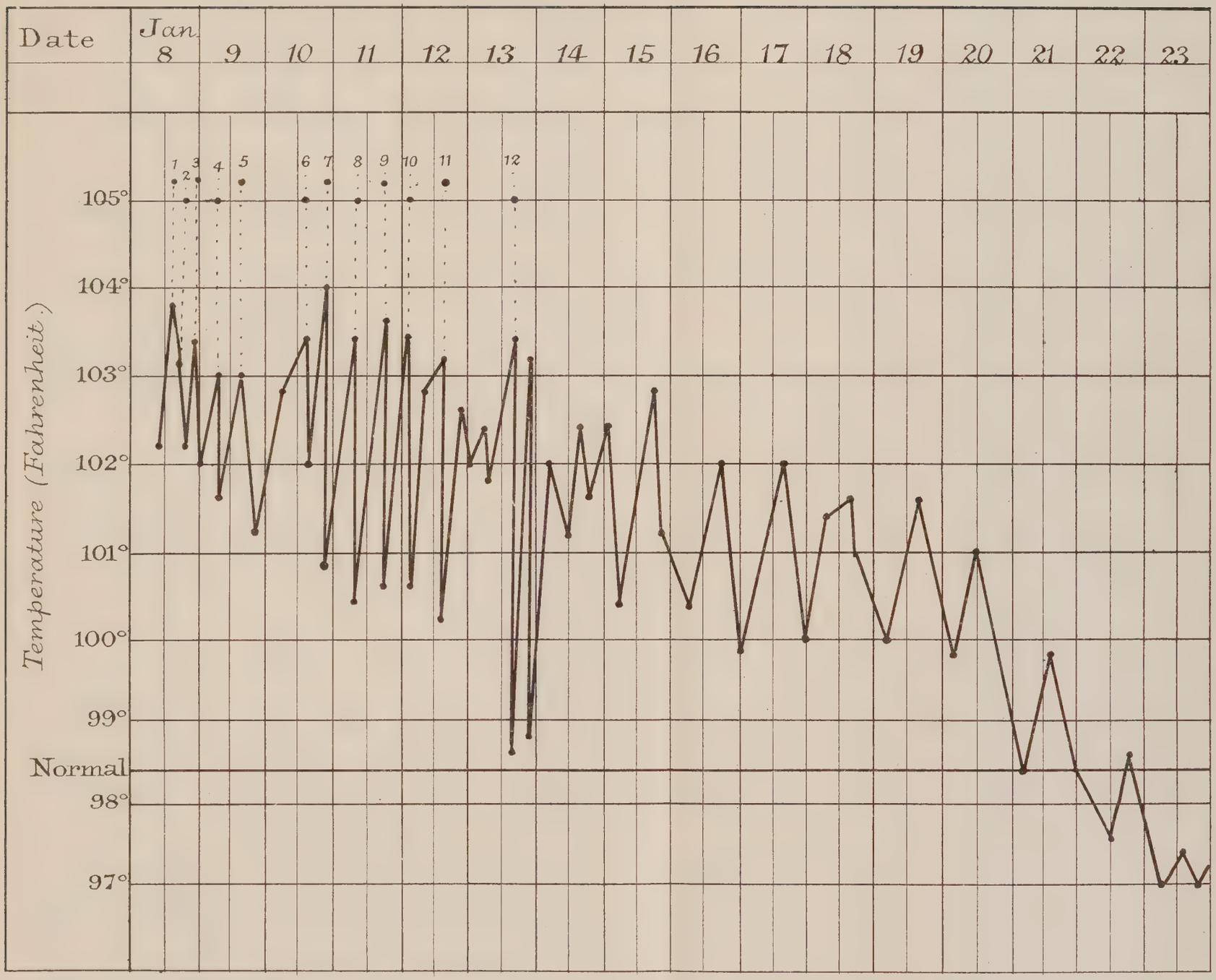


Plate IX. See page 244. CASE 12.



West, Newman & C° lith.

CASES BATHED.

No.	Sex, age.	Date of admission.	Day of the attack on which admitted	No. of baths.	Day of attack on which bath or baths given.	Day of attack on which sponging employed.	Day of attack on which compresses applied.	Day of attack on which quinine administered.	Day of complete defervescence.	Days of relapse.	Result.	Duration of stay in hospital.	Total duration of illness.	Remarks.
10	M. 15	Jan. 22, 1883	4th d.	14	5, 29, 30, 31, 32, 33, 34, 37 39, 41	5, 27, 28, 29, 33	...	...	18th	24th—44th	Rec.	74	78	Mild primary attack; severe relapse.
41	F. 26	Sept. 11, 1883	,,	2	4, 5	...	...	...	7th	...	,,	28	32	Abortive.
42	M. 17	Oct. 25, 1883	,,	2	4	...	...	...	49th?	...	,,	83	87	Prolonged pyrexia; sequelæ:—abscess and synovitis of elbow-joint.
50	F. 16	Nov. 27, 1882	,,	1	7	4, 7	...	4, 5	14th	...	,,	45	49	Slight diarrhœa.
11	M. 17	Sept. 22, 1880	5th d.	13	5, 6, 12, 13, 14, 15, 16, 18	...	...	...	22nd	...	,,	51	56	From 8th to 11th day morning temperature normal; 12th to 15th high fever; constipation; otorrhæa.
36	M. 15	Sept. 5, 1882	,,	3	20, 21	5, 6, 7, 9, 10	...	...	31st	54th—60th	,,	71	74	Relapse set in 2 days after placed on fish diet, fresh roseola.
37	F. 30	Mar. 20, 1883	,,	3	15, 16, 18	5, 6, 7	8, 10, 11, 12	5, 7, 9, 12	57th	...	,,	78	83	Prolonged, severe; commencing with pleuropneumonia.
40	M. 25	Oct. 22, 1881	,,	2	10, 11	11, 12, 13, 14	...	10	28th	...	,,	45	50	Began with pneumonia; 5th to 10th days temp. 103° to 104°; before antipyretic treatment.
46	M. 21	Oct. 25, 1881	,,	1	5	...	5	...	15th	...	,,	45	50	Mild; slight diarrhœa and bronchitis.
25	F. 16	May 19, 1882	6th d.	6	6, 7, 8, 10, 14	...	...	6, 7, 8, 9, 10, 11, 14, 17	28th	...	,,	48	54	Slight diarrhœa; rose spots till 20th day.
27	M. 12	Feb. 16, 1880	,,	5	6, 7, 8	...	...	...	15th	...	,,	37	43	Marked cardiac debility.
28	F. 14	May 11, 1882	,,	5	6, 8, 9	...	...	7, 8, 11	15th	...	,,	45	51	Diarrhœa marked; bronchitis marked.
30	F. 16	Oct. 9, 1883	,,	5	6, 7, 10	...	8, 9	...	21st	...	,,	31	37	Fair amount of bronchitis.
20	M. 11	Apr. 20, 1879	7th d.	9	8, 9, 10, 11, 12, 13, 16, 18	14, 16, 17, 18, 19	...	8, 9, 10, 11, 12, 13, 16	23rd	...	,,	62	69	High fever from 7th to 15th day, temperature over 104°; quinine given after each bath, baths tepid; slept well, no complications.
39	M. 25	Sept. 27, 1880	,,	2	8	...	...	...	36th	...	,,	43	50	Prolonged irregular fever; constipation.
43	M. 11	Nov. 7, 1883	,,	2	7, 21	21	8, 9, 10, 11, 12	...	27th	...	,,	42	49	On 21st day temp. 103·6°; ? relapse.
47	M. 4	Nov. 11, 1881	,,	1	10	7, 10	...	...	22nd	...	,,	36	43	Moderately severe; much eruption.
56	F. 32	Dec. 17, 1883	,,	1	8	7, 8, 9, 10	...	...	15th	...	,,	44	51	Mild.
1	M. 12	Nov. 8, 1880	8th d.	48	8, 9, 10, 11, 12, 13, 14, 15, 16, 17, 18, 19	17	...	8, 10	20th	...	,,	43	51	Persistent pyrexia; no delirium; bronchitis; twice slight hæmorrhage.
4	F. 31	Oct. 25, 1880	,,	19	8, 9, 10, 11, 12, 13, 14, 15, 16, 17	...	...	8	25th	...	,,	46	54	14th day, appearance of rash, like rötheln, fading on 16th; great prostration; slight bronchitis, no diarrhœa.
6	F. 21	Jan. 24, 1882	,,	17	8, 9, 10, 11, 12, 13, 14, 16, 17, 37, 38	9, 10, 38	...	9, 10, 12, 39, 41	27th	34th—50th	,,	85	93	Severe as to pyrexia, both in primary and in relapse; bronchitis; feeble pulse; slow convalescence.
14	F. 11	Feb. 11, 1882	,,	12	14, 15, 16, 17, 18, 19, 20, 21, 22	18	...	...	27th	...	,,	44	52	Moderate pyrexia.
15	F. 23	Dec. 22, 1883	,,	11	10, 11, 12, 14, 15, 16, 17	9, 10, 11, 12, 13 15, 16, 17, 18, 19	...	12, 13, 14, 15	27th	...	,,	40	48	Rather severe; constipation; headache and insomnia; no complication.
23	M. 9	Aug. 3, 1881	,,	7	15, 16, 18, 19	8	...	...	26th	...	,,	49	57	7th to 14th day, evening temp. 102·6° to 103·6°; slight bronchitis; constipation.
44	F. 17	April 7, 1879	,,	1	38	...	...	42	18th	28th—49th	,,	78	86	Rheumatic symptoms at first, with mild pyrexia till relapse.
45	F. 24	May 26, 1880	,,	1	15	11, 14, 15, 16, 17, 18	...	11, 14	25th	...	,,	44	52	Severe; hæmorrhage on 18th day.
48	M. 40	Dec. 7, 1881	9th d.	1	18	11, 12	...	...	35th	...	,,	44	53	Marked diarrhœa.
24	M. 19	Sept. 14, 1883	,,	7	10, 11, 12	18, 23	10, 11, 12, 13, 16, 17, 20, 23	...	36th	...	,,	66	75	Severe; hæmorrhage on 20th to 24th day; bedsore and sequela of gangrene of heels delayed convalescence.
2	F. 33	Sept. 22, 1880	10th d.	29	10, 11, 12, 13, 14, 15, 16	16	15	10, 12, 13	48th	...	,,	100	110	Severe case; much hæmorrhage, prolonged fever.
9	M. 15	Feb. 20, 1880	,,	14	10, 11, 12, 13, 14	13	...	...	20th	...	,,	41	51	Attacked whilst convalescing from acute pneumonia; feeble rapid pulse; no diarrhœa.
18	F. 15	Mar. 24, 1882	,,	10	10, 13, 14, 15, 16, 18, 23, 24	...	...	11, 12, 15, 16, 20, 22	39th	...	,,	65	75	Prolonged fever; much pulmonary congestion.
31	F. 22	Jan. 20, 1882	,,	4	18, 21	12, 43 to 50	10, 15, 16, 19	...	33rd	43rd—50th	,,	76	86	Much bronchitis.
38	M. 18	Nov. 21, 1883	,,	3	10, 11, 15	16, 17, 19	...	...	20th	...	,,	27	37	Diagnosis obscure at first; he was suffering from otorrhœa and severe headache with high fever.
3	F. 24	Aug. 27, 1883	12th d.	26	12, 13, 14, 15, 17, 18, 19, 20, 26, 27, 28, 29, 30, 31	12, 30	22, 23, 24, 25, 26, 31, 32, 33	13, 16, 26, 27, 28	58th	...	,,	106	118	Remarkably severe and prolonged; much cardiac feebleness; convalescence retarded by periostitis of tibia.

TABLE **A** (*continued*).

No.	Sex, age.	Date of admission.	Day of the attack on which admitted	No. of baths.	Day of attack on which bath or baths given.	Day of attack on which sponging employed.	Day of attack on which compresses applied.	Day of attack on which quinine administered.	Day of complete defervescence.	Days of relapse.	Result.	Duration of stay in hospital.	Total duration of illness.	Remarks.
5	M. 26	July 12, 1881	12th d.	18	30, 31, 32, 33, 34, 35, 36	12	...	13, 27, 28, 29, 30	40th	?	Rec.	37	49	Hæmorrhage on 13th day (temp. 104·2° to 100°); prolonged pyrexia without abatement; rose spots appearing as late as 33rd day; marked bronchitis throughout; left at his own request, very feeble.
8	F. 11	Oct. 24, 1883	,,	17	14, 15, 16, 17, 18, 20, 21	17, 18	14, 15, 16	Kairin (5 doses), 13, 14	28th	...	,,	68	80	A phthisical subject; some anxiety on this score.
19	F. 22	Nov. 13, 1882	,,	10	16, 17, 18, 19, 21	12, 13, 14, 15	...	...	26th	...	,,	60	72	Baths 85°; prolonged constipation; rise of temperature on 42nd day, falling after enemata.
22	M. 15	June 3, 1881	,,	7	13, 16, 18, 19, 42, 44	42	...	41	30th	39th—46th & 51st—60th	,,	62	74	Prolonged pyrexia and relapses.
34	M. 23	Sept. 15, 1881	,,	3	14, 15, 16	...	...	15, 16 ?	24th	...	,,	33	45	Considerable diarrhœa.
53	F. 28	Oct. 9, 1883	,,	1	14	12, 13	...	...	51st ?	..	,,	76	88	Prolonged 'low' pyrexia; considerable diarrhœa; convalescence retarded by apparent loss of control over sphincter ani, and mental enfeeblement.
54	M. 33	Nov. 3, 1883	,,	1	16	...	12, 13, 14, 15, 16, 17, 18, 19, 20, 21, 22, 23, 24	...	30th	50th—55th	,,	54	66	Prolonged pyrexia; relapse due to indiscretion of diet.
29	F. 10	Oct. 8, 1883	13th d.	5	13, 15, 17, 18	...	16, 19	...	27th	...	,,	40	53	Constipation.
49	M. 25	Feb. 10, 1882	,,	1	15	...	...	...	20th	...	,,	46	59	Mild case.
26	F. 16	Sept. 8, 1882	14th d.	6	18, 19, 20	14, 40	...	30, Sod. sal., 22, 41	23rd	36th—46th	,,	60	74	Relapse rather severe.
52	M. 26	Sept. 21, 1883	,,	1	14	...	17, 19, 20, 21, 22, 23, 31, 32, 34, 35, 36, 38, 40	...	52nd	...	,,	88	102	Debilitated by alcoholism; constipation; slight rise in temperature 61st to 64th day; on 62nd day iliac thrombosis, which greatly prolonged his stay in hospital.
Cases admitted in 3rd week:														
17	M. 19	Feb. 2, 1882	? 18th day	10	18, 19, 20, 22, 23, 24	18, 20, 21	...	...	...	...	Died	13	31 ?	Severe diarrhœa on 23rd day; signs of perforation on 26th; *P.M.*, ulcers large, denuded, few; one had perforated.
33	F. 40	Sept. 19, 1881	? 20th day	3	23, 25	...	...	21	36th	...	Rec.	53	73 ?	Much delirium at first; pulmonary congestion and pneumonia (right lung).
51	M. 9	Sept. 18, 1883	? ,,	1	20	...	...	...	30th	...	,,	31	51 ?	Mild, no diarrhœa after admission; some bronchitis.
Cases admitted in relapse in 4th week of illness:														
7	M. 17	Oct. 26, 1882	? 25th day	17	26, 27, 28, 29, 30, 31, 32	31, 32, 33	28	27, 28, 30, 32	...	...	Died	9	34 ?	"Ambulatory typhoid;" mild primary attack; very severe relapse, setting in about 24th day; hyperpyrexia; pulmonary congestion; *P.M.*, bowel lesions in various stages, intense pulmonary congestion.
12	M. 19	Jan. 8, 1881	? ,,	13	24, 25, 26, 27, 28, 29, 30	...	...	...	...	6th week	Rec.	52	77 ?	Mild relapse, but great debility; axillary abscess following.
16	F. 35	Dec. 27, 1881	? ,,	10	30, 31, 32, 33, 34	...	...	...	...	...	,,	46	70 ?	Much bronchitis; diarrhœa; "typhoid state" marked.
21	F. 34	Oct. 2, 1883	? ,,	9	25, 26, 27, 28, 29, 30, 31, 32 33	29, 39	30, 31, 34, 35	Kairin, 36, 37	...	...	Died	25	50 ?	Severe; much diarrhœa; profuse hæmorrhage in last week; *P.M.*, numerous denuded and sloughing ulcers in colon as well as ileum.
35	F. 22	Jan. 21, 1882	? 28th day	3	28, 29	28	...	30, 31, 32	...	...	Rec.	65	93 ?	Severe case; much diarrhœa; hæmorrhage; pulmonary congestion; delirium.
Cases admitted in relapse in 5th week of illness:														
13	M. 16	Jan. 16, 1881	? 30th day	12	30, 31, 32, 33, 34	...	...	30, 32, 34	...	...	,,	51	81 ?	Pulmonary congestion; bronchitis; delirium.
Cases admitted in relapse in 6th week of illness:														
55	F. 21	Nov. 21, 1883	...	1	Day of admission	One day after	...	...	...	...	Died	3	?	Never recovered strength after primary attack; perforation; *P.M.*, a few ulcers; denuded; one perforated.
Apparently contracted in Hospital:														
32	M. 30	Feb. 15, 1883	...	4	28, 30	...	35	...	20th	26th—37th	,,	...	...	Admitted with acute rheumatism; endocarditis; slight albuminuria; perforation.

CASES NOT BATHED.

TABLE B.

No.	Sex, age.	Date of admission.	Day of attack on which admitted.	Day of attack on which sponging employed.	Day of attack on which compresses applied.	Day of attack on which quinine administered.	Day of complete defervescence.	Days of relapse.	Result.	Duration of stay in hospital.	Total duration of illness.	Remarks.
86	M. 8	Nov. 20, 1883	3rd d.	3	...	...	23rd	...	Rec.	31	34	A very mild case, for first week almost apyrexial.
59	M. 19	Dec. 15, 1879	5th d.	...	...	...	10th	...	„	30	35	Ditto, but contracted typhus, temperature rising on 28th day. Sent to Fever Hospital.
75	M. 24	Nov. 9, 1882	„	6	...	...	13th	...	„	19	24	Discharged at his own request.
71	M. 24	Feb. 1, 1882	6th d.	...	...	...	14th	...	„	34	40	Mild; abortive.
72	M. 29	Aug. 28, 1882	„	...	6 (to arm), 7, 10	Sod. sal. 7	20th	32nd to 43rd	„	96	102	Marked by constipation.
74	F. 4	Oct. 20, 1882	„	...	...	...	27th	...	„	21	27	Severe case; bronchitis; diarrhœa; removed by mother just as defervescence apparently set in.
60	M. 8	Sept. 4, 1880	7th d.	...	...	...	17th	...	„	24	30	Mild case, but weakly emaciated subject.
69	M. 7	Dec. 29, 1881	„	...	...	...	18th	...	„	49	56	Moderately severe.
77	F. 28	Dec. 10, 1882	„	...	...	...	16th	...	„	46	53	Slight diarrhœa; no bronchitis.
57	F. 6	Apr. 1, 1879	8th d.	12, 13, 14	...	...	20th	...	„	51	58	High fever (over 104°) from 8th to 15th day.
61	F. 28	Aug. 1, 1880	„	...	...	...	24th	...	„	44	52	Considerable diarrhœa.
68	M. 22	Dec. 5, 1881	„	..	...	...	23rd	...	„	47	55	Morning temperature normal from 13th day; considerable diarrhœa.
70	M. 13	Jan. 31, 1882	„	...	...	...	21st	...	„	35	43	Mild case.
78	F. 32	Dec. 20, 1882	9th d.	...	...	...	24th	...	„	57	66	Vomiting; high fever; no antipyretics.
63	M. 26	June 1, 1881	10th d.	...	...	From 40th day; often whenever temp. 103°	24th ?	...	„	69	78	No true relapse, but owing to extensive bedsore and abscess there was much pyrexia; delirium from 25th to 50th day.
65	M. 36	Sept. 22, 1881	„	...	...	...	35th	...	„	44	54	Mild only occasional temp. 102°, but protracted.
83	M. 27	Oct. 10, 1883	„	...	10, 11, 12, 13, 14, 15, 16	...	21st	...	„	29	39	Moderately severe.
85	F. 4	Nov. 15, 1883	„	11, 12, 13, 14	...	...	27th	...	„	43	53	Moderately severe; constipation mainly.
88	M. 24	Dec. 15, 1883	„	10, 11, 12, 13, 14, 15, 16, 17, 18	...	15, 16	22nd	...	„	25	35	Began with cerebral symptoms (fit followed by slight hemiplegia and aphasia), and pneumonia.
67	M. 24	Nov. 28, 1881	12th d.	14, 15, 16, 17, 18	...	...	...	...	Died	8	20	High fever (temp. 104°); pulmonary congestion; much diarrhœa; perforation; peritonitis; sloughs in process of detachment.
84	M. 20	Oct. 11, 1883	„	...	13	...	21st	...	Rec.	28	40	Mild.
73	M. 20	Oct. 18, 1882	14th d.	...	...	...	20th	...	„	29	43	Mild.
76	F. 17	Dec. 1, 1882	„	...	...	Mist. sod. sal. 24 to 28	32nd	...	„	52	66	Constipation.
Cases admitted in 3rd week.												
64	F. 12	Sept. 21, 1881	16th d. ?	...	...	Throughout pyrexial period	45th	...	„	47	63 ?	At first thought to be tuberculosis. In convalescence she contracted scarlet fever.
89	F. 24	Dec. 31, 1883	„ ?	16, 17, 18, 19, 23	...	16, 17, 18, 20, 21, 22, 23, 25	31st	...	„	42	58 ?	Pregnant 5 months; aborted 19th day; melancholia followed.
87	M. 8	Dec. 7, 1883	17th d. ?	18	...	...	20th	...	„	21	38 ?	Diarrhœa before admission; none after.
58	F. 19	April 7, 1879	21st d. ?	...	...	...	33rd ?	...	„	45	66 ?	Constipation.
62	M. 25	Aug. 20, 1880	„	21, 22, 23, 24	...	...	35th ?	...	„	46	67 ?	Irregular evening rise in temperature till 50th day.
66	M. 23	Nov. 19, 1881	„	...	...	22	37th ?	...	„	28	49 ?	Uncomplicated.
80	M. 28	Aug. 28, 1883	„	...	...	...	29th ?	36th to 41st	„	34	55 ?	Mild relapse with sorethroat and constipation.
81	M. 27	Sept. 20, 1883	„	...	...	...	31st ?	...	„	30	51 ?	No complication.
Admitted in 4th week.												
79	M. 13	Mar. 6, 1883	28th d. ?	29, 30, 31, 32, 33, 34	..	...	—	45th ?	„	52	80 ?	Probably a relapse.
Admitted in 6th week.												
82	F. 21	Oct. 10, 1883	„	...	...	Took quin. and sod. sal.	—	...	Died	44	—	Not diagnosed; at first thought to be tuberculosis; then pyrexia abated, and mitral disease detected; iliac thrombosis; death from syncope (heart disease); intestinal ulcers granulating.

N.B.—(1) The "day of the attack" here given is of course only approximate, but as much care as possible has been taken to ensure comparative accuracy in the reckoning. (2) The "day of defervescence," which, it will be seen, was often much delayed, signifies that day upon which the evening temperature first fell to the normal and remained so on the following days. It may be observed that in several cases the *morning* temperature fell to normal some days prior to this. (3) As a rule a *bath* was given whenever the temperature rose to 103° or above. The temperature of the bath was on an average 70° F.; the time of immersion being from ten to fifteen minutes. *Cold sponging* (for ten minutes) or the continuous application of *compresses* (*i.e.* cloths wrung out in ice-cold water, and renewed every ten minutes) to the abdomen, until the temperature fell to 99°, was mostly prescribed at temperature of 102°. *Quinine* was frequently given, in doses of ten or twenty grains, immediately after a bath, with the view of prolonging the period of apyrexia. Hence the frequency with which these two measures are recorded on the same day. (4) In many cases these alternative measures were taken prior to having recourse to bathing, as well as employed when bathing was contraindicated.

Summary.

A. Cases in which baths were employed.

		MALES.	FEMALES.	RECOVERED.	DIED.
23 baths and over	3	1	2	3	0
10 „ and under 20	16	8	8	14	2
5 „ „ 10	11	5	6	10	1
Less than 5 baths	26	15	11	24	2
	56	29	27	51	5
B. No baths in	33	22	11	31	2
Total	89	51	38	82	7

Mortality on the whole number 7·8 per cent.
„ „ bathed cases 8·9 „
„ non-bathed cases 6·0 „

Causes of death in fatal cases:

Of bathed cases—Intestinal perforation 3
„ hæmorrhage 1
Hyperpyrexia—Pulm. congestion 1 = 5
Of non-bathed cases—Intestinal perforation 1
Cardiac disease—Syncope 1 = 2
7

ON THE COLD-BATH TREATMENT OF ENTERIC FEVER.

By J. S. Bristowe, M.D., F.R.C.P.

My first duty is to apologise to the meeting for venturing to read a paper to-night. I do not apologise to the Secretaries, though I thank them; for they did me the honour of asking me some two months back to take part in the present discussion, and after mature consideration I determined in response to their invitation, although I had little special knowledge on the subject, to place that little at your service; but to my surprise I received from them a week or ten days ago an intimation that my paper was second on the list for this evening, and I have since seen an announcement to the same effect in the medical journals. Well, gentlemen, I confess I was taken aback, for there is a wide difference between reading a paper which should contain the results of tabulated experience and mature thought, and simply rising to make any casual remarks that may strike one for the purpose of promoting discussion and evolving truth from the conflict of opinion; there is a wide difference in appearing before you as the author of a something on which discussion is to be based, and appearing among you simply as a critic; and undoubtedly, if I had foreseen what was in store for me, I should have respectfully declined the proffered honour. I trust that under these circumstances I shall be forgiven if my remarks appear crude and insufficient when compared with the more elaborate addresses of the gentleman who precedes and the gentlemen who follow me. Indeed, I should like at once to acknowledge that my original intention in coming here this evening was to learn from those who know better than myself, and I doubt not that in this respect I shall not be disappointed.

There is nothing more difficult than to determine of medical treatment whether it does good or whether it does none. And of all methods of arriving at a conclusion on this point, the statistical is the most unsatisfactory and the most misleading. I suppose few, if any, doubt the efficacy of iodide of potassium and mercury in syphilis, of quinine in ague, of iron in chlorosis, and of salicylic acid in acute rheumatism. But the universal trust in these remedies does not depend on the flourishing of yards of statistical details before our eyes, but on the daily evidence presented to us of the cutting

short or cure of these maladies as soon as ever specific treatment is employed. In most cases we base our judgment of the efficacy of particular plans of treatment on the belief that the diseases for which they are employed are rendered somewhat less severe than they otherwise would be, or that their duration is shortened by a few hours. No treatment of disease was, in its time, believed to be more successful than that of pneumonia by antimony and calomel. At a later period alcohol in overwhelming doses was regarded by many distinguished physicians as a panacea for all kinds of inflammatory and febrile diseases. Alkalies are still believed in by many as the best of all cures for acute rheumatism. The efficacy of all the above remedies in the cure of the diseases with which I have associated them has been proved over and over again by elaborate statistics; the explanation of their efficacy has been made apparent both by chemists and by physiologists; numberless practitioners in all walks of the profession and all over the world have attested their value. Does anyone seriously believe in them now? And yet if they possessed one tithe of the virtue which has been ascribed to them their value ought to be wholly beyond dispute, and their neglect at the present day is little less than a scandal. What must be said about the cold-bath treatment of enteric fever? Is this a remedy of unmistakable value? Does it relieve? Does it injure? Does it cure? Does it kill? Or does its use, like that of many other remedies, simply prove that within certain limits many illnesses take their course uninfluenced by the skill or want of skill brought to bear upon them?

We must admit that strong *a priori* arguments may be adduced in favour of the cold-bath treatment. An abnormally high temperature is in itself injurious, and may be fatal. Excessive heat is at once a consequence of undue disintegration of body tissue, and a cause of further disintegration. And as a result of such disintegration effete matters accumulate in the blood and poison the system; and presumably therefore, if we can by the application of cold diminish these injurious conditions, the patient must to that extent be benefited. Indeed, there can be no doubt patients do seem, at any rate temporarily, benefited by immersion in cold water. But then it will be recollected that there were good *a priori* grounds for believing that the injection of saline fluid into the veins of cholera patients would prove curative, that such injections have in most cases been followed by temporary amendment, and that many

patients have recovered after its use; yet the practice has not in the long run commended itself to the profession.

On the other hand, there are surely theoretical reasons for fearing the cold-bath treatment might prove injurious. With all due respect for the omnipotent bacteria, I presume that there are good grounds for believing that inflammations and congestions of internal organs are largely due to exposure of the surface to cold and wet; that bronchitis, pneumonia, and pleurisy, inflammation of the peritoneum and bowels, nephritis, and many other like affections, are generally caused by such conditions. Does it not seem probable that such internal congestions and inflammations in typhoid fever might be excited or aggravated by cold bathing? Again, have we any actual proof that the specific processes of the disease are kept in abeyance or arrested by lowering the patient's temperature? Are the living organisms which are presumably its cause rendered less venomous or less prolific, or killed thereby? Do the intestinal lesions subside or heal more rapidly than they would otherwise do? Is death from intestinal hæmorrhage or perforation reduced in frequency? Is the rash modified in any degree?

Well, gentlemen, I have no doubt that statistical evidence may be adduced giving a satisfactory explanation or response to all my questions. But, as I have already stated, I have very little faith in the application of statistics to such questions, and before I could accept them should need to weigh the value of all the facts on which they were based, and even then should probably not be influenced by them, unless they were of overwhelming weight in themselves and supported or confirmed by other considerations. Dr. Cayley will recollect that a few years ago, on the occasion of a discussion in which the cold-bath treatment of enteric fever was involved, he adduced the then recently published results of the treatment of the disease by some German physician, which showed statistically that by means of it in his own hands the mortality of enteric fever had been reduced to 2 or 3 per cent. I think Dr. Cayley did not attach much importance to those statistics. Nor did I.

My personal experience in the treatment of enteric fever by the cold bath is not extensive, and for some years past I have rarely, if ever, had recourse to it. I have undoubtedly seen patients apparently benefited by it, undoubtedly benefited at the time, and making later a good recovery. But I have never felt perfectly satisfied in those cases that I have carefully watched that the benefit

to the patient was real, or that his recovery was determined or hastened by it. But I had two cases, occurring within a short time of one another, which impressed me strongly, and the results of which were the main cause of my giving up the habitual practice of the treatment. I have not had time to search for the notes of these cases, but I have no difficulty in recalling the main features of them. The first was that of a young man who had a very severe attack of enteric fever, who had had much diarrhœa, a little pulmonary complication, and in the second or third week was in the typhoid state, with a temperature reaching at least 105°. A few days before his death the cold-bath treatment was adopted *secundum artem*, and at first with a little apparent benefit. Then he almost suddenly fell into collapse, with livid surface, rapid, shallow breathing, and distension of the abdomen. It was thought that he had perforation of the bowel. At the post-mortem examination all the usual lesions of enteric fever were present, with very extensive ulceration of the bowel, but there was no perforation, and no sign whatever of peritonitis. The lungs, however, were in a condition which I never recollected to have seen before in enteric fever. They were small, slate-coloured, and almost empty of air throughout. They were in a state of universal collapse, without any trace of actual pneumonic consolidation. I could not help regarding the condition of the lungs as a consequence of the repeated application of external cold. A few weeks after this I had another case of enteric fever of about the same standing, and equally ill, and the question arose of using cold baths; recollecting the other case I decided not to employ them. A few days later the patient died, and I was present at the autopsy. The bowels were much ulcerated, but there was no perforation and no peritonitis. The lungs were exactly like those in the former case, and I at once remarked, "Well now, if this patient had been treated as the other was, by cold baths, I should have attributed his death to them; as it is, my explanation of death in the other case needs correction." The resident assistant physician, however, who was present, then informed me that after my visit the cold-bath treatment had been had recourse to. I have little doubt that my explanation of the condition of these patients' lungs was correct, and that the immediate cause of death in both cases was the lung affection. Of course I do not mean to say that they would have recovered even if the baths had not been given to them.

I have recently been speaking on this subject to a young Austrian physician who has been for several weeks past in London, and he tells me that at some large hospital in Austria—I am not sure whether it is at Vienna—the use of cold baths, which were formerly largely in vogue, has been discontinued owing to the heavy mortality and to the frequent occurrence of intestinal hæmorrhage among the patients thus treated. I should be interested to know what is the experience of other practitioners in London as regards the occurrence of congestion and collapse of the lungs, and congestion and hæmorrhage of the bowels as a consequence of cold bathing.

When any plan of treating disease is introduced on high authority it is usually largely adopted, and finds many enthusiastic advocates. There is one reason, no doubt, why the cold-bath treatment should be an exception to the rule—namely, that it is difficult and expensive of application. Nevertheless I believe that it has been very largely adopted abroad, and even to no inconsiderable extent in our own country and in this metropolis. It was once much employed at St. Thomas's. Some of my colleagues were much more enamoured of it than myself, and some at any rate have written strongly in its favour, but for several years its use has only been exceptional. My impression is that it has been largely discontinued in most of the hospitals of London. The German gentleman whom I just now quoted assures me that its employment has almost died out in Austria, and some other parts of the Continent; and that even in Prussia it is scarcely in use, excepting by two or three enthusiasts who have identified themselves and their reputation with it.

If the view above expressed of the liability of the cold-bath treatment to induce dangerous internal complications, and if its large discontinuance by those who formerly employed it be correct, they present strong arguments against its value as a curative agent. I acknowledge, however, my lamentable ignorance on the subject. What I have written I have written mainly to draw the advocates of the cold-bath treatment. I have come to talk and learn, and not to teach; and if I have treated the subject flippantly, my excuse must be that I never anticipated writing a paper, that I have had no time to collect material to write one of any value, that, after all, I have taken up only a little of your time, and that though I have taken the opposition side of the question, I am

ready in the most cowardly way at any moment to strike my colours, or to desert to the enemy.

Dr. CAYLEY first pointed out that much of what had been brought forward was not antipyretic treatment; by thorough antipyretic treatment was meant the employment of means for reducing the temperature from the first, and continuously throughout the course of the case. The failure of heart and respiratory power, which so much tended to produce collapse of the lungs, of which Dr. Bristowe had been so frequently frightened, was really much less frequent in cases treated systematically with cold. He further criticised Dr. Bristowe's remarks. Was it more scientific to use a chemical balance or to infer from muscular impressions, or was the microscope of no value because some observers had originally made errors therewith? These questions were asked in part reply to Dr. Bristowe's objection to statistics. Dr. Cayley contended that if statistics conformed to certain canons, if they extended over a sufficient length of time, included large numbers, and dealt with like cases, they could certainly be trusted. Dr. Bristowe had said that the cold-bath treatment of enteric fever had fallen into disuse in Germany. But was it not true that the Minister of War for Prussia had ordered its employment in the Prussian army? And Dr. Sagetzky had said, "We only here and there meet with a solitary opponent." Dr. Cayley next proceeded to give statistics from the London Fever Hospital. Murchison had shown that the disease was much more fatal among the well or overfed classes—the rate of mortality was much lower among the Irish both in Ireland and England. The mortality for six years after 1865 showed an average death-rate of 14 per cent.; from 1872 to 1878 it was 17 per cent.; and from then onwards it was 14 per cent. During the last period the cold treatment had been in vogue. When it was remembered that during the first period the number of poor patients was considerably greater than later on, and when it was understood that among the better classes the mild cases would be treated at home and only the bad cases be sent to the hospital, we might see some reason for believing that even the reduction of 3 per cent. told something in favour of the cold treatment. The tables also showed, at first sight, an increase in the number of deaths from hæmorrhage and perforation under the cold

treatment; but this was probably a fallacy, and due to insufficient numbers and to the fact that bad cases came into the hospital late in the disease. At Middlesex Hospital the percentage of fatal cases under the cold treatment was 11·8 per cent. But, lest these numbers be considered not numerous enough and not to extend over a sufficiently long period, the statistics of the Charité Hospital, at Berlin were quoted as of unbiassed source and as fulfilling the conditions of reliable statistics which he had previously laid down. From 1848 to 1867, 2228 cases were treated with 405 deaths, or 18 per cent., being pretty much the same as in our general hospitals. From 1868 to 1876, 2086 cases, with 267 deaths, or 13 per cent., this being under the employment of cold-water treatment. Again, the Prussian army statistics showed from 1868 to 1874 a mortality of 15 per cent.; whilst from 1874 onwards, under the cold treatment, the percentage was 9·7 per cent. From French sources, information is obtained from Jaccoud that from so large a number as 80,000 the percentage mortality was 19 without and 11 with the application of cold. No doubt there were a large number of cases in which the treatment was not applicable. He thought the bath better than large and repeated doses of antipyretic drugs. With regard to the mode of action of cold he considered that it answered to physiological indications, viz. to diminish metabolism, the excessive character of which Murchison believed gave rise to deleterious products which induced the symptoms of the typhoid state. The stimulating effect of the bath on the vaso-motor system counteracted that conspicuous want of vascular tone and tended to prevent granular degeneration and softening of parenchymatous organs. Further, the symptoms of the disease were materially alleviated, the delirium, dry brown tongue, &c., ceased to form prominent features of typhoid fever when genuine antipyretic treatment was adopted. The opinion (that typhoid was robbed of its terrors) of Dr. Austin Flint, who was not an ardent advocate of the system, was a piece of valuable testimony. It was further contended that the complications were not rendered either more severe or more numerous. Lastly, it was to his mind incontrovertibly proved that the mortality was greatly reduced.

THE TREATMENT OF TYPHOID FEVER BY BATHS.

By Frederick Taylor, M.D.

Mr. President and Gentlemen,—I have been asked to bring before the Medical Society the results of the treatment of typhoid fever by the bath as practised at Guy's Hospital.

I am glad to have the opportunity of doing so because I have always been inclined to look favorably upon this method of treatment, and I first used it myself in a case in Guy's Hospital in the year 1874. In giving the experience of Guy's Hospital, I have gone back to that time, and have analysed to the best of my ability in the time at my disposal, all the cases treated by cold applications from that time to the present, *i.e.* during the years 1874—1883 inclusive. I have since found that Dr. Wilks treated a patient in 1873 by this method, four baths being given to a patient on the temperature reaching 104°.

The subject for discussion this evening has been announced as the treatment of enteric fever by the cold bath, and I should have been willing to limit my remarks to that alone, but I find that the field of observation at Guy's Hospital is more than doubled by including all other methods of application of cold externally for the purpose of carrying off heat from the surface of the body; and as this is alone the *rationale* of the bath treatment, I think, inclusion of the other methods is justified.

I may say that these are the methods of reduction of heat which have been in use at Guy's Hospital.

The bath.—At different temperatures from 90° to 70° F.; the patient being sometimes immersed at once in a bath of the lower temperature or below 80°, sometimes placed in a bath at 90°, the temperature of which has been at once lowered by the introduction of pieces of ice.

In the majority of these cases the temperature of the patient has been taken every three hours, and the bath has been used when the thermometer has marked 103°.

2. *Sponging* has been freely used, mostly with cold or iced water. I need not describe the method in detail.

3. The *cold wet pack*—or ice pack as it has been called, has been employed.

4. *Ice-bags* have been applied to the axillæ. I believe the first introduction of this practice was due to one of our students,

Mr. W. H. Strachan, who devised an arrangement for cooling the blood in the main arterial trunks, so far as they were accessible, by a continuous current of cold water. Small reservoirs were placed in the axillæ and groins in contact with the axillary arteries and femoral arteries respectively, and to each reservoir were attached two tubes, one for the introduction of water, the other for its discharge. It was thus hoped that the temperature of the blood in the axillary vessels would be lowered and the cooler blood distributed through the body, while, of course, a constant change of water was required to keep the effect continuous. Ice-bags changed from time to time, as the ice disappeared, would have a similar effect with much less trouble.

5. *Leiter's coils* of tubing for the continuous circulation of water have been used in a few cases, applied either to the chest, or abdomen, or to both.

All these are methods of cooling the body, though the degree of cooling is apt to be different in different cases.

I must at once confess I feel that the material before me is not entirely satisfactory, so far as it is expected to provide an answer to the question. "Is the cold bath a valuable method of treatment in typhoid fever?" and that feeling arises from the fact that so few cases over the whole period of ten years have been systematically and thoroughly treated by this method. It is true that out of 440 patients with typhoid fever during that time, 100 were submitted to some form of cooling treatment, either as bath, or sponging, or wet pack, or one at one time, another at another, in the course of the illness; but it will be seen that the number in which frequent applications of the bath were made is comparatively small, while the variation of treatment in the same case rather tends to complicate the conclusions.

I may here say at once that though only 100 individuals had cooling treatment, nine of these suffered from definite relapses, in which they were again submitted to a similar treatment. I have therefore, considered such cases as double, and include in my analysis 109 cases as corresponding to the 100 individuals. Of this number—

72 had cold-water baths, and of these 27 were otherwise cooled as well.

43 had cold spongings; of these 15 were otherwise cooled as well.

13 had ice-packs; of these 7 were otherwise cooled as well.

13 had ice-bags; of these 9 were otherwise cooled as well.

2 had Leiter's coils, in addition to other means.

Hence it is seen that in forty-five cases the method of cooling was varied in the course of the illness. To what extent is this of importance? Can the same effect be obtained from the different methods?

The *rationale*, as I have stated, is the same, and there does not appear any good reason why, even if the bath is most quickly effectual, the temperature should not be reduced to a similar extent by a sufficient duration of the sponging. As a fact, however, it is quite clear from the notes in the reports that the spongings were less effective. The bath constantly reduced the temperature 4° or 5°, and in some cases 6° or 7°; whereas sponging often gave a depression of only 1°, 2°, or 3°. Some cases of ice-pack were as successful as the bath, reducing the temperature 4° or 5°. Leiter's coils and the ice-bags were much less satisfactory to judge from the reduction of temperature alone. On the whole, I have preferred to consider all the cases together, though not losing sight of the minor differences in the kind of treatment employed.

Besides variations in the method we have to consider the persistence with which each has been carried out, or, at any rate, the number of baths or coolings that have been employed, whether this was determined by the severity of the pyrexia, by the date of the disease at which the patient came under observation, or by the occurrence of complications. No one can suppose much good has resulted from the single bath in those cases where only one has been given. If the temperature is only above 103° on one or two occasions, the disease is but mild, and will almost certainly get well of itself, and, at any rate, the complications which are fatal, not being dependent on severe pyrexia in such a case, would not be preventable by antipyretic means. Again, a patient who is admitted in the third week of a moderate or mild typhoid may have his temperature at 103° only on one or two evenings for a few hours, and here again there is little scope for the bath; if harm was to be done by the pyrexia it has been done already; and though even here a little reduction of temperature might prove a turning-point in the case it can never be felt that such cases are so conclusively in favour of the treatment as those in which the disease has been severe and the treatment often repeated. They are, however, rightly included in any statistical statement, as will be shown presently. Were these deducted, the

number of the cases of bathing, that is, those in which the baths or spongings were frequent, would be found relatively small.

Thus of 72 cases the number of baths is roughly given below:

17	cases had	only 1 bath.
29	,,	from 2 to 5 baths.
16	,,	from 5 to 10 baths.
9	,,	more than 10 baths.
1	,,	unrecorded.
72		

But 27 of these were cooled by other means, and if we take the remaining 45 cases, treated by bath alone, we find the following:

	12	cases had	1 bath.
22	4	,,	2 baths.
	7	,,	3 ,,
	6	,,	4 ,,
	5	,,	5 ,,
4	3	,,	6 ,,
	1	case had	7 ,,
7	2	,,	11 ,,
	1	case had	14 ,,
	2	cases had	15 ,,
	1	case had	31 ,,
	1	,,	37 ,,
45			

In a similar way 43 cases sponged (including 15 treated in other ways also) were divided as follows:

3	cases had	1 sponging.
19	,,	2 to 5 spongings.
7	,,	6 to 10 ,,
10	,,	more than 10 spongings.
7	cases unrecorded.	
43		

The above will show the material before us, and I will now proceed to the conclusions which, I think, may legitimately be drawn from it with reference to the value of the antipyretic treatment.

When we desire to estimate the value of a treatment of this kind of disease we may consider it from three points of view:

1. The effect upon mortality.
2. The effect upon complications and sequelæ, including relapses.
3. The immediate effect upon the symptoms and general comfort of the patient.

In typhoid fever the mortality is of the first importance; the complications and sequelæ that do not imperil life are much less important, and the main test of any treatment that may be introduced for typhoid fever will be the test of mortality.

The mortality of this disease is very different in different years, and in different epidemics. From returns that have been published the percentage mostly varies from 10 to 24; but much lower and much higher mortalities have also been recorded.

Now, I propose to treat of the mortality of the cases treated at Guy's Hospital, dealing with those treated by sponging and ice-packs, as well as those treated by the bath alone; first giving the bare results and then examining how far they really represent the influence of the treatment in the disease. The following table gives the mortality from typhoid fever at Guy's Hospital during the last ten years, both amongst all the cases, and amongst those in which cooling methods were employed.

Years.	Total cases.	Deaths.	Percentage mortality.	Cases bathed and sponged.	Mortality.
1874	26	3	11·5	2	1
1875	31	3	9·7	2	0
1876	30	6	20·0	4	1
1877	37	3	8·1	5	1
1878	70	14	20·0	11 and 1 relapse.	2
1879	53	13	24·5	4	2
1880	30	9	30·0	9 and 1 relapse.	5
1881	48	7	14·6	13 and 2 relapses.	1
1882	60	11	18·3	12 and 1 relapse.	5
1883	55	9	16·3	38 and 4 relapses.	9
	440	78	17·7	100 and 9 relapses.	27

It will be seen that the percentage mortality was 17·7. It was different in different years, ranging from a low figure in 1874, 1875, and 1877, to a very high figure in 1879 and 1880. In 1882 and 1883 the mortality differed but little from the average of the whole.

To compare with these we have the mortality of those cases amongst them which were treated by cooling. Of 100 patients

so treated 27 died, making a mortality, of course, of 27 per cent. This appears at first sight to be a strong argument against the treatment by the bath, but a little consideration will show that the comparison, so stated, is not a fair one. As cases are only submitted to the bath when the temperature has reached a certain elevation, it is clear that some of the mildest cases really requiring no treatment but care and nursing will not have baths. It may be said that nearly every case of typhoid fever reaches a temperature of 103°, but even if this is true, it must be remembered that there are two or three ways in which such cases may have got off without a bath in hospital practice.

Firstly, as I have said, the temperature of 103° has not always been selected, but a higher one, namely 104°. Secondly, many cases come in at the end of a fever when the temperature after the first day does not rise above 103°, and for the first twenty-four hours the diagnosis may be uncertain, or the treatment may have been suspended till the attendance of the physician, and then the necessity for it may have passed away. As a fact, with a relatively high temperature for bathing, this treatment is applied actually only to the more severe cases; and the comparison should be drawn between these more severe cases in both categories, and not between all cases, mild or severe, of the unbathed, and the bathed cases, which include such an undue proportion of a severe form. This objection has, of course, less weight where the temperature at which a bath is administered is fixed comparatively low. The most just comparison would be where all cases admitted to one physician or to one ward or set of wards were treated in the old lines of expectancy, and all cases admitted to another physician or ward were at certain temperatures submitted to a cooling plan of treatment. A comparison of the same institution in different years is possible, but it is not very trustworthy, as the severity is so very variable at different times. Such a comparison may be attempted with the cases at Guy's Hospital, but the smallness of the figures renders any conclusion unreliable.

It is sufficient to say that the cases bathed or cooled in these years have been increasingly numerous, from two each in 1874 and 1875, to thirteen and twelve in 1881 and 1882, and thirty-eight in 1883; yet the mortality in those years has been curiously different, and practically it is impossible to say that the bathing has done good or harm from that consideration alone.

I have attempted to get the mortality of cases at Guy's Hospital prior to 1874, but do not feel sure that the results are accurate. Still, dealing with them as best I can, it appears that there were in the years 1866 to 1873 inclusive 176 cases with 27 fatal cases, a mortality of 15·3. Four cases were partially treated by bathing. I find that one case was bathed in 1873; four baths were given when the temperature reached 104°.

We may next consider the causes of death in the bathed and in the unbathed cases, to see whether the cooling treatment averts danger from one source more than another. It would, of course, be no great satisfaction to feel that patients are saved from one kind of death only to die by another; and it is therefore very desirable to inquire whether the treatment by bathing has any special influence upon the mode of death, whether in diminishing the number of cases which seem to die by the severity of the fever, or in increasing those which die of lung complaints or hæmorrhage from the bowels. Here I must confess the figures become so small that they have but little positive value. To take first all the fatal cases in the decennial period. I have arranged them in four groups, according to the cause of death, a classification which is perhaps not perfect, but will serve our present purpose:

A. *Exhaustion.*—That is death from the severity of the disease, at the end of the second or in the third week, or, of course, later in some cases, without any other complication than severe ulceration of the intestine, or hypostatic pneumonia or broncho-pneumonia.

B. *Peritonitis.*—Almost always due to perforation, but occasionally without any discoverable rupture.

C. *Hæmorrhage.*—This is not quite a satisfactory group; it includes fatal cases in which hæmorrhage has been a prominent symptom, and not only those in which hæmorrhage was the sole immediate cause of death. Perhaps some of these should go under Group A, but it is difficult to form an opinion from reports alone, and the interest of hæmorrhage in relation to this special mode of treatment justifies the separation of these cases.

D. *Other causes.*—Occasional complications, such as sloughing, gout, cystitis. Of these, however, a very small number occurred.

Taking the classification for what it is worth, and with every reserve as to the propriety of placing C as a separate group, I find that over the ten years we had 78 deaths, of which there occurred:

From exhaustion	33,	of which	9	had been cooled.
From perforation and peritonitis	32	„	9	„
From hæmorrhage	9	„	5	„
From other causes	4	„	3	„
	78	„	26	„

Among bathed cases there were actually 27 deaths; but one of these was not examined post mortem.

This shows that exhaustion and perforation were nearly equal in the two groups. Perforation was a little less frequent in the cooled cases by 34·6 per cent. to 41 per cent., not a very marked difference; hæmorrhage, on the other hand, was more frequent in the cooled cases by 19·2 per cent. to 11·5 per cent., a more notable difference, but less to be relied on.

One must next consider whether the treatment has in all cases had a fair chance; that is to say whether it has been applied sufficiently early, and pushed with sufficient energy to achieve a result. There must, of course, be a certain relation between the disease and that which is used to antagonise it; and as no one would expect a severe case of tertiary syphilis to be cured by two or three grains daily of iodide of potassium, so it must not be charged against the use of the bath that it is valueless, if cases receive but little benefit from one or two immersions.

Theoretically, it is of course desirable to make use of the bath as early as possible in the disease, and one must, therefore, in these cases consider how often the patient has come under treatment early enough in the disease for a fairly thorough bath treatment. As is well known, doubt often attaches to the estimate formed of the date of the illness. Sometimes the history is entirely vague; sometimes two dates are given, the first the date when the patient first felt ill, but not ill enough to require him to give up work, the second the date of his keeping to the house, or taking to his bed. Where it is quite impossible to say positively which of these days really represent the commencement of the illness, I have assumed the later to be the true commencement; if I am wrong in any given case the error will be that of assuming that the bath was given earlier in the disease than it really was. Where there has been a definite relapse of a case under observation, and the patient has been treated with cold in both fever and relapses, I have considered the relapse as a distinct case; and so out of 100 patients there are

109 cases, of which in 99 there are fairly good data for fixing the date of the commencement of the illness, and so the earliest day of the fever at which the bath was administered; though among these there are six in which it is probable that a relapse of uncertain date was being dealt with.

In 64 of the 99, about two-thirds, the treatment was commenced on the fifth to the fifteenth day; the mortality of these was 13 or 20·3 per cent. Thus, the percentage of mortality was less for these than for the whole number.

The relation between early bathing and mortality is well shown by a further examination of the figures, as follows:

Thus, of 30 cases in which the treatment was begun on or before the seventh day, 5 died, giving a mortality of 16·6 per cent.; whereas, of 69 begun after the seventh day, 20 died, or 28·9 per cent.

Cases.		Day.	Died.	Percentage.
30	began on or before	7th	5	16·6
69	,, after	7th	20	28·9
Again—				
49	,, on or before	10th	10	20·4
50	,, after	10th	15	30

This shows that the proportion of fatal cases was much greater amongst those the treatment of which was begun late in the disease.

Another way in which this question may be approached is by taking only the fatal cases and noticing the period at which bathing was commenced in each case. We find—

2	on or before the	4th	day.
2	,, ,,	5th	,,
1	,, ,,	7th	,,
3	,, ,,	8th	,,
2	,, ,,	9th	,,
2	,, ,,	12th	,,
1	,, ,,	14th	,,
2	,, ,,	15th	,,
2	,, ,,	16th	,,
1	,, ,,	17th	,,
1	,, ,,	18th	,,
2	,, ,,	19th	,,
4 (each 1)	20th, 22nd, 23rd, 28th		day.
25	(2 doubtful)		

This gives 5 out of 25, or only one-fifth, begun on or before the seventh day; 8, or one-third, on or before the eighth day.

