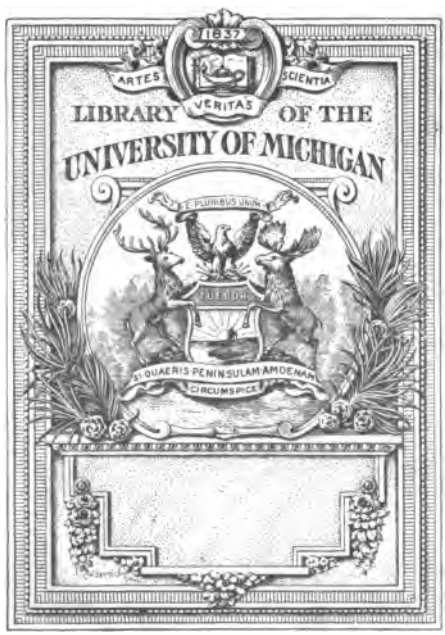
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TRANSACTIONS

OF

THE CLINICAL SOCIETY.

VOL. XVIII.

TRANSACTIONS

OF

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OF

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NOTICE.

THE present Volume comprises the Proceedings of the Society during its Eighteenth Session, October, 1884, to May, 1885.

The Council think it proper to state that the authors of the several communications are alone responsible for the statements, reasonings, and opinions contained in their respective papers.

53, BERNERS STREET, OXFORD STREET;
October, 1885.

CONTENTS.

LIST OF ILLUSTRATIONS.

PLATES.

WOODCUTS.

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1883 ANDERSON, WILLIAM, Assistant Surgeon and Joint Lecturer on Anatomy, St. Thomas's Hospital; 13, Welbeck Street, Cavendish Square, W. *Trans.* 2.

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1880 BALL, B., M.D., Professor to the Faculty of Medicine of Paris; 3, Faubourg St. Honoré, Paris.

1878 BALL, JAMES BARRY, M.D., 29, Belgrave Road, S.W.

1883 BALLANCE, CHARLES ALFRED, M.B., Assistant Surgeon, West London Hospital; Demonstrator of Anatomy, St. Thomas's Hospital; 56, Harley Street, Cavendish Square, W. *C.S.* 1.

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1875 BARLOW, THOMAS, M.D., Physician to University College Hospital, to the Hospital for Sick Children, Great Ormond Street, and to the London Fever Hospital; 10, Montague Street, Russell Square, W.C. (C. 1880–82.) *Trans.* 8, *C.S.* 1.

Orig. Memb. BARWELL, RICHARD, Surgeon to, and Lecturer on Surgery at, the Charing Cross Hospital; 55, Wimpole Street, Cavendish Square, W. (C. 1872–5, V.P. 1877–9.) *Trans.* 16.

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1882 BATEMAN, ALFRED G., M.B., 13, Canonbury Lane, Islington, N., and 64, Longridge Road, South Kensington, S.W.

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1875 BECK, MARCUS, M.S., Professor of Surgery, University College, London, and Surgeon to University College Hospital; 30, Wimpole Street, Cavendish Square, W. (C. 1880–81.) *Trans.* 1.

1880 *BEEVOR, CHARLES EDWARD, M.D., Assistant Physician to the National Hospital for Paralysed and Epileptic; 33, Harley Street, Cavendish Square, W.

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1878 BRIDGES, ROBERT, M.B., M.A., The Manor House, Yattendon, Berkshire. *Trans.* 1.

1868 BRIGHT, GEORGE CHARLES, M.B., 29, Lüttichen Strasse, Dresden.

1868 BRIGHT, JOHN MEABURN, M.D., Forest Hill, S.E.

Orig. Memb. BRISTOWE, JOHN S., M.D., F.R.S., Physician to, and Lecturer on Medicine at, St. Thomas's Hospital; Medical Officer of Health for Camberwell; 11, Old Burlington Street, W. (C. 1869–70, V.P. 1879–80.) *Trans.* 2.

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1876 BROWNE, GEORGE BUCKSTON, 80, Wimpole Street, Cavendish Square, W.