With regard to frequency of application I have already said that in my cases the applications were so few as to almost invalidate the case for the purposes of this evening's inquiry. Thus, simply to take the figure 10 as an arbitrary number, I find that only nine cases had more than ten baths, of which two were fatal. Only ten cases had more than ten spongings, of which three were fatal.

But counting all the cold applications, whether bath, sponging, or ice pack in any given case I find that thirty cases had more than ten cold applications—and of these seven were fatal—a percentage of 23·3, greater than that of the total cases, but still less than the mortality of the cooled cases generally.

Whether baths or sponging give better results is perhaps a question that cannot be decided from such figures as one has here. As a general rule it has been found that the temperature has fallen lower after a bath than after a sponging, but I have little doubt that with sufficient perseverance and with water cold enough a sponging could be made as effectual. Another question is, whether any injury has been done by bathing, in the transference of the patient from the bed to the bath, in the struggling that some semi-delirious or simply nervous patients indulge in, or in the strain which, with insufficient bathing apparatus, some patients put themselves to in order to keep their heads above water or to prevent themselves sinking.

To compare results one must, of course, take the cases in which one or other method has been alone tried.

Of baths alone I find 45 cases, with 9 fatal cases, a mortality of 20.

Of spongings alone 28 cases, of which 6 were fatal, a mortality of 21·4.

It must be remembered here that, under the varying conditions of treatment at a hospital under several physicians, with occasional assistance on the part of house physicians, the choice of the remedy may be determined differently at different times, and it is on the whole probable that very bad cases would be simply sponged, and the bath would fall in a larger proportion to those that were just not so bad. On the other hand, it has not unfrequently happened that sponging has been considered sufficient for a moderately high temperature, and the bath brought in only when, so to say, the sponging did not seem to be doing much good.

The relation of frequency of baths to mortality is rather strikingly shown in the list of cases treated by baths alone.

For instance, amongst these:

34 cases had only 1 bath, 4 were fatal.
4 ,, 2 ,, 1 ,,
7 ,, 3 ,, 0 ,
6 ,, 4 ,, 3 ,,
5 ,, 5 ,, 1 ,,

11 cases had more than 5 baths, no deaths.
3 ,, 6 ,,
1 ,, 7 ,,
2 ,, 11 ,,
1 ,, 14 ,,
2 ,, 15 ,,
1 ,, 31 ,,
1 ,, 37 ,,

34 cases had 5 baths or *less*, 9 died = 26 per cent.

11 cases had more than 5 baths, no deaths.

Any analysis of the cases treated by ice packs, ice bags, or Leiter's coils would be of little value, as more than half in either case had some other method of cooling applied also.

Half of the ice-pack cases were fatal, and of 6 cases ice-packed only 3 died. It is here right to say that the fatal cases were mostly severe before treatment, and the treatment was mostly begun late.

If, finally, we take both these points into consideration together, namely, early application and frequent application, we shall find that a very small number of cases are available. For instance, taking as our standard commencement of treatment on or before the seventh day, and number of applications of cold not less than ten, we find only 10 cases of which 2 were fatal; taking the commencement of treatment on or before the tenth day, and the same number of applications, we have 19 cases with 4 fatal. These give percentage mortalities of 20 and 21 respectively, still much higher than the percentage mortality of the total number of typhoid cases.

Of the influence of a cooling method of treatment on the complication of typhoid fever I can speak only in very general terms. I should say, from the reports of cases at Guy's Hospital, that this influence is not very apparent; but I must allow that I have not investigated the subject with all the care I should have liked to give to it, and am unable to support my opinion by figures, and references to individual cases. Some complications are, of course,

represented in the mortality statistics, and among them the most important of all, perforation. The other complications which one might expect would be especially affected by a cooling method of treatment are hæmorrhage, bronchitis, and albuminuria. As a kind of complication we may also consider the occurrence of relapses.

With regard to hæmorrhage, I find that it happened to 12 patients out of the 100 treated by cooling measures. To one patient it occurred in the relapse as well as in the original attack; and thus, whether patients or illnesses be considered, it may be said that hæmorrhage occurred in 12 per cent. I have not the facts with regard to the unbathed cases, but, comparing these results with the few figures given in Murchison's work, hæmorrhages appear to have been more frequent. According to Murchison Louis had 8 cases of hæmorrhage in 134 cases of typhoid fever, and Murchison himself counts 58 cases in 1564, or 3·77 per cent., but hæmorrhage of not less than six ounces in quantity is not included here, so that the total percentage of his hæmorrhagic cases must have been much higher. But before condemning the bath treatment for what difference there appears to be to its discredit, let us inquire into the circumstances of the individual cases, and we shall see that in some, at least, there are grounds for regarding the hæmorrhage as likely to be independent of the bath. In one case certainly, for it appeared before anything was done; in another, it only occurred five days after baths and other cooling measures had been stopped; in another, four days after the single ice-pack employed. In another there had been sponging for two days, which only reduced the temperature by 1° Fahr.; and in another case the fever was extremely high, and the case was very severe before any treatment was adopted. Of the 13 cases in which hæmorrhage occurred, 5 recovered, 2 died from perforation, 1 died mainly from exhaustion (the hæmorrhage occurring only once, some days before death), and 5 may be regarded as having died chiefly from this accident. So far as my own practice is concerned, I have always looked upon this occurrence as a call for the baths to be stopped; but the bath has occasionally been continued for six or twelve hours longer in the absence of instructions.

In a few of these cases, cold spongings have been continued throughout, regardless of the existence of hæmorrhage.

As to *bronchitis*, I can see no sure evidence that this complication has been more frequent or more severe in consequence of the bath.

But here again I am speaking rather from my general impressions after reading the reports than from special notes made as to the condition. But in any case one would have to draw inferences with very great care; bronchitis is certainly one of the most frequent complications of typhoid fever, and as it frequently exists when patients are admitted, and may get worse even on expectant treatment, it ought not to surprise us if it got worse under a bath treatment, or even first appeared under such circumstances. As to its frequency in typhoid fever, Dr. Murchison says that he once noted it twenty-one times in 100 cases, but my own experience would certainly be that it was much more frequent, and, indeed, in the 109 cases here alluded to, bronchitis was noted as present in different degrees *before* the use of any cooling measures, in 64 cases. I can only say I have not seen any such severe increase as to regard it as necessarily the result of the baths; on the other hand, I have seen much improvement in a few days after the use of the bath. The above remarks refer to the signs of bronchitis not localised to the bases behind, but I have also seen the evidences of hypostatic congestion rapidly clear up under the same conditions.

Of albuminuria, I can give very little information. In the cases here analysed it occurred but rarely, and I did not especially note it.

As to relapses they appear to me to have been no more frequent in the cooled cases than in the uncooled. Here, again, I cannot compare the figures in the two classes occurring at Guy's Hospital, but can only give the results in the cooled cases and leave them to be compared with standards derived from other sources. Thus in these 100 patients there were 10 cases followed by relapse. No doubt some of these cases were themselves relapses. I scarcely know how to deal more accurately or certainly with such cases than to consider them with the others.

Of the 100 cases I find the date of admission is doubtful. Eighteen are represented as having come in after the fifteenth day, and the majority of these no doubt stated that they had been ill three weeks, eleven cases being attributed to the twenty-first and twenty-second days. Whether some of these were not relapse cases must remain uncertain. Two of them, however, contributed definite relapses which occurred afterwards in the hospital. From this I am inclined to regard the cooling treatment as not chargeable with the production of relapses.*

* From Murchison's work it appears that in the London Fever Hospital from

As to minor complications, thrombosis of the femoral veins, abscesses, &c., I find a very small number indeed in these bathed cases—so insignificant that the cases occurring at Guy's would not support any charge of the kind against the cooling treatment.

Now I come to what is really a minor point, that is, the immediate influence on the patient's condition and comfort. I suppose everyone who has seen cases bathed will allow the great improvement that is manifest in the symptoms present and that in the great majority of instances. For instance, the lessening of delirium, the cleaning and moistening of the tongue, the increased tendency to sleep, and the general feeling of greater comfort experienced by those who are still sensible. Many patients have expressed a desire for the bath to be repeated, and I remember one girl who again and again asked to be placed in the bath.

As a rule the first bath is disliked by the patient, and it seems to me that the amount of collapse, chilling of the extremities and shivering, are much more marked in the first two or three baths than with subsequent ones. The circulation and the nervous system seem to adapt themselves to the change of temperature much more readily after a little experience.

Now that I have brought forward the main facts about the use of the cooling treatment at this hospital as clearly as I could, without going into unnecessary and wearisome detail, it is my duty to state what I believe are the conclusions to be drawn from them.

I must again repeat my opinion that the material is by no means entirely satisfactory, because no combined effort has been made to test in a crucial way the treatment under consideration; and the conclusions, therefore, must often be rather reserved, or negative, in character.

It must, further, be remembered that they are drawn from cases the majority of which came under treatment late; and in the majority of which the cooling treatment was not vigorously pushed. I think it would be right to say:

1. That mortality has not been obviously decreased by the use of cooling measures.

2. That the larger percentage of deaths amongst cooled cases

1862 to 1868 the relapses were only 80 in 2591 cases, or 3·08 per cent.; but Griesinger had 6 per cent. in 463 cases at Zürich; Human, 8 per cent. in 548 cases at Leipzig; and Maclagan, 10 per cent. in 128 cases at Dundee.

may be, in part at least, explained by the greater original severity of the cases so treated.

3. That the deaths from hæmorrhage have been proportionately more frequent.

4. That deaths from other causes have not been strikingly different in cooled and uncooled cases.

5. That complications other than hæmorrhage have not been increased.

6. That relapses have not been more frequent.

7. That in proportion as the patients came early under treatment, and were thoroughly treated, the results were better.

8. That patients are mostly much improved for the time in comfort and general condition by the bath.

9. That the bath is the most efficient means for the reduction of temperature.

I may, perhaps, be permitted a few general remarks apart from the cases occurring at Guy's Hospital.

I must say that the results I have put before the Society have somewhat surprised me; and, perhaps, I have laid undue stress upon the less favorable aspect of the case. I should be glad to take the more favorable view, and, as I have shown, when one's consideration is limited to cases treated early, and to those in which several applications of cold have been made, the result is much more satisfactory. Still, the cases then would be but few in number, and though I have attended to them I think it is not right to disregard the other cases, because it may still be a question how far even a single bath with a great reduction of temperature may do harm.

By some the mechanical disturbance involved in the transference of the patient from bed to bath and back again is regarded with apprehension, and charged with the risk of causing perforation. This, no doubt, may be entirely obviated by the use of a proper apparatus, such as Dr. Coupland has shown in use at the Middlesex Hospital. This is not generally available in private, and the patient will often use much muscular exertion from dislike of the cold and from fear of what is going to happen, or to prevent himself from sinking, or from getting his head or face under the water. No doubt some expenditure of power, too valuable to waste, may thus be incurred, but I doubt if much injury is likely to result in the abdomen, as, I take it, perforation occurs rather from movements of gas in the intestine, or from contractions of the intestinal

muscular coats, than from the uniform contraction, which is really protective, of the muscular abdominal walls.

As regards the selection of a temperature, I have nearly always had the temperature taken every three hours, and when the patient has reached 103° the bath has been administered. Temperatures of 102·5° and 102° have been taken as standards by some. This is, of course, entirely arbitrary at present; and the advantage of one or the other can be, I suppose, only determined by the observation and comparison of many cases. Some patients no doubt bear—or seem to be less affected by—a higher temperature than others, and one is naturally inclined to let those patients suffer it, with more complacency than those who are much upset by comparatively slight fever. This, I may say, of itself shows that there is another factor besides the temperature which we do not touch with the bath, or, at least, only indirectly.

The method of treatment by employing a cold bath only when the temperature reaches 105°, is the very mildest form of the kind that can be used, and practically would leave a number of dangerous cases untreated. I believe that the risk of death by a single sudden elevation of temperature, such as occurs in acute rheumatism, is infinitesimally small. I have once seen the temperature rise to 108°, but the patient was already moribund, after high fever and hæmorrhage, and it appeared quite obvious that no reduction of temperature would save his life; and it was not attempted.

Then it occurs to me to ask whether it is necessary to bathe a case that comes in quite late in the illness, at a time when the pyrexia has taken on the intermittent type. The theory of the bath is that, since continuous high temperature damages the viscera, and tends to accidents hereby, the average temperature should be kept down by occasional interposition of cold application.

Now, in ordinary cases, by the fifteenth or sixteenth day, the temperature falls every morning and rises every evening only. The average temperature, therefore, is already comparatively low, and the patient in such a condition is on the road to recovery. One does, however, now and then see such a case continue with an intermitting temperature for some time, and perhaps, on the whole, assuming the value of the treatment, there can be no harm in submitting cases in this stage as well as those in an earlier stage to the treatment.

Lastly, with regard to complications, though I am unable to record

any special increase or occurrence of them in the cases at Guy's Hospital, an interesting case came under my care at the Evelina Hospital, in which an unusual complication did follow the employment of the bath.

In 1877, a boy, nearly ten years of age, was admitted under my care on the sixth day of typhoid fever, he was rather severely ill with a temperature of 104·2°, pulse 120, enlarged spleen, slight cough, no spots. For the first eleven days the bath was given at 104°, and subsequently at 103°. During these eleven days he had thirteen baths, and in the next six days he had twenty-three baths; this brought him up to the twenty-fourth day of his illness, and he died on the twenty-fifth. He went on without complication, but had much pyrexia, great weakness, and rapid pulse.

On the evening of the twenty-third day, he had a black motion; a bath was given the following morning, and then there was some oozing of blood from the gums, close to the teeth, which was only stopped by the application of tincture of perchloride of iron. During the day he lay on his side, but was blanched; not drowsy; abdomen moderately full.

The baths were stopped. Early the next morning his temperature was 104·6°, his breathing was very rapid, and became increasingly so, and short like that in pneumonia. The bowels had acted three times, the motions being loose and black. He sank and died at 8 a.m.

Post-mortem.—The upper lobe of the right lung was quite consolidated, with the exception of a narrow portion along the lower edge. The consolidation was quite uniform in character, and apparently due to effusion of blood into the structure. The pleural surface was dark from the subjacent blood. A similar patch of smaller size was seen at the base.

The heart was normal. There was much ulceration of the ileum, and many ulcers were healing or healed. The mesenteric glands were enlarged, one full of yellow creamy pus. There was no peritonitis.

The explanation of this case is no doubt that it is a hæmorrhagic infarction due to embolism of branches of the pulmonary artery. The occurrence of these is comparatively rare in typhoid fever. Hoffmann (Liebermeister in 'Ziemssen's Encyclopedia') observed them fifteen times in 250 cases, and Murchison also speaks of infarctions as occurring in typhoid fever. Such an event can be

scarcely referred to the prejudicial effect of the bath, being due, indeed, to a local diminution of blood pressure, and not to a general increase within the interior of the body.

REMARKS UPON THE TREATMENT OF TYPHOID FEVER BY THE COLD BATH.

By Samuel West, M.D.

The points to which I desire to direct attention are chiefly two:—first, I wish to speak briefly of the doctrine of fever, upon which this method of treatment is based; and secondly, to refer to the statistics which have been published, and to consider the value of the conclusions which have been drawn from them.

Liebermeister commences his pamphlet* upon this subject with the following paragraph: "The longer I am engaged in the careful study of fever the more confirmed I become in my conviction that the manifold phenomena, the sum of which constitutes the complex of symptoms of fever, are in chief part only the results of one of these phenomena, viz. the increase of body temperature. It is the increase of body temperature which, according to my conception, is not only the pathognomonic symptom of fever, but also the proximate and sufficient cause of most of the other symptoms peculiar to fever, or characteristic of it; and, finally, it is also the increase of body temperature which in the highest degree produces the danger of fever." In other words, the temperature is practically the cause of the symptoms of fever, and of the dangers which arise in it. That I am not straining Leibermeister's meaning is quite clear from numerous other passages in the same pamphlet.

It would be quite beyond the field of this discussion to criticise this statement with reference to fever generally, but we may with advantage consider its applicability to typhoid fever only.

Clinical observation is, I think, quite opposed to the acceptance of this proposition, as it stands, in the case of typhoid fever. It is certainly not our experience in this country that the dangers of typhoid fever vary in direct proportion to the height of the tempe-

* Liebermeister und Hagenbach, 'Anwendung des kalten Wassers bei fieberhaften Krankheiten.' Leipzig, 1868, p. 83.

rature. Numerous cases with high temperature recover, and many with low temperature die. Diarrhœa, exhaustion, hæmorrhage, perforation, and peritonitis, each has its own victims, and produces its own dangers quite irrespective of the temperature. And although it has been asserted that a high temperature increases the risks of these complications, and it may be so, still the assertion has to be proved, and must not be assumed.

Liebermeister's statement, therefore, as it stands is incorrect, without so many reservations and conditions that it becomes practically of little value when applied to any individual case. I need hardly refer to other diseases, such as phthisis, or suppurative fever, in which the temperature is high for long periods together, and in which no symptoms arise which call for the same active treatment as is demanded by the advocates of cold bathing, with similar temperatures, in typhoid fever.

I would not, of course, deny that high temperature may in itself be a source of danger, but such cases belong to the general group of hyperpyrexiæ, a state the pathology of which we do not comprehend, and which may arise under so many divers conditions, that it is difficult to find a factor common to them all. Even supposing that it were proved, and this it is not, that a temperature of 103° or 104° were in itself a danger to life, and that by cold bathing the temperature could be permanently reduced to its normal height, knowing so little as we do of the exact way in which a cold bath acts upon the body, we should not be justified in assuming without further proof that the improvement was due simply to reduction of temperature.

The facts known about fever seem to indicate that we have in fever a complex of co-ordinate symptoms, each of which may vary within wide limits—and this is true even of that symptom of which we are specially speaking now, viz. temperature—and that each and every one of these symptoms is due probably to some common cause as yet undetermined.

We must give account, to obtain any satisfactory explanation of fever, of the increased heart's action, of the sensorial and gastric disturbances, of the chills, of the increase of chemical metabolism, and of the diminution of secretions, as well as of the rise in temperature, of which each of the other phenomena is, it appears, essentially independent. This, however, is not the time for a dissertation upon the general question of fever. I have said this much,

only to show that the theory, which appears to be often assumed by the chief advocates of this method of treatment, is one which is in all probability insufficient, and that, therefore, the treatment based upon this theory will be probably also found insufficient.

But it might be urged, if the results of the treatment are good, they become an argument in favour of the theory upon which the treatment is established. This is a specious, but very dangerous argument to use in medicine, and it is hardly necessary to point out the many fallacies which it embraces. As, however, the results to which reference is made are obtained from statistics, I am brought naturally to the consideration of the second point of my paper, viz. the applicability of statistics to the solution of this problem.

The fallacies to which all statistical conclusions are exposed, are chiefly three.

1. Inaccurate observation and grouping of facts.
2. Insufficient number of facts.
3. Insufficient elimination of, or allowance for, disturbing causes.

In the particular case with which we have to deal, we may assume the correctness of the observations, and turn to the consideration of the last two sources of error—insufficient data, and disturbing causes.

The problem to be dealt with is one of the highest complexity. If it were possible to get two sets of observations, in all respects similar, except in regard to the treatment by cold bathing, the one having this method of treatment and the other not, the comparison would be just and the conclusions final, but these conditions are clearly impossible of fulfilment.

The unreliability of comparative statistics in typhoid fever is, I think, generally recognised, and the disturbing causes are so great that it is impossible to make sufficient allowance for them. It is not possible, except in the most general way, to compare the statistics of one epidemic with those of another, or, indeed, of the same epidemic in different countries, or even in different parts of the same country, still less of different epidemics in different places. This is true, even of the same epidemic in different parts of the same town. I have drawn out upon the following table the mortality statistics of the last twenty-three years at St. Bartholomew's Hospital. The figures demonstrate very clearly the points to which I am referring :

TABLE I.—*Statistics of Typhoid Fever at St. Bartholomew's Hospital.*

Year.	No. of cases.	Deaths.	Percentage.
1860	29	2	7·6
1861	49	10	20·4
1862	63	5	7·94
1863	41	3	7·32
1864	37	7	18·9
1865	55	9	16·36
1866	57	9	15·8
1867	61	14	20·3
1868	61	15	24·36
1869	47	14	30·0
1870	61	9	14·75
1871	72	10	13·9
1872	53	6	11·7
1873	80	13	16·25
1874	87	11	12·64
1875	46	3	6·52
1876	59	11	18·64
1877	77	13	16·9
1878	167	20	12·0
1879	103	19	18·45
1880	91	21	23·08
1881	126	32	25·4
1882	146	16	10·96
Total . .	1668	272	16·96

The total number of cases is 1668 with 272 deaths, giving a percentage of mortality of 16·96, which agrees closely with that given by Murchison. When we compare the mortality year by year, it is surprising to find how great the variations are, from 6·52 per cent. up to 30 per cent. In fact, there are hardly any two, and certainly no three, consecutive years in which the percentage mortality is even approximately the same. Even in consecutive periods of five years the mortality differs widely, 14·6, 13·88, 19·8, 12·33; and the difference over periods of ten years, though of course less than for short periods, is still considerable, 17·6, 14·28 (Table II). The statistical tables of the Middlesex Hospital, brought forward by Dr. Coupland; those of Guy's, by Dr. Frederick Taylor; and those of the London, by Dr. Gilbart Smith, show similar wide variations, and demonstrate the extreme need for caution in the comparison of typhoid fever statistics.

TABLE II.—*Percentages over Periods of Five Years.*

Period.	No. of cases.	Deaths.	Percentage.
1860–4	219	27	12·33
1865–9	281	61	21·7
1870–4	353	49	13·9
1875–9	452	66	14·6
1881–3	363	69	19·01
Percentages over Periods of Ten Years.			
1860–9	500	88	17·6
1870–9	805	115	14·28

If any results could be comparable with one another, it might be thought to be those of the different physicians in the same hospital, during the same epidemic. These statistics are shown upon Table III for the last three years at St. Bartholomew's Hospital.

Of the four physicians—whom I may designate by the letters A, B, C, and D—one, viz. A, practised the cold-bath treatment with activity; the other three, B, C, D, used, if I may so call it, the modified cold-water treatment, endeavouring to reduce the temperature by cold-sponging, wet-packs, light clothing, drugs, &c., and using only the cold or tepid bath when the temperature was persistently very high. I thought, by comparing the results of the one with those of the other three, I might discover some facts of interest. The results are shown upon the Table III, in which are put out the number of cases, with the number of deaths in each year, and the percentage reckoned.

We find that A had under his care 110 cases, with 18 deaths, giving a percentage of 16·36; but the percentage in different years varies very widely, and in 1882 is more than double that of either of the other years.

Taking the practices of B, C, and D together, we find that there were 255 cases under treatment, with 45 deaths, giving a percentage of 17·65, which is not very different from the percentage of A; but when we analyse the figures of the B, C, and D group, and compare them with one another, we find the same great variations and irregularity, to which I have already referred, as occurring in every statistical table published upon the subject.

TABLE III.—*Statistics of the four Physicians during the years:*

A.

Sex.	1881.		1882.		1883.		Grand Totals.	
	Total number.	Deaths.	Total number.	Deaths.	Total number.	Deaths.	Total number.	Deaths.
Male . . .	22	7	25	4	26	3	73	14
Female . . .	12	2	11	0	14	2	37	4
Total . .	34	9	36	4	40	5	110	18
Percentage . .	11·1		26·5		12·5		16·36	

B.

Male . . .	17	3	24	2	16	4	57	9
Female . . .	8	0	16	1	12	2	36	3
Total . .	25	3	40	3	28	6	93	12
Percentage . .	12·0		7·5		21·48		12·9	

C.

Male . . .	13	8	27	2	16	5	56	15
Female . . .	12	4	7	1	10	1	29	6
Total . .	25	12	34	3	26	6	85	21
Percentage . .	48·0		8·82		23·1		24·7	

D.

Male . . .	13	2	11	2	9	1	33	5
Female . . .	13	3	21	3	10	1	44	7
Total . .	26	5	32	5	19	2	77	12
Percentage . .	19·2		15·62		10·53		15·6	

These tables thus only accentuate one of the chief difficulties which we have to meet in dealing with medical statistics, and show, I think, conclusively the complexity of the disturbing causes for which, if logical conclusions are to be drawn, we must make adequate allowance.

Such statistics as these latter are, of course, open to the objection, that the numbers are small; but they are not smaller than some which have been published, and which are supposed to justify certain conclusions.

But all who are familiar with the working of a hospital know how largely such results as these may vary, in consequence of accidental causes, quite irrespective of treatment, depending, as it is often said, upon good or bad luck—one ward having a run of severe and another of light cases. How greatly chance enters into such results, is illustrated by an authentic tradition of St. Bartholomew's Hospital, upon the authority of the late Dr. Jeaffreson, who, for the first five years after he entered upon charge of the wards in the hospital, never had a single death from fever, and that when there was no isolation fever-ward in the hospital, but all cases of fever alike were admitted into the general ward indiscriminately. A similar experience befell also the late Dr. Black, who, for a period of eighteen months, never lost a single fever case. Such facts as these are very extraordinary, though, of course, quite accidental, but their bearing upon the present subject of discussion is clear.

The same evidence of disturbing causes is seen when the results of the various advocates of the cold bath are compared with one another, the percentage varying from 3·1 to 13·2.

Now, on referring to the general table which Dr. Coupland quoted, giving the results of foreign observation, two strange facts strike me. First, the high rate of mortality taken as the average upon treatment other than cold-bathing; and secondly, the extremely low mortality with that treatment. Such a wide divergence is, I venture to think, suspicious. In the first place, the average mortality assumed is excessive; a percentage of 30, 27, 26, 24 represents a mortality which is most unusual, and can be explained only on the assumption that either the numbers taken are too small, or that other diseases of a severe kind are included, for it is far in excess of our average in this country, which is about 16 to 18. Out of the twenty-three years at the London Fever Hospital (1848

—70), from which Murchison draws his statistics, on only four occasions did the percentage exceed twenty, viz. 20·55, 26·97, 27·83, and 28·4. The possibility of error is openly acknowledged even by Liebermeister in the early statistics which he gives, where the mortality is stated to be 27·3.

Another striking fact, viz. the lowness of the percentage on cold-bath treatment, suggests the opposite possibility, viz. the inclusion among typhoid fever cases of a large number of mild or even doubtful cases, many of which we classify in this country under an entirely different heading, such as ephemera febricula, &c., and which would seem to correspond with Jürgensen's "typhus levissimus." With reference to these light or mild cases, Liebermeister himself puts the percentage mortality of cases not treated antiseptically, in which the temperature did not rise above 104°, at the remarkably low figure of 9·6. That the question of diagnosis assumes an important position in this consideration is also acknowledged in a very careful paper by Dr. Goltdammer, of Berlin, whose conclusions recommend themselves the more strongly because the subject is discussed in a more critical and less enthusiastic manner. This paper is a very important one: first, on account of the large number of cases with which it deals; and secondly, on account of the caution which the author displays. The percentage given of the mortality of cases not treated by cold bathing is 18·1, a number which closely approximates to our usual average in this country. By cold bathing the mortality was reduced by 5 per cent., *i.e.* to 13·2. But in criticising his own results, he makes several remarks which may throw light upon some of the other foreign statistics. He explains the comparative highness of his percentage upon the cold-water treatment, by stating that he has included all cases indiscriminately, and has not excluded those which came to the hospital in a hopeless state—this, I need not say, is a very suggestive remark. He adds further that, so far as he is aware, his results are not surpassed by those of any other hospital in Berlin. But even the difference of 5 per cent. in the mortality is small, and, as I have shown, comes well within the range of periodical variations. Dr. Cayley claims even a smaller percentage improvement than this, about 3 per cent.

I may refer in illustration to the statistical tables of heart disease as a consequence of rheumatic fever given by various authors. One would have thought that, if any disease did admit of tolerably

accurate diagnosis and classification, that disease would be rheumatic fever and its cardiac sequelæ, and yet the percentages differ so extraordinarily as to defy explanation. They range from so low a figure as 3 per cent. up to 50, 60, or even higher.

The only possible way of eliminating the fallacy of disturbing causes in statistics is by dealing with very large numbers, and even when the numbers are large the difficulties in excluding cases which do not really belong to the class become almost insuperable. Indeed, I do not see how it is possible in the case of typhoid fever to obtain sufficient data of the right kind. For these reasons I venture to think that the statistical method is not applicable to the solution of the problem we have before us.

By what means can this question then be solved? Believing, as I do, that it must be answered by individual experience, and not by statistics, I have thought I could best contribute to the discussion by eliciting the opinion of the physicians at St. Bartholomew's Hospital, who responded most readily to my request and have given me their opinions in writing to communicate to this Society.

Dr. Andrew writes thus: "I should not use the bath in any ordinary case of typhoid fever in which the temperature did not rise above 105°. It is impossible for me, in reasonable compass, to lay down the considerations which guide me in such cases. The duration of the hyperpyrexia, as well as its height, the period of the fever, the special tendency to death in the particular patient, his age, and the characters of the epidemic at the time, would have to be all taken into account. There are also gentle means of reducing the temperature in fever, the early employment of which may prevent the necessity for the bath arising, *e.g.* attention to the temperature of the sick room, diminishing the amount of bedclothes, and especially by the use of cradles preventing the bedclothes being in actual contact with the body. This last is a most efficient means of reducing temperature; and from observation of its effects I am convinced that in fever, unless its natural crises be close at hand, it is often injurious to reduce your patient's temperature much below 102°. For the bath, even when necessary, is not without dangers of its own. Pulmonary symptoms not infrequently develop, or become more serious after its use. In short, I look upon the bath as necessary in hyperpyrexia, needless and often harmful in ordinary cases."

Dr. Church's opinion I will quote from an article published by

him in the 'St. Bartholomew's Hospital Reports' for 1881:—"Although I have made pretty frequent use of the cold bath, I do not feel sure of the propriety of using it as a routine instrument for typhoid, after the manner of Dr. Brand, of Stettin, Prof. Liebermeister, and others, as recommended by Dr. Cayley in his 'Croonian Lectures.' The difficulty which applies to all statistics, of estimating the probability of the circumstances under which they were compiled being identical with those with which you yourself are dealing, applies with increased weight to the statistics of foreign observers, and I must confess that foreign statistics have but little weight with me. Dr. Cayley's own statistics are, however, very satisfactory. He claims to have reduced the mortality of the severe cases to the level of the average mortality of the disease of the London Fever Hospital. The mortality at St. Bartholomew's very closely corresponds with that of the London Fever Hospital, and has not become less during the few years in which cold baths have been more or less in use there. Dr. Alexander Collie, at the Homerton Fever Hospital, came to the conclusion that cold bathing had no effect on the mortality there; but the number of cases in which he used the bath is not sufficiently large for any reliable deduction to be drawn from them. Another point which renders it necessary to be careful in forming any opinion from a comparison of statistics taken from a limited period, is the remarkable fluctuation in the rate of mortality from typhoid in different years.

"To give every patient a cold bath as often as the temperature rises to 102°, appears to me a most unnecessary amount of fatigue, both to the patient and his attendants. I have no statistics on the subject, but I should say the mortality from typhoid in cases where the temperature does not reach above 102° is extremely small, and if to them he added all the cases of pyrexia, which may be classed as Professor Jürgensen's 'typhus levissimus,' I can conceive that very favorable statistics might be obtained for any form of treatment not directly hurtful.

"During the last four years, *i.e.* up to 1881, I have used the cold bath on adults whenever the temperature has remained for twenty-four hours or so persistently at or about 104°, and have repeated the bath three times in the twenty-four hours, but not oftener. The utility of cold bathing in cases of extremely high temperature is well illustrated by Case 1 (quoted in the paper).

On the other hand, little or no benefit was obtained from its use in Case 8. Cold sponging, which is almost invariably grateful to the feelings of the patient, is constantly used, though it appears to me to have but a transient effect on the temperature. All my patients are kept very lightly covered with bedclothes."

This opinion, stated in 1881, the further experience of the last three years has only confirmed.

Dr. Gee writes thus: "Thinking over the promise I made in answer to your request, I find that I have very little to say, and that the value of that little is still less. My febrifuge treatment is no more than to let the patients lie as lightly clad as possible. Sometimes, but seldom, if the fever rise very high, they are sponged with lukewarm water once a day, or oftener if need be.

"I have followed the question of cold bathing in typhoid fever ever since its first introduction in Germany, and I have been waiting for better proof than I have yet found, that such treatment is beneficial, before I cared to subject my patients to what seemed to me to be a severe method of cure."

Dr. Southey states his opinions thus:—"Cold baths answer well as 'stirs,' are admirably suited to cases of enteric, which are early overwhelmed with typhus-like symptoms, and present a very ataxic condition, cases with much subsultus, delirium, and passive lung congestion. I dipped several patients into cold water, temperature of ward 55°—65°, but only in and out again twice or three times in the twenty-four hours. I continued this in some cases for two or three days. It loses effect, this cold dipping, after too frequent repetition, but is capital in procuring quiet sleep, when resorted to in suitable cases. Employed after the third week it was found too often succeeded by hæmorrhage from the bowels, to doubt its ill effects in this direction.

"Any bathing is good in the first two weeks, cold or tepid, but the baths are after a short time succeeded by an elevation of temperature, unless protracted so as to require warmth and stimulants. Indeed, when I used cold baths, longer than mere dips, I doubted their being so efficacious in lowering temperature as tepid baths taken at 95°, and protracted for fifteen to twenty-five minutes, or until the patient shivered. The tepid baths were not in a single case that I had followed by hæmorrhage, so far as I can recollect."

It is noteworthy that with so considerable an experience of the

cold-water treatment, Dr. Southey's opinion should still be so cautious and so far from enthusiastic.

Dr. Duckworth has sent me the following communication, in which his opinion is stated:—"I have had experience of the cold-bath treatment of typhoid fever. For some time cases with hyperpyrexia were thus frequently treated by several of my colleagues, and by myself, at St. Bartholomew's. I think I may say that this practice has now become almost obsolete with us. Not that, as a therapeutic measure, it is absolutely discarded, but it is held in reserve for cases which appear to require it.

"Had I been present at the recent discussions, I should like to have remarked that in the treatment of any disease I regard it as always essential to make a distinction between treating the malady and treating the patient, and I wish this all important point were oftener borne in mind than it is. Next, I make a clear distinction between what I may call ordinary cases of well-marked typhoid fever and those in which hyperpyrexia occurs. I consider that a temperature of 105° indicates the beginning of hyperpyrexia. Below that I do not consider a case hyperpyretic, or as exciting any undue anxiety as to the ultimate issue of the disease. I am perfectly satisfied that it is quite unnecessary to employ the cold bath for cases which are not hyperpyretic. I never have any trouble with such cases, and find that the upward pyretic tendency can commonly be checked by what we term the air-bath—that is, by a cradle placed over the limbs, the bed-clothes being raised, and a current of air allowed to pass over the patient's body. This is very effectual in moderating undue thermic tendency. If this fails, I order cold sponging to the limbs, with or without the addition of a bag of ice to the head. Certainly, the great majority of cases, in my experience, need no more in this direction. If, however, the temperature shows persistent disposition to rise in spite of these measures, I employ five or ten-grain doses of quinine, or instead, a few doses of salicylate of soda, always preferring the former, however, and by this means commonly find the pyrexia under control. I am fully satisfied that for the great majority of cases no further measures are required, or, I may add, justifiable. Hyperpyrexia, as I understand it, is not a common complication. When it does occur, I am prepared to use the bath with the ordinary precautions, and as often as may be necessary, and I have no fear of inducing internal congestion or hæmorrhage in such cases. To use it in the other class

of cases, I should consider *nimia diligentia*. The cold-bath treatment is practically available for but few other than hospital cases. The process is a very troublesome and fatiguing one for the ward staff, even with the best modern arrangements for moving the patient. Nothing but its absolute necessity would justify such an addition to the labour and anxiety of the resident and nursing staff.

"I am quite sure that it is often practised when it is unnecessary, but I do not affirm that it is injurious in such cases. I think other frigorific measures short of it suffice for most cases. Cold bathing I regard as a most valuable therapeutic agent in true hyperpyrexia, however induced. I reserve to myself the employment of it whenever I require it in practice, and relegate it to the same position as the lancet, the cupping glass, the use of calomel or alcohol, or, in short, any other potent and well-recognised agent for the treatment of disease."

The experience of St. Bartholomew's Hospital, therefore, is very clearly in favour only of the modified cold-water treatment, *e.g.* of the use of bathing in cases where the temperature is persistently high, as an adjunct to other antipyretic methods; and here I may remark that the use of the cold bath in typhoid fever patients with a low temperature, say 102°, without harm accruing, proves no more than that the risks of constant bathing are not so great as have been anticipated, but does not prove that the chance of life is in any way increased by it.

Although I would speak with diffidence of my own personal experience, still, as I have been for some years much interested in this question, I may be allowed, without presumption, to say, that I agree entirely in the results which experience has arrived at at St. Bartholomew's Hospital, and I confess at once, that I cannot reconcile the enthusiastic statements of the advocates of cold bathing with my own observations and experience.

It appears to me that when the temperature does not rise above 102°, or probably 103°, the cold bath, if it does no harm, does at any rate no good, and is, therefore, I think, unnecessary. Of the cases with higher temperature, there are two classes in which antipyretic treatment is of great service: first, that in which the temperature runs up to an unusual height, say 106° or higher, even though this level be maintained only for a short time; and secondly, that in which the temperature, though never so high at any one

time as in the preceding group, still preserves a constant high level, say of 104°, and in which the usual daily remissions are small or absent. But these are the very cases in which all writers seem to agree, that treatment by cold baths involve too much fatigue and excitement to be at all times applicable, and under these circumstances accessory measures must be adopted, such as cold sponging, wet-packing, light clothing, and antipyretic drugs, remedies which though not in all cases of equal efficacy with cold bathing, are, at any rate, many of them, free from its special disadvantages.

In conclusion, I would restate the points which I have endeavoured to establish in this paper:

1. That the thorough cold-water treatment of typhoid fever is based upon what appears to be a one-sided view of the process of fever.

2. That the question is one which cannot be satisfactorily determined by statistics.

3. That a general opinion has gradually developed, which, whatever statistics may appear to prove, cannot be disregarded, against the indiscriminate use of bathing, and in favour of its use in appropriate cases.

4. That while not denying that in hyperpyretic cases cold bathing is the most valuable of the known methods of treatment, in the less severe cases it stands only on the same level as many other antipyretic remedies; and

5. That the success of the treatment depends chiefly upon the clinical skill which recognises the proper circumstances for its use.

Dr. Mahomed.—The Society has already had the advantage of hearing this evening the statistical results of the treatment of enteric fever by cold bathing at the London Fever Hospital, from my colleague Dr. Cayley, who has done so much to introduce the antipyretic treatment of fever into this country. I shall not, therefore, have to deal with this part of the subject, but as nearly all the cases of enteric fever which have fallen under my care during the last ten years have been more or less treated by cold bathing, I feel that I am able to formulate my opinions very

definitely as to the value of this treatment and also as to its supposed dangers.

In the first place, I may say that I believe that the mortality of the cases of enteric fever that are admitted into hospitals, and receive good nursing and treatment, but without cold bathing or any equivalent, has been, and will continue to be, about 16·0 per cent. Whereas I believe, that in similar cases which are submitted to a routine and thorough system of cold bathing, the mortality may be reduced to 10 per cent. Of course I need not point out that I am now speaking only of hospital cases. There can be little doubt that the mortality of enteric fever, as seen in private practice, is very much less than that observed in hospitals, inasmuch as the mild cases are usually kept at home, while the severe ones are often sent to the hospital. The mortality of this disease in private practice is a point on which information is very much needed, and I believe this information can only be obtained by a carefully guarded system of collective investigation.

When I place the possible hospital mortality at 10 per cent. I believe that I am placing it at the lowest attainable figure. Any series of cases in which the mortality is much below this, I should regard as merely a fortunate and exceptional series and, unless they consisted of upwards of a thousand cases, I should place no dependence upon them. It is notorious how frequently in fevers we may have a fortunate series of cases followed by a train of disasters.

There is another fertile source of fallacy underlying all statistics of enteric fever, namely, the *short* cases, those that used to be, and still are, often styled cases of "febricula." I believe that in considering the effect of cold bathing upon the mortality of this disease it is desirable that the cases should be limited to those whose duration has been three weeks and upwards, or that have proved fatal before this time; for if we admit the short attacks of pyrexia, lasting seven, ten, or fourteen days, we can never be sure that we are dealing with the real disease; moreover, these mild cases are never fatal.

I do not think that Brand's statistics can be compared with those of our hospital cases, for the low mortality attained by him and by the other German physicians whom he quotes, includes all cases, long or short, which they felt justified in calling enteric fever. Moreover, his mortality is practically that of private practice under

the best possible circumstances; for they are chiefly composed of the results of treatment in military hospitals, and the patients have been under observation from the commencement of the disease; I believe that it is quite impossible to obtain such results in any English hospital. The circumstances which chiefly influence the mortality of enteric, or *any other fever*, are the malignancy of the germ, the susceptibility of the soil in which it is sown, and the general environment of the patient; by the latter I mean the purity of the atmosphere in which the patient is treated, the nature of the food, and the skill and experience employed in the management of the case. A certain number of cases of enteric fever are certain to die on account of the type of the disease which they have developed and from the receptivity of the patient; a certain number are also certain to recover, for similar reasons. There still remains a considerable number of cases midway between these whose chance of life or death may be distinctly influenced by treatment, and it is the treatment which these receive that will chiefly determine the variations in the percentage of deaths; I feel fully convinced that by the judicious use of the bath we can save about six lives in every hundred cases of enteric fever.

I do not say that the bath should be our only antipyretic agent, or that its employment should be a matter of slavish routine. Although the rule "bathe whenever the temperature is over 102°," gives us the full advantage of the bath, and does but little harm when it is intelligently carried out, nevertheless I believe that equally good results can be obtained with less annoyance to the patient and less strain upon the nurses. So preferable is it that patients should have a few more baths than necessary, rather than too few, that I feel some hesitation in calling the rule in question; I only do so because I know the difficulty of carrying it out in practice, especially in a private house, and I believe that equally good results can be obtained without adhering to it.

That a cold bath will greatly comfort and refresh a patient suffering from fever no one can deny. That it will often remove delirium, give sleep, moisten the tongue, and reduce the pulse-rate as well as the temperature, must be the experience of all who have used it. If, therefore, in severe conditions it can be seen to produce such good results, it may be concluded that in less grave states it may also be used with much advantage though its results cannot be so clearly traced.

As this discussion is limited to the treatment of the disease by cold bathing, I must refrain from discussing other methods, but I may, perhaps, be allowed to say that I should always prefer to adapt my methods to the requirements of the particular patient under consideration at the time. In some, cold sponging, or the occasional use of Leiter's tubes, or ice-bags in the axillæ, are quite sufficient to obtain the end required. It is true that these methods of applying cold have not the powerful effect upon the nervous system produced by a cold bath, but when such an effect is not called for the use of the bath seems to me unnecessary, it is like using a steam hammer to crack a nut, no doubt it will do it efficiently, but it is unnecessary.

In some severe cases in which the temperature is not controlled by these simpler means I believe the bath is invaluable, and when the bath is not sufficient, its effect may be increased by the use of quinine in large doses, or, if you can insure obtaining it pure and fresh, by kairin; this latter remedy is by far the surest and most powerful antipyretic with which I am acquainted, but unfortunately the preparation is unreliable and its results sometimes disastrous.

It has been said that the use of cold baths in the early stages of the disease is especially important, as they diminish the severity of the local lesions. I am quite inclined to believe that the early use of the bath is of great value; it not only economises strength for the prolonged illness the patient may have to encounter, but it brings him with comparative comfort through what is often the most severe period of the fever. More than this, I believe that, by checking the pyrexia at this period, it assists to form a good *pyrectic habit* for the remainder of the illness; this I conceive to be of the highest importance, for all of us must have observed how much habit has often to do with the course, and especially with the prolongation of the temperature curve; habit tends to regulate the production of heat as it does all other functions of the organism, and it is in the formation of good habits in the course of the fever and in the correction of bad ones, that I esteem the bath as most valuable. As to its supposed effect upon the local lesions I am very sceptical; the degree of pyrexia has but little to do with this it must be within the knowledge of all that the most severe lesions are often associated with comparatively low temperatures, often with exceedingly low ones; I have known patients go through their

illness and terminate by perforation, whose temperature has never been above 101°.

It has been stated that cold bathing produces a variety of evils, especially by inducing congestion of the internal organs. I have no doubt that this may occasionally be so, but when such effects are produced it is by the injudicious and improper use of the bath. I have known patients bathed for twenty minutes in ice-cold water; although some might undergo even this without perceptible injury, I am certain that it would injure many by the profound depression it produces and the prolonged internal congestions to which it must necessarily give rise. The bath should never be lower than 70°, and by preference not lower than 75°; a period of five minutes is an amply sufficient time. If the desired result is not produced by this, it is very likely that more severe measures will do more harm than good. I believe that I have once or twice seen the condition of the lungs which Dr. Bristowe appears inclined to attribute to cold bathing, in cases of enteric fever which have not been bathed. A large amount of collapse of the lung is common in this disease, from the bronchitis which so constantly accompanies it. Plugs of muco-pus are frequently drawn into the smaller tubes and act in the valve-like manner so apt to produce collapse in the lungs of children; collapse occurs more frequently in this disease than in the ordinary bronchitis of the adult, because the patient lying on the back very imperfectly expands the lower and posterior part of the lungs, the exhaustion produced by the fever makes both respiration and coughing feeble and inefficient and allows mucus to be retained in the tubes which would otherwise be expelled, while the feeble circulation induces extreme congestion of the lungs.

I have not found either bronchitis or pneumonia any contra-indication to the use of the bath; patients with these complications often benefit greatly by it. Indeed the bath is often used in ordinary cases of acute pneumonia, and with great advantage, as it may be in other cases of severe pyrexia, including scarlatina, a disease in which, on *à priori* grounds, its evil effect would seem likely to be very great.

In conclusion, I may say that I believe the properly regulated bath may always be used freely and fearlessly in all cases of enteric fever, except for old people, for those having much abdominal distension, any sign of intestinal hæmorrhage, remarkably severe bronchitis, or any cardiac or renal complication. It is our best

weapon wherewith to combat the evil results of high temperature; I should always prefer to have a bath available in the treatment of this disease, but in many cases milder and more convenient measures will enable us to obtain equally good results.

Dr. A. T. Myers.—I have in the first place to thank the President and this Society for the courtesy with which they have allowed a stranger and visitor like myself to make a few remarks to-night, and I must add that I should have thought it most impertinent in me to take advantage of such courtesy if I could not make certain that I should only detain you for a very few minutes, and further, what is more important, if I had not had placed at my disposal a large mass of observations made by your medical secretary, Dr. Isambard Owen, which are of their kind so conspicuous for their care and accuracy, that I can hardly think you would wish them entirely passed by. They form parts of the reports made by him as Medical Registrar to St. George's Hospital during the years 1877, 1878, 1879. I have endeavoured, in looking through them again, to correct the observations on a few points and to add to them the record of the succeeding four years at St. George's Hospital, for the last three of which I must admit that, as Medical Registrar, I am entirely responsible.

I may seem very rash in bringing forward a few statistics, especially after what Dr. West has been saying, but I would for a moment beg to remind Dr. West that the rejection of arguments from statistics is logically the rejection of methods of inductive philosophy; for it amounts to the rejection of the data of observation, and the falling back upon the seductive methods of intuition. Statistics, I admit, lead up historically to most abundant fallacies, but that is due to the defects of the reasoning based on these statistics; it is due, in fact, not to the statistical basis but the logic of the superstructure.

You will remember that the death-rate in Dr. Coupland's series of eighty-nine cases under his own care which was detailed here last week was a little less than 8 per cent. In this summary of seven years at St. George's there are 281 cases and 69 deaths, *i.e.* a death-rate of a little more than 24 per cent. That, at any rate in comparison, is a striking result. The treatment under which it

was obtained may be roughly described to have been one by expectancy and alcohol. No bath or any general application of cold water was used during these seven years, so that it is only by comparison, or perhaps I should say contrast, that the results bear upon the cold bath treatment which is our main subject to-night; but I venture to bring them forward very briefly because no treatment can be judged otherwise than by comparison.

There was throughout these seven years occasional moderate use of antipyretic drugs, especially of sodium salicylate, given generally in single evening doses of twenty or thirty grains, and as a rule producing slight temporary lowering of the temperature, but no very noteworthy result for good or bad; and also quinine has been very frequently used, not in antipyretic quantities, but generally in doses of two, three, or four grains at a time as a tonic.

I think I am justified in calling the treatment on the whole alcoholic; though I have not had an opportunity of any very accurate comparison with other hospitals on this point. But I understand that the administration of eight ounces of brandy in a day is very rare at Middlesex Hospital and not common at St. Bartholomew's or St. Thomas's.

Now at St. George's during the past seven years, more than half, in fact three fifths, of the patients with typhoid fever have had at some time or other in their illness as much as eight ounces of brandy a day or more; and what is perhaps more remarkable, nearly one fourth of the whole number, adults and children, have gradually risen to have at some time as much as sixteen ounces of brandy or more in a day; in some cases, indeed, of extreme danger from exhaustion after hæmorrhage or other calamities, as much as thirty or thirty-six ounces in a day; and I must say in a few such I have thought I could trace great good to it. But to attempt a judgment without any sufficient knowledge of other non-alcoholic modes of treatment would be most unreasonable. I have merely to lay the facts before you.

There are two points at least on which I should be glad to compare notes very briefly with Dr. Coupland; first, as to the comparative safety of the cases which come early under treatment, and secondly, as to the immediate causes of death.

It will have struck those who heard Dr. Coupland's paper or saw his table of cases, that, with the exception of a man who contracted typhoid in the hospital—which I am very sorry to think

should have been possible in such a hospital as the Middlesex—not one case out of the 52 admitted in the first eleven days of the disease proved fatal, whereas 6 died out of the remaining 37. That is a point which I am surprised was not more pressed upon your attention by Dr. Coupland, for it gives some force to the remark that typhoid fever if early taken in hand by antipyretic methods may be tolerably efficiently controlled. One could only wish that Dr. Coupland had had ten times as many cases in which he could show the same result to hold; for I cannot help feeling that 89 cases with 7 deaths is a very small number to argue from in such a subject. Turning now to cases without antipyretic treatment, such as 281 in St. George's, what do we find? That the cases admitted during and after the first eleven days bear about the same numerical ratio to one another as in Dr. Coupland's series, *i.e.* that there are at least twice as many of the earlier dates as of the later, but that the death-rate is quite different, being rather greater in the cases of earlier admission than in the later, 28 per cent. in the earlier as against 22 per cent. in the later, instead of about 2 per cent. in the earlier and 16 per cent. in the later. I have endeavoured to be honest and have excluded a small group of cases where I was incompetent to determine the date of admission even roughly, but they are too small to affect the main result. There can be no doubt that between Dr. Coupland's series and the series I have to give there is not only a great difference in the total death-rate, but a difference also in the relation of the mortality to the time of admission. To determine whether this is due in whole or part to antipyretic treatment would need abundant statistics and good logic; to see that the worst cases will probably be admitted earliest needs only a little common sense.

The second point that I mentioned, the analysis of causes of death, presents many difficulties, for death is rarely the result of a single cause. Even perforation of the bowel, which often seems a single and immediate cause of death, may be determined by many small accidents—movement, coughing, delirium, &c., which are themselves due to other and various groups of causes. Dr. Coupland has attempted a division of the causes of death into four groups, of which the first two, and far the most important, are the febrile and the intestinal; and if anything can be clear in such a difficult classification it is that hyperpyrexia must come among the febrile. Under antipyretic treatment one naturally expects to see hyper-

pyrexia banished at all costs, and in fact there is only one case of Dr. Coupland's where it seems to have been admitted to have been present in conjunction with pulmonary congestion as a cause of death. But in my series of 281 cases there are 17 in which the temperature arose above 106°, which may fairly be called hyperpyretic; 4 rose above 108°, 1 above 109°, and in all but one of these 17 death followed. These are certainly febrile enough, and if I include also under the febrile group the cases where exhaustion from prolonged fever and pneumonia were the chief causes of death, I should say that at least half the total cases of death were due to febrile causes, and nearly all the remaining half to intestinal causes —hæmorrhage, peritonitis, and perforation. That is a contrast to Dr. Coupland's summary of the total results of antipyretic treatment during the last five years at the Middlesex Hospital, where he says that the deaths from febrile causes have been only one third as numerous as from intestinal, whereas my experience under expectant alcoholic treatment is that, as far as I can arrange the deaths under these headings, they are about equally numerous.

Dr. Coupland naturally gave no account of the maximum temperatures reached by his patients, for after they had got up to 103° they were doomed to fall. In looking through my series of 281 I find that out of the 69 who died 36 went over 105° and 33 did not, so that 105° may be taken as very near to their average maximum; and of the total recoveries the average maximum is 103·5°, so that you will see they had ample opportunity of suffering from whatever pyrexia can do.

So far I have merely attempted to give very short evidence that cases of enteric treated by expectancy and alcohol do no better when admitted into hospital early than when admitted late, and that hyperpyrexia is a frequent accompaniment of death among them—an accompaniment by itself of nearly a quarter of the deaths, and along with pulmonary symptoms of at least another quarter. I must add a final word as to the high death-rate that I have described; a death-rate in fact of 24 per cent. I cannot pretend to account at all satisfactorily for its difference from what I am assured again and again is the general hospital mortality in London, but I imagine that it may, at least in part, be due to an adoption of an unusually stringent definition of enteric fever as contrasted with various forms of febricula. There is no case included among these 281 where the evidence that it is enteric fever is not, I think, strong.

My most careful predecessor, Dr. Owen, has put down in the three earlier years some dozen cases as "possibly enteric" which I have not included, and I have as many more to add of a similar sort, enough perhaps to reduce the death-rate to 20 or 21½ if they were all genuine, and I have certainly seen some cases admitted as genuine which I should not have recognised. But it is quite out of the question that I should detain you longer with tabulated figures at this period of the evening or attempt to drag down your attention to consider the lower limits of typhoid, or whether, indeed, it has any lower limits. I can only hope to add a small quota to the evidence of the natural history of the disease as compared with its history under antipyretic treatment, and perhaps to bring out the high lights of the antipyretic speakers by the addition to the picture of a little shadow.

Dr. GILBART SMITH showed that out of 456 cases there were 68 deaths, or a mortality of 14·9 per cent., in the London Hospital. During 1883 the treatment by cold was still in vogue at the London Hospital, and had not fallen into disuse, as Dr. Bristowe had imagined, in all the metropolitan hospitals. Dr. Gilbart Smith was in favour of employing antipyretic means, but preferably some other form than the bath. He thought the employment of the apparatus for a hot-air bath might be utilised so as to give a cold-air bath. This method of "cradling" he had found at times distressing, but he was of opinion that some method for its management might be devised. The influence of nationality on typhoid fever was alluded to. At Milan the Italians were unable to digest milk during typhoid fever, whilst English people treated at the same hospital were able to take a milk diet.

Dr. BROADBENT said that the case for the cold bath had never been better stated than by Drs. Coupland and Cayley. He was decidedly in favour of the employment of the cold bath. He would use sponging if the temperature reached 102° F., and bathing if 104° F. He thought the severity of the disease was proportionate to the temperature. High pyrexia during the first few days was a strong indication of the future course of the fever. The form of enteric fever known as "bilious" was no doubt an exception, and the temperature might remain low in that variety. He gave his personal unbiased opinion that many lives were saved by the bathing, and he had never seen evil consequences follow its use. He had seen pulmonary complications and albuminuria clear up immediately with subsidence of the large abdomen after cold bathing. Perhaps the cold bathing protracted the fever. The only contraindication was the occurrence of intestinal hæmorrhage.

Dr. MACLAGAN could not coincide with Dr. Coupland's views. The blood would be driven away from the surface by the cold, and so the tendency to bowel complication was increased, and with it the danger to life. And Dr. Coupland's figures showed that the mortality was greater. He did not think that a high temperature was the only source of mischief, for it was not found at the period of greatest danger. The intestinal lesion was of the greatest moment in typhoid fever. He

thought that the cold bath was going through the same phases as the employment of other remedies—*e.g.* quinine, salicylates, &c., in medicine.

Dr. SANSOM had employed the cold treatment since 1871. He sponged if the temperature reached 102°, and bathed if it attained 104°. He considered that the statistical method was the only useful one for the great body politic of medical men. He had no hesitation in believing that the mortality had been in some sense reduced since the introduction of cold applications. His individual experience proved to him that the employment of cold was free from danger and from the occurrence of complications. He treated pneumonia with cold water. He had found pale lungs at the autopsy of enteric cases so treated.

Dr. KINGSTON FOWLER had had considerable experience in the treatment of typhoid fever, and believed that the application of cold was of undoubted value.

Sir JOSEPH FAYRER had treated cases of hyperpyrexia in India with ice and the cold douche with great success.

Dr. COUPLAND, in reply, said that the statements made by Dr. Cayley, Dr. Sansom, and many other speakers, had sufficiently met many of the objections urged against this method of treatment. He fully concurred with the interpretation given by Dr. Cayley of the fatal cases, the occurrence of which had been the means of inducing Dr. Bristowe to abandon the practice; and he trusted that Dr. Bristowe, whose freedom from anything like bias or prejudice was well known, might be so persuaded by what he had heard of the experience of others as to once more give this line of treatment a trial. For his own part he was so convinced of its value that he looked forward to pursuing it with greater diligence, and would undertake to communicate the results of his further experience to the Society. He could assure Dr. West that he did not wish to lay too great stress upon statistics, knowing well the fallacies that underlay them; but at the same time statistics were of value when considered in detail, as he had endeavoured to do. He was glad, however, to find that neither Dr. West nor the physicians whose opinions he quoted, could gainsay the advantage of antipyretic treatment, although they either condemned or deprecated the use of cold bathing to effect this in typhoid fever. And admitting that there were objections, mostly on the side of difficulty in application, to the cold bath, any measure which should be equally efficacious in reducing the fever would be welcome; for—and here he must join issue with Dr. Maclagan—he must confess that the heightened temperature was *per se* an element of considerable gravity in any case of typhoid fever. This he had attempted to show by contrasting the fatal cases in two different periods, viz. in that when but few measures were adopted to reduce temperature, and that when antipyretic treatment was more in vogue. The contrast would have been far more striking had it not happened that in the latter period the mortality from intestinal perforation had been so high, an increase which he had already explained could not be attributed to the use of cold, since the majority of these cases were not so treated. As to the alleged difficulties in the way of carrying out systematic bathing of fever patients he thought these had been exaggerated. In hospitals, where the nurses were imbued with the fact of its efficacy, the labour involved was really but slight; and to show that it could be carried out with similar ease in private practice he might be allowed to read a note received from Dr. E. H. Lendon, of Uxbridge Road. Dr. Lendon writes:

"February 24th, 1884.—In answer to your inquiries I have to say that I have found no great difficulty in carrying out the bathing of typhoid patients in their own homes. I have done it in all my bad cases. A good nurse is essential, and when I find that she thoroughly understands her duties I give her orders to bath the patient even in my absence if the temperature rise to 103°. I find that however much opposition there is on the part of friends at first, they are soon convinced of the beneficial effects of the bath, and the patient usually asks for it when the temperature begins to rise. I shall be happy to give you any further details you may wish."

March 3rd, 1884.

ON THE ENDEMIC ORIGIN OF THE LATE EPIDEMIC OF CHOLERA IN EGYPT.

By Sir W. Guyer Hunter, K.C.M.G., M.D.

After a brief description of the physical characters of the country, its soil, meteorology, and water supply, reference was made to the insanitary conditions constantly present and to the prevalence of bowel complaints including gastric and typhoid fever, dysentery, and gastric catarrh, the mortality from which alone in Cairo in 1881 reached as high as 33·7 per cent. and in 1882 38·3 per cent. of the total number of deaths.

Reasons were given for grouping the diseases termed cholerine, choleriform, cholera nostras, cholera, and choleraic diarrhœa, into one class, viz. cholera, and stress was laid upon the many points of resemblance in clinical symptoms and pathological anatomy of these various diseases.

The history of the late epidemic in Egypt was given in detail, and it was shown that many statements which had been brought forward to prove the importation of the disease from India were groundless.

A strong opinion was expressed that the late epidemic was the outcome of the diseases which already existed in the country in an endemic form, and that cholera itself was not a disease resulting from the entrance into the organism of a specific germ.

March 10*th*, 1884.

ON THE CONNECTION BETWEEN DISEASE AND DEFECTIVE HOUSE SANITATION.

By W. H. Corfield, M.A., M.D. (Oxon.), F.R.C.P.

Professor Corfield said that his object was not to put forward any new or startling theories, but to bring before the Medical Society a short account of a few of the cases of disease that had come under his own knowledge as being caused through defective sanitary appliances and arrangements in houses. By such defective appliances foul air from the drains and sewers was allowed to escape into the houses, thus contaminating the air breathed, the water drunk, and the food eaten, either with the results of fæcal and other organic decompositions, or, with these, accompanied with the poison of some specific disease.

The result of this admission of foul air into houses was, in the first place, malaise; and it was quite a common thing for persons to observe that they and their families enjoyed better health generally after their houses had been put into proper sanitary condition. When the contamination of the air was marked the most frequent result was the production of sorethroat or of diphtheria, and frequently diarrhœa would be found, and occasionally attacks of pneumonia had been believed to be attributed to the same cause. Puerperal diseases, too, were frequently traceable to the same source.

The air of the house was contaminated by means of defective drains pervious to air, often directly connected with the sewers or with cesspools, and from which rats found their way into the basements of the houses, the foul air following them. Sometimes rats bored their way from the defective drains of one house, under the party wall, into the basements of the neighbouring houses; and he gave a remarkable instance of disease which had been produced repeatedly in a house in this way, until the cause was found out and remedied.

Frequently, too, there are direct connections with the drains from the basement floor of the house through water traps of some kind or other, but these in such situations frequently got dry, and no such connections with the drains and the basement floor of the

house should be allowed. Many instances of disease had been observed by him to arise from this cause.

Unventilated soil-pipes directly connected with the drains and sewers frequently became perforated by foul air, which thus escaped into the house. Numerous cases of sorethroat, diphtheria, and occasionally typhoid fever had been traced by him to this cause. In one instance he had found a soil-pipe with an open head inside the house in a cupboard on the nursery floor; in this house there had been frequent cases of illness. In another house, where the children were always ailing, the soil-pipe of a w.c. was found to discharge its contents underneath the school-room floor, where there was a kind of extemporised open cesspool. The ventilating pipes of soil-pipes were made to end too near to windows, and he had recently had brought under his notice some cases of diphtheria and several sorethroats which had been produced in this way. Bad forms of water-closet apparatus also tend to make the air of the house foul, and to produce disease. The "pan" closets with their foul containers are chiefly to blame in this respect. Professor Corfield mentioned the case of a house where diarrhœa was produced in most of the inmates from the foul air from the containers of the pan closets which had ventilating holes in them, but no ventilating pipes attached to them, so that the foul air was driven into the house.

It is important to have water-traps under water-closets, but the D-trap, which is most commonly used, is in reality a small cesspool, as its section shows, and is frequently the cause of bad smells in the water-closets. The connection of the waste pipes of sinks with the drains is another fertile source of disease, and any number of instances of it might be produced.

Rain-water pipes and rain-water gutters connected with the drains are very frequently the cause of sorethroat and diphtheria in houses, and sometimes of typhoid fever. Several instances of these were given. In one instance a case of diphtheria occurred in a room the balcony of which drained into a rain-water pipe directly connected with the house drain and with the main sewer, the trap on the house drain having been removed because it was liable to get blocked up.

In another house where sorethroats were very frequent many defects had been remedied without any good result being observable, but it was at last found out that a rain-water pipe from the

roof passed down through the best bedroom and the drawing-room into the drain, and that the joints were defective.

Frequently cases of diphtheria or of sorethroat are found in rooms in the upper storeys of houses when the windows are near the heads of rain-water pipes, if the latter are connected with the drains; and even when they are trapped at the foot they require to be disconnected. Several instances were also given of the ill effects produced by the travelling of foul air about houses by means of bell-wire tubes, floor spaces, spaces behind panels, &c. A slight escape of coal-gas must not be mistaken for an escape of foul air, as is not unfrequently the case. The smell, too, of an old flue, especially if it has been on fire recently, is very liable to be mistaken for the smell of foul air from a soil-pipe or drain. With regard to contamination of drinking-water, either by reason of leakage of foul matters into it, so frequent in the case of wells, and occasionally happening in the case of cisterns where the soil-pipes are near them, the result was the production of diarrhœa or of typhoid fever and occasionally of diphtheria. The same results were produced by contamination of water in cisterns by means of overflow pipes connected with the drains or soil-pipes; instances of these are very numerous. An instance was also given of a case of typhoid fever which was produced by the contents of a soil-pipe, which was out of doors, leaking through a defective joint and running down the outside of the soil-pipe into a cistern which supplied a tap. Instances of the contamination of food in larders by foul air were also given. One in which a dried bell trap directly connected with the street sewer was found inside the larder in a house where there had been frequent cases of illness of various sorts, and one of enteric fever. Another instance of a soil-pipe in the wall of a larder into which a nail had been driven and was found loosely hanging in it, the foul air escaping around into the larder. Another in which the w.c. opened directly out of the larder. In dairies, too, there are often connections with the drains, generally trapped, but sometimes even untrapped. The danger of disused sanitary appliances was also pointed out, and an instance given of a school where there had been several cases of sorethroat owing to the fact that there was a disused sink in the basement connected with the drains, and from which foul air was escaping fast into the house. When the sanitary arrangements of this house had been put to rights (as was supposed) this sink was

not interfered with as it was not used. In another quite recent instance a case of enteric fever was produced in a house by the fact that the trap of a disused w.c. in the basement had become dry, and allowed foul air to escape into the house.

The paper was illustrated by specimens from the Parkes Museum of Hygiene and by diagrams. In conclusion, Professor Corfield remarked that people were fast becoming wiser in these matters, and having their houses put into proper order before they went into them instead of waiting for disease to warn them that there was something wrong. It was very important for medical men to understand at any rate the general principles of house sanitation, as they might by a timely warning prevent much disease and death.

The PRESIDENT said the subject was one of wide and varied interest.

Sir JOSEPH FAYRER thought the lessons which had been given ought to be more generally promulgated than they were. He hoped that the International Health Exhibition would help to dispel the alarming ignorance which prevailed in these matters. The diminution of mortality and the alteration of the type of disease were what he anticipated from the practical adoption of Prof. Corfield's lessons.

Dr. HERON regarded the defects in houses as still of very common occurrence, not only in towns, but also in health resorts, where he had heard that the inhabitants did not like to make sanitary improvements for fear of attracting the attention of the public.

Mr. BOWREMAN JESSETT could bear out the remarks of Dr. Corfield on the danger of bell-wire tubes. An instance was given in illustration. He did not think the Hopper closet was the best.

Dr. ISAMBARD OWEN said he would like to draw an authoritative declaration from Dr. Corfield on two points. The first was as to whether competent inspection ought not to be made of the work of the British workman. Secondly, whether he thought children and old people were much more liable to be affected by sewer gas than adults. Sir JOSEPH FAYRER had found the atmosphere of Amsterdam in a horrible condition, and had been surprised that disease and the death-rate were not heavier.

Mr. PURCELL and Mr. WELLS made a few remarks.

Dr. CORFIELD, in reply, said that the syphon-trap was self-cleaning, and therefore unlike the D-trap. Lavatory basins in bedrooms were a great mistake when they were directly connected with the soil-pipe. A modified Hopper closet, with vertical back and flushing rim, was thought to be the best for the basements of houses, and some people preferred this form throughout the house. Every pipe and every joint of a drain ought to be inspected by a competent person. Children were certainly markedly affected by sewer gas, but he did not know that old people were. The lecture was illustrated by specimens kindly lent by the Parkes Museum of Hygiene, Margaret-street, W.

Dr. SANSOM showed some specimens of Solid Liniments, and

Mr. FLEMING showed an Electric Lamp.

March 17*th*, 1884.

A CASE OF SPORADIC CRETINISM WITH APPEARANCE OF MYXŒDEMA.

By Amand Routh M.D., B.S., M.R.C.P.

Eliz. H—, aged twenty-five and three quarters, is an inmate of the Home for Female Incurables, Marylebone Road, and was born near Southampton, in Hampshire. She was the eldest of four children, both parents being quite healthy when she was born, being respectively twenty-four and twenty-two years of age. The father, however, died twelve years afterwards, and the mother sixteen years afterwards, both of consumption—a disease which also caused the death of the patient's own sister, aged seventeen, and her mother's sister, aged twenty-seven. The father was born in Hampshire, the mother in Berkshire, and there was no blood-relationship between them.

Up to the age of seven years the patient was just like other children, but plumper than usual, and always looked like a "large white pudding," being "fat, flabby, and of a doughy-white colour." Whilst pregnant with this patient the mother was frightened by a bull. At seven years of age the girl ceased growing taller, but continued growing stouter and flabbier, becoming slow in all her movements, and so stupid that all attempts at teaching her to read and write were fruitless. At eight years of age the child came to London, and beyond getting less able to help herself and more silly-looking, the friends say there has been little or no change, with the exception that latterly she has become thinner.

Family history.—Beyond the apparent heredity of consumption, there is no family history of any disease, such as syphilis or Bright's disease, and no history of alcoholism, neither is there any history of insanity or cretinism in the family. A sister of the patient, aged twenty-three, is a fairly healthy-looking girl, well developed, and regular in all her functions, though somewhat anæmic. She has, however, an enlarged thyroid gland, now somewhat diminished under treatment, and there is a systolic murmur to be heard at the heart's apex and left axilla, dating from an attack of acute

rheumatism three years ago. The patient also has a healthy brother, aged nineteen, now in the army. All the children were born in Hampshire.

Present state.—Height, 3ft. 5in., the average height of a child of five years of age. Weight, 4st. 2lb. 12oz. She is much stunted in growth, and her bony skeleton is very considerably altered by rickety changes. Thus, there is a lateral curvature of the spine in the dorsal region, with the ribs thereby secondarily distorted. Lordosis is well marked. The tibiæ are curved. The epiphyses of the bones of the lower limbs are somewhat enlarged, and the ribs beaded at their junction with their cartilages. The head is very dolicho-cephalic—a condition of the skull which Dr. Gee has stated is often due to rickets. Some of these bony changes are not unlike those described by Dr. Barlow and Mr. Bowlby as occuring in fœtal rickets (sporadic cretinism as it occurs in the fœtus?), and it is possible that one reason why they do not coincide more exactly is that in this case these changes may not have occurred till seven years of age, when the sporadic cretinism first became apparent. The face is absolutely expressionless, and the lines obliterated. The features are swollen; the lips thickened, high coloured, and shapeless; the mouth usually open; the nose is flattened and broadened between the eyes, and the nostrils dilated; the ears large and flat. The head does not hang forward, but the neck appears shortened, being lost in the supra-clavicular tumefaction.

The *skin* is everywhere waxy-looking and pallid, with the exception of the face, where there is a slight flush, absent at times, over the flabby, pendulous cheeks, and on the feet and legs, where the circulation is so poor that a state of chronic chilblain exists. The skin is dry and rough in parts, but the palms of the hands are occasionally moist. The hands are spade-like in form. There is no true œdema.

The *hair* is jet black, and fairly plentiful, but very harsh.

The *temperature* of the body averages 98·8° F. in rectum, but the surface temperature is always subnormal, being usually between 95° and 96° in the axilla or groin. Once only was it as high as 97°, and that was after the patient had been covered up in bed for some hours.

The *thyroid body* cannot be felt. Above the clavicle on either side, and to a less extent immediately below that bone, are well-

marked *tumefactions*, which slip away when grasped, and though somewhat resembling lipomatous or myxomatous tissue to the fingers, also remind one of the sensation conveyed by grasping a varicocele. A clear respiratory murmur can be heard over the swellings, but no venous bruit. Similar subcutaneous swellings can be felt over the ribs in posterior axillary line.

The *abdomen* is tumid and pendulous, owing partly to the shallow pelvis.

The *umbilicus* is protruded, and the recti are non-adherent in the middle line.

The patient can walk slowly and totteringly for short distances, but often falls suddenly, so never walks without assistance being at hand. The legs seem unable to bear the weight of the body, and give way suddenly at the ankles or knees. She has free movement of her arms, but does everything very slowly and methodically, yet quite unhesitatingly. She does not dress herself, because she would be all the morning doing so, but can put on or off her clothes if allowed to take her own time.

The *speech* is slow and monotonous, and pitched in a high key. She answers questions correctly enough, but her answers are delayed. She appears to take a longer time than usual to understand a question, and then to require a still longer time to frame the reply.

Nervous system.—Patient can neither read nor write, and every attempt to teach her anything has failed; in fact, it is impossible to keep her attention concentrated. She cannot remember recent events, but can recall events which occurred before she left Hampshire at seven years of age, the growth of both body and mind seeming to have been then arrested. She sleeps well and heavily. She is almost absolutely indifferent to pain, yet often complains of feeling cold. The "knee reflexes" are exaggerated; and Dr Angel Money drew my attention to Gowers' "front tap contraction," which is also well marked. Nothing appears to interest her for more than a few seconds, but when cheerily spoken to a momentary gleam of pleasure flits across her face. Otherwise, her expression is always motionless, callous, and somewhat sad.

The *eyesight* is good, and ophthalmoscopic appearances normal. Pupils are somewhat dilated, and rather sluggish.

Urinary organs.—Patient passes on an average 25 oz. of urine in

twenty-four hours. Sp. gr., 1015. There is no sugar, albumen, or casts. The urea is deficient, being only about one third of the normal quantity, viz., 10½ grammes, or 172 grains per diem (percentage 1·3). This result was obtained by the method devised by Drs. Russell and West.

Digestion, Organs of.—The tongue is not notably enlarged, nor indented by the teeth; the faucial arch is broad; the teeth are badly formed, and several have been removed, being decayed. The alveolar processes are unusually prominent, and the mucous membrane of the gums much thickened. Patient's appetite is good; her breath is usually foul; her bowels constipated. She constantly complains of a nasty bitter taste in her mouth.

Organs of circulation.—Pulse 72. It is very difficult to feel the pulse at the wrist owing to its distance from the surface, and its weakness. The heart-sounds are normal but weak.

The *blood.*—The blood was very kindly examined for me by Dr. Montagu Murray by means of Dr. Gowers' hæmocytometer and hæmodynamometer, as well as microscopically. Dr. Murray states that the proportion of white to red corpuscles is about 1 to 250: that the percentage of red blood discs to the normal standard is 86; whilst the percentage of hæmoglobin is 77. There was no obvious change in the appearance of the blood-corpuscles, red or white.

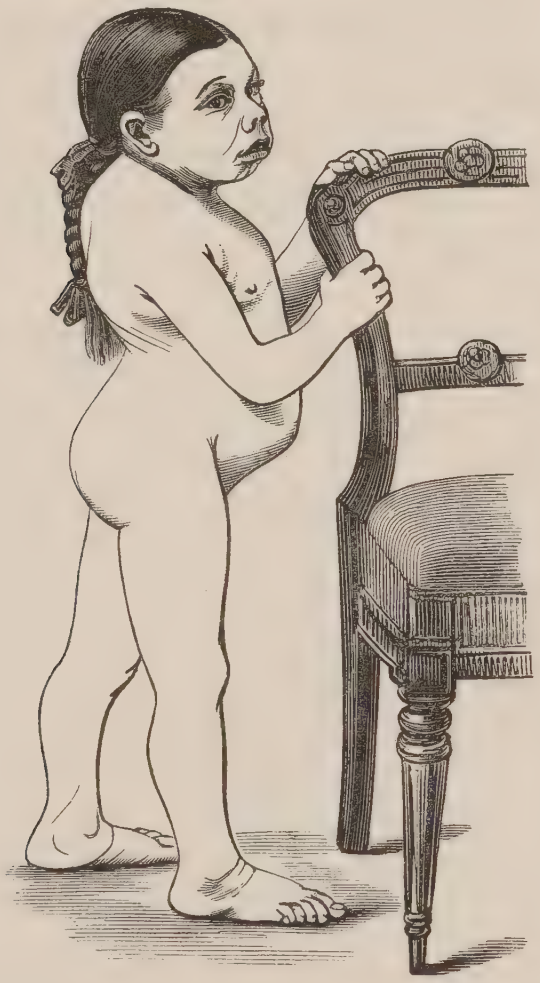
Organs of respiration.—Quite normal in every respect.

Liver and spleen.—Nothing abnormal is to be made out in these organs. They are not perceptibly enlarged.

Generative organs.—Patient has never menstruated. There is no leucorrhœa. The external organs are quite undeveloped and destitute of hair. The labia minora are elongated and thickened, protruding some distance from the vulva. The mammæ are quite infantile. The vagina and os uteri are normal. Per rectum, the pelvis is found to be infantile in shape and size, and the uterus is half its normal adult size, but normal in shape and position. The ovaries lie against the sides of the pelvis, lower and more posterior than usual, and are at least twice their normal bulk; and are softer than usual, and cause no sense of pain when grasped between the finger in the rectum and the hand on the abdomen. It is probable that the ovaries have undergone a change analogous to myxœdema, which would account for the persistent amenorrhœa,

and the infantile condition of both external and internal organs of generation.

Remarks.—I first saw this patient on account of her chilblains, and because the other inmates of the "Home for Incurables," where she is staying, complained of her bad breath. I believed the case to be sporadic cretinism, with myxœdema, and this diagnosis was kindly confirmed subsequently by Drs. Gowers, Ord, Barlow, and my father. Dr. Ord pointed out that the bony deformi-



ties appeared to be of rickety nature. The case agrees in most of its features with the description given by Sir William Gull and the late Dr. Hilton Fagge, in their papers on sporadic cretinism—especially as possessing the subcutaneous symmetrical swellings, as first pointed out by Mr. Curling, and considered by many, and by Dr. Ord in particular, to be similar to myxœdema, a disease named and described by that gentleman in 1878. In his paper read before the Royal Medical and Chirurgical Society in 1871, the late Dr. Hilton

Fagge laid stress upon the fact that sporadic cretinism was probably due to atrophy of the thyroid, and he further stated that he believed that a healthy, and still more an enlarged, thyroid was to be regarded as a safeguard against cretinism—in other words, that a healthy thyroid exercised a protective influence over the production of cretinism. He showed that in countries where cretinism was endemic those who had the largest goitres were not cretins at all; and that a large goitre protected the individual from the more severe effects of cretinism. He further showed that in England the cause of cretinism, whatever it may be, only acts with a low power, which is expended in producing goitrous enlargements of the thyroid, but that if this cause of cretinism should act upon a person with an undeveloped or absent thyroid no such protective influence could be afforded, and cretinism would result. This case lends some little support to this theory, which has not hitherto been much entertained.

The exact relationship between myxœdema and sporadic cretinism is not yet made out. Sir William Gull and Dr. Fagge showed that sporadic cretinism was invariably associated with the presence of symmetrical tumours in the supra-clavicular and other regions; but their relative priority is unknown. In Dr. Hilton Fagge's four cases, those in which the cretinism had lasted the longest had these symmetrical swellings the least well marked, and *vice versâ*. Hence it would seem that the myxœdema, if such it be, decreased as the cretinism increased. The case I am showing this evening also seems to indicate this, as the girl has latterly been getting thinner and losing weight. The cretinoid change does not appear to have commenced in this case till seven years of age, up to which time the patient was much as other children, and this is in accordance with Dr. Fagge's remark that sporadic cretinism may arise as late as the eighth year. It would, however, seem, from the aunt's description of the case that in this patient the myxœdema had commenced much earlier; for she says the patient, as a baby, always looked "fat and flabby," and like a "doughy white pudding," a very fair popular description, I think, of a myxœdematous baby. This description is also in accord with that given before the Sardinian Commission on Cretinism, by Foederé, who states that those infants destined to become cretins have a body extraordinarily voluminous, and are mostly œdematous. There is reason, therefore, for believing that myxœdema precedes, or, at all events, may precede

the development of sporadic cretinism. It seems now also clearly made out that non-development or atrophy of the thyroid gland precedes both myxœdema and sporadic cretinism. In his paper on myxœdema read before the Royal Medical and Chirurgical Society in 1878, Dr. Ord suggested that the mucous œdema might precede the atrophy of the thyroid, but the cases of Mr. Curling, the papers of Sir William Gull and Dr. Fagge, together with Kocher's remarkable results following complete extirpation of the thyroid gland, as narrated by Dr. Felix Semon, have led Dr. Ord lately to admit that evidence is now tending to show that atrophy of the thyroid gland precedes the myxœdema.

Résumé.—No date can be fixed in this case for the atrophy of the thyroid gland, but it seems likely that it is a case of intra-uterine non-development of the gland, tending first to myxœdema, and secondarily to cretinism, for we have a clear history that the mother, whilst pregnant with this child, was frightened and chased by a bull, and maternal fright during pregnancy was given by Dr. Hilton Fagge as having occurred in one of his four cases. After birth, up to seven years of age, the child's general appearance was that of a myxœdematous baby. At that age sporadic cretinism supervened; whilst quite latterly the mucous œdema has been showing signs of subsidence. The girl continues to be under medical supervision at the above-named establishment.

Dr. Miller Ord thought that myxœdema and sporadic cretinism had many points in common. The present case began a little after infancy, and some time before adolescence. A mixture of conditions was to be observed. The absence of the thyroid, the condition of the skin generally—dry, harsh, anæmic, with diminished sensibility and without uniform swelling were alluded to; there was less swelling of the tongue than in myxœdema, and an absence of swelling of the eyelids; the lowering of temperature of external parts was present; the supra-clavicular swellings were better seen generally in cases of sporadic cretinism; the reduction of weight of the whole body and disappearance of the fulness and tension of the skin as a whole were important features, as were also the arrest of intellectual development and of the development of the sexual organs. The thyroid body varied considerably in cases prior to the full development of myxœdema. Sometimes exophthalmic goitre had preceded the condition of myxœdema and atrophic thyroid body. The case of a lady which presented all the symptoms of myxœdema was related, and it appeared that exophthalmic goitre developed so rapidly later on that the patient lost one eye. It was curious to observe what different sets of symptoms were associated with each enlargement of the thyroid in exophthalmic

and the other chief form of goitre. We want to know more of the condition of the thyroid body prior to the development of cretinism and myxœdema.

Dr. Felix Semon said that Bal, of New York, had observed after extirpation of the thyroid body the symptoms which Kocher had described as ensuing some time after the operation of thyroidectomy. Schiff had shown that death ensued in dogs in from four to six or eight weeks after removal of the thyroid gland. The animals got very sleepy and died of convulsions. After death there was anæmia of the brain. Schiff and Liebermeister looked on the thyroid gland as a blood regulator for the circulation of the cerebrum. Recently Zesas had asked the question whether thyroidectomy was a physiologically permissible operation. The spleen could be removed without harm, but not the thyroid gland. Of Dr. Routh's case he would say that he could feel a suspicious swelling on each side of the thyroid cartilage, but hesitated to pronounce the swelling to be the thyroid gland.

Dr. Hadden related the outlines of a case of mixed myxœdema and sporadic cretinism which he had seen in France, and which tallied very closely with the one shown by Dr. Routh. He said that in the present case there was exaggeration of the knee reflexes; these were not exaggerated in myxœdema in his experience. He spoke of the retardation of body-waste, and mentioned that a foreign observer had found undoubted lesions of the sympathetic trunk in the neck and abdomen of a case of myxœdema. The dogs mentioned in Schiff's paper lived only three weeks, and so it was hardly justifiable to draw conclusions as to their mental and physical state.

Dr. Angel Money said that the knee-jerks were decidedly exaggerated; and, further, the "front tap contraction" of Dr. Gowers was to be obtained.

Sir Joseph Fayrer spoke of the cures of goitre to be effected by the ointment of the biniodide of mercury and exposure to a tropical sun. He failed to see what advantage could accrue from a surgical operation when such simple means were so efficacious.

Mr. Sydney Jones related briefly the result of his operation of the removal of the isthmus of the thyroid gland in three cases—viz. great atrophy of the lateral lobes. It was a safe operation, and continental experience was like his own.

Dr. Cavafy said that it seemed to him that myxœdema resembled sporadic cretinism rather than sporadic cretinism resembled myxœdema.

Dr. C. H. Routh had seen but little good result from the iodine treatment in any form. He asked what was the mental condition of Mr. Jones's cases.

Mr. Jones replied that the time since the operation was too brief to allow of any positive statement.

Dr. Radcliffe Crocker had used the biniodide ointment in the treatment of goitre, using a strong fire instead of the sun. His success had not been marked.

ON IMPETIGO VEL PORRIGO CONTAGIOSA.

By T. Colcott Fox, M.B. (Lond.), M.R.C.P.

The affection known as *impetigo contagiosa*, though not very formidable in itself, is of some importance and interest on account of its frequent occurrence, contagiousness, and disputed nature. I bring the subject forward with the hope that the facts I have observed may conduce to a more exact knowledge of the disease, and tend to clear away some of the vagueness and confusion which in my experience surround it. I believe I am correct in saying that, although the affection was certainly known to some few observers here, it was by the writings of Dr. Tilbury Fox that attention to the disease was first drawn in America and subsequently Germany. Hence many writers have awarded to him a priority in the description of the disease, which is also claimed, I believe, for the late Mr. Startin by gentlemen who have worked at the Hospital for Skin Diseases in Blackfriars. I propose to open this paper with a short historical account, which I hope will place this matter on sure ground.

Willan and the English dermatologists immediately following him distinguished a group of diseases under the generic term *porrigo* from another group of non-contagious pustular and scabbing affections denominated *impetigo*. The several varieties of *impetigo* (excepting i. rodens) viz. i. figurata, sparsa, scabida, and erysipelatodes, were gradually rearranged and brought by various writers into relation with eczema, and the seal was conclusively set on this classification by Hebra. So too the heterogeneous group included under the term *porrigo* was gradually broken up; p. larvalis and furfurans were relegated to eczema, p. scutulata we now know as tinea trichophytina or ringworm, p. decalvans as alopecia areata vel circumscripta, and p. lupinosa as tinea favosa or favus. One variety alone survived as a distinct disease with some English writers, viz. the porrigo favosa of Willan and A. T. Thomson, or, as Bateman preferred to call it, impetigo favosa, and even this phase of disease was absorbed into ezcema by Hebra and confounded by Rayer and Biett by reason of the name with the parasitic affection now known as favus. It is to this impetigo or porrigo favosa of the earlier English writers on skin diseases I would specially draw

your attention. The contagious nature of this eruption was insisted upon by these writers, as I will show by a few short extracts. Thus Plumbe ('Practical Treatise,' 4th ed. 1827, p. 268,) says, "Like ringworm the p. favosa spreads rapidly by infection through families of children, and it is not uncommon to see several of them inoculated from one child, around whose mouth one or two pustules may have appeared, and the contents have been applied to the lips and cheeks of its brothers and sisters in kissing them. The breasts of the nurse are not unfrequently inoculated in the same manner." Bateman speaks to the same effect and says that the acrimonious discharge is both auto-inoculable and contagious. He writes (8th ed. 1837, p. 199), "A sudden eruption of impetigo contagiosa, accompanied by fever, occasionally takes place also in children. A considerable alarm was excited by such an occurrence in a family which I was requested to see in which the disease was deemed to be some new or anomalous contagion. The first patient, aged five, was seized with severe fever, in which the pulse was at one time 140, and continued at 110 for several days; at the same time clusters of favous pustules appeared behind the ears, which were speedily followed by others on the scalp, and about the apertures of the nostrils, which they plugged up as the scabs were formed. A few days after the commencement of the attack, a younger child, aged two, was seized in a similar manner. . . . ."

In his lectures in the 'Medical Times' for 1846, Startin identified the affection called by him porrigo simplex with Willan's p. favosa, and wrote thus, "It is not uncommon to see whole families suffering from p. simplex or favosa, and I have known nearly every individual in a school of young children thus affected." Mr. Startin's colleagues and successors continued to recognise the affection as described by him, and Mr. Hutchinson illustrated it in the 'Sydenham Society's Atlas," but I think they overlooked the previous descriptions I have mentioned. In 1863 ('Brit. Med. Journal,' 1864,) and in 1869 ('Journ. of Cut. Med.') Dr. Tilbury Fox independently redescribed the affection at some length under the name impetigo contagiosa, and states that he understood some such disorder was recognised by Mr. Startin under the name porrigo contagiosa. Mr. R. W. Dunn, also writing about the same time, said the disease was not described in books or only defined in a vague and unintelligible manner. From that time numerous papers on the subject have appeared in America and on the Continent.

It is not my intention to trouble the Society with any exhaustive description of the eruption, which varies widely in extent of distribution and severity, and in some degree according to the site, nor shall I go into the differential diagnosis, for all are now agreed that the affection is distinct from vesicular and pustular eczema, though liable to be mistaken for it when the eruption is confluent. Those who include the artificial inflammations of the skin, as excited by the acarus scabiei and croton oil, with eczema might possibly retain impetigo contagiosa in that category. Briefly the eruption is a vesico-pustular one, and the inflammation very superficial. It appears as isolated acuminate or rounded projecting vesicles about the size of pins' heads, just tinged at their bases with a little redness and usually accompanied by only a slight degree of irritation. The vesicles, unless destroyed by scratching, enlarge to the size of a split pea, but the walls rarely remain tense as in the ordinary pemphigus bullæ, but get flaccid and collapsed. As this collapse begins in the central parts the vesico-pustules are not unfrequently umbilicated, and this character is very much more marked in some epidemics and at some seasons than others. These umbilicated vesico-pustules present such a striking resemblance to the vaccination eruption that it has suggested the probability of an intimate relation of the disease to vaccinia. Indeed, such an eruption has more than once been described as "bastard vaccinia." Then, again, in certain groups of cases the individual bullæ cover a much larger area than usual and spread rapidly by a raised, exuding border, whilst the central parts are collapsed just as we see in some cases of chilblains and festerings about the fingers. Last year I saw in succession two sisters, aged respectively fifteen and twenty-one, with an extensive eruption of this character covering areas larger than a penny-piece. The contents vary in character with the state of nutrition of the tissues, the age of the subject, and the virulency or special features of the outbreak, and hence the scabs also vary as the contents are serous, honey-like, or more or less puriform. The contents are, however, usually sero-purulent, and the scabs straw coloured, flat, dry, and granular looking, with a special "stuck-on" appearance (Tilbury Fox). The eruption is nearly always disseminated in greatest profusion about the face, but very commonly extends to the scalp, the neck, the fingers and hands, and less frequently to the limbs and trunk. Its extent of distribution varies from a few lesions in one locality to an almost universal

eruption, which has to be carefully distinguished from varicella or some anomalous vesicating eruption. The mucous membranes of the eyes and nostrils may be involved. About the fingers the lesions assume an unhealthy festering state associated with onychia, as seen in pustular scabies, and about the limbs again take the ecthymatous aspect. I. contagiosa is chiefly an affection of children, but adults brought into immediate relation to them are attacked. I have never seen an aged person with it. It is far more common amongst those who live overcrowded, uncleanly, and surrounded by insanitary conditions, but in rarer instances it may attack the well-to-do and cleanly, though in my experience it has not then the same tendency to rapid spread unless the health of the household, or school, or locality has been lowered from defective draining or some special cause. The eruption is described as evolving in crops, but I think there is rather an irregular appearance, and I believe its course is not terminated spontaneously, but rather that the disfigurement at a certain stage brings about the adoption of remedial measures. That the disease is contagious there can be no doubt. I have already quoted the experience of Plumbe, Bateman, and Startin, and, indeed, there is a consensus of opinion on this point. From time to time we may come across isolated cases, or in twos or threes, and at another time numerous cases present for treatment, and epidemics occur in houses, courts, streets, schools and other institutions. Such have been reported in the journals, and, indeed, it is a common reply on the part of a patient to our questions as to whether there are other cases about, "Oh yes, Sir, there are a lot of children just like it in our street." I may refer here to the epidemic reported in the 'Lancet' in 1871, attacking 100 men of the 2nd Battalion of the Foot Guards at the Beggar's Bush Barracks at Dublin, and originating amongst the children at the Curragh. I have also met with several outbreaks in schools and other institutions.

Now, what is the nature of this affection? Is it a specific affection running a definite course and comparable to varicella? Is it only inoculable like vaccinia and acquired syphilis? Is the eruption due to a systemic infection, or is each individual vesico-pustule merely a superficial inflammation excited by the inoculation or contact of irritant discharge at that spot? I venture to say my brother's views were not entirely clear on this point. At one time he considered the affection one *sui generis*, that a highly conta-

gious and specific quality resided in the secretion of the vesico-pustules, and that the extension of the disease was due to the contagion of this secretion, but he further remarked in reference to the antecedent malaise and febrile excitement, which he recorded in many cases, and the appearance of the bulk of the eruption in crops early in the course of the disease, that "there is clearly an affection of the system at large." He, however, added that it could scarcely be ranked amongst the "acute specific diseases," and he held that in addition to the eruption from the systemic infection there was a local extension of the lesions from spot to spot by direct inoculation. Mr. Hutchinson writes ('Syd. Soc. Atlas') that porrigo contagiosa "may originate from any cause which induces the formation of pus, such, for instance, as a scratch. In the preseut instance it resulted, as it not unfrequently does, from suppuration and the scab left by vaccination." I understand Mr. Hutchinson to hold that it is purely a local disease, and that each lesion is a local inflammation excited by the contact of pus from some source. Now, this problem is surrounded by many difficulties, and I think the majority of observers assume something of Hillier's attitude when he said that he was fully satisfied some cases of impetigo were contagious and quite different from ordinary impetigo (*i.e.* pustular eczema), but he could not make up his mind whether impetigo contagiosa was a distinct disease or a mixture of many pustular diseases.

Now, there can be no doubt that pus from many sources is capable of setting up a local inflammation or vesico-pustule when inoculated in the skin. Take the pustular eruption of the posterior third of the scalp excited by pediculi, which by the way were not recognised as the cause of an eruption in the first half of this century, and see how commonly vesico-pustules are excited about the face, fingers, hands, neck, and even trunk. We notice the same thing in purulent catarrh of the ear and nose. It is unquestionably true that a similar eruption may be excited by the pus from beneath a vaccination scab. Tilbury Fox at the time he first wrote on the subject was struck with the great frequency with which i. contagiosa followed in the wake of vaccination, and he states that it "could be traced by contagion through a series of cases to some one subject in whom the disease had followed close upon vaccination." There seems to have been a suspicion excited that the eruption was in some measure connected with vaccinia. Moreover, it is

not uncommon to find an unhealthy child excoriate its skin and the watery pus from the festering sore readily excites a vesico-pustular eruption whenever it is inoculated. And we may go a step further and show that such an eruption is inoculable on other children in the same house or locality. In localities or institutions where the nutrition of the individuals is much lowered through bad or insufficient food, or malhygienic conditions, such sources of pus formation are very rife, and epidemics of impetigo arise. In the cases I have examined into I have almost always determined some source of this kind. Such an eruption is confessedly indistinguishable in aspect from that of impetigo or porrigo contagiosa, and Dr. Tilbury Fox eliminated, certainly in his later years, such cases, but contended that there were still others occasionally met with where no such origin could be traced. For some time I have systematically endeavoured to trace such cases as presented themselves to their source and have very frequently been able to do so satisfactorily. As might be expected, failures are not infrequent, but the conclusion I have come to is that the cases included under the term i. contagiosa are entirely of local origin and extension, and not the consequence of systemic infection, although I allow that now and again cases are met with, more difficult to account for thus, of wide distribution on the surface rapid in evolution, and in appearance analogous to varicella. By those who think that the disease is one *sui generis* it is argued with the late Dr. Oscar Simon that, though any pustule might be contagious, it would only produce another like itself, not a complete disease with characteristic symptoms and course. But it is this very point of which I am sceptical, and with regard to the occurrence of an antecedent febrile state we want more exact observations. There is some uncertainty about it, and observers are not agreed. Moreover, it must be proved to be a constant and special feature because it is not improbable that when pus is inoculated some irregular systemic excitement is produced, but the eruption itself may nevertheless not be due to a systemic infection. Even allowing that the eruption is entirely local in origin and spread, it would seem probable that pus from the several sources described acquires more irritating qualities at some seasons than others, either from what Bateman calls acrimony or from the presence of some micro-organism in especial abundance, such as Drs. Piffard and Crocker have described. Kaposi and others, it is well known, have described a fungus and considered the affection an

impetigo parasitaria, but Kaposi has, I believe, now abandoned the idea that this fungus is a constant feature. I have sought over and over again for a fungus and have only found it in an interesting case which turned out to be one in which the trichophyton fungus had excited some pus production in the scalp, and thence pustules containing the fungus had arisen about the face.

P.S.—Since the reading of this paper I have met with a second case of vesico-pustular ringworm of the face simulating i. contagiosa.

Dr. Radcliffe Crocker was not disposed to draw any marked distinction between impetigo contagiosa and cases of ordinary pus inoculation. He thought the presumption was strongly in favour of their being identical. He narrated a case in an adult of most extensive impetigo contagiosa which appeared to ensue on irritation from bugs. In the clear contents of the vesicles he had found chains of micrococci, which were more abundant when the eruption became pustular, and were to be seen in the pus-cells, especially at their periphery. He believed in the external origin of impetigo contagiosa, and that it was curable by external agencies. The discrete character of the lesions was pointed out. He would not positively assert that the micrococci were the cause of the disease.

March 24th, 1884.

CASES OF FACIAL ERYSIPELAS WITH LOW TEMPERATURE.

By John Cavafy, M.D., F.R.C.P.

There is probably no disease which presents so great a variety as erysipelas in the extent, severity, and character of the inflammation of the skin, which forms its essential symptom; and accordingly an extensive nomenclature has been devised in which some attempt has been made by authors to define the different forms of the disease. Thus we meet with the terms erysipelas erythematosum, and e. phlegmonosum, distinguishing the depth of tissue affected; e. glabrum, diffusum, and marginatum, according to the character of the surface and edge of the inflamed part; the presence of vesicles, bullæ, pustules, and crusts is denoted by the terms e. vesiculosum, bullosum, pustulosum, &c.; while the name e. gangrænosum points to what is fortunately a rare termination.

Again, if the affection remain limited to the parts first attacked it is called e. fixum; while if it extend to fresh tissue, the term e. migrans marks this character; lastly, the names e. multiplex and erraticum signify the simultaneous or successive implication of distant parts of the skin.

It may be doubted whether much is gained by the employment of so many names to designate varieties of what is essentially the same disease; no light is thereby thrown on the nature of the erysipelatous process, and variability, although very striking in erysipelas, is at least equally so in many other diseases; but it may perhaps be admitted that the particular character of the affection is emphasised by the adoption of these diverse appellations, and that an important clinical fact is thereby stamped more firmly on the memory.

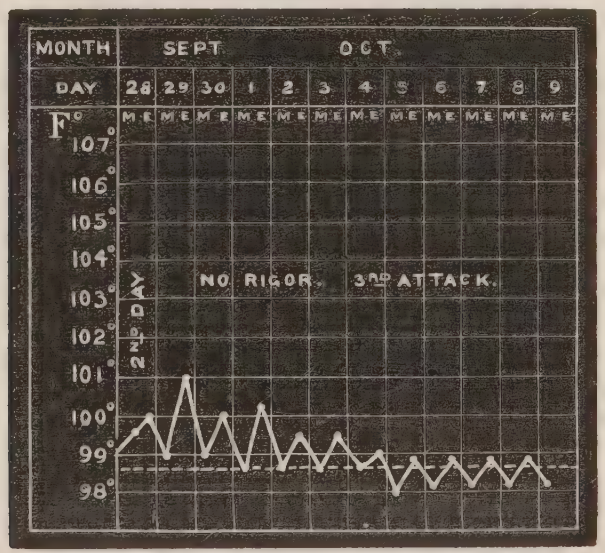
Be this, however, as it may, it is not only in aspect and extent of cutaneous inflammation that erysipelas varies widely, but also in the severity and character of the fever which accompanies such inflammation. Cases may be preceded by a rigor or rigors, and the temperature may rise to 104° or more in the first twenty-four hours, or it may reach this point gradually, in the course of two or three days; it may be maintained at a high pitch, with very slight remissions, or the remissions may be as marked as in enteric fever; and there may even be cases of an intermittent type, a morning temperature of normal height being followed by an evening rise to 104° or more, for several days in succession. It may be said that, as a general rule, the fever is directly proportional to the skin-affection, any extension of the process being accompanied by a rise, while quiescence or temporary diminution of the inflammation, is expressed by a more or less marked fall. Defervescence usually takes place by crisis, a fall to normal or subnormal occurring as suddenly as the rise; or, more rarely, there is a gradual subsidence, extending over several days.

But, although the above mentioned varieties may be said to include the majority of cases, instances of still greater divergence may be not unfrequently met with. Thus, there may be no initial rigor; the fever may precede the skin-affection by one or two days, or it may follow the inflammation; the remissions or intermissions may last several days; there may be high fever with comparatively slight inflammation; and lastly, the fever may be very slight throughout, and there may be an extension of the inflammatory

process in the skin during defervescence, even when a normal temperature has already been reached.

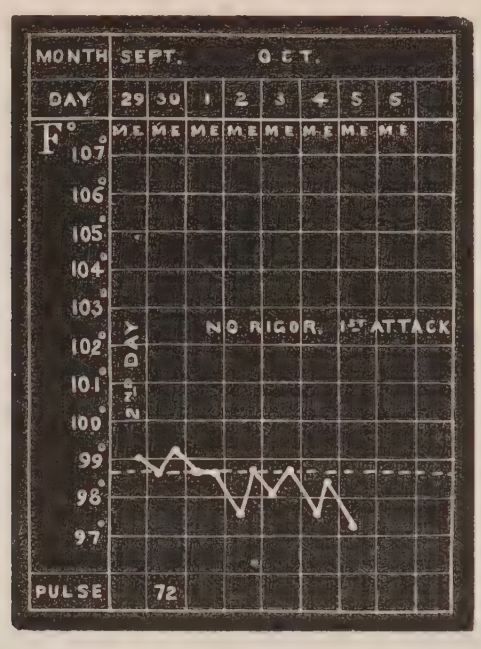
I have had recently a few cases of the latter mild type under my care in St. George's Hospital, which seem to me not without interest in some particulars, and I have therefore thought that a condensed account of them might be worth a little attention.

Case 1.—A man, aged sixty-five, admitted on September 28th, 1883, had had two previous attacks of erysipelas; the first, and severest, five years ago; the second, two years ago. In the afternoon of the day before admission, while feeling quite well, he noticed that the bridge of the nose was slightly swollen and red. The same evening, the swelling extended to the left cheek, and during the night to the right cheek, with slight burning pain; no rigor. On admission (second day), there was circumscribed swelling and redness over the bridge of the



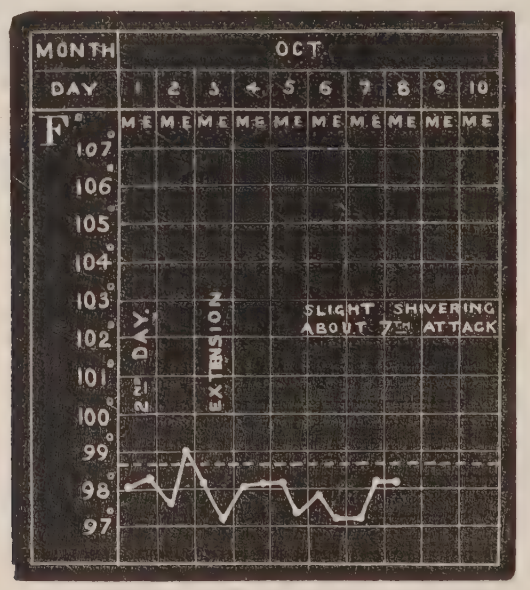
nose, and on both cheeks below the eyes; the affected parts were tense, tender, and slightly painful; evening temperature, 100°. The temperatures were:—September 29th, morning, 99°; evening, 101°. 30th, morning, 99°; evening, 100°. October 1st, morning, normal; evening, 100·2°. 2nd, morning, normal; evening, 99·5°. 3rd, morning, normal; evening, 99·5°. 4th, morning, normal; evening, 99°. 5th (ninth day), morning and evening normal, and continued so. There was no extension, and the swelling and redness steadily subsided.

Case 2.—A man, aged thirty-nine, admitted on September 29th; no previous attack of erysipelas. On the day before admission he felt quite well until the evening, when there was some heat and tingling of the left cheek; on the following morning swelling and redness had appeared, and extended to the nose; no rigor. On admission (second day), circumscribed redness and swelling of left side of nose, and neighbouring left cheek, on which are two bullæ near the nose; no pain; evening temperature, 99°. September 30th, temperatures; morning,



normal; evening 99·2°. October 1st (fourth day), morning and evening normal, and continued so. No extension, but slow subsidence, with much branny desquamation.

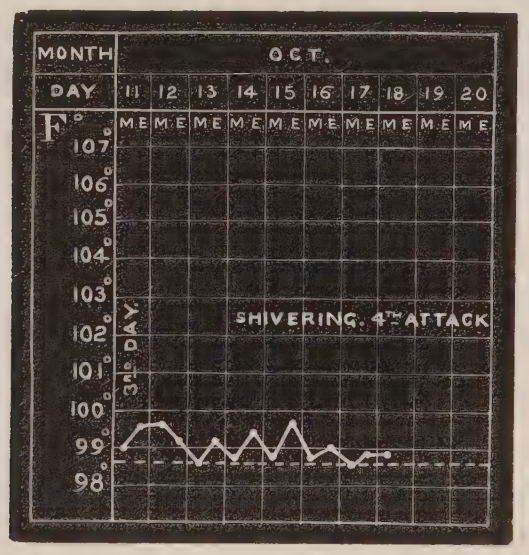
Case 3.—A woman, aged thirty-two, admitted on October 1st. About six previous attacks of erysipelas during the last seven years; the last nine months before the present one. On the day before admission she had slight shivering and headache, with smarting and tingling of the face, which began to swell; the swelling had increased and extended next day. On admission (second day), circumscribed redness and swelling of both cheeks and bridge of nose, most marked on left side, where



it extends to the margin of lower jaw; a good deal of aching pain. Temperatures, morning and evening, normal. October 2nd, morning,

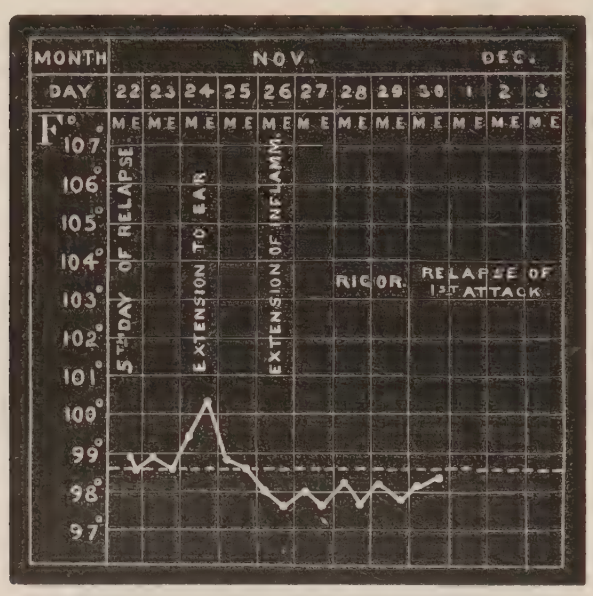
subnormal; evening 99°. No change in face. October 3rd (fourth day), morning and evening subnormal, and continued so. On this day, however, the redness and swelling extended over the upper and inner two thirds of the left concha, where it lasted three days before subsidence, the temperature never rising above 98°. All swelling and redness afterwards steadily diminished, with branny desquamation.

Case 4.—A girl, aged twelve, admitted on October 11th. Three previous attacks of erysipelas. Two days before admission she had a fit of shivering, and the face and nose swelled. On admission (third day), swelling and pale redness of nose and neighbouring cheeks; margin not very sharply marked; very slight sensation of pain and heat. Tem-



perature, morning, 99°; evening, 99·6°. October 12th, morning, 99·7°; evening, 99°. 13th, morning, normal; evening, 99°. 14th, morning, normal; evening, 99·5°. 15th, morning, normal; evening, 99·7°. 16th (eighth day), morning and evening, normal, and continued so. Steady subsidence throughout; very slight branny desquamation.

Case 5.—A woman, aged thirty-five, admitted on November 22nd. A month ago, having had no previous attack of erysipelas, the right ear became swollen, red, and painful; this subsided in a few days. Eight days ago swelling and redness of right eyelids, increasing so as to close eye; this subsided completely in four days, when she had a rigor, followed by redness and swelling of left side of nose and cheek, with much pain. On admission (fifth day of relapse), slight branny desquamation of right eyelids and cheek; bright redness and swelling of left side of nose, both left eyelids, and cheek, sharply circumscribed downwards and inwards; more diffused over outer side of cheek. Temperatures; evening, normal. November 23rd, morning and evening, normal. 24th, morning, 99·5°; evening, 100·2°. Headache; fresh patch of redness and swelling at back of left ear; bulla on cheek. 25th (eighth day), morning and evening temperatures were normal, and on following day subnormal, and continued so. But on the 26th, there was an extension of redness



and swelling to left side of forehead and temple. From this time steady subsidence, with desquamation.

Such are the cases which I have observed, and probably many will think that there is not much to be said about them; they are simply mild or abortive attacks of erysipelas, and there is an end of the matter. If this view be held, it still seems to me not without interest that in one of them (Case 5) a rigor, and in two others (Cases 3 and 4) slighter fits of shivering, preceded so mild an attack; further, that two (Cases 2 and 5) were first attacks, which are usually severe; and, lastly, that in two (Cases 3 and 5) an extension took place while the temperature was subnormal.

But another view of these cases may be taken. It may be doubted whether they are to be considered examples of true erysipelas, and some may prefer to rank them as instances of spurious erysipelas, or erythema, or non-specialised dermatitis. As the contagiousness of true erysipelas has been placed beyond question by the experiments of Koch, Tillmanns ("Erysipelas," 'Deutsche Chirurgie,' p. 9), and especially Fehleisen ("Deutsche med. Wochenschrift,' 1882, p. 553), as well as by many clinical facts, an accurate diagnosis is obviously of importance. It has been shown that the virus of erysipelas may be collected, cultivated by appropriate methods, and transmitted to animals and to man by inoculation, the resulting disease being often of great severity. The question then arises, can the virus, when inoculated, give rise to a mild affection similar to that under consideration? The experiments of

Tillmans (loc. cit., pp. 14, 18) seem to show that it can. Out of twenty-five inoculations on dogs and rabbits, five were followed by marked erysipelas with high fever; in two there was erysipelatous redness and swelling, but only a slight rise of temperature, while in several others (the number is not stated) the inoculation was followed by spreading redness and swelling, which resembled erysipelas in every particular, but ran their course without fever. Now Tillmanns considers that as these cases were apyretic they were not genuine erysipelas, but I must admit that I fail to see the force of his reasoning. The selection of high fever as the pathognomonic sign of the true disease seems to me altogether arbitrary, and, even if it be accepted, impossible to apply with accuracy. It is surely true that the cases with the highest fever and those with the lowest, or even with none, are connected by transitional forms which establish an insensible gradation between the two extremes, and I am quite unable to see where the line should be drawn, on one side of which we are to place all the genuine cases, and, on the other, all the spurious ones.

If, then, we admit that true erysipelas may occasionally run a mild course, practically without fever, and if we also recognise the fact that some forms of erythema, and even urticaria (*e.g.*, the "acute circumscribed cutaneous œdema" of Quincke), may have a close resemblance to erysipelas, we have to inquire by what means we may distinguish between these affections. I think there is one character of importance which is present in erysipelas and not in the others, and that is rapid spreading at the margins. Mr. Jonathan Hutchinson, in an able paper on "Certain Diseases allied to Erysipelas" ('Medical Times and Gazette,' January 1883), insists strongly on this point. After pointing out that erysipelas may be often wanting in vesication, in abrupt margin and even in hyperæmia, he defines as erysipelatous any inflammation which *travels* and is attended by œdema. But, even if we pay attention to this valuable sign, I think we shall still meet with instances in which diagnosis must remain uncertain. Tillmanns, referring to such cases (loc. cit., p. 196), says that it must remain a question how far they are true erysipelas; and, further, that a correct diagnosis has no practical importance; but I do not think it would be wise to accept this view without reserve. Erysipelas is unquestionably contagious; and, although this may not be equally true of all cases, the severe ones being probably far more dangerous in this

respect than mild examples, we cannot do wrong in taking all necessary measures to avoid communication of the disease to others. The severe cases shade off into the mild and doubtful ones by such gentle transitions, that it is certainly not always an easy matter to decide as to the rank which should be occupied by any particular case under notice. The difficulty is, perhaps, best solved by Mr. Hutchinson, who concludes that there is a family of erysipelatous affections, rather than a single disease erysipelas—some closely, others remotely, connected with the typical complaint. He holds that, in all, a virus is probably produced, which enables the disease to spread by contagion; and, whenever transmission to another person occurs, the disease, he believes, is intensified.

If these views are correct (and we shall at least be on the safe side if we adopt them), it is plain that there may be a danger of infection from any case, however mild; and caution will be always desirable. This possible danger of infection should, I think, be especially guarded against by those who are engaged in the practice of midwifery, and in the performance of vaccination and other trivial operations. The close relation of erysipelas to a grave form of puerperal fever is well established, and the slightest wound may become the starting-point of erysipelatous inflammation; indeed, it is nearly certain that some breach of surface, however trifling, is a necessary antecedent of all erysipelas. Under these circumstances, I have no doubt whatever that he will be the best practitioner who takes the minutest precautions against the possible conveyance of infection, however remote and improbable such a contingency may appear.

Mr. Robert Parker had seen cases in children which presented all the characters of erysipelas without fever; further, the amount of constitutional disturbance was not in direct relation with the amount of skin inflamed.

Dr. Cullimore mentioned some examples of inflammatory disease unattended with pyrexia.

Dr. C. H. Routh narrated facts tending to prove that all exanthematous eruptions might at times be apyrexial in their course.

Sir Joseph Fayrer had long ceased to believe that rigors and pyrexia were necessary to the diagnosis of every abscess of the liver. He was of opinion that some cases of spreading facial erysipelas were not attended with a rise of temperature.

Dr. Wynn Williams narrated a case of "nervous shock" associated with rise of temperature.

Mr. Robert Parker said children frequently presented high fever.

Mr. Pearce Gould asked whether Dr. Cavafy had heard of cases of traumatic erysipelas running their course without fever. He considered that some individuals could resist the causes of the febrile process much more readily than others.

Dr. Cavafy explained that "erysipelas fixum" did not spread after it had reached a certain point. Albuminuria was present in one or two cases. Facial erysipelas might sometimes be preceded by a minute traumatism.

A CASE OF OPIUM POISONING.

By David W. Finlay, B.A., M.D., M.R.C.P.

The record of the following case is interesting and instructive chiefly on two grounds—firstly, as showing how near may be a fatal termination, and yet recovery ultimately take place under vigorous and persistent treatment; and secondly, on account of the apparently antidotal effect of subcutaneous injections of atropia.

Alfred S.—, a stableman, aged thirty-six, was admitted into the Middlesex Hospital, under my care, at 10·15 p.m. of Nov. 17th, 1883. Twenty minutes before admission he had swallowed in mistake for porter, a draught intended for a horse, which contained a quantity of tincture of opium and compound tincture of camphor, equal to seventeen grains of dry opium. When brought to the hospital he was unconscious, but was breathing fairly, with a good pulse; his pupils were contracted to the size of pins' heads. While the stomach-pump was being got ready, the breathing, which had been gradually becoming slower, ceased altogether, and he became deeply cyanosed. Artificial respiration and faradaism were then resorted to, which had the effect after a few minutes of restoring respiration, and with this a return of natural colour to the face. The stomach-pump was then passed and the stomach well washed out, the returning fluid being at first of a brown colour, and containing but little solid material. After this his breathing failed a second time and rendered a return to artificial respiration necessary. This was kept up for an hour and a half, and its effects aided by faradaism and flicking the face and chest with wet towels. The stomach was again washed out and about half a pint of strong coffee introduced into it. An enema of brandy was also administered. Improvement again ensued, and he was walked briskly

up and down the hospital corridor for about three-quarters of an hour, after which he was able to swallow some coffee and brandy. Even then, however, he relapsed if left alone for a moment, and he was accordingly walked up and down the garden for half an hour. Till now his pulse had been pretty good, but on returning from the garden it was found to be small and weak, numbering 140 in the minute, and he relapsed into a condition of profound unconsciousness. The respirations were now only two in the minute. The condition of his breathing, however, was speedily improved by a further recourse to artificial respiration, and his pulse by a subcutaneous injection of ether. As the respirations speedily sank again to two in the minute when artificial respiration was omitted, it was decided to try the effect of atropine, and accordingly one-sixtieth of a grain of the sulphate was injected subcutaneously and repeated in half an hour. His respirations, a few minutes later, were found to be fully doubled on the average. They were irregular however, as he had several respirations one after another, followed by a period of apnœa. The pupils also were soon noticed to be less contracted. If left alone he immediately lapsed into a deeply soporose condition, but could be roused by two or three sharp slaps on the face. After this compelled exertion was again employed, and about 4 a.m. (Nov. 18th) he was taken into the ward and put to bed, an enema of beef-tea and brandy being given. He was now able to tell his name, and he swallowed about an ounce and a half of beef-tea and brandy. His respirations after this varied between six and ten per minute, and he was prevented from falling into a deep sleep and freely stimulated with brandy. At 11 a.m. he was found to be much improved, and could answer questions intelligently although still sleepy. The respirations were then from ten to twelve per minute. From this time till 2 p.m. there was little change, except that in the interval he had several attacks of vomiting. The note made at 5 p.m. states that he had vomited again, and the respirations were fourteen per minute. At 9 p.m. the temperature was 99·4°; respirations 20, and he had again vomited. On the following morning (Nov. 19th) his temperature was 99·2°, he had vomited only once during the night, and had slept well. He complained, however, of a dull headache, and general aching and soreness. His urine was acid, had a specific gravity of 1015, and contained one-eighth of albumen. His pupils were noticed to be abnormally large. The evening temperature was 100°.

Next day he was much better although still somewhat drowsy; his temperature was normal, and his pulse 76. On the 22nd his bowels were opened naturally and his appetite was improving, the temperature being rather below the normal, 97·8° in the morning, and 97·2° in the evening. Next day he suffered a little from headache, but the bowels were open and the urine was found to be free from albumen. He was now able to be up out of bed, and he improved rapidly, his pupils gradually became smaller, and he was discharged well on Nov. 28th.

The symptoms which followed the man's recovery from immediate danger were such as are commonly found in cases of opium poisoning. He had nausea and sickness, with headache, anorexia, and slight albuminuria. Contraction of the pupils was replaced at first by slight and afterwards by marked dilatation, which lasted for some days, and was no doubt due to the atropia injections. This opens up the question of the atropia favorably influencing the course of the case, and it seems more than probable that it had this effect. It is certain, at all events, that a marked improvement in the condition of respiration almost immediately followed the injection of atropia, and from the subsequent dilatation of the pupils there could be no doubt that it had produced some physiological action. It was obvious that the patient was in danger of death more from apnœa than from cardiac failure, and was consequently in the state most likely to be benefited by the antagonistic action of atropia; for many observers are agreed that there is a real antagonism between the two poisons, in the human subject at least, in their action upon the respiratory centre. It is worthy of note in connexion with this view that recourse to artificial respiration was not required after the injections.

I should mention, in conclusion, that the satisfactory result of the case was due mainly to the prompt and energetic action of the resident medical officer, assisted by the other resident officials of the hospital.

Dr. CHOLMELEY thought that injections of atropia ought to be employed very early in cases of opium poisoning. Some cases were so collapsed that walking was out of the question.

Dr. C. H. ROUTH said that the simultaneous injection of atropine with morphia prevented sickness.

Dr. ISAMBARD OWEN said he believed that morphia paralysed the respiratory centre in the medulla oblongata.

Dr. FINLAY replied that it was impossible to say how much of the

poison had been absorbed, and how much got rid of in other directions. Atropia was used as a *dernier ressort.*

Dr. CULLIMORE showed a case of Post-Diphtheritic Paralysis.

INFLUENCE OF THE WEATHER ON THE BODY TEMPERATURE, AS SHEWN BY FIGURES COLLECTED IN DAVOS.

By ARTHUR WM. WATERS, F.G.S., F.L.S.

I APPEALED about the middle of September, 1881, through the local paper and privately applied to a number of people remaining in Davos to measure their temperature three times a day in order that we might be able to see how patients were influenced by the weather.

I considered that the body temperature would give a fair indication as to the general alterations in the state of health, and this is one of the very few symptoms which can be used for statistical purposes, though fortunately, from our present stand-point it is one of the most important that a doctor can have under his consideration. I undertook the investigation in the hope that anything which enables the medical men to judge how the patients were affected would also enable them the better to decide for whom the climate is unsuitable and who should not remain here; for having known this health resort directly and indirectly for a number of years I am *most* decidedly of opinion that there are a large number of people who can derive great benefit from these mountain climates, but on the other hand I am painfully persuaded that a large number come here annually in various conditions of illness who are not at all likely to derive any advantage, many of whom might get on much more satisfactorily in other places and some who have not found this climate suit them I know have got on well elsewhere, and one such case has now for twelve years followed his profession in a warm resort.

As long as so little true scientific examination of the action of this climate is being made, it may seem almost hopeless to expect that it will ever be possible to prognosticate, with any reasonable probability, who may expect to find the climate suit them. Yet although there may seem little light in front, I do believe

EACH PATIENT ABOVE OR BELOW THEIR 5P.M. MEAN

ABSOLUTE MOISTURE ABOVE OR BELOW THE MONTHS MEAN IN M.M.

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OCTOBER 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 26 27 28 29 30 31

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MEAN ABS. MOIST

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that whenever the question of climatic suitability is attacked from all possible points it will yield material results, and the present attack on one point seems to show that it is a subject worthy the fullest investigation, and what I have now done should only be looked upon as a small beginning.

For the measurements, I fixed upon the hours of 7—8 in the morning, 5—6 in the afternoon, and 9—10 in the evening. I laid much the greatest weight on the 5—6 p.m. temperature, and let all know that in case it should be impracticable to take all these observations regularly, that this one should if possible never be neglected and as naturally might be expected this was, as we shall see, the time at which people were most regularly and simultaneously affected. In the instructions which I wrote out, among other things, I asked all to take the temperature in the mouth for about ten minutes, always using the same instrument, and measurements not to be taken immediately after drinking extremely hot or cold liquids, nor within ten minutes of a stool, also to note whether taken in bed or dressed, any diarrhœa, constipation or excessive perspiration, also to note when there was any unusual exertion, if kept indoors, variation from usual amount of stimulant, cold water applications, cold baths and fever-reducing medicines.

Although many very kindly furnished me very regularly with their temperature, the value of the series is very materially reduced by the fact that a large number did not give any notes as above indicated, and thus I have been unable to eliminate special causes. On this account such observations ought to be collected conjointly by all the medical men in practice.

I obtained fifty-one promises, of whom thirty-seven (living in different hotels and with various meal-times) sent me observations, some for short periods, but others with great regularity, during several months. Among the number there were for a short time for comparison, six who considered themselves quite well, and the others varied from those with very little the matter to extremely serious and hopeless cases. Out of the thirty-seven I applied personally to twenty-two, and beside these Drs. Unger, Spengler, and Ruedi each very kindly induced some of their patients to furnish me with figures, and they and Dr. Boner gave me confidential diagnoses of all their patients on my list. Those found by the doctors were, as a rule, more serious cases than those I chose. In those I applied to myself I gave special consideration to the reli-

ability of the person, and chose as far as possible those who had had experience in exact observations, and I had among the number four medical men, three professors or doctors of chemistry, two pharmacists, and a large number of the others men of scientific or professional education, and I certainly believe that the figures were as a rule, taken with great care and may be relied upon, though of course out of over 3000 observations it would be rather Utopian to imagine that none may have been occasionally scamped.

The first question to which the figures had to answer was, *Is there an influence of the weather* so that with a number of individuals the body temperature generally, or that of the majority, rises or falls on the same day? 2. Next, having found that there is such an influence, the question is *How are healthy people and invalids comparatively affected?* 3. Then having found that the general effect is similar in the healthy and invalids, only that the invalids are affected to a much greater extent, the question is, *Are individuals as a rule affected in the same way,* or are some such more sensitive than others, or even affected in a different manner? With regard to this last point we found that some individuals almost invariably follow the general curve and that others do not do so, that one patient will month after month increase the amount of the general curve, while another person month after month will appear not to be sensitive to the same causes as the majority.

As we have said, at those times where the rise or fall of the temperature in the invalids is most marked, we also find a similar change among the healthy individuals, yet the number of healthy persons who took measurements was too small to allow of a full analysis of the healthy curve, which was not so marked as that of the invalids, and more figures are necessary before we can make a trustworthy comparison. However, it is shown that the amount of sensitiveness increases with the amount of disease, so that the most seriously ill give the most marked curve, the seriously ill a decided curve, but with a smaller range, which is again still less in the curve of those who are slightly ill.

I made a special effort to have as large a number as possible in October, during which month the average was about twenty observers. I was unable to keep this number up, though during November it was about eighteen; during the early part of September it was only six, though towards the end it was fifteen; in February the number was ten. Knowing that there would be many

disturbing causes before and after Christmas time, and that it was only likely that a great many would be unable and unwilling to carry out the measurements regularly at this festive season, I wrote to those on my list, letting them know that though I should be glad of any figures taken, yet I was not attaching the same importance to the observations of this period, and therefore the number of patients who sent records from the middle of December to the middle of January was very small, but I shall have to return to some interesting facts concerning these months. During December and January the number varied from eight to fourteen.

In consequence of October being the most complete month I look upon that as a test case, and as the meteorological curves happened to be decidedly pronounced, it makes this month very favorable for comparison, and it is certainly striking to see the way in which the curve of the mean of the 5 p.m. clinical temperature and that of the temperature of the air or of the absolute moisture, keep parallel, but striking as this is we must allow that it *might* be accidental if we had only one month to rely upon, but during the rest of the winter the two sets of curves follow one another in the same sense as we found to be the case in October, though not always in the same striking way as when we were dealing with more complete figures, but they fully establish the fact that the laws brought out in that month could have been deduced from the less perfect part of the series.

Having found that most people were simultaneously affected, the next question to solve was what is the influence, and this is by no means an easy problem, for one meteorological condition interacts upon the others so much that there may be marked changes in several at the same time, and therefore it is not easy to separate out each cause. I, however, made monthly curves of the barometrical and thermometrical changes, of the relative and of the absolute moisture both for the mean and for the 1 p.m., and of the wind.

Upon comparing these with the patient's curves I did not find that there was an indication of influence by barometrical conditions, and this was to me a matter of some surprise, nor could I attribute much to the wind or the relative moisture; we now come to a very great difficulty, for the curve of the air temperature and of the absolute moisture is almost the same. With these two curves and the clinical curve there is, as can be seen, great

similarity, but there is not enough to guide us as to which is the influencing cause.

In October we find almost every change of absolute moisture shown in the patient's temperature. There is, however, the rise of temperature on the 5th which at first sight looks like an exception as the mean absolute moisture falls, but when we turn to the absolute moisture at 1 p.m. we find a rise in the midday observation, making the irregularity more apparent than real. It must not be forgotten that we are most affected by the atmospherical influences of the daytime, and there is much to be said for comparing the midday observation instead of the mean. Again, some people are no doubt more influenced at the commencement of a change, while others will feel the ill or good effects later, and taking all this into consideration the resemblance of the curves is very striking.

As the measurements at 5—6 p.m. would always be taken in the rooms, which would not vary considerably in temperature from day to day, we cannot attribute any of the results to physical causes; and, as showing this to be the case, I prepared curves of the *range* of the body temperature between 8 a.m. and 5 p.m., and find that the curve thus given approximately corresponds with the absolute moisture, varying in October from an average difference of 0·70° C. on the 16th, when the weather was cold and dry, to 1·05° C. on the 21st, when it was warm.

It is known that the temperature of people in warm climates is greater than in cold,* and that the difference of colour between arterial and venous blood is greatest in cold climates and least in warm, and when an animal is brought into a warmer space there is a rise of temperature, but in laboratory experiments the change of condition is usually rapid and extreme, and therefore such figures cannot be used in comparing those caused by gradual meteorological changes. These facts would give a *prima facie* reason for considering the influence to be the temperature of the air, but, on the other hand, the curve of the absolute moisture much more closely corresponds to the curve of the patients, and from careful examination of the figures this would certainly seem to be the influencing cause, but the question as to whether the temperature

* Wünderlich, 'Eigenwarme in Krankheit,' says, p. 117, "temperature about 0·1—0·2 Cent. higher in summer than winter," and Davy (*vide* Wünderlich) says, "the temperature is 1° Fahr. higher in the tropics than in temperate zones."

or the absolute moisture is to be looked upon as the factor producing changes in the clinical temperature is one which should be definitely settled by further examination, taking summer and winter months, and it should be done in various climates.

Besides the 5—6 p.m. observations, to which we have been alluding, there were the 7—8 a.m. and 9—10 p.m. The 9—10 p.m. pretty well follows the afternoon temperature, but here the changes are much less marked, and the average rise and fall is by no means so great. The 7 p.m.—1 a.m. changes are again much less marked, and we hardly find a definite curve, but at the same time it is often clear that the causes which have to a greater extent influenced the afternoon and evening temperature have also been at work here.

In the majority of cases the temperature was (as given by the text books) higher at 9—10 p.m. than at 7—8 a.m., but there were five exceptions, and these were prophylactic, or cases in whom the disease had not made extensive progress, and all these five have since continued well. Those who considered themselves healthy were almost regularly slightly higher at 9 p.m. On the other hand, the difference between the temperature at these two hours steadily becomes greater as we go down the list, and when we reach the most serious cases the difference is greatest, the month's mean in one such case being 1·6° C. higher at 9 p.m. than at 7 a.m. Besides seeing where there was a rise or fall of the total temperature, I also examined to see if there was agreement in the rising or falling from the previous day, and in some cases this is very marked, in others less so, but the curve thus obtained shows the same influence of the weather here. In October, on the 25th at 5 p.m., out of eighteen cases the temperature of four rose from the previous day, while that of fourteen fell. On the 8th, at 9—10 p.m., out of twenty, three rose, while the temperature of seventeen fell from the previous day. An examination of the series of these figures shows that the general results do not arise from accident.

We must not forget that we are throughout dealing with incomplete statistics for the meteorological figures are only taken three times in the course of the twenty-four hours, while in order to completely study the influence of the weather we ought to have figures taken at frequent intervals throughout the day, as otherwise some changes of temperature and moisture cannot be brought into consideration, besides which as I have already said the number of

patients is not sufficiently large, and therefore undue influence from special individual circumstances cannot be avoided. While speaking of the incompleteness of the statistics, I should like to point out that no observations were made as to the amount of electricity in the air, nor have any as yet been made, and this may influence the condition of health to a considerable degree. There were also no ozone observations, but these I took one winter,* but as I did not consider that the results were very valuable I have not done so since. The measurement of the amount of moisture is in winter very difficult,† and it does not seem that any system is entirely reliable. The figures which I used for the moisture were taken by Mr. Steffen with a hair hygrometer, and I have calculated the absolute moisture from his figures of the relative.

There is another source of incompleteness, which has been brought out clearly by the figures I have had before me. I refer to the fact that when a physiological derangement takes place causing an unusual rise of temperature then the time of maximum temperature is earlier, often as much as five hours, so that a patient who in what may be looked upon as his present normal condition would have his highest temperature at 5 p.m. will, under aggravated disease of a temporary or even of a permanent character, have the highest temperature at 12 noon. Only a few, and those among the more serious cases, took the temperature in the middle of the day, but I have had some opportunity of seeing the same thing in other cases, and the few figures had under examination lead me to believe that the time of highest temperature gives about as valuable an idea as to the progress of the patient as the degree of temperature attained. In one or two cases where the two curves do not correspond I have, in cases where the material was available, tried replacing the 5—6 p.m. changes by those at 12 midday, and have found that the new curve thus obtained approximated very closely with that of the moisture. This shows that to carry out thoroughly the investigations now commenced the maxima should be obtained, but until a thermometer is devised which can be readily carried about fixed in position this is impracticable. Various such causes as these make it impossible for me to always show by any diagram or by a few figures how clearly there is an influence of one of the two

* 'Klim. Notiz. ü. Winter im Hochgebirge,' Basel, 1871, p. 19.

† See "Preliminary Remarks on Observations made in Davos, 1881–2," by A. W. Waters, 'Proc. Lit. and Phil. Soc., Manch.,' p. 155, 1882.

mentioned meterological factors, but the detail examination of the mass of figures collected has during the last two years made it clearer to me than any diagram could.

Among the irregularities I was surprised to find a decided fall in the patient's temperature about the 13th—15th February without there being any fall in the amount of absolute moisture as shown by the official figures, but upon referring to my observations, of which several were taken in the course of the day, I found that there was an average fall on the 14th which was not shown in the one official observation. There was, however, no fall in temperature of the air.

We have seen that it was necessary to allow the end of December and beginning of January to be very incomplete, and I am sure that it could not have been otherwise, though it was unfortunate seeing that towards the end of December there was a very considerable amount of illness not confined to one hotel or one district, but more or less general, and in my opinion arising from various causes, among which I attribute some of the illness to fish that had travelled some distance, some to newly-built and not fully-dried buildings, and a large number to the natural results of Christmas festivities. The illness included several cases of acute gastritis and colds of minor and serious severity. Among the very few figures received at this time there was a general marked rise in the individuals' temperature about the 25th—29th December, while the meterological conditions did not indicate such a material rise.

If I had been a medical man an analysis of the cases and the results would have been of great interest, but this is beyond my province, and the classification based on the medical diagnosis was made largely for my own guidance. Out of the thirty-seven six were well, seven I called slight cases, twenty very ill, seven extremely serious, fifteen had more or less mischief in both lungs, and at least eleven had cavities. Of the whole number I know that a few months ago all who were comparatively not very ill are now fairly strong and getting on well, while all the most serious cases have died. I was able to hear about most, eighteen months after the figures were taken, and at that time I knew of the death of twelve of the observers. I do not wish to dwell on personal matters, but perhaps it may be well to say that though I have not had previous practical experience with clinical temperatures I tried to qualify myself for the investigation by a study of the works of Claude

Bernard, Wünderlich, Buss, Liebermeister, &c., in order that I might as far as possible know the results already obtained.

Although scarcely a part of the present investigation, there is one point to which it is well to call attention, as it may have some bearing on the influence of temperature and absolute moisture. The variation of the absolute moisture is naturally in winter very small, and in the living-rooms extremely slight, and this I take it is partly the reason why colds are so much rarer here in winter, whereas we all know that in England consumptive people are very liable to them. On the sea the daily variation of absolute moisture is also very small, and the universal report seems to be that colds are very rare during voyages. Again, when we get up to the mountains of Peru there is a very small variation of temperature throughout the year, and in consequence a small range of absolute moisture. This is a factor which is common to these three restorative climates, and there is scarcely any other meteorological condition common to them.

The figures are too few to help us as yet in judging as to who should remain in the high climates, but to my mind they suggest various ways in which they should be extended. Differences in the body temperature may at times arise from serious organic modification, or at other times may arise from different activity of the regulatory apparatus, that is to say, may depend upon the vigour of the sympathetic nervous system, and this may be variously affected in different constitutions, and may explain some being sensitive while others are not. It has been considered by several doctors that these high climates are not suitable for consumptive people with eretic constitutions, and the climate is not found beneficial for most people suffering from nervous excitability or nervous disease, whereas there are forms of nervous exhaustion and even nervous disease, which are recommended to try the high altitudes, and all medical men do not confirm the rule that no eretic consumptive should be sent here. It may be seen that the great influence of the nervous system has been recognised, and seeing how this affects every part of the organism it may seem too general to talk of turning attention to the nervous condition of each consumptive patient, but nevertheless I cannot help thinking that the knowledge gained as to the action of the climate points to the necessity of such a study, giving the chief attention to the sympathetic system, and perhaps before long it may be possible to bring

in electrical measurements as one indication of what climate a patient should resort to.

Explanation of curves of body temperature and absolute moisture. The observations of each individual were added together, and the month's mean for each person was thus found. The variations from their mean was tabulated and added up for each day for the whole number of observers, and from this the average amount above or below the mean was found for each day. The curve therefore starts from the mean and shows the average variation above or below this, and in order to make the two curves comparable I found out the mean absolute moisture for each month, and took this in the same way as the starting-point showing the variations above or below this.

March 31*st*, 1884.

FEBRIS EXANTHEMATOSA ORIENTALIS, OTHERWISE BERIBERI FEVER.

By Norman Chevers, C.I.E., M.D.

I am led by a careful inquiry into the ætiology of beriberi, and especially by a review of the history of the epidemic which visited Bengal and Mauritius in 1877 and 1878, to regard this malady as a specific fever, *sui generis;* and, consequently, to be of opinion that physicians will remove it from its present anomalous position in our nomenclature of disease, as the last and least defined in the roll of general diseases, and place it next to scarlatina, with which fever it has certain marked features in common.

We are told* that the word beriberi is derived from the Hindustani name of a sheep (*b'here*), from a fanciful notion that persons affected with the disease walk like a peculiar species of that animal, *i.e.* with a kind of jerking motion; but the name by which it is known to the Gentoos of the coast, *Ooboo-waioo*, simply means rheumatism combined with dropsical swelling.†

* 'Madras Topographical Reports,' Russel Condah, p. 89.

† Those who may be desirous to trace out fully the etymology of beriberi will find ample data in Fayrer's article, 'Quain's Dict.,' p. 104, and in vol. ii, O. S. of the 'India Journal of Medical Science,' p. 343.

The writer adds, "This disease, though endemic in many situations, frequently also appears as an epidemic after the setting in of the rains, or from July to the close of the year, when fevers, dysenteric complaints, and cholera also break out, to a greater or less extent annually."

Morehead justly objects to the unscientific vernacular designation of this malady; but the name beriberi has been so long accepted that it would now be useless to attempt to abandon it entirely, although our present knowledge of the ætiology of the disease renders it needful that we should modify it—as by calling it "beriberi fever."

The form of acute general anasarca, generally recognised under the name of beriberi, occurs as an endemic and as an epidemic. Its principal endemic habitats are Ceylon and portions of the Madras Presidency. According to Hamilton, the limits of the Indian *endemic* were the Malabar coast and that tract of country which extends from Madras to Ganjam, being confined to these parts, and extending no farther inland than forty miles. Later observation has considerably extended this endemic area, the limits of which it may have transgressed in recent times. Its ascertained *epidemic* range is exceedingly wide; its field occupying a very large expanse of the globe's surface—the South Pacific Ocean and its islands; the southern part of Australia and Mauritius in the southern hemisphere, and, in the northern hemisphere, as high as the Kasia hills, above the parallel of 25°. If we are to regard the epidemic which invaded Lower Bengal, in 1877, as true beriberi, as I think we must, the influence of this malady, when it becomes epidemic, may be expected to extend, in a plain country, as far as the sea breeze does, *i.e.* about 250 miles inland—Calcutta standing 110 miles, and Shillong some 140 miles from the sea. Both places suffered in the recent epidemic.

Beriberi is most common among natives, and it attacks half-castes. European soldiers have often suffered; their officers occasionally, but far less frequently. It spares neither age nor sex.

Hitherto the scientific definition of beriberi has always appeared to be one of the most obscure problems in ætiology. Probably feeling this, the College of Physicians have, in their nomenclature, placed beriberi next to general dropsy, but have refrained from giving it any definition. Although its manifestations are numerous

and complex, I think that, on carefully studying its phenomena, we cannot fail to arrive at the conclusion that true acute beriberi is not, as some have thought, a group of diseases, but a malady quite distinct from all others—a fever of varying type, but characterised by a very marked individuality. According to their opportunities of observing it, and to the type of disease with which each authority has been most familiar, authors have assigned various causes to this malady. Thus (1) Ranking held that it is renal anasarca; (2) Malcolmson viewed it mainly in its paraplegic and scorbutic aspects; (3) Morehead considered it to be an expression of scorbutic cachexia; while (4) others have regarded it as an outcome of paludal cachexia. Each of the leading authorities on the subject adduced very cogent facts in support of his opinion. I think, however, that an analysis of the characters of the epidemic disease which first appeared in Calcutta in 1877, and Mauritius in 1878, enables us to declare (5) that *acute beriberi is an exanthematous fever, hitherto best known by its sequelæ—acute general anasarca, and frequently but not invariably morbus Brightii and paraplegia.*

I shall review these five doctrines *seriatim*, save that I shall give precedence to the consideration of the fifth.

Fifth.—Acute Beriberi an Exanthematous Fever characterised by general dropsy, effusions into the serous sacs, and other grave sequelæ.

I have mentioned above that, in 1877, a disease having all the characters of acute beriberi made its appearance in Calcutta, and became rather extensively prevalent. No European is proved to have been affected. Eurasians and Armenians suffered. A very full account of this outbreak will be found in the 'Indian Medical Gazette,' *passim*, for 1880, as contributed by various good observers, who were not at all unanimous regarding the nature and origin of the malady. None but those practically well acquainted with beriberi in its homes can judge with absolute certainty whether we ought to agree with one or other of the only two Calcutta men who had seen beriberi—Dr. Coates, who had observed what was called beriberi in the Madras regiment at Cuttack, and who holds that the new Calcutta disease is not identical with beriberi; and Baboo Rammay Roy, who had recently

treated some 500 cases of veritable beriberi in the Madras district of Coimbatore, and had, since his return, seen some 150 cases of the new disease in and around Calcutta, who asserted that "they were identically the same malady." My own conviction, arrived at after a very full survey of the whole question, clearly is that the Calcutta disease was true acute beriberi.

In early accounts of beriberi we frequently meet with such entries as these: "The disease commenced with fever, followed by swelling and numbness of the feet and legs." "Had fever last night, skin dry, pulse excited." In the following quotation the italics are mine: "In detailing cases of beriberi, the cause, supposed or probable, should be first given, stating whether it was preceded by or followed any epidemic visitation of disease in the district or neighbourhood, such as cholera, *fever*, or dysentery; and also distinguishing particularly the *acute form of beriberi, attended with increased vascular excitement*, and coming on as a primary affection, from the chronic form which is frequently the sequela of rheumatism, *fevers*, &c."*

"Beriberi occurs both in an acute and chronic form, the acute form being usually attended by fever of an intermittent type, and of four or five days' duration, whereas the chronic form is more frequently the sequela of rheumatism or of fever, either of the intermittent or remittent form."†

In an abstract given by Malcolmson of nineteen cases of men, admitted with various diseases, who subsequently became the subjects of beriberi, five were admitted for "fever," and the eleven who were admitted for the four diseases—phlogosis, two; rheumatism, five; diarrhœa, one; anasarca, three—probably had commencing beriberi when they went into hospital. Malcolmson, and many who followed him, threw over this disease a darkness which has never yet been dispelled, by commencing their description of its symptoms at the beginning of the end. I cite Malcolmson's words (the italics are mine), "It (beriberi) usually *commences* gradually with a feeling of numbness, sense of weight and slight weakness and stiffness below the middle of the thighs." We might as well attempt to describe splenic cachexia by saying "it usually *commences* with a pain and sense of weight in the left side." They fell into the great error of not inquiring strictly into the preceding

* Editorial Remarks, 'Madras Medical Journal,' p. 472, vol. i, for 1839.

† 'Madras Medical Topographical Report,' Northern Division, p. 90.

fever. Doubtless their patients, not being properly questioned, did not enlighten them on this material point. Natives pay little attention to a single attack of fever, but usually seek relief when œdema and still graver symptoms occur. The severity of fever varies greatly in the exanthemata, and so does the amount and character of the exanthem, especially in the dark races. Judging by the light which the recent outbreaks in Bengal and Mauritius afford us, I am confident that Malcolmson and others erred in overlooking the fever and its exanthem, and by regarding the complications as the disease. In the epidemic which made its appearance in Calcutta in 1877, the initial fever and its exanthem were frequently overlooked, but it was generally noticed that the attack was ushered in by febrile symptoms of a few days' duration. It must be borne in mind that a malarious taint is to be suspected in the constitution of every Calcutta native; but it is clear that this fever was not the common paludal fever of Bengal. Many observers stated that there was no periodicity. Elsewhere, we read of evening exacerbations and morning remissions. In February, 1880, Dr. McLeod said that "fever sometimes preceded and sometimes succeeded the œdema, and sometimes did not occur at all. It seemed to be of quotidian type when present, and well-marked rigors ushered in the attacks."

Being a general practitioner, Dr. Chambers had an advantage over nearly all of those who observed the disease, in seeing cases from the commencement, while they, being consultants, were doubtless, in most instances, called in late. Indeed, Dr. Harvey remarked that, although he had seen many cases, he had not been able to have any one case under observation throughout. Dr. Chambers is the only reporter who says much of the cerebral conditions. He noticed marked head symptoms. He speaks of "intense frontal headache," and insomnia, and says that "when the disease is ushered in with great severity there is a tendency to stupor and wandering delirium, generally confined to one particular subject. The patient is, however, rarely insensible, but is always roused up from the stupor and delirium by being loudly spoken to; sometimes furious delirium and violence occur."

Frequent mention is made in the reports of an exanthem. Thus, in a girl expressly stated to have escaped fever, Tumez Khan says, "The limb swellings were stated to have been preceded by intoler-

able itching of the whole body, and the subsequent appearance of a rash all over it, and which, from the very vague description given, I think to have been of an urticarious kind." In 1879, Dr. Juggobuudhoo Bose had two cases in which "a redness of the skin, like scarlatina, preceded the swelling." In February, 1880, Dr. McLeod noticed, from large experience, that "the skin had been carefully examined, and in only one case was any abnormality found. This was a claret-coloured discoloration of the legs, disappearing on pressure." But he also stated, doubtless from the patients' own imperfect accounts of what had occurred to them, that the œdema "was preceded by burning and painful sensations, which seemed to be confined to the skin and soft parts." In a case of Dr. Nicholson's "there were purpuric spots." In March, 1880, Dr. Chambers, in describing the leading symptoms, mentions a "sensation of burning heat in the body, as though the skin were rubbed over with ground chillies, preceded or followed by repeated sensations of chill; a scarlet efflorescence from the beginning of the disease, especially confined to the face, neck, upper part of the thorax, and the extremities, or petechial spots with circumscribed measly eruptions on face or trunk, or large purpural or mulberry patches. In infants a marbly, mottled, purplish or red tinge of the skin, especially of the extremities, neck, and cheeks; a flushed puffy countenance, with suffused glistening eyes." These observations become very noteworthy when we are told by Dr. Lovell that in 1878—79, the Mauritius epidemic of "acute anæmic dropsy," as it was termed, was ushered in by "slight fever, and in most cases by a rubeolar skin eruption, disappearing under pressure, sometimes ending in petechiæ or phlyctenæ and desquamation." In Calcutta the fever was succeeded by general anasarca. The œdema was by some described as being "hard and brawny." It usually commenced in the lower limbs, sometimes in the hands, spreading more or less to the trunk and head. "Œdema," said Dr. McLeod, "was doubtless the most constant symptom." "The œdema," said Dr. Harvey, "is general, not only throughout the connective tissue of the muscles, but the connective tissue of solid and visceral organs, in every cavity of the body, is bathed in fluid."

Dr. Harvey noticed that "in two cases there was an appearance of paralysis of the lower limbs, as if from effusion of serum into the spinal canal, and in a good many more there has been great difficulty

in walking, apparently not due to paralysis, but to the local condition of the limbs."

Tumeez Khan's expression, that, in one of his cases, there was "inability of locomotion owing to excessive tension and stiffness of the lower extremities," appears to represent correctly the symptom in question in many cases. Dr. Chambers notices the occurrence of epistaxis in several instances. Capillary bronchitis was frequently present; pneumonic consolidation in some cases. Death commonly resulted from large, but apparently not inflammatory, effusions of clear serum into the pleuræ and pericardium. Consequently, there were palpitation and great præcordial distress and orthopnœa. Anæmic murmurs were frequently present. Dr. McLeod, speaking from large observation, stated that "death where it occurred would seem always to be sudden and associated with dyspnœa." Many early writers described the suddenness of death in beriberi. Malcolmson and McLeod use nearly the same words. The former wrote, "There is oppression and weight at præcordia, dyspnœa on slight exertion, diffused and irregular pulsation in the cardiac region." . . . . "The patient is often found dead in bed, or sinks after several fainting fits or throbbings of the heart." The urine was often noticed to be high coloured, scanty (not always), almost suppressed, but there was no albuminuria or other evidence of Bright's disease. In a solitary case, which I think goes for nothing, there was "a small quantity of albumen." In some cases the urine was of a very low specific gravity. Here and there a case is reported to have commenced or terminated with diarrhœa, or there was diarrhœa at the height of the disease, or dysentery later on; but, in most cases, there was constipation; in several agonizing pain in the lower bowel in defæcation. In many cases, in persons who were not sailors, the state of the gums gave evidence of scorbutus. In some the gums were healthy. In examining a considerable number of Bengalis we may always expect to find that some are scorbutic, but it does not appear to be shown that scurvy was generally present among these patients. Still, Dr. McLeod observed that "in a few cases the gums were rather swollen and bled when the teeth were cleaned." "Neither an anæmic nor scorbutic condition appeared to be an essential precursor of the malady, though a certain degree of both dyscrasiæ seemed to be developed in its progress."

Most of those who observed the disease at its outbreak, in 1877, met with many cases which did well. In 1880, there were numerous deaths. In February, Dr. Cayley reported four deaths, and two persons in a dangerous condition in one family. In that month, Dr. McLeod, Health Officer of Calcutta, said that the town returns showed 266 cases and 51 deaths, and the suburban 364 cases and 163 deaths. It was observed by Dr. Harvey and others that the disease was not confined to the poor and ill-fed; rich and well-to-do people, well-fed and living in well-raised and very dry houses, were quite as freely attacked as the poor, living in damp huts on the ground level. "The disease," says Dr. McLeod, "lasted for two or three months in well-developed cases." In the huge mass of evidence which I have analysed, I see no mention of the duration of very rapidly fatal cases, save in one, by Dr. Chambers, in which death appears to have occurred in three days.

There are only two reports of autopsies, made in February, 1880, by Tumeez Khan. The most remarkable appearances were general œdema. Integuments of the lower limbs "hard and brawny," not pitting on pressure, and loaded with effused sero-sanguinolent fluid. There do not appear to have been any head symptoms during life, and no marked cerebral lesions were discovered. Large effusions of limpid serum, without traces of inflammation, in the pleural and pericardial sacs. Considerable œdema and carnification of the lungs. Heart flabby, normal in size (in one or both cases?), fatty. No peritoneal effusion, no noteworthy renal lesion. In both of Tumeez Khan's cases, as in most of the other reported autopsies, there was marked hepatic congestion. Blood thin and watery, with a marked excess of white corpuscles.

Much was said about the contagiousness of this disease, but no evidence of contagion is given, beyond the frequent occurrence of several cases in one family—which, standing quite alone, is, of course, no valid proof of the agency of contagion—and one very suspicious case mentioned by Dr. Harvey. Dr. Chambers, who was one of my most esteemed pupils and house physicians, considered that this "new disease" was relapsing fever. He does not appear to have found support from any other observer. He shows that a tendency to relapse marked several of his cases; but I cannot think that his patients suffered from true relapsing fever. I think that he saw an exanthematous fever; but he appears to place his fever cases apart from those which he considered resembled beriberi.

This epidemic was observed at Dacca and Sylhet, and at Shillong in the Kasia hills.

There does not seem to have been any appearance of the disease in Calcutta after the hot weather of 1880.

Between November, 1878, and June of the following year a similar epidemic prevailed in Mauritius. It was stated to have caused 729 deaths. Dr. Lovell, the chief medical officer, sent a report on this disease, which he termed "acute anæmic dropsy," to the Calcutta Medical Society, in July, 1880. I have already spoken of the exanthem which generally characterised this malady. In the worst cases, there were hydro-pericardium, hydrothorax, and ascites. Signs of scorbutus are not mentioned. "Europeans were entirely exempt." "The patients, generally Indians, were very anæmic, the blood watery, with marked diminution of the red blood-cells, and increase of leucocytes and granules." "The urine was seldom albuminous." Enlargement of the spleen was not found to be a feature of the disease. Opinions of medical men were divided regarding the contagiousness of this malady; the majority were opposed to the view of contagion.

A report which appeared in the first volume of the 'Madras Quarterly Medical Journal,' page 70, shows that, late in the year 1829, beriberi, which had previously been unknown in that locality, attacked the Madras sepoys at Singapore, while "*the convicts remained without a single patient with the complaint.*" True beriberi prevailed in the criminal prison at Singapore in 1875–76–78–79, and 80. *It did not spread among the community at large.* No European or native female prisoner suffered. In 1878, the death-rate was 16·20 per cent. of admissions, and rose to 20·63 in 1879. There is no mention of fever or eruption. Rheumatic pains and paraplegia were frequent symptoms. The great serous cavities, peritonæum, pleuræ, pericardium, were filled with serum. Scorbutic symptoms appeared in some cases. *The urine was scanty, high coloured and albuminous.* In a leading article in the 'Indian Medical Gazette,' for September, 1880, Dr. McLeod observed that "as regards symptoms and mortality, the Calcutta disease presented a sort of transition between the pure beriberi of Singapore, with rheumatic and paralytic phenomena and albuminous urine, and the acute dropsy of Mauritius, which presented many of the features of a contagious exanthem."

I must repeat that my own inference from the evidence is that

the "new disease," as it occurred in Bengal and Mauritius, was an exanthematous fever. Its mode of invasion, when epidemic, would alone declare beriberi to be a fever. No one who has studied the natural history of fever will believe that the epidemic disease which, as we have seen, invaded Lower Bengal in 1877, and Mauritius in the following year, was either paraplegia or scorbutus, or consider that it was renal anasarca.

First.—Beriberi regarded as Renal Anasarca.

In a valuable but very rare volume,* 'The Proceedings of the Hyderabad Medical and Physical Society,' Mr. J. L. Ranking published, in 1853, a series of reports, tending to show that the previously obscure disease, beriberi, depended mainly upon the existence of a form of *morbus Brightii*. Mr. Ranking said that his limited experience led him to adopt the opinion that beriberi is primarily and essentially a renal disease; that deterioration of the blood is caused by lesion of the kidney, and that the "numbness," staggering gait, or more confirmed paralysis of motion (pointing to affection of the spinal cord), the "irritable" condition of the heart, the endo- or pericardial "inflammation" [?] and effusion (which latter is generally the immediate cause of death) are secondary affections. He showed that, in sixteen cases of beriberi, the urine was more or less albuminous in eleven; in eight, the microscopic elements characteristic of kidney disease existed. Microscopical examination was made in these eight cases only; so that, in every case of well-marked beriberi which fell under Mr. Ranking's notice, the microscopic evidences of kidney disease were discovered when sought for. In nine, there was general anasarca; in one, the anasarca existed with ascites; in two, there was delirium; in one, coma; in fourteen, numbness without actual anæsthesia; in two, impaired sensibility with numbness; in six, there was more or less loss of motor power; in seven, symptoms of pericardial and endocardial affection; in five, signs of œdema of the lungs; in two, hepatitis; in two, nausea and vomiting; in two, diarrhœa. It was noticed by Malcolmson that there were certain points of resemblance between

* I only know of one copy, possessed by my friend Dr. Waring, C.I.E. I gave extracts from its contents in the "Bibliographical Record" attached to No. 2 of the 'Indian Annals of Medical Science' in 1854.

beriberi and *morbus Brightii*. This resemblance forced itself upon Mr. Ranking's attention before he had access to Mr. Malcolmson's work. Upon commencing the examination of the urine of all beriberi patients, it was found that, although the quantity excreted, the reaction, specific gravity, colour, &c., differed according to the stage and form of the affection, the microscope revealed the presence of blood-discs and epithelial scales. In two cases there were also tube casts, and mucous and organic globules. The urine was often coagulable by heat and nitric acid, though never to a high degree albuminous. He represented that, in the earlier stages of beriberi, the state of the urine points only to simple congestion or sub-acute inflammation of the kidneys. He found it to have a deep red colour, owing partly to the admixture of blood, partly to concentration; a specific gravity at or somewhat above the natural standard. A deposit of lithic acid and lithate of ammonia sometimes occurred, and abundant and large crystals of lithic acid. These morbid conditions of the urine will, Mr. Ranking considered, be found to precede the anascarca, numbness, and other symptoms of spinal affection. Unless the state of renal congestion, or sub-acute inflammation, upon which it depends, can be overcome, the anascarca continues, effusion "into the spinal cord" [?] takes place, the heart becomes involved, and effusion into the pericardium and pulmonary œdema carry off the patient. The urine remains scanty, in such cases, to the end, or is even totally suppressed. In more chronic cases, or in individuals who have struggled through the acute stage, the urine was found to alter in character—it increased in quantity, but decreased in density. It seldom, however, reached the healthy standard even as to quantity—it acquired a pale opalescent colour, had an alkaline reaction, a specific gravity of 1010, and coagulated more or less by nitric acid and heat. "Here," said the author, "we have all the characters of the urine of 'Bright's kidney.'" "The alkaline reaction is due doubtless to the existing lesion of the spinal cord which, although in itself a secondary affection, reacts upon the urinary organs, the mucous membrane of the bladder especially, causing an increased secretion of mucus, or even pus, and consequent alkalinity of the urine. The microscope still reveals blood-discs; a great abundance of epithelial scales with mucus or blood-globules entangling crystals of triple phosphate, or, if any deposit occurs, it is made up of mono-basic or bi-basic phosphates, amorphous phosphate of lime and mucus."

Others who observed beriberi nearly at the same time, noticed the renal complication.

In the middle of August, 1852 (dates are self-evidently of great importance in the correlation of cases of beriberi, as showing agreement or difference in type in the same or in other epidemics of the disease), Dr. George Mackay* admitted to hospital, at Rangoon, a Madras sepoy, who died, in about twenty-six days after sickening, with typical symptoms of beriberi. "The urine presented a copious deposit of mucus, with altered blood-discs and crystals of the triple phosphate." Many other cases of a nature similar to this came under Dr. Mackay's notice during the same (second Burmese) war, but none in which the symptoms so nearly resembled those of beriberi. His attention in those cases was early directed to the state of the urinary organs. "Epithelial scales, altered blood-discs, crystals of the triple phosphate being almost invariably met with; in some cases oxalate of lime crystals, and in one or two tubular casts were observed."

The 'Hyderabad Proceedings,' in which Mr. Ranking's paper appeared, also published a case by Mr. Maillardet. Dr. Van Someren also discovered "casts of uriniferous tubes, epithelial scales, altered blood-discs, and octohedral crystals of the oxalate of lime." We have here descriptions, by three observers, of the conditions of the urine in (epidemic ?) beriberi as the disease presented itself within a short space of time—apparently a period of a few months —on the continents of Madras and Burmah. Although the above facts, being published in the 'Indian Annals of Medical Science,' must have come before many observers in India, I do not find that Ranking, Van Someren, Mackay, or anyone else recorded other cases of albuminuria, cast desquamation, &c., in beriberi. Was this because, during that particular epidemic and that one only, the kidneys suffered nearly as they do in the cognate but very distinct (from epidemic acute beriberi) exanthematous fever scarlatina? At any rate, regarding beriberi as a fever of this genus I am quite prepared to find that, when the exanthem is checked by the "chill," so frequently mentioned as a prominent exciting cause in this disease, albuminuria may occur. It is clear, however, that albuminuria was not a marked feature in the late Bengal and Mauritius epidemic, although it is to be regretted that none of the reports

* 'Indian Annals of Medical Science,' No. 5, p. 349.

which I have seen take any notice of the microscopic condition of the urine.

It will have been noticed that in the Singapore outbreak, which commenced in 1875, "the urine was scanty, high coloured, and albuminous."

Undoubtedly the incidence of beriberi is especially directed to those who are the subjects of renal disease. It has always been observed that a very considerable proportion of the victims of this disease are drunkards. Intemperate European soldiers are liable to this malady; their officers are far less so, although they have not always escaped. Christie says, in his report on beriberi: "I have remarked that a very great proportion of the patients seized with this disease were men accustomed to lead a sedentary and debauched life, such as tailors, shoemakers, &c., who, when working at their trade, are often excused the duty of the field, and by their double earnings are enabled to procure a larger quantity of spirits than the other men." He adds, "I have never met with an instance of this complaint in a woman, an officer, or a boy under twenty." I was told at Aden, about ten years ago, that European soldiers posted there, who are temperate, generally escape; but that, when a man becomes a sergeant, he is not unlikely to take to free-living, and then probably is attacked with beriberi. At the same time it is clear that, while beriberi sometimes resembles Bright's disease it by no means always assumes that character. Certainly it is not produced by the ordinary causes of renal anasarca, and, if some of the cases of beriberi, such as those observed by Ranking, Mackay, and others, may be designated as examples of this form of general anasarca, ordinary renal anasarca is in no sense beriberi. The paraplegic element of beriberi is not characteristic of common *morbus Brightii.*

Second.—Beriberi viewed in its Paraplegic aspect.

One of the leading sequelæ of beriberi being anasarcous and dropsical effusion into the areolar tissue and serous cavities, the occurrence of spinal paralysis, in various degrees, as one of its complications, is clearly accounted for. As I have shown, in describing the Bengal epidemic, the stiffness of the lower limbs is frequently due to the presence of hard œdema, not to spinal

paralysis; but, in many cases of beriberi, distinct impairment of motor and sensory power in the lower limbs has been observed.

In an abstract of Malcolmson's prize essay on beriberi, Sir G. Ballingall says: "In dissecting patients who have died of this disease, serous effusions, more or less extensive, into the cellular substance, as well as into the great cavities, would seem to be the more prominent appearances; and, in addition to this serous effusion, there often exist in the lower region of the spine, about the origin of the lumbar and sacral nerves, symptoms of congestion; and in one very remarkable case, of which the details are given by Mr. Malcolmson with an illustrative engraving, an effusion of reddish coagulable lymph had taken place on the posterior surface of the theca, at the fourth dorsal vertebra, and the same in the region of the sacrum."

Although the form of chronic paraplegia, termed by Bontius and other old writers on the diseases of the East, "barbiers," is, as shown by Henry Marshall, quite distinct from beriberi, it can hardly be doubted that much of that which was formerly regarded as barbiers was, in reality, beriberi with spinal paralysis.

Lathyrism, paralysis of the lower limbs caused by habitual feeding upon the *dâl* of the *Lathyrus sativus*, prevails extensively in Upper and Central India, especially near Allahabad and in Upper Scinde. M. Proust has observed it in the Jurjura mountains of Algeria, and it is stated to be far from uncommon in France. A recent writer having stated that lathyrism and beriberi are identical diseases, M. Pierre Marie* has undertaken to prove that such is not the case. It appears to me not improbable that the writers have confused beriberi with barbiers. At p. 113 of the 'Indian Annals of Medical Science,' No. 23, Dr. James Irving has contrasted the symptoms and other conditions of lathyrism and barbiers. I should have considered beriberi and lathyrism as little congeneric as Monmouth and Macedon—beriberi being a very acute disease, lathyrism, as it occurs in India, a chronic one. But, since the question has arisen, M. Marie's paper calls for notice from medical men working in those districts, far separated as they are, in which lathyrism and beriberi are most common; lathyrism prevailing far inland beriberi occurring almost entirely within the influence of the sea breeze. It appears singular that, in giving long and minute

* 'Progrès Médical,' No. 43. An abstract of this paper is given at p. 606, 'Medical Times and Gazette,' vol. ii, for 1883.

descriptions of the conditions of the lower extremities in the contrasted diseases, M. Marie appears to have overlooked œdema in beriberi.

The loss of power in the lower limbs, so frequently noticed in beriberi, appears to need clear definition. In what cases is it dependent upon spinal lesion, and in what others is it due to hard œdema of the extremities and debility? With the regard to the former cases, one point ought to be cleared. Sir Joseph Fayrer has suggested the probability that filarious disease may be in some way concerned in inducing beriberi.* A form of spinal paralysis in the horse, *kumree*, which prevails within the Madras area of beriberi, and in Eastern Bengal, has been ascribed by Twining and others† to the presence of entozoa in the cord. Do spinal lesions in beriberi ever arise from this cause?

Undoubtedly, although spinal paralysis is a recognised sequel of beriberi, it cannot be considered that beriberi is, in any sense, paraplegia.

Third.—Beriberi regarded as an expression of Scorbutic Cachexia.

Marked evidences of scorbutus are so often noticeable as prominent symptoms in cases of beriberi, that at least one of our best Indian observers has considered that beriberi is essentially a form of scurvy.

Wishing to give fair play to this, as well as to all the other leading theories upon the causation of beriberi, I may cite the following rather striking example of this coincidence, as observed by Tumeez Khan, in Calcutta, in 1878, and others which I find in the 'India Journal of Medical Science,' vol. v, N. S., p. 75, as occurring *circa* 1839 on a passage from Java.

1839.	1878.
A Dutch barque arrived in the Madras Roads, from Java, on the 12th May, having left Malacca, where she lay only three or four days, all being well, on the 7th April. The crew were Javanese, who had made objections about coming to the Coromandel	A Mahomedan ship-captain of one of the Arab sailing vessels which ply between Calcutta and Jeddah, a native of some part of the Coromandel coast, his son, and a third member of his family—both said that they had returned hale and hearty from a trip to

* 'Quain's Dictionary,' p. 105.

† Vide 'Calcutta Med. and Phys. Trans.,' vol. i.

coast, as they said "many of their countrymen had died of a similar disease on former voyages."

"About twenty days before reaching Madras, many of the crew were seized with swellings and numbness of the legs and feet, but yet were able to perform their duty; when they arrived within 50 or 60 miles of the coast, and within the influence of the land winds, in three cases the symptoms became much aggravated, they were unable to walk, dyspnœa and pain in the præcordia supervened, and they died the day after the ship arrived.

"They were a wretched looking set, and were very badly fed, their rations consisting only of rice, and of that a very limited quantity. The water was bad, and they were on very short allowance."

Jeddah, the third person had died previous to Tumeez Kahn's visit.

A short time after their arrival in Calcutta "both were nearly simultaneously attacked with febrile symptoms, which lasted for a few days, and were followed by œdema of the lower limbs, to which succeeded cough and dyspnœa" (the son had hæmoptysis). The "œdema extended up to the loins." When visited they had "orthopnœa, palpitation, and inability to lie down." It is not said whether the father and son died, but they probably did.

"Both had spongy and discoloured gums."

In connection with the above observations, all who are desirous to investigate the nature of beriberi ought to study Morehead's chapter on this disease. His data are mainly derived from histories of "beriberi as it occurred among the Lascar crew of the ship *Faize Allum*, in May, 1853, when in lat. 10° north, within sixteen days' sail of Bombay; and from an account of an outbreak of 'epidemic ascites,' or 'peritonæal dropsy,' in thirty of the crew of H.M.S. *Juno*, on her arrival at Sydney from a lengthened cruise among the islands of the Pacific, in January, 1856."

The native subjects, four cases of beriberi, from the ship *Faize Allum*, whom Morehead treated, were doubtless scorbutic; the men of the *Juno* were probably so. Morehead does not appear to have met with any other cases of beriberi. He considered that "beriberi, more particularly in its acute form, occurs usually in persons favorably circumstanced for the development of a scorbutic taint, and subsequently exposed to cold, dry or moist winds, or to lying on the ground wet with rain or dew." With the judicious circumspection which characterises all his reasoning, he observes that to him "it seems that beriberi is a general dropsy, and that, in regard to each instance, the question ought to be—what is the pathology of this case of general dropsy?" He adds, "generally it will be

found that a scorbutic diathesis and external cold or wet, are the determining conditions.* Dr. Peet adopts this view without qualification. He says, "When individuals who are scorbutic are exposed to cold and alternations of temperature, they are not unfrequently attacked with general dropsy. It has been customary to designate the disease now referred to as beriberi."† Here is, I think, another example of the common error of regarding as cause and effect two circumstances which are merely associated as frequent coincidences. Is is to be borne in mind that beriberi is often epidemic, which scorbutus cannot possibly become. There was no beriberi among the scorbutic mariners of last century; and in many cases of sea scurvy, which I have treated, there was not the very slightest evidence of its existence. Beriberi occurs in some localities notorious for scurvy, as Aden; but it does not prevail in certain of those districts of Hindustan—as the province of Behar, and in Scinde—in which scurvy is extremely prevalent. Scorbutus, as we have already seen, occurs in Lower Bengal, but not so extensively as in some other Indian districts. The recent Bengal epidemic frequently attacked the scorbutic, but it was noticed that many of the sufferers from the epidemic were free from evidences of scorbutus. I think that when we state the fact that wherever beriberi prevails it especially selects the scurvied, we are free from the error of supposing that acute beriberi is an expression of scorbutic cachexia, although scorbutus should be looked for in all cases of beriberi, and be treated, when present, as a grave complication.

Fourth.—Is Beriberi, as some have considered, a form of Malarious Cachexia?

I think that this question must be answered in the negative. Beriberi prevails in localities, such as Aden and Mauritius, where the paludal poison does not abound in great intensity. Until 1877, the Delta of the Ganges, that hot-bed of marsh influence, was never visited by this pest as an epidemic, and has never generated endemic beriberi. I believe that up to the time of my leaving Bengal, eight years ago, it was never suggested that beriberi could originate there. Considering that a vast number of sailors pass through the Calcutta hospitals, I think it noteworthy that only one

* Op. cit., pp. 707-8,

† 'Principles and Practice of Medicine,' p. 570.

not unquestionable case of this disease, in a half-caste seafaring-man, made its appearance in my wards in fifteen years. No one acquainted with the natural history of the marsh poison would, in studying the outbreaks of beriberi in Calcutta and Mauritius in 1877 and the following year, consider that there could have been any interchange of paludal influence between these two widely separated localities. Nevertheless, seeing how generally and how often gravely prevalent malarious cachexia is in most of the regions which beriberi visits, the necessity of searching for it, as a most serious complication, must never be overlooked. Few constitutional states can be regarded as more perilous than that of the multitudes of unfortunates who, being the subjects of the combined cachexiæ of marsh-poison and scorbutus, are attacked with beriberi.

I think that we are justified in concluding that beriberi is not a malarial fever, but that the victims of malarious cachexia are especially liable to suffer from its attacks.

Treatment.—If I am right in the firm belief which I hold that true beriberi is an exanthematous fever which, although wholly *sui generis*, pursues a course which, in more than one of its leading actions, much resembles that of scarlatina, it is evident that here our first object should be, as it is in scarlatina, to endeavour to see each case *ab initio*, and to prevent the occurrence of renal and other visceral mischief, anasarca and dropsical effusions into the serous cavities. If it could be my duty to have charge of military and jail hospitals within the endemic area of beriberi, every European soldier, sepoy, and prisoner, would be warned to seek relief from my assistants *immediately upon being attacked with even the very slightest symptom of fever.* I would myself examine all fever cases early, within twelve hours if possible, carefully searching for the exanthem on the lighter skins, and considering that, if I frequently discovered it there, its presence might be suspected in those of darker complexion. Then the chief indications would be :—To admit all fever cases as in-patients ; to avoid the occurrence of that CHILL so frequently assigned as the cause of general anasarca in beriberi; to promote healthy cutaneous action and to subdue fever; to discover and deal with pre-existing renal and other visceral disease, and with malarial and scorbutic cachexia. After remaining for two or three days under observation, the cases of ordinary intermittent

fever would explain themselves, and would then demand less close attention. When patients do not seek relief until general anasarca has become established and, being too weak to go about, are desirous to come to the hospital as a refuge, the same principles of treatment ought to guide us. With the deepest respect for the memory of a great Indian physician, I cannot agree in the validity of his maxim, guarded as it is, "remove the dropsy by purgatives or diuretics, being guided to the use of the one or the other by the state of the pulse." I would no more give direct diuretics in the anasarca of beriberi than I wonld in that of scarlatina. My great effort would be to obviate renal congestion in all possible ways, and to make the work of the kidneys as easy as possible. I would not give anything more drastic than the mildest laxatives in these cases, liable as they are to end fatally by diarrhœa or dysentery. If we can only remove the other complications of beriberi, the anasarca will undoubtedly disappear as constitutional power becomes re-established. No purgative or diuretic will, except by promoting exhaustion, touch a pericardial, arachnoid, or pleural effusion which threatens life.

I believe that, fifty years hence, the futile and destructive practice of endeavouring to remove dropsical effusions by the use of drastics and direct diuretics will be viewed as one of the most barbarous therapeutic errors of the present century.

I think that the whole subject of acute and chronic beriberi, especially as they occur in the Madras Presidency and in Ceylon, ought to be thoroughly reinvestigated by sound pathologists. Work out the subject of beriberi is one of the best pieces of advice that can be given to a young physician entering upon a career in the Madras Presidency.

The field of observation is large. In 1881, there were 102 cases of beriberi, with twenty deaths, among the native troops in the Madras Presidency, in a strength of 30,394. No cases occurred in the Bengal and Bombay Presidencies.

Especial care should be taken to ascertain the precise nature of what is termed the "chronic" form of beriberi. Are these cases of malarious and scorbutic cachexia, not allied to true beriberi, or are they examples of repeated attacks of beriberi and consequent ill-health? Christie observed that "men who have once had the complaint are the most subject to it in future." In the epidemic and acute form, light is needed with regard to the

precise type of the fever, the character of the exanthem, the range of temperature, the chemical and microscopical conditions of the urine, &c. All observers have noticed that, although the œdema of the lower limbs is hard, the serous effusions, arachnoid, pericardial, pleural, and peritonæal, the pressure of which tends to cause death in beriberi, are distinctly *non-inflammatory*. A little sifting would clear this question, enabling us to judge whether we ought or ought not to draw off the fluid from the pleuræ and pericardium, and with what prospect of success.

The PRESIDENT said he had listened with great interest to the paper.

Sir JOSEPH FAYRER had regarded beriberi as a condition of profound cachexia rather than a specific disease. Beriberi signified sheep, and the resemblance to the gait of a sheep was marked in the disease. Many authors had thrown doubts on the real origin of the term. There is an Arabic word very like beriberi which signified weakness, and again in Singalese a similar term existed. All the terms pointed to a condition of weakness and exhaustion. Notwithstanding what he had written, he was now inclined to accept Dr. Chevers' conclusions. So far as he remembered, the epidemic which began at Singapore finally spread over the Malay Peninsula. In Singapore the insanitary state had a decided influence in producing the disease and in increasing the rate of mortality. In one house built over an alluvial soil and badly ventilated the mortality was certainly remarkable.

Dr. GORDON handed round a report of the disease as it appeared in Japan, where it is known as "kakké." The mode of spread of the disease was touched upon. In an archipelago the inhabitants of some islands were exempt from the disease, though it was generally prevalent in the archipelago. In Japan the natives chiefly suffer, whereas in India the foreigners are more liable to it. Dr. Simmons showed that those who lived in stone houses were largely affected. "Triakferouk" (the Persian name for Venice treacle) was a secret remedy for the disease; and Malcolmson advocates this medicine, which appears to contain a *powder of vipers* (London Pharmacopœia, 1746). Democritus, the physician of the Emperor Nero, seems to have employed this Venice treacle.

Mr. PALM said his own experience was almost confined to the chronic form of the disease. The œdema was then not generalised, but limited to the fronts of the tibiæ. He was inclined to look on the disease as primarily one of the spinal cord. The disease had been known in Japan for at least 100 years. The Japanese method of squatting might play a part in the etiology of the disease. It was a rare thing for Japanese to die of the wet form of beriberi.

Sir W. GUYER HUNTER had never seen beriberi in an endemic or epidemic form. He thought the malady was the result of malarial cachexia and scorbutus combined. He had not met with the dry form of the disease. The paralysis was looked upon as of mechanical origin and due to the large amount of water in the lower extremities. Anasarca was of universal distribution.

Dr. NORMAN CHEVERS, in reply, maintained the positions he had advocated in the paper.

The Death of H.R.H. Prince Leopold K.G.

At the close of the Ordinary Meeting of the Society on March 31st, 1884, the following resolution was proposed by the President, Arthur E. Durham, Esq., seconded by Sir Joseph Fayrer, K.C.S.I., M.D., and carried in silence:

"That the Fellows of the Medical Society of London desire to express their sincerest and deepest condolence with Her Majesty the Queen and with Her Royal Highness the Duchess of Albany on the death of His Royal Highness the Duke of Albany."

This resolution was forwarded by the President and Secretaries, and the following reply was received on April 24th:

Whitehall; 24*th April*, 1884.

Sir,—I have had the honour to lay before the Queen the Resolution which has been adopted at a Meeting of the Fellows of the Medical Society of London on the occasion of the death of His Royal Highness Prince Leopold, the Duke of Albany, K.G. And I have the satisfaction to inform you that Her Majesty was pleased to receive the same very graciously.

I am, Sir,

Your obedient servant,

W. V. Harcourt.

April 7th, 1884.

ON COMPENSATORY HYPERTROPHY OF VARIOUS ORGANS.

By Joseph Coats, M.D.

The subject on which I venture to address you to-night is that of compensatory hypertrophy. I have chosen it chiefly because, while in regard to some organs this condition has received a well-recognised place in medical science, yet in the case of other organs where its occurrence may possibly be not infrequent, it has hardly received any attention. In order to clear the ground it will be

necessary to define exactly what is meant by hypertrophy and compensatory hypertrophy.

I would define hypertrophy as the new formation of tissue which, in structure and function, is essentially similar to the normal tissue of its own kind, and which forms an addition to the existing active tissue of that kind. When we call the hypertrophy compensatory, we mean that the new tissue has been called into existence to supply some defect; the existing tissue has been insufficient to perform the physiological functions required of it, and new tissue has been formed to supplement it. The necessity for this increase in the amount of a particular tissue may arise in two ways; in the first place, there may be from atrophy or otherwise an actual loss of tissue and new tissue is formed to bring it up to the normal amount; or, in the second place, the circumstances of the individual or of the organ may be such as to call for an excessive exercise of some particular function, and, in relation to the increased need, the existing tissue is defective in amount. In this case the new tissue constitutes by so much an absolute excess over the average normal amount.

The first of these two kinds may appear to come most strictly under our definition, but, as in most cases there is a defect relatively to the work to be done, and as the process by which this defect is made up seems to be in both cases the same, we may properly include them under the same category. This applies especially to the case of the heart, where the great majority of cases ordinarily grouped under the designation compensatory hypertrophy, really belong to the second class.

It is here necessary to refer to the distinction which has been drawn by Virchow between hypertrophy and hyperplasia. Hypertrophy in this sense includes cases of absolute increase in size of the tissue elements, while hyperplasia means a numerical increase in the elements. In actual cases it is impossible always to keep up this distinction, and although it is important to bear in mind these two different ways in which tissues increase in bulk, yet it is more convenient to include both conditions under the common term hypertrophy, using the term hyperplasia when necessary to designate a numerical hypertrophy. The essential thing is that new tissue is formed capable of normal function, and it is not so important whether this occurs by enlargement of the existing elements or the formation of new ones.

In hypertrophy there is usually a fresh start in the growth of the tissue concerned, and it will be useful at the outset to consider its relation to normal growth. I suppose that it must remain a mystery that in the growth of the body the new formation of tissue stops at the particular points at which it does in each person. Taking the skeleton as the fundamental structure, we do not know how it is that the bones grow to particular sizes and shapes, and stop growing just at the time at which they do. We can only speak in this regard of inherited powers inherent in the tissues and in the body as a whole. While this is entirely obscure, I think we may say that in regard to certain of the organs their growth is to some extent proportioned to the needs of the organism, and that the actual size attained by these organs is, partly at least, related to the amount of function which they are called upon to perform. The size of the lungs in different persons is not altogether dependent on inheritance, but is, to a certain extent, dependent on the exercise of the respiratory function during the period of growth. The same may be said with safety in regard to the muscles, and we may infer that it is so also in regard to the liver, kidneys, and other glands. In other words, the new-formation of tissue in these organs does not cease at the end of growth entirely because the inherent stimulus which the embryo possessed has exhausted itself, but also because sufficient tissue has been produced to fulfil the function of the organs concerned.

The proof of this lies in these two facts, namely, that during the period of growth, should occasion arise for the excessive performance of a particular function, the required excess of tissue is readily produced, and still more that, even after the completion of the period of growth, if necessity arises for the increased performance of the function, new formation of tissue in many cases begins anew, and that by a process perfectly analogous to the original growth. It is this increased or renewed growth which we distinguish as compensatory hypertrophy. It will be remarked, therefore, that the process is not to be compared with development, but with growth. There is not here anything comparable with the restoration of lost parts, but simply an overgrowth of existing tissue. In order to this renewed growth an important requisite is that the general nutrition of the body should not be seriously defective. As defective general nutrition interferes with normal growth, so, but to a larger extent, does it interfere with this compensatory growth.

From what has been said it will appear that compensatory hypertrophy will occur most readily during the period of growth, where it will be effected by an increase in a perfectly normal process. After the completion of growth, however, the renewal of the process is a more serious matter, and we may expect that it will not be so readily effected.

In studying compensatory hypertrophy of the individual organs we have to distinguish the two forms already alluded to, namely, those in which, from an excessive performance of a normal function, there is an absolute enlargement, and secondly, those in which, from a partial loss or destruction of the tissue, there is an excessive growth in order to make up the tissue to the normal.

Compensatory hypertrophy of the *heart* is perhaps the commonest and most generally recognised example of this condition. It may perhaps be said that all hypertrophies of the heart are compensatory; they arise in consequence of the muscle of the heart being called on to exercise more forcible contraction than normally. In the vast majority of cases this does not arise from any defect in a portion of the muscular apparatus, from any loss of muscular substance which the remaining muscle has to make up, but rather from an absolute increase in the amount of work required of the heart. In relation to the work to be done the muscle is defective, and so a new formation occurs so as to fulfil the requirements, the result being that the total amount of muscle produced is in excess of the normal.

There are, however, a few cases in which the hypertrophy presumably arises, in part at least, from loss of tissue, from actual defect in the amount of existing muscle. As a result of obstruction of the coronary artery in its finer branches (a somewhat frequent lesion), or of embolism in these vessels, there may be an infarction with necrosis of the muscular substance, and the piece of dead muscle is ultimately absorbed, a cicatrix taking its place. Later on a tendinous structure is found interrupting the muscular substance, and to this extent there is an actual defect. Under these circumstances the surrounding muscle may undergo hypertrophy so as to bring up the sum of muscular tissue to the normal. Even in this somewhat unusual case, however, the problem may hardly be so simple as I have stated it. Besides the mere loss of substance, the tendon in the midst of the muscular substance may derange the action of

the muscle, and it may be that the hypertrophy is rather the result of deranged action such as I shall mention by-and-by. The case is similarly complicated when, in consequence of pericarditis resulting in adherent pericardium, the external layers of the muscular substance are encroached upon by new-formed connective tissue. The resulting hypertrophy is not altogether or even chiefly due to absolute defect in the amount of existing muscle.

Leaving aside these somewhat complicated cases, we may divide the cases of hypertrophy of the heart into two groups. In the one group an undue strain is put on the circulation, calling for an habitual over-exertion of the heart's muscle, and in the other there is some defect in the circulatory apparatus, generally in the heart itself, which necessitates increased force in the muscle of the heart in order to carry on the circulation.

In all severe muscular exertions, as in climbing a hill, there is a demand for a rapid renewal of blood in the working muscles and in the lungs, and the heart has to work harder, its contractions being both more frequent and more forcible. The heart in its normal state is capable of meeting an occasional demand for increased exertion, but when this is frequently repeated at short intervals and the heart is not allowed to recover itself, then, if the heart is to succeed in carrying on the circulation, it must hypertrophy. The immediate effect of overstrain of the heart is to cause the blood to accumulate in the cavities, especially the ventricles, whose contractions are not sufficient fully to empty them. Dilatation of these cavities is the result, and an increase in their muscular substance is needed in order that they may deal with the excess of blood. This kind of hypertrophy seems to have been frequently met with in soldiers, and it may apparently be of comparatively rapid development. Fräntzel,* a surgeon in the Prussian army, refers to hypertrophy occurring in the course of campaigns in which there are frequent and long marches within a limited time. In the Danish war of 1864 there were few such rapid marches, and the Austro-Prussian war of 1866 lasted such a short time that there was not time for any prolonged strain. This author accordingly found that few cases of hypertrophy and dilatation of the heart occurred in these campaigns. In the Franco-Prussian war of 1870, however, when the Prussian battalions were called upon to make excessive exertions, sometimes marching with little intermission for a week, these con-

* Fräntzel, 'Virchow's Arch.,' vol. lvii, p. 215.

ditions were much more frequent. In one battalion alone he found fifteen cases of simple hypertrophy and dilatation. Among these there were ten cases of hypertrophy of the left ventricle, two of the right and two of both.

Several British army surgeons have called attention to the frequency of hypertrophy and dilatation of the heart, especially in young recruits, and have related it to the constraint of the chest which the clothing and accoutrements produce, while the soldier is going through the somewhat severe exertion of drill. Dr. Maclean* has frequently called attention to this matter. Myers† also, in a special treatise, strongly expresses the opinion that the accoutrements of the British soldier are a serious cause of simple hypertrophy and dilatation of the heart. The young recruit has frequent attacks of palpitation at drill, so much so that the "irritable heart" is quite a recognised condition, implying a frequently recurring overstrain of the heart, with dilatation. Such cases may recover with hypertrophy which completely compensates, but in some the frequently repeated over-dilatation leads to incompetency of the valves and the usual results of cardiac disease. As confirmatory of these views Dr. Maclean calls attention to the rarity of rheumatism as a cause of valvular disease in the army. In 151 cases taken from the records of Netley Hospital he found that only in six had the patients ever suffered from acute rheumatism. Dr. Parkes, at the request of Dr. Maclean, examined all the patients at a given time in Netley who suffered from heart disease. There were seventy of these, and in only two or three was there a history of any previous illness at all.

Peacock,‡ in considering the causes of heart disease, refers to the case of the Cornish miners. He believes that mitral disease with hypertrophy is a frequent result of overstrain of the heart. These workmen have (or had when Peacock wrote) to return to the surface after their work is done, by ladders. After working in foul air for the day, they have an hour's climbing to reach the surface. In such persons there will be prolonged dilatation of the right ventricle and general engorgement of the circulation with resulting dilatation and hypertrophy of both ventricles. For a time the

* Maclean, 'British Medical Journal,' Feb. 16th, 1867, &c.

† Myers, 'On Diseases of the Heart among Soldiers,' 1870.

‡ Peacock, 'On Some of the Causes and Effects of Valvular Disease of the Heart.' London, 1865.

hypertrophy may compensate, and the circulation approach to the normal, but through time the mitral valve becomes incompetent in consequence of dilatation of the left ventricle and the regular train of symptoms of mitral disease sets in.

In a somewhat interesting paper, entitled "Das Tübinger Herz," Münzinger* refers to the great frequency of heart disease in the town of Tübingen and district. He accounts for it by the laborious nature of the employment of the workpeople, who are mostly their own carriers, both men and women. As the country is hilly and the employment chiefly tending the vineyards, the loads the people carry cause frequent overstraining of the heart resulting in dilatation and hypertrophy. The hypertrophy may compensate, but frequently it fails to do so, and by-and-by the dilatation induces mitral incompetency. Jürgensen adds a note to Münzinger's paper, in which he says that on first coming to Tübingen he was struck by the number of cases of simple hypertrophy of the heart, and he fully agrees with Münzinger that the explanation is to be found in the laborious character of the occupations of the people.

It is not necessary to do more than refer to the cases of hypertrophy of the heart which result from derangements of the circulation. The whole series of hypertrophies resulting from valvular disease on the one hand, and impediments in the systemic or pulmonary circulation on the other, are familiar to us all. These have, all of them, this in common, that, on account of some interference with the blood-current the existing muscle is defective in relation to the work to be done, and so a compensatory hypertrophy takes place.

The question has frequently been asked whether, in hypertrophy of the heart, there is a true hypertrophy of the muscular fibres or a hyperplasia, and measurements of the diameter of the muscular fibres have been made. Some observers have made out that the muscular fibres are thicker, and some that there is little appreciable difference in thickness. Considering that from hypertrophy a heart which normally weighs ten ounces may increase to thirty, we may safely infer that this great addition does not result merely from thickening of the individual muscular fibres, but that there is an increase in the number of the fibres, a proper hyperplasia.

Turning now to viscera having smooth muscle in their walls, we have first to consider hypertrophy of the *urinary bladder*. We have

* Münzinger, 'Deutsch. Arch. f. klin. Med.,' vol. xix, p. 449.

seen that in the case of the heart over-exertion leads to hypertrophy, which is sometimes called simple or idiopathic. A very interesting simple hypertrophy of the bladder occurs in some cases of diabetes insipidus. In this condition there is an excessive secretion of urine, which is, as it appears, usually congenital and often hereditary. In order to meet the excessive requirements the bladder dilates and the muscular coat hypertrophies. In a case of this disease recorded by my friend, Dr. Finlayson,* a boy was in the habit of passing about 400 oz. of urine daily, and 35 to 55 oz. at a time. Here the bladder was greatly enlarged, extending, when full, an inch above the umbilicus. It is an interesting fact, brought out by Dr. Finlayson in this case that "the enlarged bladder with its hypertrophied muscular coat required periods of rest to contract thoroughly on its contents; and the analogous phenomena in the uterus during labour, and also of a distended rectum expelling large fæcal masses, at once suggest themselves." Thus, the boy with a full bladder passed 40 oz., but percussion showed that the bladder was not empty, and after a rest for five minutes he passed 12 oz., and again, after three minutes 6 oz., making in all 58 oz.

In the very remarkable instance of hereditary diabetes insipidus recorded by Weil,† many persons in the affected family had great enlargement of the bladder, which reached, in some cases, to the umbilicus and above it.

These are cases of hypertrophy from excessive performance of a normal function without any proper disease of the apparatus, and are comparable with simple hypertrophy of the heart. But we are all familiar with the much more frequent cases of hypertrophy of the bladder from obstruction of its orifice or of the urethra. Here, increased force is required to expel the urine, and in relation to the requirements the normal amount of tissue is defective, and so new-formation occurs.

In the *alimentary canal* I do not know that hypertrophy occurs from over-exertion due to a simple increase of work, but it is of occasional occurrence in consequence of obstruction. It is very common to find, in consequence of obstruction of the pylorus, a hypertrophy of the muscular tissue of the *stomach*. The difference between the function of the muscle of the stomach and that of the bladder is evidenced by the different distribution of the hypertrophy

* Finlayson, 'Glasgow Medical Journal,' vol. xv, p. 17, 1881.

† Weil, 'Virchow's Archiv,' vol. xcv, p. 70, 1884.

in the two cases. In the case of the bladder the muscular coat expels the contents by contracting on them and pressing them out. In the case of the stomach the muscle has a vermicular action by means of which it churns up the contents, and it is only the pyloric portion that is directly concerned with expelling the contents. In stricture of the pylorus, therefore, it is in the pyloric portion of the stomach that we find hypertrophy of the muscular coat, whereas at the same time the fundus may be dilated without any trace of such hypertrophy.

In the *intestine* we do not so frequently meet with hypertrophy of the muscular coat. In order to its occurrence we require to have a prolonged partial obstruction, and this is much less frequent than a temporary complete obstruction. If the obstruction be complete, then, if it lasts even a few days, the patient is liable to succumb. There is, however, one very interesting case in which there is a prolonged partial obstruction and resulting hypertrophy of the muscular coat: I refer to twisting of the sigmoid flexure. In this condition, which I believe to be of very frequent occurrence, there may be considerable narrowing of the calibre at the points where the two limbs cross, but I do not think there is much likelihood of complete closure. In these cases there may be enormous enlargement of the flexure. In two of the cases which I have examined post mortem, I found this piece of intestine entirely filling the abdomen, extending up to the diaphragm, and having a diameter like that of the thigh. There was great hypertrophy of the muscular coat, which seemed largely to monopolise the thickened wall of the intestine.

In all these forms of hypertrophy of smooth muscle there is some enlargement of the individual fibre-cells, but there is also, as in the case of the enlargement of the uterus during pregnancy, a hyperplasia or numerical increase in the cells.

In the next place the *kidney* is liable to compensatory enlargement. Little is definitely known as to a simple hypertrophy from increased secretion of urine, but in cases of diabetes insipidus we may, I think, expect to find a hypertrophy to some extent. Some of the enlargement which is usual in diabetes mellitus may be a true hypertrophy, but I am not aware that careful observations have been made in this regard.

Hypertrophy of one kidney from loss of the other is by no means uncommon, a complimentary or vicarious hypertrophy.

This matter has been frequently put to the test of experiment in animals, one kidney having been extirpated, and the condition of the urine and of the other kidney carefully observed. There have also been cases in the human subject, in which one kidney having been destroyed by disease or congenitally absent, the remaining one has been hypertrophied. As this whole subject has important practical bearings, I may be allowed to go into it more particularly.

Rosenstein* excised one kidney in rabbits and dogs, and at certain intervals afterwards weighed and measured the remaining kidney. It may be remarked that the animals stood the operation in general fairly well, and after a few days' illness returned to their ordinary diet. In one case, where a dog was operated on, the general health was hardly at all disturbed, and the animal on the second day after the operation took its normal amount of food. In this case the secretion of urine seemed hardly affected by the operation, and on the second day the amount of urine and the percentage of urea were equal to what they had been for three days before the operation. It seems, therefore, that the one kidney may completely compensate almost immediately after the removal of the other, although it is not to be inferred that it always does so. If the animal lives the remaining kidney enlarges, and in Rosenstein's cases the increase in weight seems to have begun immediately after the operation. At first the increase in weight is slight, and for some time there is not a corresponding increase of size. Through time the weight increases, although, in the different cases, not in exact proportion to the time which has elapsed since the operation. It is clear that at first the increase in weight is due essentially to addition to the amount of blood and lymph in the organ, but I think Rosenstein is in error in ascribing the whole increase which occurs at a more remote period after the operation to the same cause. He states that "in all cases the enlargement is essentially an increase in weight, and to an incomparably less extent an enlargement in volume;" and yet in examining his protocols we find in one case where a rabbit survived for 102 days, that whereas the excised kidney measured 3 cm. long, 2 broad, and 0·5 thick, the hypertrophied one measured 3·8 cm. long, 2·5 broad, and 1·2 thick; the weights of the respective kidneys being 5 and 10·7 grams. Again, in another case, where the subject of experiment was a dog, the excised kidney measured

* Rosenstein, 'Virchow's Archiv,' vol. liii, p. 141, 1871.

4·8 cm. long, 2·7 broad, and 1·5 thick, while after sixty-nine days the hypertrophied kidney measured 5·5 cm. long, 3·3 broad, and 2 thick; the respective weights being 18·6 and 31·7 grams.

A very interesting series of experiments was made by Gudden,* in which he extirpated the kidney in newly-born rabbits, and examined the remaining kidney when the animals were full grown. He compares the cubic contents and the weight of the remaining kidney with those of the two normal kidneys in a full-grown rabbit, and finds that in both respects the one kidney very closely corresponds with the two normal ones put together, being slightly greater than in the actual normal case chosen. These observations by Gudden have been generally confirmed by Grawitz and Israel,† and they have been so far amplified that, according to them, while in young animals the remaining kidney completely compensates and rapidly increases in size, in older animals the increase in size is much slower, and the compensation less complete. Thus in a young animal the left kidney excised weighed 4·7 grm., and in nineteen days the remaining right kidney had attained a weight of 7·6 grm., whereas in an old animal the extirpated right kidney weighed 7·7 grm., and the remaining one in eighty-two days only weighed 11·3 grm.

Another very important result was brought out by the observations of Grawitz and Israel. They succeeded, by obstructing the renal artery for one hour and a half to two hours and then letting it free, in inducing acute parenchymatous inflammation of the kidney. This was usually followed by contraction of the kidney, but in the case of weakly animals by a chronic parenchymatous nephritis. When by this means contraction of the one kidney was induced, there occurred a complimentary hypertrophy of the other, and in this case also as in the former one the distinction between young and old animals was marked.

In the human subject the observations are very numerous in which, in consequence of disease or defect of one kidney, there has been hypertrophy of the other. In a case of my own, in which the right kidney was congenitally entirely wanting, the remaining one weighed twelve ounces, or at least double the normal weight of one normal kidney. Including one observed by himself, Beumer‡ has

* Gudden, 'Virchow's Archiv,' vol. lxvi, p. 55, 1866.

† Grawitz and Israel, 'Virchow's Archiv,' vol. lxxvii, p. 315, 1879.

‡ Beumer, 'Virchow's Archiv,' vol. lxxii, p. 344, 1878.

collected no less than forty-eight cases, in forty-four of which one kidney was entirely awanting, while in four it was only rudimentary. In twenty-six of these the remaining kidney was healthy, and in all the twenty-six it was large and heavier than normal, the increase varying from "somewhat larger" to "double the normal size."* In addition to these cases of congenital defect resulting in complimentary hypertrophy it is by no means uncommon in cases of disease of one kidney to find the remaining one enlarged, the commonest causes of such unilateral disease being hydronephrosis and calculus in the pelvis of the kidney. In a case of my own, an old pyelitis had resulted in the closure of the pelvis of the kidney and the destruction of the entire kidney tissue, which was replaced by a congeries of cysts, some of them filled with pultaceous matter, the result of old dried-in abscesses. In this case the other kidney weighed $8\frac{3}{4}$ ounces. It is noteworthy that the hypertrophy here did not reach the same extent as in my other case where the defect of the kidney was congenital. Perls† records nine cases in which with atrophy or destruction of one kidney the remaining one was hypertrophied. Of these, eight were cases of hydronephrosis and one of contracted kidney.

In considering now the details of structure in these cases there are several points of considerable interest. The condition of the ureters and blood-vessels is one of these points, and another is the exact method by which the enlargement of the kidney takes place.

The condition of the ureters was particularly attended to by Gudden in his observations in animals in which the one kidney was excised immediately after birth. The ureter of the extirpated kidney was shorter and thinner than normal, but it still possessed a lumen, and, when stimulated by faradisation, contractions were visible although not propagated down the tube as in the normal ureter. The persistence of the ureter, however, and the retention of its muscular coat although atrophied, seems to show that the development of the ureter takes place separately from that of the kidney. In cases of congenital defect of the kidney it will often happen that the ureter will be destroyed by the same cause as that which interfered with the kidney, just as we sometimes find the right lobe of the liver and the suprarenal capsule defective along with

* In one case it is noted as three times as large as normal, but there was acute interstitial nephritis.

† Perls, 'Virchow's Archiv,' vol. lvi, p. 305, 1872.

defect of the right kidney. In most of the recorded cases it has been defective, and in Beumer's collection of cases the ureter was in most cases awanting while in others it was partly present. In one case, however, it extended from the bladder to the second lumbar vertebra having a lumen all the way. In another it extended from the bladder to the bifurcation of the aorta, and was wider than that of the remaining kidney. In my own case of congenital defect of the right kidney the ureter was preserved, and although thinner than that on the other side it could be traced up to the normal position of the kidney, where it divided into three small branches which ended in some matted tissue from which issued a vein that passed to the vena cava.

The blood-vessels were commonly absent in the cases of congenital defect and when present were extremely small.

We have now to inquire how the marked increase in bulk and weight in the hypertrophied kidney occurs. We have already seen that there is a marked increase in bulk as well as in weight, a definite addition to the substance of the kidney, and the details of this addition have now to be considered. The secreting structure of the kidney may be divided into the glomeruli and the uriniferous tubules, and in a considerable number of cases careful measurements have been made as to the sizes of these in the normal and hypertrophied kidneys. Most of the authors cited above have made such measurements, and I myself have made some, in the two cases already mentioned.

It is generally agreed that the enlargement is mainly of the cortical substance of the organ, and the chief measurements have been made of the glomeruli and the convoluted tubules. Everyone seems agreed that the glomeruli are not increased in number, and although slightly increased in size yet this is so slight that it can contribute but little to the increased bulk of the kidney. Thus Gudden found by two methods of calculation, that in cases of extirpation in newly-born rabbits, the glomeruli were not increased in number, although they were increased in size in the proportion of 41 to 30. Perls found the glomeruli very little increased in size. Grawitz and Israel found the glomeruli larger in the proportion of 8 to 5, but they were very much further apart than normal, so that whereas in a normal kidney eight or nine glomeruli would be in one field of the microscope, there would only be four or five in the same field with a hypertrophied kidney. This would indicate that

the main increase is in the tubular tissue between the glomeruli. In my own cases the glomeruli were slightly enlarged, in the case of congenital defect in the proportion of $22\frac{1}{2}$ to 20, and in that of atrophy from disease in the proportion of 26 to 20. We have to look, therefore, to the uriniferous tubules as the great cause of the increase in bulk in the kidney in these cases.

As the glomeruli are the expanded distal ends of the tubules into which the tufts of vessels are invaginated, and as these are not increased in number it follows that there can be no absolute increase in the number of the tubules. Their increase must be either in thickness or in length, and if in length then each tubule must make more convolutions than formerly in order to accommodate its increased length. According to most observers there is some increase in thickness and in the bulk of the epithelium, but it is comparatively slight. Perls asserts that this enlargement is considerable, but others have found it comparatively slight. In my own cases the convoluted tubules appeared to be larger than the normal in the proportion of about 6 to 5. It is clear, on the whole, that the increase in bulk is mainly owing to lengthening and increased convolution of the tubules, involving new formation of basement membrane and epithelium, as well as of the surrounding capillaries.

I think these results are not unimportant in relation to the function of the kidney. From them we may infer that the structures on which the principal weight of the secretion of urine falls are the convoluted tubules. When one kidney is removed in a healthy animal the remaining kidney is able at once to take up the function of both, but obviously there must be excessive exercise of function, and this leads to a gradual new formation so as to accomplish satisfactorily the increased work. When we find that the new formation affects mainly one constituent of the organ, then we are forced to the conclusion that this is the most important constituent. We may infer, therefore, that in the secretion of urine, while the glomeruli separate the water and are capable of separating double the usual amount without much increase in size, the convoluted tubules separate the more essential constituents, and are the proper secreting structures of the kidney.

It may be added that, although a single hypertrophied kidney is capable of perfectly performing the function of the two normal ones, yet that it is more exposed to disease than the latter. In his forty-eight collected cases Beumer found the kidney healthy in

twenty-six but diseased in twenty-two, the disease being usually chronic inflammation. The history of the cases also shows that the disease was worse borne than if there had been two kidneys, its duration was shorter before the fatal issue, any suppression of urine led to serious symptoms more quickly and so the fatal issue was more rapid. This doubtless arises from the fact that in disease common to both kidneys the disturbance is not equally distributed between them, and the one may partly relieve the other on a sudden emergency, whereas with only one kidney any disturbance say of the circulation cannot be compensated but produces its full results.

Judging from what has gone before, I think we may infer that when there is a still more partial loss of renal tissue, such as we so commonly find as a result of embolism, there will be to some extent a compensatory hypertrophy. We know that these embolic infarctions lead ultimately to the formation of deep cicatrices in the kidney tissue, but I do not know that kidneys with such cicatrices have been found to weigh less than the normal.

Compensatory hypertrophy of the *testicle* occurs when one testicle is awanting or defective in its development. Curling* relates two cases which occurred to Mr. Page, one of which is described in the 'Transactions of the Pathological Society.'† Curling gives the normal weight of the testicle as 6 drachms, being the average between Meckel's statement of 4 and Sir Astley Cooper's of 8 drachms. In one of Mr. Page's cases the left testis was retained in the groin and undeveloped, while the right, which appeared to be quite healthy, was enlarged, weighing 1 ounce 95 grains. In the other case the left testis was entirely absent, and the right weighed 2 ounces 160 grains. Curling also refers to two cases in which atrophy from disease was followed by hypertrophy of the other testis. A young man presented atrophy of the right testis in consequence of orchitis following mumps, and the right was enlarged. A soldier whose left testicle had been punctured on account of acute orchitis, had only a solid nodule on the left side, the seminiferous structure having all escaped by the wound; he presented a distinct enlargement of the right testicle.

I am not aware whether excessive exercise causes hyper-

* Curling, 'A Practical Treatise on Diseases of the Testis, &c.,' London, 1878.

† 'Pathological Transactions,' vol. ix, p. 317.

trophy of the testes, but should expect these organs in Turks and Mormons to be larger than normal.

Compensatory hypertrophy of the *lungs* is the next subject that concerns us, and we have first to consider the question whether increased exercise ever leads to hypertrophy. In persons living at a high altitude it is clear that the same bulk of air contains less oxygen than at low levels. Without going into the question whether this influences the absorption of oxygen in the lungs or not, which appears to be a matter of dispute at present, there seems tolerably complete evidence that in persons living habitually at high altitudes the lungs are larger than in those nearer the sea level. Williams* has so recently discussed this question that I may content myself with some quotations from his elaborate paper. He says: "The large size of the chest of dwellers in high regions has been noted in various parts of the world." "Armieux found a considerable increase of chest circumference in eighty soldiers after a residence at Barèges [in the Pyrenees, 4200 feet above the sea level] after a residence of forty-three days, and a still further increase at the close of four months' stay. The average increase was twenty-five millimetres (or an inch). In the Himalayas Kellet found the European soldiers at Landour (7300 feet) increase in chest circumference at least an inch during their stay of six months." A similar effect has been noted by Williams himself in the elevated regions of South Africa in one instance.

It may perhaps be questioned by some whether this increase in the size of the chest indicates a real hypertrophy of the lung, and not rather a mere dilatation of the air vesicles, a kind of emphysema. I think the condition is not properly designated by the term emphysema, which implies an over-distension of the air vesicles to such an extent as to interfere with their function. There is really increased function and a permanent enlargement to meet the continued increase of function. This is just what we find in other forms of hypertrophy. The organs generally are capable of meeting temporarily a great increase in their function, but when such increase is continued there is a hypertrophy to meet it. And it is consistent with this view that the actual enlargement of the chest is of gradual development. In Kellet's cases the increase was noted

* Williams, 'Transactions of the International Medical Congress,' vol. ii, p. 164, 1881.

after six months, and in Armieux's the increase which was visible after forty-three days was more marked after four months. We may say, I think, that the air vesicles permanently expand without undergoing atrophy and so expose an increased surface to the inspired air. Of course the increase of breathing surface is not in exact proportion to the increase in size, as a small cavity has in proportion to its size a larger surface than a small one, but there is considerable increase of surface, and probably also an elongation of the capillaries and increase of the supporting tissue. There is, in the terms of our original definition, an addition to the existing active tissue of its kind.

We come now to the question of a vicarious or complimentary hypertrophy of the lung, such as we have been studying in the kidney, and due to defect or disease in another part of the same lung or in the other lung. Laennec,* whose opinion on any matter where the morbid appearances are concerned, is of pre-eminent weight, mentions that Morgagni observed hypertrophy in connection with compression of the lungs from empyema, and asserts for his own part that it is much more general than this author imagined. I am afraid that this matter has rather fallen out of sight among pathologists, as it commonly receives hardly a bare mention.

Hypertrophy from defective development or growth has received still less notice than that from disease on one side, and as, so far as I know, a case which occurred to myself is the only one on record I may be allowed to describe it somewhat fully. The following is a note of the appearances as seen at the post-mortem:

"The left lung is greatly shrunken, forming a fleshy structure lying in the posterior parts of the left thoracic cavity. It measures seven and a half inches from apex to base. Although there is no morbid condensation it is seen on section that no crepitant lung tissue exists except in the middle regions of the lung where there is a very limited amount of it, and this has the usual grey pigmentation of the adult lung. The upper lobe of the lung is entirely unpigmented and presents no proper lung tissue at all, being entirely converted into a series of sacs, which have membranous walls. These sacs can be felt just beneath the pleura, and they stand in open communication with the bronchial tubes, being obviously greatly dilated bronchi. On section they are found to

* Laennec, 'A Treatise on the Diseases of the Chest,' English translation, 4th edition, London, 1834.

present a diameter of half an inch to an inch. The upper part of the lower lobe, forming the crepitant and pigmented part already mentioned, contains two of the sacculated cavities. The lower part of the lower lobe, although absolutely devoid of pigment, is more fleshy than the upper lobe, and presents some appearance of lung tissue. It contains several sacs similar to those of the upper lobe, but is not, like it, converted into a congeries of sacs.

"The right lung is greatly enlarged, and its anterior part projects beyond the mediastinum so as to a great extent to fill up, along with the hypertrophied heart, the space left by the atrophied left lung. At the time of the examination the anterior margin of the right lung reached two inches to the left of the left nipple. The lung presents somewhat the appearance as if a special projection of its anterior parts had taken place, there being a bulky piece lying in the left side of the thorax, and this piece presents at its junction with the lung a shallow groove corresponding with the mediastinum, and most marked at its upper and lower extremities. The appearance of this projecting anterior portion is not that of emphysema. The tissue is firm and deeply pigmented, and there are no bullæ or special emphysematous projections. On section it is seen, however, that the air vesicles throughout the lung are larger than normal.

"The main bronchi of the two lungs are nearly equal in diameter, but the vessels are about half the size on the left side, the pulmonary artery and vein being each about three eighths of an inch in diameter on the left side and three quarters of an inch on the right.

"The heart is greatly enlarged and considerably displaced towards the left. The enlargement is entirely of the right ventricle, the left being probably smaller than normal, and very remarkably removed upwards from the apex, the whole apex region being formed by the right ventricle. The wall of the pulmonary artery is thickened, being at least two thirds as thick as that of the aorta. The tricuspid orifice admits six fingers and the mitral three.

"The case was that of a man, æt. 46. No observation of these conditions was made during life. He had a slight cough and spit since boyhood, and considerable hæmoptysis, fourteen years before death. Six months before death he had swelling of the abdomen and legs, which disappeared but returned four weeks before admission. Latterly he complained of cough and dyspnœa."

There are several points of interest in this case. I think we may

infer from the absence of pigment in the upper lobe and in the lower part of the lower lobe that these parts had never expanded after birth, the middle part of the lower lobe having expanded partially. The condition of the bronchial tubes is peculiarly interesting. The main bronchi were equal in size to those of the other lung, and the extremities of the bronchial tree expanded into sacculated pouches. These facts would seem to indicate that the bronchi are distinct in their development and growth from the lung parenchyma, and it will be remembered that we were led to infer in regard to the kidney that the ureter is also of separate formation. The vessels of the atrophied lung were much smaller than those of the other, this consisting with the general fact that blood-vessels follow in their formation the structures which they supply.

In regard to the enlarged lung, I regard the condition as a true hypertrophy. As noted in the report the appearances were not at all those of emphysema, but the lung was firm and pigmented in the added on part of it as well as in the more normal portion. At the same time a careful examination showed that the lung alveoli, not only in the added on part but in the whole of the lung, were considerably larger than normal; there had been a general expansion of the whole vesicles, so that even to the naked eye the appearances approached that of the turtle's lung. There had apparently been no proper new formation of air vesicles, but a simple hypertrophy of them; and although the lung could not be weighed without injuring the specimen, there could be no question that it was much heavier than a normal lung, and that great new formation of tissue had occurred. It may be observed that this mode of hypertrophy is exactly analogous to that which occurs in the kidney. Just as there is no increase in number of glomeruli and tubules so there is no increase in air vesicles. Doubtless throughout life this man had less breathing surface than ordinary people, and the very striking hypertrophy of the right ventricle shows that the heart was excessively exercised in carrying on the pulmonary circulation. He had a cough and spit throughout life, and at the last seems to have died with symptoms chiefly referrible to the heart.

Looking to this case we may perhaps explain what has often been regarded as somewhat inexplicable. It is known that in post-mortems of weakly children, even a considerable time after birth, there is often seen a partial non-expansion or atelectasis of the lung. When this condition has lasted some weeks it is hardly con-

ceivable that expansion will ever occur, and yet there must be such atelectasis in many children who survive. It has been a difficult matter to explain what becomes of these collapsed portions, and how cicatrices or depressions such as we should expect from them are so seldom seen in the adult. I think we may infer that a hypertrophy—not an emphysema—probably occurs in the inflated parts of the lung, and that in the growing lung of the child the lung accommodates itself to the conditions, and all trace of the atelectasis is lost.

This leads us to consider whether a partial disease of one lung may lead to a hypertrophy of the remaining sound parts. It is well known that emphysema is common in the neighbourhood of collapsed phthisical cavities, &c., but this is a different thing to hypertrophy, In order to the occurrence of a true hypertrophy we should expect it to be a pre-requisite that the affected person should recover to a considerable extent from the disease, because, for two reasons, hypertrophy is not likely to occur while the disease is advancing. In the first place, for such new formation a somewhat vigorous state of health is requisite, such as a phthisical patient hardly possesses, and, in the second place, such persons, being invalids, do not exercise their respiratory organs so much as healthy people and so there is not so much call for hypertrophy. In the paper by Dr. Williams, already quoted from, the expansion of the chest shown by phthisical patients who have been treated at high altitudes is fully considered. At Dr. Williams' request Dr. Ruedi, of Davos, made careful measurements in 105 cases, and found enlargement in ninety-five (90 per cent.). From measurements made by himself in patients treated at Davos Dr. Williams tabulates his conclusions, of which for our purpose the following are the more important:

"1. That as a rule the portion of chest wall overlying the healthy lung more frequently undergoes dilatation than that overlying the diseased lung."

"5. That the length of residence required to produce this thoracic expansion varies in different cases, but that, as a rule, some months are necessary; . . . ."

"6. That the duration of this expansion after a return to lower levels varies, but, in the majority of patients, is permanent. . . ."

It will appear from what has gone before that the hypertrophy here is related, on the one hand, to the recovery or partial recovery

of the patient, implying increased general vigour, and, on the other, to the influence of the high altitude. We should expect that a certain amount of hypertrophy would occur even at low altitudes where recovery takes place, and we believe that this is the actual experience of those who have observed cases before and after a residence in the Riviera and other southern health resorts near the sea level.

That we have to deal with a true hypertrophy here there can hardly be any doubt. It is certainly different from emphysema, as the lung tissue is more active than normal, and it is really an enlargement of the organs from increased healthy exercise.

Compensatory hypertrophy of the liver is also a condition which to a large extent has been overlooked. Considering how obscure the function of the liver is, we are hardly prepared to expect a hypertrophy from simple over-exercise of its function. And yet the enlargement generally observed at the beginning of diabetes, which, according to Cornil and Ranvier,* is due to enlargement of the hepatic cells, may be regarded as such. In this case there is an active congestion of the liver, and the enlarged cells contain an excess of glycogen; apparently they are over-exercised in their normal function.

Of more certain occurrence is hypertrophy of one part of the liver from defect or destruction in another. My attention was forcibly directed to this subject by a case which has already been referred to in another part of this paper. In the case of congenital absence of the right kidney already alluded to, in which the left kidney was hypertrophied, the following was the condition of the liver as described at the time:

"The liver appears, at first sight, reduced in size, and remarkably rounded in form; its weight is $43\frac{1}{2}$ ounces. The right lobe is greatly atrophied, as evidenced by the position of the suspensory ligament and the gall-bladder. That is to say there is much more of the liver to the left of the ligament than to the right, and the gall-bladder is close to the right border, partly presenting over this border. The liver tissue appears to be normal."

In another case which I met with there were deep cicatrices in the liver, one of them in the situation of the gall-bladder almost dividing the liver into two separate pieces, which could be folded on one another. The right lobe was nearly globular in form, and

* Cornil and Ranvier, 'Manuel d'histologie pathologique,' 1873, p. 872.

measured four inches transversely, while the greatly enlarged left lobe measured six inches. In this case the atrophy of the right lobe may have been due to syphilis, the cicatrices indicating this as a possible cause. But more probably it was congenital, as the right kidney was very much smaller than the left, weighing only $2\frac{1}{2}$ ounces, while the left weighed $5\frac{3}{4}$. Both kidneys showed amyloid disease and interstitial inflammation.

In the first of these cases some injury or disturbance during fœtal life has destroyed the right kidney and neighbouring part of the liver and a hypertrophy of the remaining parts of the liver has been the result. In the second case there has probably been a similar origin, but the interference with the right kidney has not been sufficient to destroy it.

Hypertrophy also occurs in consequence of destruction of liver tissue from disease. Frerichs * refers to enlarged cells and lobules as occurring in consequence of destruction caused by the deep cicatrices in syphilis, by obliteration of the portal vein, or by any other cause. Goodhart † relates a case in which "the right lobe of the liver was entirely destroyed by a gummatous and fibroid deposit, the whole lobe not weighing more than two or three ounces. The left lobe weighed forty-six ounces and thus formed a new liver not far short of the average bulk."

As to the mode in which such hypertrophy takes place, we may infer from the analogy of the kidney and lung that there will not probably be any new formation of hepatic lobules, and that the hypertrophy will be due to enlargement of them involving multiplication of the hepatic cells and other constituents of the lobules. From some measurements in my second case I am led to infer that the lobules are somewhat enlarged, but I am not in a position to say that this is the entire cause of the hypertrophy.

In concluding this paper, I may be allowed to sum up the general conclusions which the facts adduced entitle us to draw. We have not passed in review all the organs of the body, but we have considered a sufficient number to show that where there is an increased call for the performance of a normal function

* Frerichs, 'A Clinical Treatise on Diseases of the Liver,' translated by Charles Murchison. 1861. Vol. ii, p. 210.

† Goodhart, "Summary on Diseases of the Liver," in Sydenham Society's 'Atlas of Pathology,' 1882.

there is, if the growth be still incomplete, an accelerated formation of tissue, and if the adult form have been reached, a renewed growth to meet the requirements of the body. In this process there is not, apparently, a new formation of complicated structures such as we find in many of the lower forms of life, where lost limbs or even larger parts of the body may be restored, but the process seems to confine itself to multiplication of the finer elements, such as muscular cells, hepatic cells, renal epithelium, with their accessory blood-vessels and supporting structures.

Dr. Douglas Powell differed from the author in respect of compensatory hypertrophy, as it was seen in phthisis; he considered that in such cases hypertrophy could occur. No doubt in some instances the lung remaining did not become hypertrophied but only dilated, and then the patient was breathless and weak. Hypertrophy of organs beset us in all directions. Instances in the heart and stomach were mentioned.

Dr. Fancourt Barnes spoke of the hypertrophy of the heart in pregnancy as a typical example of physiological compensatory hypertrophy.

Dr. Kingston Fowler alluded to cases of hypertrophy and dilatation of the ascending colon associated with ulceration, but no obstruction. Was there general cirrhosis in the case of syphilitic hepatic disease?

Dr. Joseph Coats, in reply, thought that he had admitted in the paper the possibility of the occurrence of compensatory hypertrophy in cases of chronic phthisis. In diabetes insipidus he considered that all the secretory structures were at work, and so tended to become hypertrophied. There was no general cirrhosis in the instance of syphilitic disease of the liver which he had mentioned. He had no experience of cardiac hypertrophy in pregnancy, but regarded the line between physiology and pathology as by no means a hard-and-fast one.

RETROVERSION OF THE GRAVID UTERUS.

By Clement Godson, M.D.

When invited to contribute a paper this session I selected as my subject "Cancer of the Neck of the Womb," which I have been working at for several years. I have found it impossible, however, to complete this in the time at my disposal, and so, rather than bring it forward in its imperfect state, I have, with the kind permission of your secretaries, replaced it by a totally different subject, viz., "Retroversion of the Gravid Uterus."

In a Society such as this, composed not only of surgeons and physicians, but largely of that most important and useful department of the profession—general practitioners, I felt that this subject

would be acceptable, as it treated of a complaint frequently met with, and often beset with great difficulties and complications. It has also another and a special claim to be discussed here, for it was in 1770 that Dr. William Hunter, at a meeting of this ancient Society, described it and gave to it the name it now bears; an account of this paper will be found in a book published in 1772, entitled 'Medical Observations, and Inquiries, by a Society of Physicians in London.'

It is stated by Martin de Jeune in his 'Mémoires de Médecine et de Chirurgie pratique,' 1835, p. 137, that this displacement was known to Hippocrates and Philumenus. I do not know his authority for this statement. The earliest account of it I have met with is in a book published at Basle in 1535, containing the writings of Aetius Amidenus, 'De re Medica,' in liber xvi of which, cap. lxxix, "De revocatione, aversione, et recursâ uteri, ex Aspasia" there is not only an account of the displacement, but a plan of treatment is proposed, which has been very little improved upon at the present day. This will be referred to later on. Some accounts of this complaint and deaths resulting therefrom are recorded by Deventer, 1701, J. A. Külm, Dantzig, 1732, and Walter Van Doeveren, Groningen, 1765, but it was not until William Hunter's paper that any lengthy or accurate description of it was given.

William Hunter's contribution was entitled "The History of a Fatal Inversion of the Uterus, and Rupture of the Bladder in Pregnancy, by Mr. John Lynn, Surgeon at Woodbridge, in Suffolk," with an appendix by William Hunter, M.D., F.R.S., addressed to the Medical Society.

The patient the subject of this accident was aged forty, a multipara, and had suffered previously from prolapse of the vagina, as it is termed (which almost certainly implies prolapse of the womb and retroversion), which she had been in the habit of pushing back as she lay in a horizontal posture, but on this occasion, when about fifteen or sixteen weeks pregnant, while stooping down to glean corn in a field, she felt (as she stated) "something suddenly give way at the bottom of her belly, and fall down towards her back." This was followed by great pain and retention of urine and fæces, and Mr. Lynn was unsuccessful in his attempt to pass a catheter into the bladder. A vaginal examination convinced him that it was a case of an inverted gravid uterus, such as he remembered being described in a lecture by Dr. Hunter to his pupils on the

21st October, 1754: "Many efforts were made to reduce the uterus by placing the woman on her knees and elbows with her head downwards, and by introducing one hand up the vagina, attempting to draw it forward, at the same time with two fingers of the other hand in ano he endeavoured to push up the fundus uteri according to the method recommended and attempted by Dr. Hunter and the surgeon who desired his advice." This attempt failed, and as the catheter could still not be passed it was proposed to puncture the bladder above the pubes. To this, however, the woman would not consent. Two days afterwards she felt something burst within her, which proved to be the bladder; this was almost immediately followed by abortion, and after a few hours by death. The autopsy showed a rupture near the fundus of the bladder, large enough to admit the end of the finger, and for about the breadth of a shilling around the aperture it was in a gangrenous state.

I have quoted this case at some length as it contains so many points of interest to which I will later refer. In the appendix Dr. William Hunter points out that the case referred to by Mr. Lynn, to which he was called in consultation, and which formed the subject of his lecture, was one of a young woman, four months advanced in her first pregnancy, who sought the advice of Mr. Walter Wall for suppression of urine. Mr. Wall recognised retroversion of the gravid uterus which he had heard described by M. Grégoire in his lectures in Paris. He therefore, after having passed a catheter and drawn off about seven or eight quarts of urine, "attempted to reduce the uterus by laying the patient on her back and by assisting with one finger in the vagina and another in the anus, as M. Grégoire had directed, but without success." It appears, therefore, that M. Grégoire had, previous to William Hunter, lectured on this displacement, though his lectures have never been published.

When Dr. Hunter arrived he placed the patient in the knee-elbow position, and tried, with one hand in the vagina and two fingers of the other hand in the rectum, to replace the uterus, but without success, and four days afterwards the woman died.

The post-mortem examination supplied the subject of a large plate in Dr. Hunter's celebrated work on the gravid uterus.

From this time the writings on the subject and the cases recorded have been so numerous that it is impossible to do more than allude to some of them in the course of this paper.

We will now pass on to the symptoms produced by this displacement.

As observed in the cases already quoted the first and prominent symptom is inability to pass the urine; this may be followed, and frequently is, by its dribbling away after the bladder has become distended. The period of pregnancy at which this occurs is almost always between the third and fourth months. Difficulty in defæcation and tenesmus rapidly ensue. Sometimes the distension of the abdomen by the enlarged bladder is one of the chief complaints of the patient, but it is remarkable how often this is not elicited until an examination has been made.

History and causes.—Formerly it was believed that over-distension of the bladder from want of opportunities to void the urine was the invariable cause of the womb being thrust backwards and jammed in the pelvis. Dr. Meigs,* of Philadelphia, says "The natural delicacy of a woman often compels her to resist most urgent desire to pass off the urine, as when out driving, &c. If three months advanced this is almost sure to bring on retroversion." Dr. Ramsbotham (1867) says, "The cause of this accident is most frequently, if not always, to be traced to an over-distended bladder. The woman has been, from some circumstance, unable to pass her urine for a considerable time; she has probably been engaged in society in a state of some restraint, and the bladder therefore has become more than ordinarily full."

Until Dr. Tylor Smith † in a paper before the Obstetrical Society, read November 7th, 1860, laid great stress on the fact that as a primary cause of retroversion the distended bladder is of little consequence, though such is no doubt one of the most important phenomena of complete retroversion, Dr. Rigby seemed to be almost the only writer who had expressed this view. He had remarked, "In every case which has come under our own observation the bladder had not been distended until the retroversion had taken place, in consequence of which the os and cervix uteri had been tilted up behind the symphysis pubis, and, having thus compressed its neck, had caused the difficulty in passing water." My own experience is that though generally the bladder has been greatly distended in the large number of cases I have seen, I have never been able to get a genuine history of abstinence from passing water

* 'Science and Art of Obstetrics,' by Charles D. Meigs, Philadelphia, 1863.

† 'Obstetrical Transactions,' vol. ii, paper, Nov. 7th, 1860.

for any length of time from want of opportunity, though frequently there has been a history of straining violently the abdominal muscles, as in a case reported by me in the 'Obstetrical Journal of Great Britain and Ireland,' November, 1876, in which the woman when lifting a heavy weight "felt something give in her inside and was sure something slipped out of place," some time after which distension of the abdomen appeared, and inability to pass water. Also in Mr. Lynn's case, already quoted, the woman was stooping down to glean corn in a field when "she felt something suddenly give way at the bottom of her belly and fall down towards her back."

Another important predisposing cause which this case of Mr. Lynn's affords, is the previous prolapse of the womb, and my experience has shown me that hardly ever, if ever, is there prolapse of the womb to any extent without retroversion.

The frequent recurrence of the complaint in the same patient, and the fact that retroversion is often found subsequently in the non-gravid state supports this view; and during the last few months I have seen no less than three patients who had been under my treatment for the same condition during previous pregnancies, one of these on as many as three occasions.

It seems therefore that a gravid uterus previously retroverted and probably prolapsed increases in size until perhaps by some violent straining of the abdominal muscles forcing the bladder and intestines against the body of the womb so as to depress it still further, it so jams the viscus in the pelvis that the neck is pressed to such an extent forwards as to produce inability to empty the bladder.

Under other circumstances the gradual increase in the size of the womb as pregnancy advances has the effect of raising it up from its retroverted position till it ascends completely into the abdomen.

That this action may be prevented by other causes except tension of the abdominal muscles appears clear, for frequently no history of any violent effort or straining is obtained. William Hunter laid great stress on a pelvis contracted above and large inferiorly predisposing to this. Dr. Ramsbotham also noticed in a case in which the conjugate diameter at the brim of the pelvis only measured three and a half inches, that retroversion of the gravid uterus occurred on two occasions. The symptoms which first induce a patient suffering from this displacement to seek advice have been already given. I now pass on to the examination of the patient.

First, *the abdomen* will probably be occupied by a considerable swelling, perhaps reaching above the umbilicus even as high as the ensiform cartilage, dull, elastic, with a feeling of fluid. This might be mistaken for the gravid uterus at a more advanced period, but the catheter, if it be passed, will soon show that it is not this, for after the evacuation of the urine the swelling will entirely disappear.

Per vaginam, the cervix uteri will not be found in its natural situation; as a rule it is high up and felt with great difficulty, indeed it may be entirely out of reach, as in a case recorded in the 'Transactions of the Medical Congress of Philadelphia in 1876,' in which, on account of urgent symptoms, it was decided to induce abortion, and the most dependent part of the uterus had to be punctured by a trocar from the vagina, all efforts to reach the os uteri having failed.

The pelvic excavation will be found to be occupied by a firm rounded tumour often reaching so low as almost to rest upon the perineum.

If the urine have been drawn off the body of the womb will be found on bimanual examination to be absent from the abdomen, and this is an important point, for it is the great distinguishing feature between this displacement and those complaints which have been mistaken for it, viz. retro-uterine hæmatocele, subperitoneal fibrous tumour springing from the posterior wall of the uterus, a small ovarian tumour, and extra-uterine fœtation.

Unless the hæmatocele is in connection with pregnancy, it should not be mistaken for a retroverted gravid womb, for the signs and history of pregnancy would be absent. So also with a fibrous tumour and a small ovarian tumour.

With extra-uterine fœtation it is different. There is in both the history of the cessation of the catamenia, and there have probably been in both the symptoms of pregnancy, but in extra-uterine fœtation the fundus uteri, little larger than its natural size, may generally be detected on careful examination pushed towards one or other iliac region.

That these conditions may be confounded by the most experienced and skilful observers the following case will show:

On April 16th, 1879, M. B—, æt. 38, was admitted into Martha Ward of St. Bartholomew's Hospital under the care of Dr. Matthews Duncan. She had been married thirteen years, and had one child a year after marriage, her only pregnancy. The catamenia

had been regular until towards the end of last December, no appearance since. Since 13th inst. had been unable to pass water; four pints of urine were drawn off yesterday before admission.

Examination.—Per hypogastrium, nothing abnormal.

Per vaginam.—Cervix adjacent to symphysis pubis, examining finger easily passed between them; behind the cervix the pelvis is nearly occupied by a tender, irregular, elastic mass, probably a retroverted gravid uterus.

Now, beyond the fact that the cervix uteri permitted a finger to be passed between it and the symphysis pubis, and was not barely within reach, and that no mention is made of the absence of the fundus uteri from any part of the lower abdomen, what better history and symptoms and signs could one have of a case of retroverted gravid uterus?

To proceed. An attempt to reduce it was unsuccessful. A little later an air-ball was placed in the rectum by the midwifery assistant, and an attempt made to restore the position of the womb, without success. The air-ball was left in for two days, but no difference in the position of the pelvic tumour having occurred at the end of this time, another attempt to push it up was made unsuccessfully. On May 9th the patient was discharged. It turned out subsequently to be a case of extra-uterine fœtation, the bones of the fœtus being removed through the abdominal parietes by Dr. Carter at the Hospital for Women, Soho Square.

This case better illustrates the difficulties of diagnosis than any remarks I could have made.

Treatment.—The first act should be, as has already been stated, in order to assist in the diagnosis, as well as for the relief of the patient, to pass a catheter and draw off the urine. For this purpose a long flexible male catheter is the best. I have never met with a case in which this could not be accomplished, but cases are recorded, and indeed mention has been already made in this paper, of one in which the instrument could not be got into the bladder. Under such circumstances the bladder might be aspirated from the abdomen. Dr. Münchmeyer relates a case in which he punctured the bladder, which enabled him to restore the position of the womb, and the patient recovered. Also where the catheter could not be passed, the size of the womb has been reduced by puncturing the uterus from the vagina or rectum, and thus removing the pressure forwards sufficient to permit the instrument to reach the bladder.

Such treatment would, however, be only necessary in very rare and severe cases.

Going back to the treatment of the olden times, that proposed by M. Grégoire, and that adopted by Dr. William Hunter has been given, both being a hand in the vagina to pull forward and depress the cervix and anterior vaginal wall, with two fingers of the other hand in the rectum to push up the fundus, the only difference being that Dr. William Hunter adopted the knee-elbow position. I said that I would refer again to the article by Aetius Amidenus from Aspasia, published in 1535.* The following is an extract from it, translated accurately, I believe:

"And we shall cure version of the uterus towards the anus in the following manner: first we shall order the midwife to push the uterus forward with the finger placed in the anus, and then to place a pellet of a width of four fingers in the anus besmeared with galbanum and wax, to the summit of which a string shall be attached for the purpose of drawing it out, or she shall place under it castor diluted with water and put upon wool in a similar manner, or dried bitumen or liquid pitch." Further on it speaks of the midwife directing the neck of the womb by holding the head of a probe.

There is a diversity of opinion as to whether, the bladder having been emptied, the uterus should be left to restore itself, the rectum being kept empty by enemata, and the bladder by catheterism if necessary. Dr. Gervis, in a paper read before the Obstetrical Society of London (vol. xvi), advocated immediate replacement. In the discussion which followed several speakers opposed this view. For my own part I entertain no doubt it is the proper practice.

Then comes the position of the patient in which the attempt at restoration should be made. A great many writers (among these may be mentioned Ramsbotham, Boivin, and Dujés) believe that the knee-elbow position, advocated and adopted by William Hunter, offers no advantages over the ordinary English obstetric position, lying on the left side.

Godefroy ('Gazette des Hôpitaux,' 1859) placed the patient on the edge of the bed so that the head and chest were hanging on the floor, the legs and pelvis only being on the bed.

* The only reference to Aspasia I can find is in 'Dissertatio Historica Critica de Feminis ex Arte Medica claris,' by P. F. Schacher et J. H. Schmidius, Leipsic, CIↃ IↃ CC XXX VIIJ, p. 16: "Aspasiam mulieribus quoque medicis jure meritoque annumerandam esse, præter alios, Aetium præsertim testem hanc in rem cito."

Then with regard to the preparation of the patient before attempting reduction. As a rule none is necessary, but it has been recommended that blood shall first be taken away from the cervix. Dr. E. Rigby agrees with Dewees that this affords facilities. Dr. Barnes strongly recommends that chloroform shall be administered to a surgical degree. This would, however, only be necessary in cases of difficulty. When the fingers have been found insufficient to push up the retroverted organ, various instruments have been proposed for this purpose.

Dr. Byford, of Chicago, recommends, if a colpeurynter (or air-ball) in the vagina fails, that a drumstick or ivory-headed cane be passed into the rectum*—certainly a somewhat rough kind of manipulation.

In France the tampon of Evrat in the vagina is employed; this is described as a small rod about twelve inches in length, which has, fastened to one extremity, a sort of mop made of fine old linen, well smeared with oil or fresh lard.

Dr. Halpin, of Cavan, introduced a sheep's bladder into the vagina and inflated it slowly, and thus reduced a dislocation which had defied the ordinary means.†

M. Favrot proposed the introduction of an india-rubber bottle into the rectum and inflation afterwards. Dr. Fleetwood Churchill tried this instrument in the vagina with immediate and easy success.

Dr. Barnes recommends that one of his hydrostatic bags—which is very much the same thing—should be placed in the rectum, distended with water—*not air*—and left there, and he mentions a case‡ in which after the space of an hour an acutely retroflexed uterus was by this means restored to its position.

Dr. Bond§ invented an instrument, consisting of two curved branches, one to pass into the rectum, the other into the vagina, both with a button on the extremities. By means of this he succeeded in replacing the uterus with considerable ease.

Calisen suggested, in impracticable cases, abdominal section, and seizing and replacing the fundus; and Purcell, Gardien, and Cruikshank have advised division of the symphysis pubis as affording

* 'A Treatise on the Theory and Practice of Obstetrics,' New York, 1873.

† Halpin, "On Retroversion of the Uterus," 'Dublin Journal,' 1840, p. 76.

‡ 'British Medical Journal,' 1881.

§ 'American Journal of Medical Sciences,' April, 1849, p. 408.

more room for reposition of the displaced womb. These are extreme remedies which, I am happy to say, I have as yet found unnecessary.

The treatment I have adopted when I have had the means at hand, and invariably with success, has been first to empty the bladder, then, with the patient on the left side, endeavouring, with two fingers of the left hand in the rectum and two fingers of the right in the vagina, to push up the uterine body; if this fails, to insert an india-rubber ball in the rectum, and, putting the patient in the knee-elbow position, inflate it with air and push upon it, at the same time using pressure upwards with two fingers of the other hand in the vagina. Should the reposition not be complete it will be well to leave the air-ball in the rectum, and in a short time it may effect its object. It appears to me a matter of indifference whether the india-rubber ball be filled with air, or with water as recommended by Dr. Barnes, but that the former possesses advantages in being lighter.

This is really little more than was advocated 350 years ago by Aetius Amidenus, who, after using the finger in the rectum, inserted a pellet (probably of cotton wool) besmeared with grease.

Lately in the out-patients' room at St. Bartholomew's Hospital two cases of greatly distended bladder with retroversion of the gravid uterus have come before me; they were both recent as regards the retention of urine, and I endeavoured at once to reduce the dislocation by putting the patient in the knee-elbow position, and pushing up the body of the womb with two fingers in the rectum and two in the vagina. In both cases I could only raise it so far as my fingers would reach. I wanted the air-bag, which was not at hand on the first occasion. Suddenly a happy thought struck me; I had my stethoscope in my pocket. I immediately inserted it into the vagina, and pushed it up till the ear-piece pressed on the perineum, and by this means the uterus was at once restored to its proper position. On the second occasion I adopted the same treatment with a like result. The first patient I admitted to the ward, the second I allowed to return home at once after placing a large Hodge's pessary in the vagina. In neither case was there any further trouble. I believe that this novel treatment will be found useful in cases of emergency when other appliances are not at hand. If necessary the stethoscope may by means of a T bandage be retained *in sitû*, and this will be recognised as almost

identical with the treatment now generally adopted, and with such success, for cases of chronic inversion of the uterus.

It is very important, unless the patient has been seen immediately after the first symptoms, that she shall be kept in bed for several days after the restoration, and that the bladder be frequently emptied, for, as I pointed out in 1874 in the discussion on Dr. Gervis's paper, a great addition to the ordinary amount of urine is secreted, often for several days after the first accumulation. In the 'Obstetrical Journal of Great Britain and Ireland,' Nov., 1876, I give the following amounts from a case I had under treatment: 1st day, $11\frac{3}{4}$ pints; 2nd day, $6\frac{1}{2}$ pints; 3rd day, 5 pints; 4th day, 5 pints; 5th day, $4\frac{1}{2}$ pints; 6th day, 3 pints.

Before allowing the patient to resume the upright position it may be well to introduce a Hodge's pessary to avoid a recurrence of the displacement. The great danger arising from retroversion of the gravid uterus is from cystitis; the urine is sometines very sanious, but if it have been long retained it is sure to have become ammoniacal and to contain mucus and pus, and it has been known for the whole mucous membrane of the bladder to exfoliate, as in the specimens from the museum of St. Bartholomew's Hospital, which I now show you, both of which came from patients in the hospital under my observation.

It is said that women die occasionally from rupture of the bladder, but this is probably from sloughing of the coats of the bladder following cystitis, as in the case of Mr. Lynn, where the mucous membrane round the aperture communicating with the peritoneal cavity was gangrenous.

The woman who passed the first specimen, an entire cast of the bladder, completely recovered, a contracted bladder only remaining.

The second specimen I show you of exfoliated mucous membrane is from a patient recently in St. Bartholomew's Hospital, under the care of Dr. Matthews Duncan and myself. She was admitted on January 2nd of this year, thirty-four years of age, pregnant for the third time, four and a half months since her last menstruation. Three weeks before admission she experienced difficulty in passing water, and had to strain very much in order to void it. During the last nine days she has had to have it drawn off occasionally, but for the last three days it has dribbled away. The bladder was found to be greatly distended, and the urine drawn off strongly alkaline, ammoniacal, containing a large quantity of

blood and pus, with crystallised phosphates and oxalates. The uterus was found to be retroverted. An attempt by the midwifery assistant to replace it by pressure with the fingers in the vagina, with the patient in the genu-pectoral position being unsuccessful, an air-ball was inserted in the vagina, and on the following day it was discovered that the womb had rectified itself. The great trouble now was the cystitis. On January 19th, finding the patient very ill, I decided to wash out the bladder by means of a double catheter; in doing so I dislodged a mass which made its way out of the urethra by the side of the catheter. It was found to be the mucous membrane of the bladder encrusted with phosphates; it measured three and a half by two and a half inches. Several smaller pieces passed afterwards. The following day the patient was seized with violent spasmodic pains precisely simulating labour, during which another piece of mucous membrane, the same size as that removed the previous day, was expelled. Just a week subsequent to this a large slough came away by the vagina, which was proved to be the vesico-vaginal septum. The patient recovered sufficiently to return home, the pregnancy proceeding, incontinence of urine from the fistula being the chief trouble.

This is a typical instance of what has been misnamed rupture of the bladder; fortunately in this instance the site where the slough occurred was away from the peritoneal cavity.

This patient's recovery may be truly said to be remarkable. She has recently been confined, premature labour having been induced at the seventh month, and she will in course of time enter the hospital for the vesico-vaginal fistula to be treated.

Ramsbotham states that he once opened the body of a pregnant woman, whose uterus was retroverted, in which case an aperture was made through all the coats of the bladder by the ulcerative process, and the fluid was effused into the peritoneal sac. It is doubtful if there is any authentic case of rupture of the coats of the bladder in a woman from simple splitting due to stretching.

Where death occurs it is almost invariably from exhaustion associated with blood-poisoning, or from uræmia, the matters which should be excreted by the kidneys being retained in the system. Peritonitis is rarely a cause, but it has been described.

When the womb has been replaced, if the patient be kept at rest for a sufficiently long period and carefully attended to, as a rule the pregnancy will proceed. Abortion is, however, not a very

infrequent consequence of this displacement. Where the uterus is completely retroverted, topsy-turvy as it were, the womb, though contracting violently, is unable to expel its contents. This is shown in the case of Mr. Lynn so frequently referred to by me. Immediately the bladder gave way and permitted the womb to move up, the fœtus and placenta were expelled.

A remarkable instance of this came under my observation a short time since. I was requested by Mr. Outhwaite, of Denmark Hill, to see with him a patient who was pregnant about four months, was in great agony, could not pass her water, and in whom he could not get the catheter into the bladder. She was thirty-nine years of age, had been married eight years, and had three children; with all of these, in early pregnancy, she had suffered from trouble in passing her water, having had to have it drawn.

I found her lying on the bed, calling out with pain, as with a woman in labour. The bladder was greatly distended; I could not feel the os uteri, the pelvis was entirely occupied by the uterine body. Having emptied the bladder, and drawn off a considerable quantity of high-coloured ammoniacal urine, I endeavoured, with the patient on her left side, to push up with my fingers in the rectum and vagina the displaced womb, but it would not move. Having been apprised of the nature of the case, I had come provided with an india-rubber ball, which I then placed in the rectum and distended with air and pushed on, but without effect. And so I placed the patient in the knee-elbow position on the bed, and with two fingers beneath the air-ball in the rectum and two pressing on that part of the body felt from the vagina I with but little difficulty caused the womb to revolve; the body went up and the cervix came down, and at the same moment the fœtus and placenta were violently expelled with the liquor amnii. The patient experienced immediate relief, and I have since learned completely recovered. This case also illustrates the success of the treatment I advocate, when such means are at our disposal. Otherwise I strongly recommend the stethoscope, in the top of which a piece of sponge may be inserted to overcome the sharpness of the edges in some forms employed.

Mr. GRIFFITH thought that alteration of the conjugate diameter could only influence the occurrence of retroversion of the gravid uterus by altering the period of its occurrence.

Dr. C. H. ROUTH alluded to the part played by the conjugate diameter

in the causation of the mischief, and advocated the use of the vectis in the treatment.

Dr. Godson briefly replied.

April 21st, 1884.

A CASE OF PHLEGMONOUS GASTRITIS.

By Thomas Whipham, M.B.

Martha M—, aged forty-nine, was admitted into St. George's Hospital under my care on July 4th, 1883. It appeared from the history that she had been for several years subject to bilious attacks, in which severe vomiting with occasional diarrhœa were prominent symptoms. She had lost all her teeth, and was accustomed to drink large quantities of warm liquids. The diarrhœa had been very troublesome since the beginning of 1883; and on April 24th of that year she was an in-patient under Dr. Wadham, in a state of great prostration, after prolonged diarrhœa. There was on this occasion no vomiting. With rest and light diet (fish, &c.), the symptoms improved, and she was discharged relieved in a fortnight.

About a month after she had left the hospital, the diarrhœa returned; and on July 1st an old femoral hernia came down, causing her much abdominal pain, more especially in the epigastrium. When in the recumbent position, the hernia disappeared; but this was at first followed by profuse and continuous vomiting, at first of dark matter, then of thin bilious fluid. The bowels were freely open.

She was admitted into hospital at the request of Dr. S. D. Clippingdale of 26, Ladbroke Grove, who had been called in to attend her. At first, she was placed in a surgical ward as it was supposed that the symptoms might depend upon the hernia. No rupture being discovered, she was at once transferred to my care.

The patient was badly nourished and slightly jaundiced. She had lost all her teeth. She complained of abdominal pain, referred chiefly to the epigastrium. No tumour was detected. The abdom-

inal walls were flaccid. The vomiting was almost continuous; the vomited matter was small in quantitity, fluid, opaque, green and strongly acid.

Small doses of acetate of morphia with sulphate of atropia were given to relieve the pain; while at the same time, with a view of maintaining the patient's strength, nutrient enemata containing brandy were administered. The injections were retained fairly well; but the vomiting did not entirely cease, nor did the character of the vomited matter alter.

On July 6th, the patient sank into a comatose state; the temperature gradually rose from normal to 104° Fahr., with symptoms of peritonitis; and she died soon after midnight.

Necropsy (twelve hours after death).—*General Appearances.*—Height, five feet; black hair; no teeth; jaw atrophied.

Lungs.—Posterior portions of both congested.

Heart.—Weight, twelve ounces. The mitral valve was thickened and contracted; only one finger could be passed between its flaps. The remaining valves and orifices were natural.

Abdomen.—The peritoneal cavity contained a small quantity of purulent fluid.

Stomach.—The contents were thin greenish-yellow fluid. The mucous surface at the cardiac third of the organ was very red, felt thick, and infiltrated with fluid. On transverse section, it was a quarter of an inch thick. The remaining two thirds of the stomach, so far as the inner surface was concerned, presented no abnormal appearance; but, on transverse section, the pyloric end was found infiltrated with a purulent effusion between the muscular and mucous coats. On the posterior surface of the organ, below the lesser curvature, there was an area about one inch wide by three inches long, which was of a distinctly yellow colour; and, at this place, section showed an abundance of purulent fluid between the muscular and serous coats. The remainder of the alimentary canal was natural.

Liver.—Weighed three pounds twelve ounces; its surface was smooth. The tissue was pale and fatty.

Spleen.—Healthy; weight, seven ounces.

Kidneys.—Weighed eight ounces. The capsules were adherent; the surfaces were granular; the cortical parts diminished, and mottled slightly with dark coloured spots.

Microscopical examination confirmed the naked eye appearances of

the walls of the stomach. There was, as the drawings show, an extensive infiltration of the submucous tissue, chiefly by pus-cells. The pus was most abundant around the secreting glands, the cells of which were somewhat swollen, and their contents opaque and granular. At that part of the posterior surface of the organ where the yellow discolouration of the external surface was found, as described above, all the coats of the stomach were infiltrated with pus.

After the patient's death, the friends brought a box labelled "podophyllin pills." They stated that she had taken one or two of them on the night before admission; but they were unable to say whether or not she was in the habit of taking them.

Cases of acute inflammation of the walls of the stomach have received but little attention in the ordinary English text-books. In foreign works, however, more attention has been paid to this affection. Raynaud, in the 'Bulletins de la Société Anatomique,' 1860; and Leube, in Ziemssen's 'Cyclopædia,' vol. vii, devotes a considerable space to the disease—gastritis phlegmonosa. According to the latter, it occurs in two forms: 1. Circumscribed suppuration; 2. Diffuse purulent infiltration of the parietes, extending around and between the gastric glands. In the circumscribed form, the mucous as well as the serous layers are usually intact, or at most only erythematous. "The abscesses of the stomach are sometimes single, at other times multiple." "Cases of diffuse purulent infiltration of the parietes have been observed less frequently, and less exactly." In this latter, the condition of the mucous layer varies greatly in the different cases examined; it is sometimes unchanged, at other times infiltrated with pus or serum. Occasionally the muscular layer may be destroyed by the purulent infiltration.

The serous layer may be also unaffected, according to the seat and extent of the phlegmonous inflammation. In other cases, it may take on inflammation, accompanied by the accumulation of dirty grey thick masses of exudation upon the surface of the stomach. In these cases, the subserous layer may be infiltrated with serum or pus.

Of thirty-one cases collected by Leube, twenty-six occurred in males and five in females (Ziemssen's 'Cyclopæd.,' vol. vii, p. 157.) Bamberger ('Handbuch der Speciel. Pathol. under Therapie Krankheit: des Chylöopetisch. Syst.') devotes but a single

DESCRIPTION OF PLATE

Illustrating Dr. Whipham's Case of Phlegmonous Gastritis.

FIG. 1.—Transverse section of the stomach, showing thickening and purulent infiltration of the submucosa. × 70.

a. Submucosa infiltrated with pus.

b. Portion of the muscularis mucosæ.

FIG. 2.—Showing the purulent infiltration of the submucosa. (More highly magnified.) × 450.

a. Gastric glands.

b. Purulent infiltration.

Fig. 1.

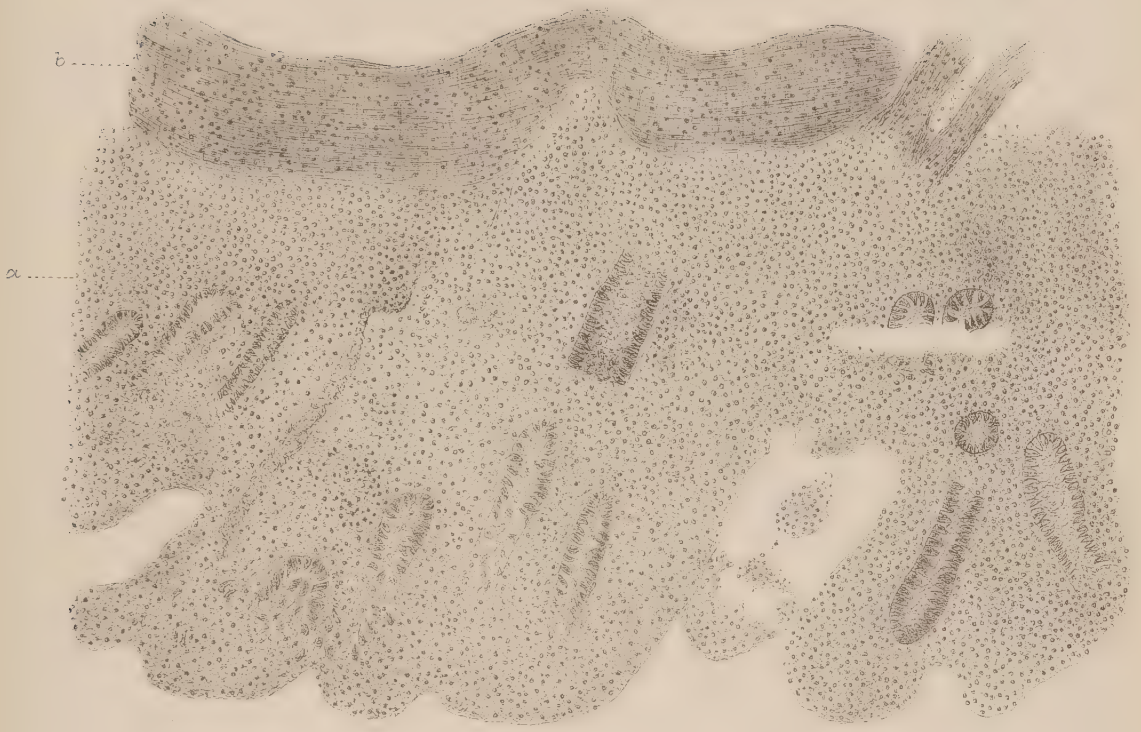
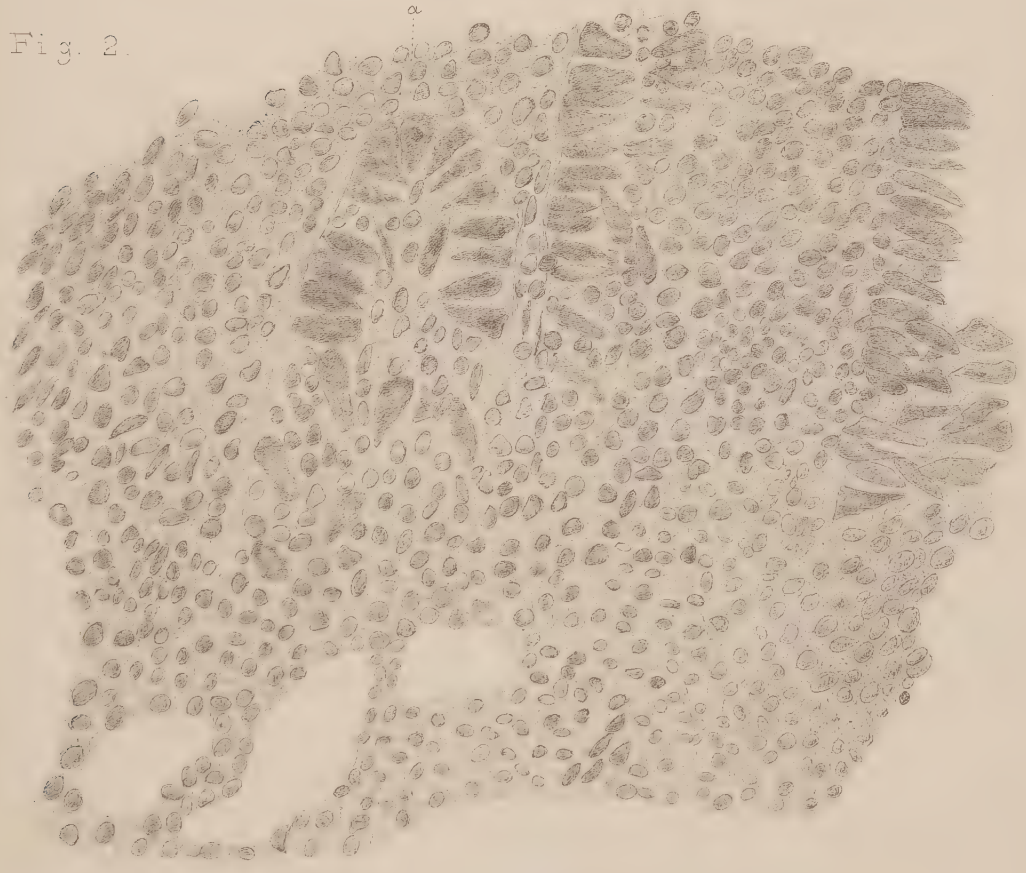


Fig. 2.



West Newman & Co sculpt

short paragraph to this affection of the stomach; his description of the morbid appearances agrees with that of Leube.

Etiology.—It appears, from the records of the cases which have been observed, that the cause is unknown. According to Leube: "In a number of cases, the origin of the affection has been attributed to excessive indulgence in alcohol; in a few, to some wound occurring in the region of the stomach; in others, again, to some error of diet." An obvious objection occurs at once to such explanations of the cause of phlegmonous gastritis—viz., that in countries such as England, to say nothing of other nations, where drinking is carried on to so great excess, and in which errors in diet are equally prevalent, instances of this disease are of rare occurrence. I purposely omit those cases in which inflammation of the walls of the stomach has followed acute diseases, such as typhoid fever, puerperal fever, etc. It is within the knowledge of all medical practitioners that, in many persons, certain articles of diet exercise a deleterions effect —*e.g.*, in some, lobster or other shell-fish invariably produce an attack of urticaria. In my own experience, a few strawberries always have this effect in a boy of about ten years of age. Podophyllin is differently tolerated by different individuals. In some, its action is most beneficial; in others, it has the reverse effect. In a patient of my own, a quarter of a grain is sufficient to render her so irritable that, as she expressed it on one occasion on which I prescribed it, she was "unable to sit still all day;" while the idiosyncrasy which renders many persons intolerant, even of a few grains of potassium iodide, is too well recognised to require comment.

It may be that, in certain persons, an attack of phlegmonous gastritis may result from the ingestion of articles of diet usually considered wholesome, or from the administration of drugs in ordinary use; hitherto, however, its occurrence under any circumstances is a rare event.

We have no evidence that the patient whose case forms the subject of the present communication had ever taken podophyllin before, and with such deficient information no conclusion can be drawn in this case, one way or the other.

Leube states that, "as regards age, those who died of gastritis phlegmonosa were, almost without exception, in middle life." The age (49) of the patient in the present instance is a case in point.

Symptomatology.—To quote Leube again, the cases have been too

seldom observed to enable an accurate description of the disease to be given, and in this he is in accordance with Raynaud.

Bamberger (loc. cit.) states that a diagnosis of the disease is, from want of observation, impossible, the main difficulty in diagnosis lying in the fact that the symptoms of the original malady are always masked by the concomitant peritonitis.

It would appear that the duration of the disease may vary from three to eighteen days, the average being nine days (Leube). The leading symptoms in the cases recorded are that, after symptoms of gastro-intestinal derangement have existed for some days, the patient is attacked with severe abdominal pain, chiefly in the region of the stomach, high fever, diarrhœa, and finally irregularity of the pulse, delirium, great prostration, and death. The rapid course of inflammation of the stomach and the peritonitis to which it gives rise is such as to render all treatment futile.

Of the recorded cases, I have selected one or two in which the notes are given in some detail; subjoined is an epitome of them.

1. Dietrich's case (abridged from Ziemssen's 'Cyclopædia,' vol. vii, p. 160). A tanner, entirely convalescent for a fortnight from pleurisy, felt quite well on October 20th, 1866, dined with good appetite, and spent the day at work as usual. On returning home on the same evening, he experienced severe chills, alternating with fever, and accompanied by pain in the region of the stomach, heart, and left thorax, so that he could not sleep that night. Next morning, there was repeated vomiting of bilious matter, while the former symptoms continued. In the evening, the patient had the aspect of a person seriously ill, with increased temperature, and rapid pulse (112). The abdomen was neither distended nor painful; he complained of nausea and eructation: there was occasional vomiting. One-twelfth of a grain of morphia was ordered to be taken four times during the night. The vomiting ceased next day; but, on October 24th, the patient was much worse in every way *i.e.*, higher temperature, flushed face, occasional delirium, rapid and soft pulse, short and laborious respiration. Early on the following morning, collapse supervened, and, in spite of stimulants, the case terminated fatally on the morning of the 25th. At the necropsy, sero-purulent inflammation of the submucous layer of the stomach was found, also peritonitis and pleurisy; the two last being probably due to extension of the inflammation of the stomach.

2. Glaix ('Berliner klinische Wochenschrift,' 1879, xvi, 565-8, epitome in 'Medical Record,' 1879, p. 303) records a case of a workman, aged 52, with rigors and violent sickness, which lasted twenty-four hours. The man was of intemperate habits. When first seen by Glaix, the patient's abdomen was flat and hard, and very tender to pressure in the ventricular and ilio-cæcal region; the spleen and liver were much enlarged; he had lost much flesh. The temperature was 104·5°; pulse, 136; respirations, 36. The treatment consisted of quinine internally, ice to the head, and galvanism applied to the neck once in two hours. On the following morning, the man was very restless; his extremities were cold, and cyanotic. Pulse, soft, 130; temperature, 102°; respirations, 24; urine very scanty. The bowels acted several times spontaneously. A hypodermic injection of Fowler's solution and pure alcohol was made, after which the pulse rose, but he became again collapsed, and died next morning.

At the *post-mortem* examination, the peritoneal cavity contained muddy stringy fluid mixed with shreds of mucous membrane. The blood-vessels of the peritoneum were much injected; the liver was enlarged and fatty; the spleen was three or four times its natural size. The stomach was enlarged, and contained a few ounces of yellowish stringy fluid; its walls were abnormally thick and soft, and fluctuating to the touch. On section of them, a whitish stringy liquid mixed with pus trickled from the submucous cellular tissue. The walls of the stomach were infiltrated with this fluid; the mucous membrane was pale, save in some places, where it was hyperæmic and ecchymosed. The case was considered to be one of idiopathic phlegmonous gastritis and general peritonitis. Glaix states that he has observed a second case of this disease, which, however, was not verified by *post mortem* examination, as the patient recovered. The etiology of this case was clear, the patient having been imprudent in eating. This was followed at once by symptoms of inflammation of the stomach. Pus was vomited, whereupon the temperature sank, facts which lead to the supposition that a submucous abscess had burst.

3. In the 'Bulletins de la Société Anatomique de Paris,' 1861, p. 63, M. Raynaud has an article on this subject, and gives the following cases, the first recorded by Cornil.

A man, forty years of age, of strong constitution, but addicted to drink, was admitted into the Charité Hospital on May 26th, 1860.

He had suffered no external injury; had been ailing for three months, but had only given up work for fifteen days. On admission he was delirious; convulsive movements of the body existed; his face was pale, without expression; his lips were red, his tongue dry; his pupils dilated and inactive. Vomiting which had been a prominent symptom for two days previously, had given place to profuse diarrhœa. The pulse was very small and irregular; respiration slow; the belly, distended and hard, did not appear tender. The patient died on the day of his admission, without treatment.

The *post-mortem* examination revealed extensive peritonitis. The stomach was adherent to the omentum, its walls were softened and thickened with an infiltration of plastic exudation and pus.

In this case, the mucous membrane was slightly injected, but not softened; the submucous tissue was much increased in thickness, and infiltrated with colourless fluid and pus cells. In short, the submucous tissue was in a condition similar to that of the subcutaneous areolar tissue in diffuse phlegmonous erysipelas.

This case during life appears to have been mistaken for meningitis.

The second case was that of a carter, aged forty-four. The patient was of a bilious temperament, strong constitution, but enfeebled by hard work and insufficient food. He had spat blood several times ten years ago, and again a month or six weeks previously to his admission into hospltal. He was short of breath, had cough, and sweated occasionally at night, but presented few physical signs of pulmonary disease. The pulse was very slow. Fifteen days later he was attacked with diarrhœa, accompanied by tenesmus and bloody stools. The left iliac fossa was tender to pressure; there was nausea but no vomiting; the tongue was red and glazed. The diarrhœa had ceased on admission, and for several days the bowels did not act. He was kept on light diet, was free from abdominal pain, and was allowed to walk in the garden. He was, in fact, considered to have recovered, and, on the seventh day after his admission, it was proposed to discharge him. However, he then complained of a feeling of uneasiness, loss of appetite, and nausea. Vomiting then set in, with abdominal pain, and, on the ninth day after admission, the belly was tender and somewhat distended, his pulse small, 120. Leeches and fomentations were applied to the belly. These symptoms having become more urgent, leeches were again applied to the abdomen, and, on the twelfth day following his admission, diarrhœa

set in, the stools being bloody. Ice was applied externally, and opium was administered by the mouth. Next day the man became delirious, with a dry tongue and weak pulse. The patient gradually became weaker, the abdomen more distended, and he died on May 29th, having been admitted on May 15th, 1860.

At the necropsy, extensive peritonitis was found. The walls of the stomach were thickened; the mucous membrane was thickened but not softened. Towards the larger *cul-de-sac* was a bright red patch, apparently the result of inflammation. Pus was found between the mucous and muscular coats, or between the latter and the peritoneal covering.

Raynaud's article, from which I have extracted the preceding cases, is the most complete of the earlier accounts of phlegmonous gastritis. In it he refers to records of such cases as far back as the year 1620, by Varandœus ('Tractatus de Morbis Ventriculi'), and again to a Dutch physician, named Henon ('De Morbis Ventriculi,' 1658), to Petri Borelli in 1656, and to the 'Sepulchretum of Bonetus. Most of the descriptions, however, furnished by these authors, and by writers of subsequent periods, are those of circumscribed abscess of the stomach, rather than of diffuse phlegmonous inflammation of its walls.

In 'Path. Soc. Trans.,' vol. xxvi, p. 81, the late Dr. Hilton Fagge has recorded, "a case of Diffused Suppurative Inflammation of the Stomach." The patient was an Australian merchant, aged 51, who returned from Brighton on the evening of Nov. 24th, 1873, not feeling very well. He appeared to have suffered from weak digestion and, in consequence, his diet for the two or three days previous to the attack, had been soup, tea, and light farinaceous food, and of these he had taken very sparingly. When seen by his medical attendant next morning, he complained of paroxysmal pain shooting up to the right shoulder, pain in the epigastric and umbilical regions, retching and vomiting. There was a yellow tinge about the skin of the neck. Under the impression that the case was one of biliary colic the treatment was opium and hot fomentations, and as the pain was relieved by the middle of the day (Nov. 25th) the former was discontinued. In the afternoon the patient was slightly delirious and sleepy, with a frequent desire to relieve the bowels; nothing, however, passed. Beyond this there was nothing in the symptoms to excite alarm; at midnight, however, the medical attendant was hastily summoned to the house and found his patient dead. The relatives expressed

their apprehensions that he had been poisoned by opium (of which he had taken three doses of one grain each), and Dr. Fagge, therefore, made a post-mortem examination. The coats of the stomach were much thickened. The seat of the thickening being mainly in the submucous tissue, but partly in the muscular coat. All the tissues of the stomach walls were infiltrated with pus cells; "they extended outwards to the subserous connective tissue; the gastric glands appeared not to have taken any part in the inflammation, their cylindrical epithelium was unaltered, but they had pus cells all round them."

Again in 'Path. Soc. Trans.,' vol. xxxiv, p. 90, Dr. A. Q. Silcock gives a case of phlegmonous gastritis which followed the operation of gastrostomy. He supposes the affection in this instance to have been due to the absorption of "virulently septic products" derived from the wound. In this case the microscopical appearances were precisely similar to those above described.

Dr. Habershon, in his 'Pathological and Practical Observations on Diseases of the Alimentary Canal,' relates the case of a woman, aged 40, who was admitted into Guy's Hospital on May 2nd, 1847, who had for a fortnight suffered from pain in the loins, and subsequently in the stomach and chest. She suffered also from anorexia, incessant vomiting of black bitter fluid, and intense thirst. Death was preceded by coma.

At the necropsy, peritonitis was found. At the pyloric end of the great curvature was a hard mass measuring four inches and a half by three inches and a half. On the inner surface corresponding to this mass was an irregularly shaped patch on the mucous membrane of a dark brown colour of the size of a shilling. On section pus was found infiltrating the submucous tissue. The pus was not fluid.

Budd, in his work on the 'Organic Diseases and Functional Disorders of the Stomach,' 1855, mentions a similar case.

In some of the recorded cases, ulceration of the mucous membrane existed; but this ulceration is considered by Raynaud to be the result rather than the cause of the disease. One may add to this that, if ulceration were the cause of such a virulent form of inflammation, we should, considering the frequency of gastric ulcer expect to meet with phlegmonous gastritis as a disease of every day occurrence.

In order to confine this paper within due bounds, I have limited

my remarks to simple purulent infiltration (if one may use such an expression) of the walls of the stomach, of which my case appears to be an instance, and have purposely omitted more than a casual reference to circumscribed abscess of that organ. As the two are, however, so intimately connected, I may, perhaps, conclude with Raynaud's summary at the end of his article referred to above.

1. The term gastritis should be restricted to the sense which custom has assigned to it, that is to say, inflammation of the mucous membrane, without any tendency to extension to the cellular tissue.

2. There are three distinct varieties of abscess of the walls of the stomach—intramucous abscess, circumscribed abscess of the submucous tissue, purulent infiltration more or less general.

3. The circumscribed abscess appears, in the majority of cases, as a chronic condition, and, perhaps, one of the pathogenic forms of gastric ulcer.

4. The purulent infiltration is an anatomical condition, liable to arise in many general diseases, characterised by a disposition of different organs to the production of pus.

Dr. HABERSHON thought such cases were rare, especially to English pathologists. Dr. Hilton Fagge had recorded a case about ten or twelve years ago. Diagnosis was seldom made during life. Direct local irritation could rarely be traced. Diffuse inflammations have nearly always been associated with diseases of the blood, and this was probably the explanation of their not being diagnosed during life.

Sir JOSEPH FAYRER had no recollection of having seen a case precisely like the one referred to by Dr. Whipham. He had met with diffuse purulent infiltration in connection with cases of sepitcæmia, but could not explain Dr. Whipham's case.

Dr. C. H. ROUTH remarked on the analogy of certain cases of septicæmia.

Dr. WHIPHAM briefly replied, and agreed that the explanation of his case was as impossible to him as to Sir Joseph Fayrer.

THE TREATMENT OF SIMPLE AND SLOUGHING DYSENTERY BY LARGE DOSES OF IPECACUANHA GIVEN MORNING AND EVENING ONLY.

By Joseph Ewart, M.D., F.R.C.P.

The efficacy of ipecacuanha in the congestive, exudative, and ulcerative stages of acute dysentery is now universally acknowledged. But both as to the dose and the frequency with which it is repeated, there would still appear to be some divergences in practice. Some practitioners still hold that five to ten grains represent appropriate doses, usually adopting the former, whilst the latter is the utmost limit attempted. Thus five grains is given repeatedly, three or four times a day or oftener. The smallness of the quantity is said to be compensated for by the greater frequency in the repetition of the drug, and the alleged depression of the vital powers from the exhibition of large doses in asthenic patients avoided. It is, it may here be premised, overlooked that it is, in such cases, the unchecked progress of the dysenteric process, and not the ipecacuanha, in the largest doses hitherto ventured upon, that is to be dreaded and combated with all practicable expedition.

When the question is tested clinically, it will be found that, of the two modes of treatment, the vital depression caused by a quick and prolonged succession of small doses is far greater than when larger ones are given at longer intervals of time. Under the *former* method, the stomach is constantly teased and nauseated, so that the general nutrition is seriously interfered with by partial cessation or marked impairment of the primary process of digestion. A sufficient interval of freedom from the action of the drug, and for the digestion of aliment preparatory to absorption by the veins of the stomach and villi of the small intestines, is not allowed. Hence, not only does the general nutrition suffer, but the reparative process, whether that is being accomplished by resolution, granulation, glazing, or cicatrisation of injured, abraded, or ulcerated parts, is liable to be materially interrupted, and the benefit accruing from even these small doses frequently repeated considerably neutra-

lised. By this plan the drug is placed at a great disadvantage. It is, moreover, the most distasteful and disagreeable way of utilising it. To be racked and tormented by tormina and tenesmus is bad enough, but to have superadded upon these almost constant nausea, with or without retching and vomiting, is to most patients intolerable. Under such circumstances, it is not at all uncommon to find in the stools the most easily digestible kinds of food, which have been unaffected by the juices poured out in the stomach and intestines. Under the *latter* method, of giving from a scruple to a drachm of ipecacuanha every twelve hours, or night and morning only, the disagreeable effects of each dose—nausea, retching, or vomiting—pass off in about an hour or so, and the stomach is left in sufficient repose for the conversion of liquid food into assimilable peptones. Thus we have proceeding in harmonious order the re-establishment of the equilibrium of the portal circulation, the moderate utilisation of nourishment, and the substitution, for a condition of extending disease, of advance towards local repair and general convalescence.

Although this simple plan of using ipecacuanha is most beneficial in most cases, there are exceptions, in the early management of which a modification has seemed to be advantageous. First, where there has been asthenia coupled with extreme irritability of stomach—very few in number. Here the full effects of the drug may be commanded by giving small enemata containing from a drachm to two drachms of ipecacuanha with bismuth and soda, night and morning, with or without laudanum, according to circumstances. When these cannot be retained so as to ensure the absorption of the active principle of the remedy, the desired result, I would suggest, might possibly be accomplished by the subcutaneous injection of emetin.* Secondly, where the

* A hypodermic solution consisting of two centigrammes of pure emetin dissolved in five grammes of water with one minim of sulphuric acid may be employed, of which the dose is half a gramme for each injection (Dr. Hager, 'Commentar zur Pharmacopœia Germanica'). This corresponds closely with the subcutaneous dose recommended by Dr. W. G. Smith, who calls attention to the fact that pure white emetin is three times the strength of the brown samples. Applied locally to the skin it produces pustules resembling those arising from the application of tartar emetic ointment (Martindale). If, therefore, it causes, when employed hypodermically, any effects approaching those ascribed to it when applied to the skin, its subcutaneous use would scarcely be justifiable. The question might easily be settled beforehand in India, I would suggest, by Mr.

sloughing or gangrene is so extensive that recovery under any method of treatment is hopeless, death usually supervening from shock and asthenia, not infrequently expedited and intensified by peritonitis, septicæmia, and liver complications. In such cases stimulants and opiates are indicated. Thirdly, in full-blooded, sanguineous temperaments and rather free livers large doses may be used, every six hours or so, during the first twenty-four hours of treatment.

In the early career of dysentery Piso, Friend, Towner, Pitcairn, Boulduc, Wentworth, Playfair, English, Bateman, Mortimer, Forbes, Scott Docker, Sir Joseph Fayrer, and others regard emesis, when it occurs, as rather beneficial than otherwise. It may be so, and I must confess that I have never seen any evil results from it. My aim, however, has always been to avert it as much as possible, to aid the retention of the drug, with a view to expedite the absorption of its active principle into the circulation and its operation upon the vaso-motor centres, and, through these, upon the parts affected, somewhat in the manner described at the end of this communication.

I herewith submit a series of cases treated in accordance with the principles indicated.

Case 1. *Simple acute dysentery.*—J. H—, aged twenty-one, a stout and healthy-looking English sailor, admitted into the Calcutta Medical College Hospital on the 8th of July, 1867. Has been ill with dysentery during the past four days, passing numerous scanty, slimy, and bloody stools daily, accompanied with much griping and straining, and tenderness on pressure over the cæcum and sigmoid flexure. Tongue slightly coated; appetite not much impaired; pulse good. He was ordered to take twenty grains of ipecacuanha and ten grains of bismuth in a little water morning and evening. Ice 1 lb., milk and tea. 6 p.m.—Passed four stools containing abundance of slime and blood; tormina and tenesmus less. Pergat.

9th (a.m.).—Had two stools since last report, one in the night and one in the morning, consisting of yellowish semi-liquid material, and yielding, on washing, a large proportion of undigested food, potatoes, and three portions of mutton, which were at first mistaken for sloughs. The recognition of striped muscular fibre settled the question as to their true nature. There was also a considerable quantity of gelatinoid exudation tinged with blood. Much tenderness over the

Vincent Richards, who is an accomplished experimental physiologist. *Dose by the mouth.*—As an expectorant, $\frac{1}{200}$th to $\frac{1}{50}$th, as an emetic, $\frac{1}{6}$th to $\frac{1}{8}$th (Martindale), $\frac{1}{18}$th to $\frac{1}{6}$th of a grain (W. G. Smith)—subcutaneously, $\frac{1}{30}$th of a grain with a little sulphuric acid to make it soluble (Smith). It is a "safe, rapid, easy and agreeable form of emetic."—(Ibid.)

hepatic flexure of the colon, no tumefaction. Has been nauseated, no retching or vomiting. The dose of ipecacuanha was now increased to a drachm, night and morning. Diet, &c., as before. 6 p.m.—Better. Nauseated, but did not vomit. Pergat.

10th.—Has had one motion, vomited several times. Motion consisted of viscid greenish fluid, and a very small quantity of gelatinoid exudation. Omit all medicine. Diet low, chicken.

11th.—One semi-solid motion, without blood, exudation, or pain. Appetite and pulse good.

12th.—Healthy-formed motion; feels perfectly well, joined his ship, which was on the eve of sailing. Discharged.

Case 2.—*Acute, supervening upon chronic, dysentery patient returned moribund.*—G—, aged sixteen, Hindoo, resident of Thontonia, admitted at 7 a.m. October 30th, 1866. He had been suffering for upwards of two months from dysentery with frequent attacks of malarious fever. He was one of the famine-stricken immigrants from Orissa, and so emaciated and cadaverous that he was regarded as moribund. There were pain and tenderness over the abdomen, and the stools, which were passed involuntarily, contained little feculence, but much slime and blood. Abdomen hollow and tense. Pulsation of aorta felt without the exercise of much pressure. Pulse exceedingly feeble; appetite bad; conjunctivæ, lips, and tongue pallid. At first ordered an ounce of stimulant mixture every three hours, and a scruple of compound kino powder four times a day. Diet, milk, sago, and pint of beef tea.

31st.—Passed his stools, many in number, in the bedclothes, and is much exhausted. He was now ordered to continue mixture and diet as before, and to take one drachm of ipecacuanha and ten grains of bismuth at once with three quarters of an ounce of camphor mixture.

November 1st.—From this date he improved daily, little having been required for the subsequent cure, but the compound chalk mixture ʒj and laudanum ♏x every four hours, or as often as necessary. Rum mixture and good liquid food. The stools gradually became feculent and consistent. Appetite improved. Food comprised fish, milk, &c., and barley was given.

On the 4th he passed five yellowish-green liquid motions in bedclothes, without blood or exudation. The chalk mixture was replaced by twenty grain doses of Pulv. Crêt. comp. cum Opio every five hours, or *pro re nata*, and diet as before.

5th.—One motion. Pergat.

6th.—Two stools consisting of formed fæces, attended by a little prolapsus of the rectal mucous membrane. Pergat.

7th.—One formed stool. From this date, he took only the rum mixture, three times a day, with good fluid and semi-solid nourishment, up to the 20th, the date on which he was discharged cured.

Case 3. *Acute dysentery, recurring after a previous attack, with intermittent fever.*—T. K—, aged twenty, resident of Lall Bazaar, admitted into the Medical College Hospital, on January 7th, 1867. He had been previously treated in the Jamalpore Hospital, for twenty-two days, and discharged well on the 31st December.

Owing to indiscretion in diet and drink, the dysenteric symptoms returned. On admission, he was feverish, had much griping and strain-

ing, and was passing frequent bloody and slimy stools. He had been ordered, by the officer on duty, an ounce of stimulant mixture every two hours, an ounce of chalk mixture every four hours, and milk diet. When I saw the patient at 5.30 p.m. he had passed six scanty stools, consisting of blood, slime, and a small quantity of liquid feculence. Tormina and tenesmus had been severe, and there was much tenderness on pressure along the course of the colon. He was feverish, very thirsty, with accelerated pulse. I ordered a drachm of ipecacuanha and ten grains of bismuth mixed with a little water to be given night and morning; an ounce of quinine mixture (grs. iij ad ℥j) every four hours during the intermission; ice 1 lb., and diet as before.

8th.—Had vomited four times and passed many scanty motions, consisting of yellowish fluid with dark-brown fæces and a small quantity of yellow-stained gelatinoid exudation. Tormina and tenesmus greatly diminished. Pergat.

9th.—The dysenteric symptoms much improved. Powder was omitted. The fever gave trouble until the 14th, when a few ten-grain doses of quinine subdued it, after which he never had a bad symptom. During convalescence, liberal diet and port wine were prescribed, and he was discharged cured on January 31st, 1867.

Case 4. *Acute dysentery with malarious fever.*—C. E—, aged twenty-seven, tailor, resident of College Street, admitted into the Medical College Hospital on January 11th, 1867. Has been suffering from dysentery during the last eight days, passing several scanty, slimy, bloody motions daily, attended with great griping and straining. Ordered, by the officer on duty, an ounce of chalk mixture every three hours, and milk diet. 6.30 p.m.—Passed three scanty stools composed of slime and blood. The tormina and tenesmus had been excruciating. He was very feverish; pulse full and bounding, and the thirst was great. I now ordered him a drachm of ipecacuanha and ten grains of bismuth, night and morning; an ounce of quinine mixture (grs. v ad ℥j) to be taken every four hours; turpentine stupes to abdomen; ice 1 lb., milk and chicken broth.

12th.—Had only one motion composed of thin feculence, with a small quantity of blood and gelatinoid exudation. Pergat. 6 p.m.—Several motions containing a little slime, and a good deal of greenish yellow, liquid, feculent matter; vomited twice; no griping or straining. Pergat.

13th.—Three motions composed of soft yellow feculence, without slime or blood. Omit ipecacuanha. Ordered an ounce of chalk mixture, with fifteen minims of laudanum and twenty grains of carbonate of soda, every four hours, or *pro re nata*, and the quinine mixture three times a day.

14th.—Doing well. From this date, patient never had a bad symptom, excepting an occasional recurrence of malarious fever, which was effectually checked by ten-grain doses of quinine. Discharged cured on January 24th, 1867.

Case 5. *Acute dysentery with malarious fever.*—Mowla Bux, aged twenty-four, Mahommedan student, admitted into the Medical College Hospital on Jannary 4th, 1867. He was seized with looseness and malarious fever about twelve or thirteen days ago. When the fever stopped he then suffered from much griping and straining, and his

motions consisted of slime and blood only. He was ordered, by the officer on duty, an ounce of quinine mixture (grs. iij ad ℥j), every four hours, and a drachm of ipecacuanha with ten grains of bismuth, night and morning; milk diet and soup Oj.

5th.—Had several stools with less griping and straining, motions composed of yellow feculence with gelatinoid exudation tinged with blood. Nauseated and vomited up bilious matter twice only, but did not bring up the ipecacuanha. No fever. Tongue moist and clean. Pergat.

6th.—Doing well. Only three motions since last report, constituted of pultaceous yellow feculence, without a trace of blood or exudation. Omit ipecacuanha and quinine. Ordered an ounce of chalk mixture with twenty minims of laudanum and twenty grains of carbonate of soda every four hours, or *pro re nata*, an enema of laudanum, alum, and babool, altogether ℥iss, at bedtime; milk diet and soup Oj.

7th.—Doing well. Had three soft figured motions.

8th.—Better. His food was gradually improved, and the medicine withdrawn. Discharged well on January 14th, 1867.

Case 6. *Acute dysentery following malarious fever.*—Adilodeen, aged nineteen, a Mahommedan military class student, admitted into the Medical College Hospital, on October 13th, 1866. He had been ailing with malarious fever and diarrhœa for about a month. The fever had yielded to quinine outside. The looseness continued, and a few days before coming into hospital, the motions became dysenteric, containing blood and slime. There were considerable tormina, tenesmus and tenderness over the course of the colon. He was much reduced, pulse weak, small and quick; tongue pretty clean and moist; no febrile heat of skin. He was admitted with these symptoms after my morning visit, and was prescribed, by the officer on duty, five grains of ipecacuanha with five grains of bismuth, three times a day, and milk diet. 8 p.m.—He had passed eight bloody and slimy stools, attended with much tormina and tenesmus. The medicine had nauseated him and caused him to retch once. He was now ordered a drachm of ipecacuanha with ten grains of bismuth immediately, and diet as before.

14th.—Passed seven stools during the night, consisting of greenish yellow fluid, and yielding, on washing, a quantity of gelatinoid exudation tinged with blood. Some portions of the exudation were free from blood. There were also several pieces of shreddy material. Complained of much tormina along the large intestine, from the cæcum to the sigmoid flexure. Repeat the ipecacuanha, low diet, pint of mutton soup, and jelly *ad libitum*. 6 p.m.—There is decided improvement; tormina diminished. There is less blood and gelatiniform exudation found on washing the six or seven stools which he had passed during the day. He had been nauseated and vomited only a little gastric and salivary secretion. Still he had taken his nourishment satisfactorily, and complained chiefly of the tenesmus. Ordered to repeat the ipecacuanha, and to have an anodyne enema at bedtime. Diet as before.

15th.—Passed three greenish-yellow stools during the night. On washing these, a small quantity of gelatinoid exudation with traces of blood was found. The tormina and tenesmus were gone, and the patient more had slept soundly. He had only been nauseated once. He took one more dose of ipecacuanha. Diet, sago, soup, and jelly. 5.30 p.m.—Passed three feculent stools during the day with only traces of exudation, but

without any blood. There had been no more griping or tenesmus. The last dose of ipecacuanha had caused nausea, and he retched, without vomiting, three or four times, about an hour after taking it. Omit the ipecacuanha. Ordered an ounce of chalk mixture with ten minims of laudanum and twenty grains of carbonate of soda, every four hours, or *pro re nata;* two ounces of port wine, milk, and soup Oj.

16th.—From this date, the patient steadily improved. The stools became entirely feculent and his strength returned. The pulse regained its ordinary fulness and force. A mild attack of fever on the 18th was successfully combated by five-grain doses of quinine. He continued to take the chalk and soda mixture occasionally as circumstances demanded, and quinine mixture three times a day as an antiperiodic and tonic. His diet was gradually changed from liquid to solid. He was discharged cured of his dysentery on November 6th. As, however, he had been much reduced, and still showed symptoms of fever, he was granted a month's leave, which completely set him up.

Case 7. *Acute dysentery.*—Three years ago I was asked to visit a lady suffering from symptoms of acute dysentery. She had passed some years in Calcutta, but had been at home upwards of four years. During this period she had often had intermittent fever and malarial neuralgia. Her present illness was ushered in, about three o'clock in the morning, with violent pain in the abdomen, temporarily relieved by free evacuation of the bowels. When I saw her at 10 a.m. she had been frequently moved. The griping and straining were severe. She was pallid and pinched. The pulse was slightly accelerated and irritable, and there was moderate elevation of temperature, 99·5° The tongue was clean and moist, and there was some thirst. She had just taken some tea and toast, which aggravated her symptoms. Her next motion was passed in the closet-stool pan for my inspection. It consisted of rose-coloured mucus, gelatinoid exudation, and blood without a trace of feculence. She declared when the pain was on it was in all parts of the abdomen, every portion of which appeared then to yield tenderness on pressure. In the intervals of freedom from tormina the tenderness could be clearly localised to the region of the sigmoid and splenic flexure of the colon. I prescribed a nurse, absolute rest in bed, the use of the bedpan, fomentations, turpentine epithems, and three powders, each composed of twenty grains of ipecacuanha, five grains of bismuth, and five grains of carbonate of soda, the first to be taken at once, the second at bedtime, and the third the following morning. Head to be kept low, and only small pieces of ice to be munched for an hour after each dose, if required to quench thirst. After the feeling of nausea and sickness had subsided she was ordered to take chicken broth or milk diluted with soda-water *ad libitum.* Arrangements were made to have each evacuation preserved separately for examination, as is done in Calcutta, and now, I believe, throughout India in cases of this description.

At my evening visit, I found that six motions had been kept as directed, disinfected by a solution of carbolic acid. The first three consisted of much the same material as the motions first examined; the fourth showed traces of feculence, the fifth more, and the sixth still more with slight decrease of blood and glairy gelatinoid exudation. The tormina and tenesmus had been severe, but were coming on at longer intervals. She had kept the powder down, but retched a few times. The skin was moist, pulse steady, and there was a tendency to

doze off to sleep. In about an hour the nausea disappeared, and she had taken a good deal of liquid nourishment.

At my morning visit on the second day it was found that she had retched again after the powder, but retained it and slept a good deal during the night. Pulse soft; skin moist; countenance less anxious. The griping and straining were much less severe. She had been moved six times. The motions all contained feculence and bile with less dysenteric material, the two last passed being characteristic "ipecacuanha stools." Still, as on decanting the supernatant watery liquid generously tinged with the colouring matter of the bile, there was a sediment of blood and slime, and as, with each evacuation, there was a certain though much diminished amount of tormina and tenesmus, the third dose was given.

Evening visit: the powder given in the morning had passed out of the stomach, causing nausea, but without retching and vomiting. She had taken a fair amount of nourishment during the day. Three brown fluid motions had been passed and preserved. On being washed in the usual way, the liquid part was seen to be largely mixed with biliary pigment, whilst the sediment yielded a small quantity of gelatinoid mucus without blood. The tenderness over the sigmoid and splenic flexure of the colon and the tormina and tenesmus had ceased. The general aspect of the patient confirmed the impression that the dysenteric process had also ceased; no more ipecacuanha required. But it was deemed judicious to administer an anodyne enema composed of forty minims of laudanum and an ounce of starch at bedtime.

The report on the third morning showed that she had slept well. No stool. No discomfort. Sago and arrowroot added to food. From this time the patient was practically well. A couple of days afterwards a small dose of castor-oil was given, after which she left her room apparently none the worse for the storm through which she had so recently passed.

Case 8. *Acute dysentery.*—On April 4th, 1881, I was consulted by a retired officer of the Indian army. He had been upwards of twenty-one years in India, chiefly in most malarious districts. He had had, during his service, three attacks of acute dysentery—all promptly cured by large doses of ipecacuanha. In his earlier service he had suffered much from intermittent fever. During the last twelve years of residence he had escaped such attacks; but a couple of years before his retirement he had passed through a severe seizure of typhoid fever, and the year before his leaving the country he was the subject of mild rectal dysentery. At the time of the illness under consideration he had been at home upwards of four years, enjoying fairly average health. During his sojourn in India he worked hard and successfully, living carefully and abstemiously, and since his return he has been practically a water drinker. A fortnight before the development of his present indisposition he undertook a long railway journey in very uncongenial weather, got chilled and contracted ague, which readily yielded to a good purgative and a few large doses of quinine. It left him, however, weak and ill able to withstand the alternations of temperature of an English spring. He was dyspeptic with evidence of an overloaded portal system, which was alleviated from time to time by a nocturnal pill of podophyllin and euonymin, and a morning dose of Hunyadi Janos water. He also doctored himself with tonic doses of quinine. But the appetite remained

capricious and impaired, whilst the indigestion and constipation underwent no apparent diminution. He had then resolved to consult me, but failed to do so until the morning of the 4th of April. He then stated that he was turned out in the middle of the night with a violent fit of purging after being much griped, that he had been moved several times since, and that, thinking he "was in for his old enemy," he passed the last motion in the chamber utensil, and that he had given orders that it be kept for my inspection. It consisted of nothing but blood and rose-coloured slime. The griping was increasing, so was the tenesmus, but this was not very severe. There was considerable tenderness over the cæcum and ascending colon, less, but still some tenderness over the splenic flexure and sigmoid. His countenance looked anxious and indicative of much depression and distress, pulse slightly quickened, with a temperature of 100°. The skin was moderately hot and dry; tongue coated but moist. The case proved obstinate, doubtless having been materially aggravated by his having driven to my house for advice, for the tenderness, tormina, tenesmus, slimy, bloody, and gelatinoid evacuations did not yield completely until he had taken eight scruple doses of ipecacuanha with bismuth and soda—a morning and evening dose—assisted by turpentine stupes, fomentations, poultices, a nightly anodyne injection of drachm of laudanum in an ounce and a half of starch, absolute rest in the recumbent position, the use of the bedpan, excellent nursing, and careful feeding with milk and chicken broth. He bore the ipecacuanha well, only the first two doses causing much retching and some vomiting. The subsequent doses only produced nausea, not lasting over an hour. The powder was always retained. The head was kept low, and nothing but a few lumps of ice were allowed until nausea disappeared. In the intervals he took a fair amount of liquid nourishment. After the fourth dose the characteristic stools were passed; but not until the eighth dose had been taken were the stools free from blood and slime. Then all general and local discomfort and pain ceased, and he afterwards made a rapid recovery. A week after the cessation of the malady the patient left Brighton for the milder climate of Torquay.

Case 9. *Acute dysentery.*—Mrs. ——, aged thirty-one, a pure European, born in India, mother of six children, five of whom are living, has spent the greater portion of her life in Calcutta, been in England for the benefit of her own health and to arrange for the education of her children, about six months. When a child she remembers having had dysentery. She has been a frequent sufferer from malarious fever. She had a sharp attack of ague on the 15th of February last. At midnight she was roused out of bed with severe griping, temporarily relieved by a copious evacuation. Between this and 5 p.m. on the 16th, when I saw her, she had had fourteen motions, four of which had been kept for inspection. The tormina and tenesmus had been severe. I found her pallid, parched, and prostrated from pain and repeated calls to stool, with a weak, slightly accelerated pulse, a temperature of 99·5°, a dry and pinched skin, a coated but moist tongue, little relish for any kind of food, moderate thirst, and distinct tenderness on pressure over the sigmoid and splenic flexure of the colon only. The griping was frequent and "excruciating." The food and drink she had taken increased this and always brought on desire to go to stool, defæcation being attended by much prolonged tenesmus. On washing the evacuations preserved,

these were found to consist of a good deal of blood, gelatinoid exudation, some ropy mucus, without any fæcal admixture. I prescribed absolute rest in bed, a large linseed-meal poultice sprinkled with mustard to the abdomen, every three hours, and twenty grains of ipecacuanha made up into five pills at once, and a similar dose in the morning, if needful, and liquid diet consisting of beef tea and diluted milk. On visiting her at 11 a.m. on the 17th she reported that she became nauseated half an hour after taking the pills, and vomited once, bringing up, she thought, some portions of them, after which she slept and has only been moved three times since, with slight griping and tenesmus each time, and has not used the bedpan during the last six hours. The whole half filled the chamber utensil and presented all the appearance of the ipecacuanha stool—brownish-yellow in colour, liquid in consistence, feculent in smell, free from blood and gelatinoid exudation, and yielding a small proportion of ropy-like mucus. At 8 she took a cupful of beef tea and another at my visit, without discomfort. Her countenance has lost all trace of anxiety; the pulse is stronger and normal in frequency; temperature 98·5°; no tenderness; no griping; skin soft. She had not taken the second dose. Ordered a tablespoonful of chalk mixture with ten grains of soda and five minims of Liquor Opii Sedativus, after each loose motion and an ounce of quinine mixture (three grains to ℥j) every four hours. Same diet as before, with sago or corn flour.

18th, 11 a.m.—Had one easy loose motion yesterday at 12 noon and another at 4 p.m., after which she took a dose of chalk medicine. Had not been moved since; no occasion, therefore, for any more chalk mixture. Slept soundly and well. The diet to be improved by addition of custard puddings and two light boiled eggs. Up and about.

19th.—Had a natural motion this morning. Quite well in all respects; but she was directed to continue the antiperiodic for a few days.

Before the late Dr. Parkes wrote his 'Remarks on Dysentery and Hepatitis in India' (1846), enlargement and inflammation of the solitary glands in dysentery had been observed and described by Hodgkin, Copland, Ballingall, Raciborski, Dr. Murray and Mr. Twining, of the Bengal, Dr. Lorimer, of the Madras, and Dr. Bird, of the Bombay Medical Service. But, as Parkes says, "Most of these writers appear to have regarded their existence as incidental, and not to have proclaimed their relation to dysenteric ulceration with sufficient accuracy and distinctness. Ballingall and Twining describe them as if they were pustules. Murray describes them very accurately, but calls them vesicles and pustules." It is, however, to the researches of Parkes whilst serving with European troops in Madras and Burmah that the true nature of these morbid conditions and their association with the solitary glands were first correctly established. Speaking of these glands he says, "I have considered them not to be large mucous crypts for the following reasons: they present the appearance of round opaque bodies, without apparent orifice, embedded in the mucous mem-

brane, and even apparently attached to the submucous cellular tissue. In the early stage of dysentery their contents are white-yellowish, and apparently thickened and starchy. They are sometimes streaked or striated on the surface, and bear on the summit, in some cases, a small black point, which looks like an orifice closed up. This is not, however, general, or even common. Under the microscope the mucous membrane presents the usual appearance of small honeycomb cells. In a dysenteric case which has lasted two or three days they are still more obvious. A minute vascular ring surrounds them, they become prominent, and a little hardened to the touch. In distribution these glands appear equally numerous in the sigmoid flexure as in the cæcum, and on this account I am disposed to regard them as perhaps the excretory organs of the colon. . . . Whatever opinions may hereafter be entertained regarding their nature and functions, one point has been fully established by my dissections, viz. that they are the seats and centres of ulceration in tropical dysentery."

At page 410 of 'Quain's Dictionary' (1882) my own observations of the changes occurring in the solitary glands, during the earlier career of tropical dysentery, are described from dissections. The *first* visible change is congestion, the vessels surrounding and penetrating the capsules being turgid and engorged with blood. The *second* change is augmentation of their contents from the accumulation of albuminous exudation, and enlargement "from the size of a millet-seed to that of a small shot" or larger (Baly). The *third* stage is, provided the inflammation advances, rupture of some of the capillaries in the interior of these little vascular glands, extravasation of blood, with the area of the ordinary dark point on the free aspect increased. The *fourth* stage is now marked by atrophy and molecular disintegration of the free aspect of the capsular wall, and escape of its morbid gelatinoid blood-tinged contents into the canal of the intestine. This is the rule, but, in very exceptional cases, the capsule may burst through the attached portion, lighting up inflammation in the neighbouring connective tissue and muscular coat. In a large number of instances the morbid process may stop short, under proper treatment, at any of the first three mentioned stages, and repair is then effected by resolution. In many cases the morbid action is cut short after the completion of the fourth stage without further extension of the disease. The adjoining follicles of Lieberkühn do not, in these

cases, necessarily participate to any great extent in the diseased process. Under these conditions, when the whole of the exudation has been expelled, the glands regain their tone and functions, and recovery—rapid and complete—ensues. It is not often possible to illustrate these conditions in the post-mortem room; because when death supervenes from dysentery alone the ravages committed upon every structure of the mucous membrane are so extensive as to destroy the earlier physical phases of the disease. In some cases, however, which have died from intercurrent affections, I have been able to demonstrate the earliest stages successfully to my students at a period prior to the implication of Lieberkühn's follicles, of which the mucous membrane is, in great part, composed, and to exhibit to them the gelatinoid exudation, termed by others "gelatinous mucus," free from or tinged with blood taken from enlarged and diseased solitary glands.* In some specimens I have been able to recognise every gradation of disease from this point to molecular disintegration of these glands with a few of the surrounding tubular glands, their enfoliation *en masse*, ulceration, more or less extensive, and sloughing in varying degrees as to extent and depth.

With reference to this question Sir Joseph Fayrer quotes Dr. McConnell, Professor of Pathology in the Calcutta Medical College and Hospital, who says, "The disease commences essentially and primarily in the gland structures. I have had opportunities of seeing several cases, *i.e.* post mortems, at a very early period; and then the only visible alteration is in the solitary glands and follicles of Lieberkühn, the former especially, which I have found enlarged to the size of small hazel nuts, and filled with that glazy, semi-transparent, gluelike mucus which we almost always find voided with the earliest evacuations during life in this disease" (1881).

In the cases cited there was specific inflammation of the solitary glands, and by extension, in some, of the tubular follicles, either stopping short of, or advancing to, ulceration to a limited extent. In all, the registration and recognition of morbid products, such as blood, gelatinoid exudation, ropy mucus, and so forth, as they are passed, in accordance with the method first systematically adopted by the late Dr. Edward Goodeve, and fully described by Professor Chevers in Sir Joseph Fayrer's "Lettsomian Lectures" (1881), are of primary importance. By the employment of carbolic acid, or

* See also 'Indian Annals of Medical Science,' p. 190, No. 23, 1868.

other colourless deodorant, the examination of the stools is deprived of the greater part of its repulsive character. The procedure is essential to accuracy of diagnosis and indispensable to the formation of a sound prognosis, whilst it enables the physician to appraise, at its true value and with wonderful exactitude, the progress of the pathological changes taking place, and the effects produced by our sovereign remedy—ipecacuanha. Hence the care with which the dejecta are washed and scrutinised with a view to note the character and quantity of the bloody mucus, gelatinoid exudation, and ropy or tenacious mucus expelled; their diminution and eventual disappearance under the administration of large doses of ipecacuanha constituting as they do, with the cessation of tormina and tenesmus, the signals for stopping the further use of the drug.

Invaluable as ipecacuanha, in large doses, every twelve hours, and associated in the intervals with the use of such other medicine as may be indicated, quinine, for example, for the correction of malarial poisoning or fever, small anodyne enemata for the relief of tenesmus or tormina, fomentations and counter-irritants to the abdomen, and with nutritious and easily-digestible food in a liquid form, is in the management of the simple and non-sloughing forms of acute dysentery, it is, *par excellence*, in ordinary sloughing, as distinguished from gangrenous dysentery, where its curative, or rather its conservative, power is conspicuously displayed. In this the immediate danger is the extension of the sloughing into the gangrenous form, and even when this is averted so much destruction of tissue as to involve subsequently cicatricial contraction and eventual lifelong embarrassment or fatal obstruction, not to mention the thickening and consequent narrowing of the lumen of the intestine, and other troubles, should the case become chronic. It therefore occurred to me, after much observation of the therapeutical action of ipecacuanha in the simpler and less severe forms of the disease in which sloughing did not happen, that, if given in the manner advocated in this communication, it might arrest the progress of sloughing dysentery, limit the area of its destructive ravages, and so place the intestinal mucous membrane, after the dead portions of tissue or sloughs had been successfully cast off, as nearly as possible in the same advantageous position as it usually occupies in an ordinary case in which ulceration has occurred; that, in like manner, the risk of transition into a hopeless condition of gangrene or into a chronic state and the dangerous results adverted

to would be materially diminished. An attentive perusal of the subjoined cases under my care some years ago in the Calcutta Medical College Hospital will show that my anticipations were fully realised. The microscope was, I may premise, in constant use to determine the character of the sloughs, which were also floated out in water and outlined daily, as they were observed, on the bed-head tickets.

Case 10. *Acute dysentery, the sequel of Asiatic cholera, with moderate sloughing of mucous membrane.*—Luckman, aged thirty, admitted into the Medical College Hospital on December 5th, 1866, with dysentery of twelve days' duration, following an attack of cholera. Has never been free from looseness since his discharge, a fortnight ago. There is a good deal of griping and straining with marked general debility. He passes blood and slime in the stools. Ordered by the officer on duty fomentations to the abdomen, and fifteen grains of ipecacuanha and milk diet. 4.30 p.m.—Has had three bloody and slimy motions with a very small quantity of feculence—passed with much griping and straining—no vomiting. There is tenderness over the sigmoid flexure. Ordered a drachm of ipecacuanha with ten grains of bismuth night and morning, and to repeat the fomentations and liquid diet.

6th.—Had six motions, with less tormina and tenesmus, consisting of a good deal of semi-liquid feculence with a smaller quantity of blood and slime. No vomiting; pulse good. Pergat. Ice 1 lb. 6 p.m.—Passed five stools of the same kind during the day. Improving. Pergat.

7th.—Moved five times in the night. Griping and straining still less. Feculence of a semi-liquid consistence abundant. A few small blood-clots and several small mucous sloughs—one or two having attached to them a few transverse unstriped muscular fibres—were discovered on washing the stools. Pergat. Turpentine stupes and an anodyne enema at bedtime. 6 p.m.—Improving. Passed three motions yielding an increased proportion of feculence and a few small superficial sloughs of mucous membrane. Retched several times. Pergat.

8th.—Had five stools during the night, composed of a quantity of brownish and greenish semi-fluid feculence which, on washing, yielded several small sloughs of mucous membrane. There was no slime, no trace of blood. Omit ipecacuanha. Ordered an ounce of chalk mixture with twenty minims of laudanum and twenty grains of carbonate of soda, *pro re nata*. Diet as before. 6 p.m.—Two motions, wholly feculent, semi-liquid. Pergat.

9th.—Bowels not moved during the night. Doing well. Pergat.

10th.—Two figured motions. Pergat. Soup Oj to diet.

11th.—No stool during last twenty-four hours. Pergat.

12th.—One formed motion. Appetite good. No tenderness on pressure over the sigmoid; no tenesmus. Patient never had a bad symptom afterwards. His diet was improved, and he eventually left the hospital perfectly well.

Case 11. *Sloughing dysentery, supervening upon a previous uncured attack of the acute form of the disease.*—Motee, aged twenty-five, Hindoo, resident of Shoba Bazaar, admitted into the Medical College Hospital on November 26th, 1866. He had been suffering from dysentery during

the past six weeks, passing several stools daily mixed with slime and blood, and attended with tormina and tenesmus. On admission, motions of this character were passed involuntarily in the bedclothes with much griping and straining. Pressure over the colon, particularly over the sigmoid, elicited marked pain and tenderness. Tongue coated with greyish fur, but moist. Skin slightly warmer than normal. Pulse moderately full, but easily compressible. Appetite indifferent. Ordered, by the admitting officer, five grains of Dover's powder three times a day and milk and sago. 6 p.m.—Has passed many scanty stools with great griping and straining. Ordered an anodyne enema.

27th.—Similar symptoms continued until I saw him this morning, when I prescribed a drachm of ipecacuanha with ten grains of bismuth, night and morning, an anodyne enema at bedtime daily, milk diet and ice 1 lb.

28th.—Retched a good deal, but did not reject the powder; griping and straining diminished. Purged more than a dozen times. Motions consist of a yellowish fluid, mixed with some semi-solid fæcal matter. There was also a piece of mucous slough and several portions of gelatinoid exudation tinged with blood. The last motions contained much less exudation than the earlier ones. Pulse feeble. Pergat. To have also an ounce of rum mixture three times a day.

29th.—Has had several stools, consisting of clay-coloured semi-solid feculence, with small portions of mucous sloughs. Amount of gelatinoid exudation still less. Has only retched once, though he has been nauseated after each dose. Pergat.

30th.—Motions less frequent, and consisting of clay-coloured fluid feculence with six mucous sloughs, one of them of a black colour, varying in size from a sixpence to a shilling. No gelatinoid exudation; no other inflammatory indication. Appetite good. Pulse better. Expression indicative of relief from oppression, anxiety, and distress. Omit ipecacuanha. Continue rum mixture every four hours. Ordered chalk mixture ℥j, half a drachm each of tincture of ginger and tincture of catechu, twenty minims of laudanum, and twenty grains of carbonate of soda, *pro re nata*. An anodyne enema, morning and evening, and diet as before.

December 1st.—Purged three times since last report, motions consisting of light-yellowish fluid feculence, in which was found, on washing the same, a soft and partly macerated slough about the size of a shilling. Better in all respects. Pergat.

2nd.—The disease has been subdued. The whole of the sloughs appear to have passed away. Reparation is in progress. Patient is in perfect comfort. Tongue clean; circulation tranquil; slept well. Expression of countenance favorable. Omit the enema. Continue other medicines, *pro re nata*. Diet as before.

3rd.—Passed three figured motions. To have now only rum mixture. Diet as before.

4th.—One solid motion; wants more to eat, not deemed advisable to comply with his request. Pergat.

5th.—One figured stool. Doing well all round. Pergat.

6th.—Had two scanty stools in the night; abdomen, on percussion and palpation, gives no indication of accumulation of fæces above the lesions undergoing repair. Hence, no castor-oil purge was deemed expedient.

7th.—Two natural evacuations. Pergat.

8th.—Doing well. Pergat.

9th.—Bowels regular, had only one good figured motion. Pergat. Ordered fish in addition to milk diet.

12th.—Has borne increased diet with impunity since the 9th. Discharged at his own request.

Readmitted on December 14th. After going out he had indulged in excesses of diet which, he said, had brought on a fresh attack. The probability is that, though his motions had been figured and natural for some time before he left hospital there still remained some ulcers imperfectly repaired. Slime and blood abounded in the scanty evacuations. There was considerable griping and straining. Ordered, by the officer on duty, fifteen grains of ipecacuanha, five grains of carbonate of soda, and ten grains of acacia gum in a sufficient quantity of water immediately. Milk diet and sago. 6 p.m.—Has been repeatedly at stool since morning with tormina and tenesmus, motions consisting of slime and blood without feculence. Ordered a drachm of ipecacuanha with ten grains of bismuth, night and morning; anodyne enema daily at bedtime; and milk diet with a pint of beef tea.

15th.—Tormina and tenesmus about the same; the latter causing considerable annoyance. Calls to stool have been very frequent, motions consisting of yellowish liquid with a little slime. Pulse weak; looks depressed. Vomited once. Pergat. Rum mixture ʒj every three hours. 4 p.m.—Motions frequent, composed of yellowish fluid with a sediment of gelatinoid exudation tinged with blood. Griping less; tenesmus still troublesome and annoying. Tenderness on pressure along the descending colon and sigmoid. Been nauseated. Pergat.

16th.—Symptoms much the same; intensity of the pain, tormina, and tenesmus diminished. On washing the stools a quantity of ropy exudation was discovered. Pergat. 4.45 p.m.—Much the same; nauseated after the morning dose and vomited once. Pergat.

17th.—Many evacuations free from blood, slime, and exudation. Little or no griping, moderate tenesmus at each call to stool. Better in all respects. Omit ipecacuanha. Ordered an ounce of chalk mixture, twenty minims of tincture of opium, and twenty grains of carbonate of soda, *pro re nata;* and to continue rum mixture, enema, diet and beef tea with four ounces of port wine. 4 p.m.—Has only had one semi-liquid and feculent evacuation during the day. Pergat.

18th.—Had several motions in the night, consisting of thick yellow feculence without exudation or blood; no griping and very little tenesmus. Pergat. 6 p.m.—Had one feculent motion in the morning, none since. Pergat.

19th.—Two motions, composed of more consistent greenish-yellow feculence. No stool during the day. Pergat.

20th.—Two formed yellow-coloured stools; no griping and much moderated tenesmus. Pergat. From this date patient made rapid improvement. The enema was discontinued on the 25th. He took the chalk mixture with soda and laudanum whenever required, rum mixture, wine and diet as before, until January 5th, when these were changed for ten minims of dilute nitro-muriatic acid mixture in an ounce of infusion of cheyretta three times a day; half diet, with fish for breakfast, but without dhal. His diet was still further strengthened by the addition of a pint of milk; and for some days before he was discharged from hospital on January 10th he could digest, with impunity, dhal and chupatties—unleavened bread.

Case 12. *Acute dysentery terminating in a very moderate amount of sloughing.*—J. A—, aged twenty-eight, West Indian custom-house officer, resident of China Bazaar, admitted into the Medical College Hospital on December 31st, 1866. Patient is tolerably stout, of fair complexion, and has been suffering from tormina and tenesmus with purging for the past five days. The stools consist mainly of slime and blood. The straining is so great that the gut protrudes after each evacuation. His tongue is coated and dry; pulse firm and rather accelerated. Ordered a drachm of ipecacuanha with ten grains of bismuth, night and morning. Anodyne enema at bedtime daily; milk diet and ice 1 lb.

January 1st.—Many motions, scanty, consisting of a small quantity of soft feculence with much slime and blood. Tormina and tenesmus less. There is pain on pressure over the sigmoid with tumefaction of the gut. The powder nauseated him, but he did not vomit. 5 p.m.—Had many scanty evacuations of a brown colour, containing slime and blood, with little griping or straining. Pergat.

2nd.—Had several scanty motions without griping, but there is still tenesmus and prolapsus after each evacuation. He also experiences pain and difficulty in micturition, from reflected rectal irritation and mischief. The motions consist mainly of feculence with a small quantity of bile-stained gelatinoid exudation. Omit ipecacuanha. Ordered an ounce of chalk mixture, with ten minims of laudanum and twenty grains of carbonate of soda every four hours, or *pro re nata;* to continue the enema, and to add to his diet sago and a pint of soup. Evening: Has got malarious fever, for which, during the hot stage, the ordinary diaphoretic mixture was prescribed. At 7 p.m. he vomited, and was purged and griped with straining, the evacuations consisting of a dark-brown liquid feculence. To have quinine mixture ʒj (gr. v ad ʒj) every four hours, to be commenced when the sweating begins or when the skin becomes cool.

3rd.—No return of fever; stools copious and watery; no griping; no straining; no blood; no slime. A drab-coloured superficial slough was discovered in the motions this morning after washing them and examining the sediment. Pergat.

4th.—No more fever; no stool; no griping or tenesmus. Pergat.

5th.—Doing well. Passed two good feculent motions. Pergat.

6th.—Doing well; no stool. Omit all medicines.

7th.—Doing well. Ordered fifteen minims of dilute nitro-muriatic acid in an ounce of infusion of cheyretta, three times a day.

10th.—Discharged cured.

Case 13. *Acute dysentery ending in sloughing.*—A. H—, aged twenty-nine, a seaman on board the Malabar, an Englishman, admitted into the Medical College Hospital on July 26th, 1867. States that he has had diarrhœa during the last three weeks. During the past nine or ten days, the stools, which were very frequent, contained slime and blood; and there has been much griping and straining with tenderness over the abdomen. He has passed, during the last three days, about nine motions daily. There is now pain on pressure over the cæcum and sigmoid with fulness over the latter, and much tormina aud tenesmus. There is abundance of slime, but no blood in the motions. Pulse soft, slightly accelerated; appetite indifferent. Tongue coated, which, with mouth and fauces, is dry. Ordered a drachm of ipecacuanha with ten grains of bismuth, statim, milk diet and sago, ice 1 lb.

27th.—Nine motions, had been nauseated and vomited four times. Griping and straining diminished. Repeat the ipecacuanha morning and evening. Diet as before.

28th.—Eight motions consisting of greenish-yellow fluid, without any blood or gelatinoid exudation, but yielding one soft, macerated, pulpy, shaggy drab-coloured slough, measuring when floated out about two inches in length by one inch in width, and constituted of altered mucous membrane, submucous connective tissue, and exudation material. Complained of much griping, but the straining is greatly decreased. There is still tenderness, though not to the same extent as before, on pressure over the cæcum and sigmoid—particularly over the former. Nauseated and retched once. From the absence of gelatinoid or dysenteric exudation and blood it was inferred that the morbid process was on the decline. The ipecacuanha was therefore omitted. Ordered an ounce of chalk mixture, with half a drachm each of tincture of kino and ginger and twenty grains of carbonate of soda, every four hours. An anodyne enema, horâ somni; pint of beef tea; two ounces of port wine and diet of milk and chicken broth.

29th.—Two motions; no griping; no straining. Still shrinks on pressure being applied to the cæcum; but the fulness has disappeared. Pulse improved. Tongue coated in the centre with a yellow-brown fur. The stools consist of a yellowish fluid, having a healthy feculent odour, and yielding, on washing, one soft, macerated and drab-coloured slough measuring two inches by one. A blister was now applied over the cæcum. Medicine and diet as before with a bottle of soda-water. 6 p.m.—Blister has risen well. Had four more feculent, though liquid motions, without slime or blood, or sloughs. Is quite free from pain, but complains of flatulent distension, due, doubtless, to spasm and consequent constriction at the sites of ulceration. Pergat.

30th.—One motion since last report; no pain, but the flatulency continues, though in a mitigated degree. Pergat.

31st.—Had three stools yesterday and one this morning, composed of soft feculence without blood and slime, and unattended with griping or straining. Pergat. 8 p.m.—No stool. Doing well.

After this the patient never had a bad symptom. It became occasionally necessary to assist nature to overcome the constrictive spasm which impeded the onward flow of the intestinal contents, at the seat of sloughing and ulceration, by the administration of small doses of castor-oil followed up by large tepid water enemata. The chalk mixture, &c., and anodyne enema were omitted on August 3rd, when the motions became formed. On the 8th, half diet without vegetables, but with eight ounces of extra bread, was allowed. Port wine as before. On the 11th, mutton chops were ordered, care being taken, as recovery advanced, to examine the motions with a view to see whether the food he took had been properly digested. From the 3rd to the 17th A. H— had from one to two healthy evacuations daily, was rapidly regaining health and strength, and quite free from any discomfort. Discharged cured.

Case 14. *Acute dysentery, following malarious fever, terminating in sloughing.*—Sheik Nusseeroodeen, aged thirty, Mahommedan sailor, admitted into the Medical College Hospital with dysentery, on December 24th, 1866. It followed an attack of malarious fever. Has been suffering from tormina and tenesmus attended with the passage of frequent, scanty, bloody and slimy motions during the last fifteen days. He is

greatly reduced and emaciated; the pulse is weak and thready. Ordered, by the officer on duty, ten grains of ipecacuanha and five grains of bismuth at once; an ounce of rum mixture every four hours, and milk diet. 6 p.m.—Passed six slimy, bloody stools during the day. Much tormina and tenesmus. Ordered a drachm of ipecacuanha and ten grains of bismuth, statim.

25th.—Had six scanty motions composed of yellow feculence with several portions of rose-coloured gelatinoid exudation, a few small blood-clots, five dark-coloured sloughs, and some undigested food. There is pain on pressure over the sigmoid flexure and descending colon, and thickening of the gut can be plainly detected through the attenuated abdominal parietes. Pulse not accelerated, but small and easily compressed. Tongue coated in the centre with creamy fur and dry. Conjunctivæ tinged yellow. Omit ipecacuanha. Ordered an ounce of chalk mixture with twenty minims of laudanum and twenty grains of carbonate of soda, *pro re nata*, an ounce of rum mixture every three hours, and an anodyne enema night and morning. Large sinapism to be applied over abdomen. Diet as before. 6 p.m.—Passed one scanty feculent motion during the day free from dysenteric products. Pergat.

26th and 27th.—Has had no evacuation. Pergat.

28th.—One soft yellow formed motion with hard lumps of brown fæces (scybalar), and a few pieces of gelatinoid exudation, one small blood-clot, and two soft feculent drab-coloured sloughs and some undigested rice. There is less pain on pressure over the sigmoid; pulse feeble; tongue clean, but dry and glazed. Repeat the ipecacuanha and bismuth, bis die; rum mixture, enema and diet as before. 6 p.m.—No stool; vomited once. Has taken and kept down all nourishment. Pergat.

29th.—Had one stool this morning. It consisted of greenish soft feculence with small lumps of yellow fæces, in which were found, on washing, gelatinoid exudation tinged with blood, and two soft, pulpy, drab-coloured superficial sloughs. Pergat.

30th.—Had three soft but formed motions in which were found, on washing, two pieces of gelatinoid exudation tinged with blood; vomited some bile an hour after taking the powder. Pergat.

31st.—Had three stools, consisting of yellow and formed feculence. On being examined, these yielded a sediment of spicular fæces with a small quantity of colourless gelatinoid exudation. Omit the ipecacuanha. Pergat.

January 1st.—No stool; free from pain; tenderness and tumefaction over the sigmoid gone. Found out that patient is an opium eater. Pergat. Ordered also two grains of opium, night and morning.

2nd.—Had one healthy-formed motion this morning; no griping; no straining. From this date the patient gradually improved and gained strength, having one or two figured motions daily. He had, however, been so debilitated by the previous attack of malarious fever and this invasion of dysentery, that it took a long time before he was considered fit to be discharged. Nitro-muriatic acid with quinine and iron, nutritious and easily digestible food were given until March 3rd, when he left the hospital restored to health.

Case 15. *Acute dysentery ending in sloughing.*—J. H. D—, aged forty-three, an English groom, belonging to one of the stable-keepers in Dhurmtollah, admitted into the Medical College Hospital on May 31st, 1867. States that he has been ill with dysentery for ten days, passing

eight or nine stools daily. There has been much griping and straining. There is considerable tenderness over the course of the colon—most marked over the cæcum. Feels very weak and depressed. Ordered a drachm of ipecacuanha with ten grains of bismuth, morning and evening. Milk diet and ice 1 lb.

June 1st.—Twenty unsatisfactory evacuations, with a small quantity of liquid feculence, much gelatinoid exudation, and one shaggy slough of mucous membrane, measuring, when floated out, about an inch in length and half an inch in width. Pergat. Turpentine stupes and fomentations.

2nd.—Thirteen scanty stools, consisting of nothing but gelatinoid exudation, preceded and passed with much griping and some tenesmus. Tenderness over cæcum less, but it is increased over the splenic flexure of the colon, and there is great rigidity of the left rectus abdominis. No sickness or vomiting. Pergat. Also an anodyne enema morning and evening.

3rd.—Thirteen scanty motions, sediment of which yielded, on washing, two small ash-coloured sloughs of mucous membrane and a quantity of blood-stained gelatinoid exudation. Nauseated after the powder, but did not vomit. Pergat.

4th.—Passed seven or eight motions attended with little or no griping. These consisted of semi-solid feculence without either blood or gelatinoid exudation. On careful washing, one ash-coloured and two bile-stained sloughs of mucous membrane, each about the size of a shilling, were found. Patient is now in comparative comfort, has a good appetite, and declares himself free from pain. Repeat enema and diet as before; to take also an ounce of chalk mixture after each loose evacuation.

6 p.m.—Has had several liquid feculent stools yielding, on washing, some firm portions of fæces without slough, blood, or gelatinoid exudation. Pergat.

5th.—Two copious evacuations consisting of liquid feculence, portions of solid fæces, with one solid slough as large as a shilling. No griping; no tenesmus; no dysenteric exudation. Pergat. Also sago and broth.

6th.—Six free motions, feculent and semi-solid, containing no blood or slime, but yielding, on washing, a tolerably large and firm slough of mucous membrane, about two inches long and an inch and a half broad. Patient says he now feels "all right" and wants more to eat. Pergat. Also four ounces of port wine.

7th.—Bowels not opened since last report. Pergat. Omit the enema. From this date patient made a steady and speedy convalescence. He was gradually promoted from liquid to solid and more substantial diet.

On the 13th his motions were figured. After this he required the occasional use of castor-oil guarded by an opiate, or a simple large warm-water enema to empty the lower bowel.

On the 22nd he was ordered nitro-muriatic acid in bitter infusion, three times a day.

He was discharged cured on June 28th, looking the picture of good health.

Case 16.—M. K—, aged thirty-three, an Irish sailor, admitted into the Medical College Hospital on July 12th, 1867. States that he has been suffering from symptoms of dysentery during the last ten days, passing many stools daily with much griping and straining. The stools contained chiefly blood and slime. There is tenderness on pressure

over the sigmoid flexure. Tongue furred but moist. Feels weak and prostrated. Ordered a scruple of ipecacuanha with ten grains of bismuth, morning and evening. Milk, sago, and beef tea, and ice 1 lb. 6 p.m.—Passed five or six motions since the morning visit, consisting mainly of slime without blood. Pergat.

13th.—No vomiting; many stools, filling three quarters of a stoolpan and composed of a greenish fluid, emitting a most offensive odour. On washing, these yield a sediment of gelatinoid exudation partly coloured with blood, some dark-looking exudation, and a soft pulpy slough, drab on one side and black on the other. The tenesmus has been severe, countenance depressed and anxious. There is still pain on pressure over the sigmoid. Nauseated after the powder and retched twice, bringing up some sour fluid. Pulse steady, though weak. Pergat.

6 p.m.—Has had twelve scanty stools with much tenesmus. Pergat. Also anodyne enema.

14th.—Eight offensive motions consisting of greenish fluid which yielded yellow-stained gelatinoid exudation, dark granular sediment, one soft macerated white slough, ragged at the margins, and showing some red thick points in the centre. This slough was constituted of altered mucous membrane, submucous connective tissue, degenerated and granular unstriped muscular fibre, and exudation matters. No griping. Tenesmus much diminished. Pergat. 8 p.m.—Six offensive motions without any more sloughs and without slime. Omit ipecacuanha. Ordered an ounce of chalk mixture, half a drachm each of tincture of ginger and kino, and twenty grains of carbonate of soda, every four hours, or *pro re nata*. Anodyne enema horâ somni. Diet as before.

15th.—Ten motions without griping and with little straining. The evacuations are composed of a yellow-green fluid, and yielded a few portions of feculent material and four whitish-looking sloughs, two small ones about the size of a sixpence being composed of altered mucous membrane and exudation, and two, varying in size from a half to a crown piece, were shaggy on the free aspect and smooth on what had been the attached surface, to which several degenerated and granular portions of the unstriped transverse muscular fibres adhered. Pergat. Also to take two ounces of port wine.

16th.—Two motions in the day and two in the night. No griping or straining; looks much better, but the pulse is weak and jerking. Motions consist of yellowish fluid with a large quantity of ropy catarrhal exudation (styled-ropy mucus) increased quantity of fæces, and four thick shaggy sloughs—drab coloured on one side and red on the other. From this date the patient steadily mended. Nitro-muriatic acid in cheyretta infusion was substituted for other medicines on the 20th, when his motions were formed. He continued uninterruptedly to convalesce till August 1st, when he was discharged cured.

Case 17. *Acute dysentery terminating in sloughing.*—W. C—, aged forty-five, a steward of the ship Medusa, admitted into the Medical College Hospital, on August 19th, 1867, with acute dysentery. Patient states that, during the last eight or nine days he suffered from looseness which continued until four days ago, when the stools contained blood and slime. There was then much griping and straining. He was also slightly feverish, the febrile condition not having been preceded by anything like a cold stage. Ordered a drachm of ipecacuanha with ten grains of bismuth, morning and evening. Milk diet and ice 1 lb.

6 p.m.—Has had five scanty motions since the morning, without feculence, chiefly consisting of blood with a little gelatinoid exudation. Liver enlarged. Pergat.

20th.—Passed several scanty stools during the night, with much griping and straining. The ipecacuanha nauseated him. Stools consisted of broken-down feculence of a greenish colour with gelatinoid exudation tinged with blood. Pergat.

21st.—Has had eight motions since last report, consisting of brownish-yellow fluid, yielding about an ounce of gelatinoid exudation, partly stained yellow with bile, and partly red with blood, and three small buff and two deep yellow-coloured sloughs, which were found to be mainly composed of altered mucous membrane and exudation matter. Pergat.

22nd.—Six motions consisting of greenish-yellow fluid with small quantity of gelatinoid exudation tinged yellow, and five soft, yellow, macerated sloughs. Pergat.

23rd.—Has had five stools composed of a quantity of yellow fluid with seven soft macerated sloughs. There is much less gelatinoid exudation slightly stained with blood. The griping and straining also much diminished.

24th.—Has had six motions of a yellowish-green fluid with a large proportion of soft feculence and four soft, pulpy, buff-coloured sloughs, without a trace of slime or blood. The patient is now free from pain, griping, and tenesmus. Omit ipecacuanha. Ordered an ounce of compound chalk mixture every four hours, *vel pro re nata*. Milk, broth, and four ounces of port wine. 6 p.m.—Has only had one soft feculent motion since morning. Weak, but free from pain. Pergat.

25th.—Two soft yellow feculent motions without any gelatinoid exudation, without a trace of blood; no pain or griping; appetite good; tongue moist and cleaning; complains of weakness. Pergat. Add eight ounces of bread to diet.

26th.—Two motions, altogether feculent, with a few consistent forms of fæces and with one small-sized blood-clot. Pergat. As it was inferred that there might be a tendency to the accumulation of feculent matter above the site of the ulcers, and its passage beyond might be impeded by spasm, half an ounce of castor-oil with ten minims of tincture of opium was administered. 6 p.m.—Passed semi-liquid motions without a trace of slime or blood. From this date the patient never had an unfavorable symptom. The chalk mixture was discontinued on the 28th, and replaced by ten minims of dilute nitro-muriatic acid mixture in an ounce of cheyretta infusion, three times a day. Diet was improved.

On September 3rd the tenesmus complained of was checked by an enema of half a drachm of acetate of lead, and twenty minims of laudanum in a couple of ounces of rice water (conjee) at bedtime. The motions now became figured.

Discharged cured on September 9th.

Case 18. *Acute dysentery terminating in sloughing.*—Bhoodhun, aged forty, resident of Jalkhana Potee, Hindoo of the Sudra caste, admitted into the Medical College Hospital on December 5th, 1866. Been suffering from bowel complaint during the last six days, passing many scanty, slimy, and bloody stools daily, with much griping and straining. Was perfectly well before, and can assign no cause for this attack. He is much prostrated. Ordered, by the officer on duty, five grains of

ipecacuanha with ten grains of bismuth, every six hours. Milk diet. 4.30 p.m.—Passed five scanty motions with great griping and straining. Slime and blood abundant. Ordered a drachm of ipecacuanha with ten grains of bismuth, night and morning. Diet as before. Ice 1 lb.

6th.—Passed several scanty motions containing blood and gelatinoid exudation, with some small portions of feculence. Complains of griping and straining. Pulse feeble; tongue dry and furred; considerable thirst. Vomited twice in the night. Pergat. 6 p.m.—Retched several times—griping much less. To have an anodyne enema daily at bedtime to restrain the tenesmus. Pergat.

7th.—Passed seven stools in the night, consisting of greenish-yellow semi-feculent fluid, at the bottom of which was found, on washing, five pieces of gelatinoid exudation tinged with blood, and a large quantity of bile-stained coagulated exudation. Had very little griping. No tenesmus. No vomiting. The pulse had improved. Tongue coated with brown fur, but moist. Pergat. Also jelly. 6 p.m.—Had three stools of the same character as those described in the morning report. Pergat.

8th.—Had four motions during the night. No griping. No straining. No vomiting, with very little gelatinoid exudation. 6 p.m.—Bowels moved once only since morning. Pergat.

9th.—Three motions. No griping. No straining. No vomiting, feels better. The motions consist of greenish-yellow fluid, with feculent sediment, several pieces of firm drab-coloured sloughs, without any gelatinoid or blood-stained exudation. Pulse 76; temperature normal. The largest slough was about three inches long by one in breadth, and was found to be constituted principally of exudation and mucous membrane undergoing granular degeneration. Omit ipecacuanha. Ordered an ounce of chalk mixture with ten minims of laudanum and twenty grains of carbonate of soda, *pro re nata*. An ounce of rum mixture every four hours. Diet as before, with four ounces of port wine.

10th.—During the last twenty-four hours patient has passed three motions, one during the day and two during the night. The former consisted of yellow-figured fæces smeared with blood, and one portion of firm yellowish slough; the latter, of dark feculence with a number of sloughs—a large drab coloured one. Part of this consisted of mucous membrane and submucous cellular tissue. The transverse lines, however, indicated the site where transverse muscular fibres existed. Most of these were found to have undergone transformation, but the identification of some involuntary muscular fibrils was complete. There were ten drab-coloured sloughs, so much infiltrated with exudation that few of the remains of the mucous membrane could be recognised. Altered connective tissue, and a few tubular glands, distended with granular matter, were observed. In nineteen others no structural characters could be detected. Pergat.

11th.—Passed two stools tolerably well formed. But, on washing them, a number of small sloughs, chiefly consisting of exudation, were found. Also, a large drab-coloured slough, in which unstriped fibres were discovered, and three of a similar colour made up mainly of exudation with mucous membrane in a state of granular degeneration. Pergat.

12th.—Had only one formed motion last night, without a trace of blood or gelatinoid exudation. Pergat.

13th.—Doing well. Bowels not opened during the last twenty-four

hours. Ordered ten minims of dilute nitro-muriatic acid in an ounce of infusion of cheyretta, three times a day. Diet and wine as before.

14th.—One motion since last report. Appetite greatly improved. From this date the amendment was uninterrupted. Bowels acted with regularity, and nothing abnormal could be detected in the evacuations.

On the 25th all medicine was omitted.

On the 29th he was placed on half diet, with fish, instead of dhal, and, in addition to his port wine, he was allowed four ounces of rum. He was put on full diet on January 6th, 1867, without dhal. He left the hospital on the 12th perfectly well.

The practice of washing the evacuations is very valuable, when it is suspected, from swollen and tender portions of the colon, that sloughing is impending; or when the provisional diagnosis is subsequently confirmed by the passage of sloughs in the dejecta. As in cases of the simpler or milder forms of the affection, so in the severe and aggravated varieties of sloughing dysentery, the existence and persistence of gelatinoid exudation in the stools is the unerring indication for perseverance with the ipecacuanha. (Here I exclude true gangrenous dysentery, which is usually fatal.) The exudation is heavier than the remainder of the stools, and is invariably found at the bottom of the utensil. That from one solitary gland seldom coalesces with that of another. Hence the number of these little masses of gelatinoid exudation, more or less tinged with bile, blood, or pus, and surrounded with catarrhal, or it may be ropy, mucus from inflamed tubular glands in the vicinity of the disease, affords some idea of the extent of mischief superadded to the sloughing going on within. It constantly happens that quantities of this characteristic—I had almost said pathognomonic—exudation are poured out during the sloughing process. The existence of this product in the evacuations, regardless of sloughing, is, according to my experience, the index for the continuance of the ipecacuanha. On the other hand, its disappearance, equally regardless of the passage of sloughs, is the indication for the cessation of the powder. It not unfrequently happens that sloughs may have been hanging on by a mere shred, or delayed in the exhausted and semi-paralysed gut, are not completely detached and expelled for some days, after all traces of the dysenteric exudation have vanished. The question to be solved, therefore, in these cases, is not whether the ipecacuanha is to be discontinued, because sloughing is suspected or known to be going on, but whether gelatinoid exudation and other dysenteric products are still discovered in the washings of the stools, in which case the drug must be admini-

stered in full doses, without much concern, as a rule, about the existing sloughing. In this way the extension of the mischief and the sloughing process are reduced to the narrowest possible limits. When the sloughs are all cast off, the resulting ulcers are usually repaired, under absolute rest, liquid nourishment, and appropriate medicines, quickly and successfully.

In these cases the ipecacuanha was not persisted in, because it was supposed to have any power to prevent sloughing already in progress, but because it placed those solitary glands in the immediate vicinity, engaged in specific dysenteric inflammation, in the most favorable position to resist the tendency to disintegration by molecular ulceration, or to undergo reparation, and the living though damaged structures connected with dying and dead tissue or sloughs also in the most advantageous condition for throwing them off with a minimum of shock and of extension. Doubtless, in some, it will be noted that the ipecacuanha was administered longer than was necessary. But it should be remembered that, to me at least, at the time, the practice was novel, and sufficient experience had not been gained to enable me to judge as to the period when it might be safely discontinued. After all, the point is not of much importance, for the drug, when given at long intervals, and in the large doses recommended, is borne with a minimum of stomach disturbance and of interference with the digestion and assimilation of mild, unstimulating, and liquid food. Although in my subsequent practice I was often able to leave it off sooner, still I feel convinced that when there is doubt, from the local indications, the subjective symptoms, and the character of the dejecta, it is safer and better to give too much of it than too little.

How does ipecacuanha act? Its effects in simple and ordinary sloughing dysentery are very striking, equally illustrated by clinical observation, and, in a measure, by physiological experiment on healthy dogs. Thus it acts (1) directly and locally as a slight irritant upon the peripheral nerves of the mucous membrane of the stomach, provoking anorexia, nausea, retching, or vomiting (not always); but when repeated, as often happens, the organ becomes more tolerant of its presence. Even when vomiting takes place the drug is seldom rejected in quantity; if it is, another dose given soon after is usually retained. Doubtless its retention is much facilitated by abstention from swallowing fluid until it has passed out of the viscus, partly by absorption of its active principles, and

partly by the passage of the remainder into the duodenum, where and lower down, the balance is taken up, probably by the intestinal veins; by the munching of small pieces of ice only, and by the maintenance of the head not much higher than the level of the heart. When I first went to India it was the custom to begin the treatment of intermittent with an emetic. My practice then was mainly among natives. I constantly found that the drachm of ipecacuanha given under such circumstances often produced no vomiting whatever. On inquiry, I discovered that this was due to my patients refraining from daughts of warm water: when these were taken, the emetic action of the drug was fully developed. (2) It augments the aqueous character of the secretion of the salivary, peptic, and intestinal glands. The flow of fluid in the mouth, in from twenty minutes to half an hour, after its administration or earlier, is marked and characteristic, and it probably exerts a similar influence on the pancreas, the tubular glands of the stomach and the intestine. (3) It increases the flow of healthy and non-irritating bile, thus depurating the blood and producing a comfortable and comforting laxative action, relieving the local congestion and portal plethora, and subsequently soothing the diseased parts by the promotion of physiological repose. Hence the speedy moderation, and eventual suppression, of tormina and tenesmus. (4) It diminishes the force and frequency of the pulse by lowering the general vascular tension. (5) It promotes general diaphoresis soon after it has been taken. As the active principles of ipecacuanha become absorbed and are brought to bear upon the vaso-motor centres regulating and controlling the functions of the chylopoietic viscera, the alimentary mucous membrane, the organs of circulation and the skin, the above-mentioned effects are promptly produced.

It is probable that, in dysentery, the extreme irritability of the seats of disease increases by reflex action the previously existing obstruction to the onward flow of blood through the liver, from an already overloaded portal system, thus opposing an obstacle to the relief of the congested and inflamed parts. This is the condition which invariably precedes and accompanies dysenteric disease of the solitary glands. Nature often succeeds in mitigating it either by hepatic catarrh, or a bilious attack, with or without purging, and the subsequent enforced starvation. In other cases the portal plethora may have been successfully rectified by simple catarrhal diarrhœa.

When, as is too frequently the case, she fails, the miasm or poison may take effect and light up the disease. The condition of pre-existing plethora is aggravated by increased disturbance in the balance of the portal circulation. Ipecacuanha, by its sedative or depressing power over the vaso-motor centres, lessens the tension of the arterioles of the portal and general circulation, promotes exosmosis from the skin and alimentary mucous membrane, equalises and restores the balance of the portal circulation, and places the affected parts in the best possible position to undergo repair, and the as yet unaffected but threatened glands to resist invasion. Hence the relief of tormina and tenesmus by prompt modification or mitigation of the morbid conditions upon which they are dependent, viz. congestion and hyperæsthesia of the muscular structure underlying the inflamed lenticular and tubular glands rendering peristalsis painful and spasmodic; the rapidity with which convalescence follows in the non-ulcerative forms of the disease; the success with which the healing process is promoted in molecular ulceration or that which has succeeded sloughing, and the prevention of sloughing from passing into gangrene.

The cholagogue action of ipecacuanha has been experimentally established on dogs by Professor Rutherford. He says, "Ipecacuanha is a powerful hepatic stimulant. It increases slightly the secretion of the intestinal mucus, but has no other apparent stimulant effect upon the intestine. The bile secreted under the influence of ipecacuanha has the normal composition" ('British Medical Journal,' February 8th, 1879). The great importance of this stimulation of the function of the liver signifying, among other things, a healthy balance in the portal circulation, always disturbed in unchecked dysentery, will be clearly recognised, especially when it is borne in mind that, as pointed out by Dr. Lauder Brunton, at the Ryde Meeting of the British Medical Association in August, 1881, "the liver has a power of regulating the flow of blood not merely through itself, but through the rest of the abdominal viscera as the stomach and intestines." If ipecacuanha did no more than restore the equilibrium of the portal circulation, its value in dysentery would stand even then unrivalled. For it would tend to remove the congestion and strain on the inflamed structures in the colon. To this great virtue we may now add Rutherford's experimental and a great mass of clinical evidence, proving its value in augmenting the secretion of healthy bile; and though his experi-

ments on healthy dogs led him to conclude that the intestinal mucus was only slightly increased, this result is scarcely consistent with the copious liquid evacuations following full doses of ipecacuanha. It is true that these are largely tinged with, and contain a good deal of, bile; but after all it forms only a small proportion of what is now known as the "ipecacuanha stool."

Sir Joseph Fayrer spoke in high terms of praise of Dr. Ewart's work on dysentery. He thought that the permanent retention of the ipecacuanha ingested was by no means necessary. In India dysentery was not now feared if patients presented themselves in the first days of the disease. In sloughing dysentery ipecacuanha was useless except to control further exacerbations of the disease.

Dr. Ewart said that he admitted in his paper that in the sloughing and gangrenous stages opium and stimulants were the only drugs of value. In the chronic local forms of sloughing ipecacuanha in large doses morning and evening exercised a beneficial result.

Dr. Radcliffe Crocker showed a specimen of "Bromide" Rash at the site of some recent vaccination scars.

Mr. Pearce Gould exhibited a case of Ulceration of a Finger, due to injury of the median nerve.

Mr. Sampson Gamgee exhibited in the ante-room his Antiseptic Absorbent Sponges. These artificial sponges are composed of absorbent gauze, cotton, and cocoa-nut fibre; in the very centre a thin capsule of glass or gelatine is enclosed, containing any antiseptic substance. When the sponge is required for use the capsule is broken, and so the antiseptic diffused. Messrs. Burroughs and Wellcome are the manufacturers.

April 28th, 1884.

A CASE OF INFECTIVE ENDOCARDITIS.

By W. Cayley, M.D., F.R.C.P., and Heneage Gibbes, M.D.

John O—, aged twenty-six, a footman, was admitted on February 29th, 1884. At the age of seventeen he had had an attack of rheumatic fever, but otherwise had always enjoyed good health. He was quite well on February 24th. On February 25th he went out driving insufficiently clothed, and came home shivering and feeling very cold. The next day, although feeling very unwell, he again went out driving, and waited for some time about the Victoria Station, and in the evening rode on the top of an omnibus without his great coat. When he got home, he was shivering, and looked very ill. He slept heavily during the night, and in the morning his mind was stated to have been wandering; he remained in bed all day, and vomited twice. The next day, February 28th, he was worse, and in the evening had epistaxis. He was now seen by his medical attendant, who, on the following morning, recommended his removal to the hospital.

His state on admission was as follows. He was a well-nourished, muscular young man, lying on his back in a state of prostration, with muscular twitchings; with a heavy expression of countenance, and in a dull obfuscated mental condition. The forehead was covered with perspiration, the eyes suffused, the lips dry and brown, the cheeks flushed; the tongue was red at the tip and edges, the dorsum was coated with a white fur, and the tip was ulcerated; there was a diffused, somewhat mottled, red blush over the skin of the trunk, and scattered over the trunk and extremities were numerous irregularly-shaped spots of extravasation, many, but not all, distinctly raised; on the back of the metacarpo-phalangeal joint of the left forefinger was a pustule of the size of a split pea, with a red halo surrounding it. The tip of the right great toe was swollen and livid, and there were livid patches over the left olecranon; the colour of the patches of extravasation differed, some being darker than others. There was no cough nor sorethroat. Examination of the thorax showed well-marked signs of cardiac hypertrophy, and

there was a loud rough systolic apex-murmur; the pulmonary signs were normal. There was no distension or tenderness of the abdomen; the splenic dulness was increased, measuring four inches and a half vertically, but the viscus could not be felt on palpation. The urine was very albuminous—about one half—and contained blood; it was acid, of specific gravity 1030, and of blood-red colour.

From the general symptoms of blood-poisoning, accompanied by multiple cutaneous embolisms, together with the signs of mitral disease, the case was regarded as one of ulcerative endocarditis.

He was ordered ten grains of quinine every four hours, six ounces of brandy daily, and to be sponged with cold water when the temperature exceeded 102°. He continued much in the same state during the rest of the day; the muscular twitchings kept recurring at irregular intervals; he lay in a torpid, dreamy condition, with wandering delirium, but roused up when spoken to.

On March 1st he was more prostrate, and took no notice of anything. The elevators of the upper lip and alæ nasi, on the right side, had been in a state of constant twitching since the early morning. The pulse was 120. The breathing 56, and shallow. The lips and teeth were covered with sordes; the tongue dry, brown, and cracked; and the breath offensive. Four pustules had formed on the patch of livid skin over the left olecranon, and several fresh petechial extravasations had appeared. The bowels had not been opened since admission. The patient rapidly sank, and died at 3 P.M.

		Temperature.	Pulse.	Respiration.	
February 29th.	2 p.m.	104·2°	124	36	Sponged.
„		102·2°	—	—	After sponging.
„	6 p.m.	104°	—	—	Sponged.
„		102°	—	—	After sponging.
„	10 p.m.	104°	112	40	Sponged.
„		101°	—	—	After sponging.
March 1st.	2 a.m.	101·8°			
„	6 a.m.	101·4°			
„	10 a.m.	102°	120	56	
„	2 p.m.	104·2°	130	65	

The pulse subsequently ran up to 180.

The necropsy was made by Dr. J. Kingston Fowler forty-eight hours after death. The body was well nourished, cadaveric rigidity

was passing off, and post-mortem congestion was well marked. On the abdominal wall were several ecchymotic patches, which had been present during life; some of these patches were removed for microscopical examination. The parietal peritonæum presented nothing abnormal, but the intestines were marked by numerous dark pinkish hæmorrhagic patches, varying in size from a pea to a florin. On laying open the intestine, these patches, which extended from the duodenum to the cæcum, were seen to correspond with hæmorrhages of the same size in the mucous and muscular coats; the patches were of a dark red colour, but in the centre of each was a raised nodule, firm, but almost purulent. In some of these were a number of smaller yellow spots, the size of pins' heads, surrounding a large nodule. There was no trace of ulceration in any part of the bowel. The spleen was extremely soft, almost diffluent, and contained several large soft infarctions. The kidneys were large, the surfaces smooth, and mottled with innumerable yellow and claret-coloured points, the site of minute infarctions. On section, a vast number of small caseous foci were seen scattered through every part of each kidney. The organs were swollen, and on the whole pale. In the pyramids, streaks of yellow marked the lines of some straight tubes, which were probably blocked. The liver was large and pale, and presented a number of small hæmorrhagic foci, principally in the left lobe. On section, some small lines of commencing suppuration were seen along the lines of the portal vessels. The bladder and rectum were not diseased. At the reflexion of the right pleura on to the pericardium, there was recent lymph with hæmorrhage. There were some pleural adhesions on both sides, and a few hæmorrhagic foci in the visceral layer of each pleura. The pericardium presented only some hæmorrhagic foci in the visceral pericardium. The right auricle contained a firm yellow clot, fibrinous, and moulded to the cavity. The right ventricle contained some similar clot. There were four cusps to the pulmonary artery, one very small. The semilunar valves were swollen and opaque; the thickening was especially marked at the free edges. The upper margin showed a few very small vegetations. The left ventricle was hypertrophied, its muscular walls firm and fibrous, and the endocardium rather thicker than normal. The left auricle was dilated; its posterior wall was roughened, and covered with small vegetations marking the line of the regurgitant stream of blood. The curtains of the mitral valve were thickened and opaque, and slightly contracted; a few of

the chordæ tendineæ were swollen, and two were broken. There were no large masses of fibrin, and the amount of disease of the valves was much smaller than would have been expected from the wide extent of the infarctions. The aortic valves were slightly thickened, but competent. The aorta was atheromatous in a few small patches. The lungs were congested and œdematous. Each lung contained from three to four black wedge-shaped infarctions; these were all upon the surface, and some were surrounded by small foci of hæmorrhage.

The following report on the microscopical appearances was made by Dr. Heneage Gibbes.

Sections through the cardiac valves and endocardium showed thickening of old standing. On the surface of this there was a homogeneous deposit projecting inwards in fine papillæ. This was covered with myriads of micrococci, which were also present in masses throughout the deposit. In some places they had penetrated to some distance into the deeper tissue, and might be seen in what appeared to be lymph-spaces. The liver had undergone extensive fatty degeneration, and there was a small amount of increase in the interlobular tissue. A number of capillaries were blocked by masses of micrococci; but in the portions of tissue examined these had not set up any inflammatory action in the surrounding tissue. The intestines showed a good deal of post-mortem change, and there were a large number of bacteria of various forms. No vessels blocked by micrococci were seen in the parts examined. In the deeper parts of the skin were accumulations of inflammatory cells, amongst which were masses of micrococci similar to those in the heart. Throughout the spleen there were a number of vessels blocked by micrococci. In some places the vessel and its branches were filled for some distance. There was, however, little sign of any inflammatory action in the surrounding tissue. In the kidney, many capillaries were filled and distended by masses of micrococci. In several places inflammatory action had been set up in small circumscribed areas, and the tissue had broken down. Amongst the débris were small groups of micrococci.

In addition to these micrococci in the blood-vessels, there were numbers of bacilli in the lymph-spaces of the different organs. They resembled the ordinary form of putrefactive bacteria. In the small veins of the deeper tissues of the heart were a number of small rod-shaped bacilli. In the kidney there were also numbers of large

bacilli resembling *B. subtilis;* these occurred in interstitial tissue, and in the urinary tubules. Those in the urinary tubules had a peculiar reaction in their behaviour to aniline dyes. If the section were stained in a dilute watery solution of mythyl-blue, to which a slight trace of a neutral rosaniline salt was added, the bacilli picked out the rosaniline and stained red.

Remarks by Dr. Cayley.—This case is a good example of a not uncommon form of ulcerative endocarditis; viz. where the disease supervenes in persons who are the subject of old-standing valvular lesion. The relation of the micro-organisms to this disease is a question of much interest and importance. In this case there was no reason to suspect any infection from without. The attack must, I think, be attributed to exposure to cold; this induced endocarditis, to which he was specially liable from having old mitral disease; and the inflammatory change in the valve probably produced a condition favorable for the development and growth of these organisms, which in their turn caused the multiple embolisms. In a large number of cases, the ulcerative endocarditis has supervened during an attack of rheumatic fever, where an infection from without would be extremely improbable. It seems, on the whole, more reasonable to suppose that, under certain circumstances, the inflammatory changes produce conditions which favour the development of germs which are probably generally diffused, than to suppose that these every now and then succeed in obtaining entrance into the body from without, and excite the disease; just as the germs of putrefaction do not cause the death of the putrid body, but the changes due to the cessation of life afford the necessary conditions for their growth.

Dr. Coupland asked whether it was right to use the term ulcerative endocarditis at all. He preferred to call Dr. Cayley's case septic, or, with Virchow, malignant. Klebs had described micro-organisms in ordinary endocarditis. A distinct septic source had been traced in many cases of "ulcerative" endocarditis. Osler, of Montreal, had pointed out that in many instances of this form of endocarditis acute pneumonia was present. Micrococci had been assigned as the cause of some cases of acute lobar pneumonia. In one case of septic endocarditis which resembled ague the patient had caught cold in a graveyard. Dr. Cayley's case ran a remarkably acute course.

Dr. Heneage Gibbes thought nothing definite could yet be said of the micro-parasitic origin of the disease. Masses of micrococci could be found in this case which had not caused the vessel to break down, or any inflammatory action to take place in the surrounding tissue. He thought that these masses of micrococci might have increased after

death, as in cases of charbon, where shortly after death the blood was teeming with bacilli. He did not think that vessels could be blocked during life without setting up inflammatory action in the tissues outside the vessels. In the skin and kidney there were areas broken down and micrococci were found; but also there were bacilli in the renal tubules which behaved peculiarly. With a half per cent. solution of methylene blue and a single crystal of a neutral salt of rosaniline the bacilli picked out the red stain. This was a curious feature.

Dr. Cayley thought the term ulcerative endocarditis was inappropriate, "infective" was, perhaps, a better term. He had seen cases which were indistinguishable, clinically, from typhoid fever.

Dr. Heneage Gibbes exhibited under the microscope specimens of the following conditions:—Micrococci in lymph from the inflamed endocardium; micrococci in diphtheritic membrane; vessel in the liver blocked with micrococci; deposit on endocardium covered with micrococci; inflammatory area in skin showing small masses of micrococci; blood-vessels in spleen crowded with micrococci.

A CONTRIBUTION TO THE PATHOLOGY OF HYDRO- AND PYO-SALPINX.

By J. Kingston Fowler, M.A., M.D. Cantab., M.R.C.P.

In the course of a discussion recently held by the Obstetrical Society of London, on the subject of hydro- and pyo-salpinx, it was remarked by more than one speaker as a curious fact that, if these conditions were so common as some would make out, they should not be met with at the general hospitals. In order to show that such cases are not uncommon, I have thought it worth while to put together the records of fifteen examples, which I have met with in the post-mortem room of the Middlesex Hospital, in the course of the last three years.

I do not propose in this communication to describe the symptoms which may arise from the condition, or the relief which may be afforded by the operation for removal of the tubes; on neither of these points have I any experience to place before you. I trust, however, that these omissions will not prevent this side of the question from being discussed by those whose practice brings them in contact with such cases.

I have also omitted to give any clinical details, partly because they are incomplete, as in none of the cases was the presence of the

condition suspected during life; partly also from the fact that nearly all the cases were complicated by some other general or uterine disorder, which would render doubtful to what degree any symptoms observed were directly due to the state of the Fallopian tubes, and also because, in most of the cases, the report of the post-mortem appearances clearly indicates the course of the disease, and the sequence of the several events, which, together brought about the patient's death.

At the head of each case I have placed an abstract of the changes found in the various organs, and have given in detail a description of the condition of the parts more immediately concerned.

Case 1. *Retroflexion of the uterus; double pyo-salpinx; abscess of left ovary; acute peritonitis.*—Hannah L—, aged twenty-nine. There was a large quantity of pus in the pelvic cavity, filling up the posterior *cul-de-sac*. The pus appeared to ooze from an abscess connected with the left ovary; this was really the dilated end of the left Fallopian tube, which had ruptured into the general cavity of the peritonæum. Both Fallopian tubes were much dilated, and contained pus. The right ovary was twice the normal size, and very vascular. The uterus was bulky, its lining membrane vascular. There were no old adhesions about it. Its posterior wall was deeply grooved, the result of the retroflexion.

Remarks.—In this case the pyo-salpinx appeared to have resulted indirectly from the condition of retroflexion of the uterus.

Case 2. *Hypertrophic cirrhosis of the liver; fibro-myoma of uterus; double hydro-salpinx; jaundice; ascites.*—Eliza M—, aged thirty-seven. The uterus was large, the cervix sharing in the increased size. A fibroid tumour, the size and shape of an unshelled walnut, was situated beneath the mucous membrane of the posterior wall of the fundus. The uterine mucous membrane was congested. The orifices of the Fallopian tubes were obliterated at both ends, and the tubes considerably dilated. They contained a clear brown fluid.

Case 3. *Fibroid tumour of the uterus; double pyo-salpinx; abscesses in left ovary; peritonitis; pyelitis.*—Elizabeth B—, aged thirty-two. The pelvis was almost filled by a fibroid tumour six inches by three inches, growing from within the right wall of the uterus; the latter had been displaced to the left of the middle line and dragged upwards. Recent inflammatory adhesions bound the structures in the broad ligament to the neighbouring parts on either side. Both Fallopian tubes were dilated to a moderate degree and filled with pus. There were several abscesses containing pus in the left ovary, the right was healthy.

Remarks.—In these two cases the condition of the tubes was clearly due indirectly to the presence of the fibroid tumour of the uterus.

Case 4. *Cancer of the uterus, vagina, rectum, and bladder; cystitis; double hydro-nephrosis; fibroid tumour of uterus; double pyo-salpinx; old fibroid changes in right lung; renal cyst (R).*—Selina L—, aged forty-five. Extensive ulceration and destruction of the upper part of the vagina

and lower half of the uterus. The uterus, vagina, and bladder formed a single cavity. The fundus uteri was infiltrated and contained an intramural fibroid the size of a chestnut. Each Fallopian tube was distended to the size of the little finger, and filled with creamy pus; they were adherent to the uterus and closed at both ends. There was no peritonitis.

Remarks.—In this case also the presence of the fibroid tumour may have caused the inflammation of the tubes, as it was situated immediately between their orifices; but the cancer alone was quite sufficient as an exciting cause.

Case 5. *Typhoid fever; perforation of ileum; aortic, mitral, and tricuspid stenosis; emphysema; right femoral epiplocele, inflamed and incarcerated; local acute peritonitis; double pyo-salpinx.*—Priscilla L—, aged thirty-six; vagina normal. The uterine cavity contained a small quantity (about ʒij) of purulent blood-stained fluid. The fundus was dilated. There was an indurated spot at the inner end of each Fallopian tube. Both tubes were dilated to the size of the ordinary calibre of the small intestine (about one inch in diameter), and were adherent to the posterior aspect of the fundus uteri. The fimbriæ were closed. The tubes contained a glairy, semi-purulent-looking fluid. The rectum was normal.

Case 6. *Chronic interstitial nephritis; pericarditis; pleurisy; œdema glottidis; pulmonary œdema; double hydro-salpinx.*—Jane L—, aged twenty-one. The fimbriæ of the left Fallopian tube were adherent to the ovary, and both tube and ovary were displaced, lying behind the rectum, where they were firmly held by adhesions to the peritonæum. The tube was distended with glairy, greyish-coloured fluid. The right tube was also distended and had fallen into Douglas' Fossa, its fimbriæ being adherent to the peritonæum. The uterus and vagina contained a quantity of recently effused blood.

Case 7. *Scurvy; extensive hæmorrhages in the skin, mucous membranes, and various organs; double pyo-salpinx.*—Kate O—, aged thirty-four. The uterus was held in a position of anteversion by bands uniting it to the parietal peritonæum, the fundus being drawn upwards and forwards. The uterine walls were normal. The mucous membrane was injected, and the cavity contained some turbid purulent-looking fluid. The Fallopian tubes were both dilated, forming two sausage-shaped tumours at the back of the uterus, to which, and to the parietal peritonæum, both were adherent. The tubes were closed at both ends, and contained purulent fluid. The walls were thickened.

Remarks.—In cases Nos. 5 and 7, the pyo-salpinx was the result of endometritis. In these cases the condition of the tubes, although a possible source of danger, in no way affected the fatal result.

Case 8. *Cancer of left ovary; secondary cancer of uterus and peritonæum; cystic disease of both ovaries; dilatation and elongation of the left Fallopian tube; emphysema; bronchitis; secondary cancerous nodules in right lung.*—M. E. P—, aged fifty-four. The pelvis was filled with a solid cancerous growth involving the left ovary; this was in size about equal to a cocoa-nut. There were also two cysts, one attached to the new growth and one springing from the right ovary, each of these was

about equal in size to an orange, and contained clear brown-coloured fluid. The vagina and uterus were displaced toward the right side; the cervix uteri was destroyed by cancerous ulceration, and the upper part of the vagina showed a similar change. The growth in the ovary was adherent to and directly continuous with the cancerous growth in the uterus. The cavity of the uterus was dilated to the size of a large walnut, and was filled with a brownish mucus; the body of the uterus was not infiltrated. The growth in the ovary on section was very soft and contained several mucoid-looking patches. The left Fallopian tube was stretched across the front of the growth, measuring thirteen inches in length; the tube became gradually dilated from about five inches from its origin, at the fimbrial end it was the size of a hen's egg. It contained a clear brownish-coloured fluid.

Case 9. *Cancer of splenic flexure of colon; peritonitis; cancer of right ovary; secondary nodules in liver; double hydro-salpinx.*—Mary Ann B—, aged forty-three. There was a tumour of the right ovary the size of a cricket ball, filling up pelvis and slightly adherent to the posterior wall. The uterus was small; it was displaced to the left, and tilted over in the same direction. The cervical canal was dilated and the os internum closed. The left Fallopian tube was dilated to the size of a pigeon's egg; it contained black grumous-looking fluid; its inner end was closed; the fimbriæ were adherent to the parietal peritonæum. The right tube was extremely dilated and spread out upon the surface of the ovarian growth. The growth in the ovary and that in the colon appeared to be entirely distinct.

Remarks.—In these cases (Nos. 8 and 9) the hydro-salpinx was the indirect result of the malignant disease of the ovaries.

Case 10. *Rupture of the perinæum; prolapse of the uterus; inflammation and ulceration of the vagina and rectum; right pyo-salpinx; suppuration in Douglas' pouch and around the rectum; cysts in the left ovary; acute pyelo-nephritis with chronic interstitial nephritis; amyloid disease of the liver and spleen; septicæmia; pneumonia; parotid abscess.*—Eliz. A—, aged thirty-two. Emaciated. The perinæum had been ruptured (twelve years previously); the os uteri was situated three quarters of an inch from the vaginal orifice. All the pelvic organs were firmly adherent to one another. The vaginal walls were inflamed and ulcerated over two areas the size of a sixpenny-piece, from the pressure of the prolapsed uterus. The os and cervix uteri were enlarged, congested, inflamed, and ulcerated on the anterior and upper aspect. The mucous membrane of the uterus was injected; the cavity contained some glairy mucus; the uterine walls thickened and indurated. There was an indurated nodule the size of a hazel nut, containing a small quantity of pus in its interior, situated at the uterine end of the left Fallopian tube, the lumen of which was obliterated. Tracing the Fallopian tube onwards it became much dilated and sacculated. At this point its contents were of a brownish colour, and purulent in appearance. Still further onward it became dilated to the size of a Tangerine orange, and twisted upon itself. The walls in this portion were much thickened, and covered internally with a greenish-grey membrane, and the contents were purulent and fetid. This sac communicated through an opening, the size of a pea, with another and much larger irregularly-shaped abscess cavity, situated beneath the peritonæum of Douglas' pouch and around

the rectum. The rectum was surrounded by the abscess cavity, through which it passed without any communication. The left ovary contained a cyst as large as a pigeon's egg, filled with clear watery fluid. The ovarian tissue was of a brownish tint, and contained some smaller cysts. The right ovary was converted into an abscess, containing about a drachm of yellow pus.

Remarks.—In this case the sequence of events appeared to be as follows: First, rupture of the perinæum during labour, then prolapse of the uterus, endometritis, and closure of the uterine end of the tube. These changes were followed probably by inflammation of the lining membrane of the tube, closure of the fimbriæ, and accumulation of fluid within it. Adhesions then formed between the lower end of the tube and the peritonæum, followed by ulceration of the peritonæum at the point of adhesion, and escape of the pus into the cellular tissue behind it. As a result of these conditions, amyloid disease and septicæmia ensued.

Case 11.—*Hypertrophy of the uterus; endometritis; double pyo-salpinx; Rupture of the left Fallopian tube; acute peritonitis; cystic disease of both ovaries; syphilis.*—Kate A—, aged forty-six. There were two pints of pus in the peritonæal cavity. The uterus was equal in size to a large orange; its walls were much thickened, measuring three quarters to one inch on section. The os externum was closed; the cervical canal was elongated, and contained a quantity of rather fetid smelling and purulent-looking fluid. The uterine mucous membrane was acutely inflamed and covered with a layer of lymph of a greenish tint from decomposition. The openings of the tubes were closed. Both Fallopian tubes were dilated to the size of the thumb, their walls thickened, and their contents purulent and offensive. The left tube was flaccid, and its lower end was widely opened. The right tube was intact below. Both ovaries were converted into a congeries of cysts containing clear fluid.

Remarks.—Dr. Hall Davis, under whose care the patient was, was of opinion that the hypertrophy and inflammation of the uterus had probably followed the closure of the os externum, subsequently the tubes had become dilated and inflamed, the rupture of the left tube into the cavity of the peritonæum inducing peritonitis and death.

Case 12. *Removal of a uterine polypus; double pyo-salpinx; general peritonitis; pleurisy; cardiac thrombosis; endarteritis; infarctions in spleen.*—Eliz. H—, aged forty-one. The peritonæal cavity contained a pint and a half of pus. The structures in the pelvis were united together by recently-formed adhesions. The vagina was normal. The os uteri was patent; glairy mucus was issuing from it; the cervical canal was dilated and globular, its mucous membrane vascular. From a rough surface within it, the size of a sixpence, a polypus had been recently removed. The cavity of the uterus was slightly enlarged, and contained glairy mucus. A second small vascular polypoid growth was attached close to the orifice of the left Fallopian tube. Both tubes were enormously distended, each measuring two and a half inches in circumference. They were filled with pus, and their walls much thickened from inflammatory action on both surfaces. The tubes were firmly adherent to the uterus and surrounding structures, and were lying within a sac formed by the uterus in front and the rectum behind, closed in laterally by the pelvic

walls and some peritonæal adhesions, and above by some coils of small intestine. An opening, large enough to admit the forefinger, led from this sac into the left Fallopian tube.

Remarks.—During the operation for the removal of the polypus, the adhesions which then existed between the dilated tube and the peritonæum, the result of a previous attack of peritonitis, had evidently been torn asunder, the result being, that its contents had escaped into the peritonæal cavity, setting up peritonitis, which was the immediate cause of death.

Case 13. *Cancer of the rectum; chronic pleurisy and pneumonia; chronic perihepatitis and fatty degeneration of the liver; general peritonitis; peri-rectal and peri-uterine abscess; double pyo-salpinx.*—Lousia A—, aged forty-one. There was general peritonitis. A soft cancerous growth occupied five inches of the rectum. Around it, but not communicating with it, were several abscess sacs; in one, formed by peritonæal adhesions between the fundus uteri and the sigmoid flexure, there was a quantity of fetid pus. The upper wall of the sac had given way, allowing its contents to escape into the general cavity of the peritonæum. A second cavity surrounded the uterus, and opened into the vagina by a fistulous channel, situated behind the posterior lip of the os uteri. The uterus contained a small quantity of purulent-looking fluid. The internal orifice of each Fallopian tube was obstructed; both tubes were dilated and formed sausage-shaped tumours which surrounded the ovaries, and were firmly adherent to the posterior surface of the uterus. Both were distended with creamy pus. No distinct connection was discovered between the abscess cavity surrounding the uterus and the Fallopian tubes, or between them and the other sac described.

Remarks.—Although no direct communication could be found between the dilated tubes and the abscess cavities, the rupture of one of which had produced peritonitis, there can be little doubt that such a connection had at some time existed, and that these were secondary to the disease of the tubes.

Case 14. *Cancer of uterus; R. Pyo-salpinx; general peritonitis; mitral stenosis; liver fatty; kidneys sacculated.*—Eliza A—, aged thirty-nine. The peritonæal sac contained a pint and a half of turbid yellow pus. The membrane was everywhere acutely inflamed, the change being most marked about the pelvis. Bladder normal. The upper third of the vagina and the cervix uteri were extensively ulcerated. The body and fundus were enlarged, hard, and infiltrated with cancer near the cervix, but not elsewhere. The right Fallopian tube was distended to the size of an ordinary sausage, and contained a quantity of pus. Its walls were considerably thickened; the internal orifice was closed. At the fimbrial extremity the tube had burst into the cavity of the peritonæum. The left tube was normal.

Remarks.—This case is interesting from the fact that, although the patient was suffering from cancer of the uterus, death was due to peritonitis, the result of the rupture of the dilated tube.

Case 15. *Double pyo-salpinx communicating with rectum (L) and ileum (R); ulceration of the rectum; endocarditis; amyloid disease of liver, spleen, and kidneys.*—Harriet T—, aged twenty-one. Extreme emaciation. Some coils of ileum were adherent to the fundus uteri; there were also

other peritonæal adhesions, but all of long standing. The uterus was very small, the os was slightly ulcerated, the cervical canal contained glairy mucus. The uterine ends of the tubes were occluded. Each tube was widely dilated, and its walls much thickened. On section, each was found to contain fetid fæcal material. On the left side the distal end of the tube opened into a sac, situated between it and the rectum, and communicating with the rectum through a perforation in its inner wall the size of a sixpence. On the right side the tube communicated with a coil of the ileum, which had become adherent to the right side of the tube and to the fundus uteri. The communication was through a fistulous channel, about one inch long. There were several ulcers on the verge of the anus, but none elsewhere in the intestine.

Remarks.—The disease of the tubes was in this case undoubtedly indirectly the cause of the patient's death, by inducing amyloid disease and gradual asthenia.

The bare recital of the cases is sufficient to establish the fact, which has been doubted by many, that this condition is a very real one and by no means free from danger. It is very probable that many cases of peritonitis which have been regarded as idiopathic, as secondary to abscess of the ovary, or pelvic cellulitis, have been really due to rupture of a Fallopian tube, distended with pus, into the general cavity of the peritonæum.

It will be seen that the state of the tubes was the immediate cause of death, in eight cases out of the fifteen ; all these were cases of pyo-salpinx. In six the affection was bilateral, in two it was limited to the right side.

Of the eleven cases of pyo-salpinx, death was due to peritonitis in seven (one of these, however, was from perforation of the intestine, in a case of enteric fever), and in one case each to septicæmia, amyloid disease, cancer of the uterus, and scurvy.

In nine cases of pyo-salpinx, and three of hydro-salpinx, there were old adhesions between the tubes and uterus, or other structures in the pelvis. In two cases of pyo-salpinx the adhesions were recent, and in one case of hydro-salpinx there were no adhesions.

There can be no doubt that any inflammatory condition of the uterine mucous membrane, however induced, it may be by extension of a vaginitis or some affection of the os or cervix uteri, by a retroflexion, by the presence of a fibroid tumour or polypus, or as a result of prolapse or malignant disease, may spread to the Fallopian tubes, and ultimately establish the condition of pyo-salpinx.

It is probable, also, that in some cases the primary origin of the disease may be tubercular, but of this I have not met with any instance.

In many instances I have noticed that there is an induration close to the uterine end of the tube, the lumen of which is at that point entirely obliterated.

The fimbriæ and the tubes themselves are usually adherent to the uterus or some other structure in the pelvis ; in chronic cases of pyo-salpinx these adhesions are generally firm, but in the earlier stages, when an operation for the removal of the tubes is possible, these adhesions have not been found by Mr. Lawson Tait, and others who have performed such operations, to present greater difficulties in their management than those commonly found in cases of ovarian tumour. It is to be hoped that as a result of the increased attention which is now, mainly through the writings of Mr. Lawson Tait, being directed to this condition, a clearer light will be thrown upon its pathology, for this must ever remain the only sure guide to accurate diagnosis and rational treatment.

Mr. Durham was somewhat surprised at the frequency with which this condition was known to exist.

Dr. Angel Money had seen a specimen of pyo-salpinx in a female child, aged seven years.

Dr. Playfair was quite sure that the independent position which Dr. Fowler had taken would prove most valuable. It was a revelation to him to learn that cases of pyo-salpinx were of such frequency as this paper showed. With regard to operation for this condition, he considered that it had perhaps been too frequently performed at Birmingham. On the other hand, to suppose that operation was never necessary was also a mistake. The *via media* was no doubt the proper course.

Dr. C. H. Routh spoke of the great value of Dr. Fowler's paper from its impartiality. He referred to Dr. Tyler Smith's suggestion that the Fallopian tubes could be reached through the uterine cavity. He advocated aspiration instead of oöphorectomy, which operation had the unfortunate result, that it unsexed the women.

Dr. Ewart had met with many cases of pyo-salpinx and hydro-salpinx in the course of three years' experience in the post-mortem room of St. George's Hospital.

Dr. Edis thought that he had seen as many as treble the number of cases recorded in this paper if he might trust to clinical examination as distinguished from post-mortem experience. The distress caused by such conditions was pointed out.

Dr. K. Fowler, in reply, referred to the frequency with which the cases were associated with cancer as remarked on by Dr. Playfair. He agreed with Dr. Ewart that the colour of the contents of these tumours varied greatly. Such patients were nearly always sterile.

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