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1869 CARTER, ROBERT BRUDENELL, Ophthalmic Surgeon to, and Lecturer on Ophthalmology at, St. George's Hospital; Surgeon to the Royal South London Ophthalmic Hospital; 27, Queen Anne Street, Cavendish Square, W. (C. 1873–6, V.P. 1879–81.) *Trans.* 7.

1868 CAVAFY, JOHN, M.D., Physician to St. George's Hospital; 2, Upper Berkeley Street, Portman Square, W. (C. 1881–83.) *Trans.* 4.

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1885 CHALMERS, JOHN, M.D., 29, Keppel Street, Russell Square, W.C.

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1874 DOWSE, THOMAS STRETCH, M.D., 14, Welbeck Street, Cavendish Square, W. *Trans.* 5.

1868 DRAGE, CHARLES, M.D., Hatfield, Herts.

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Orig. Memb. DUFFIN, ALFRED B., M.D., Physician to King's College Hospital, and Professor of Pathological Anatomy in King's College, London; 18, Devonshire Street, Portland Place, W. (C. 1872–4.) *Trans.* 6.

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1868 FAIRBANK, FREDERICK ROYSTON, M.D., 46, Hall Gate, Doncaster. *Trans.* 1.

1872 FENWICK, J. C. J., M.B., 16, Old Elvet, Durham.

1878 FIELD, GEORGE P., Aural Surgeon to St. Mary's Hospital; 31, Lower Seymour Street, Portman Square, W.

1876 FINLAY, DAVID WHITE, M.D. (C.), Physician to, and Lecturer on Forensic Medicine at, the Middlesex Hospital, and Physician to the Royal Hospital for Diseases of the Chest; 9, Lower Berkeley Street, Portman Square, W. (C. 1885.) *Trans.* 4.

ELECTED

1868 FISH, JOHN CROCKETT, M.D., 92, Wimpole Street, Cavendish Square, W. (C. 1869–70.)

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1878 *FONMARTIN, HENRY DE, M.D., Parkhurst, Isle of Wight.

1881 FOWLER, JAMES KINGSTON, M.D., Assistant Physician to, and Lecturer on Pathological Anatomy at, the Middlesex Hospital, and Assistant Physician to the Hospital for Consumption, Brompton; 35, Clarges Street, Piccadilly, W. *Trans.* 3, *C.S.* 3.

1878 FOX, THOMAS COLCOTT, M.B., B.A., Physician to the Skin Department, Westminster Hospital, and to the Paddington Green Hospital, and Assistant Physician to the Victoria Hospital for Children; 14, Harley Street, Cavendish Square, W. *Trans.* 5.

Orig. Memb. FOX, WILSON, M.D., F.R.S., Physician Extraordinary to H.M. the Queen; Holme Professor of Clinical Medicine in University College, and Physician to University College Hospital; 67, Grosvenor Street, W. (C. 1873, V.P. 1878–9.)

1868 GANT, FREDERICK JAMES, Surgeon to the Royal Free Hospital; 16, Connaught Square, W. (C. 1877–9.) *Trans.* 3.

1879 GARSTANG, THOMAS WALTER HARROPP, Dobcross, near Oldham.

1885 GIBBONS, ROBERT ALEXANDER, M.D., Physician to the Grosvenor Hospital for Women and Children; 32, Cadogan Place, S.W.

1868 GLOVER, JAMES GREY, M.D., Hon. Surgeon to the Holloway and North Islington Dispensary; 25, Highbury Place, N. (C. 1878–80.) *Trans.* 2.

1882 GODDARD, EUGENE, M.D., 106, Highbury New Park, N.

1875 GODLEE, RICKMAN JOHN, M.S., M.B. (*Hon. Secretary*), Surgeon to University College Hospital; Teacher of Operative Surgery, University College, London; Surgeon to the North-Eastern Hospital for Children, and to the Hospital for Consumption, Brompton; 81, Wimpole Street, Cavendish Square, W. (C. 1882–3, S. 1884–5.) *Trans.* 8.

ELECTED

1882 GOLDIE, ROBERT WILLIAM, Medical Superintendent, Poplar and Stepney Sick Asylum; Devons Road, Bromley.

1878 GOLDING-BIRD, C. H., M.B., Assistant Surgeon to, and Lecturer on Physiology at, Guy's Hospital; 13, St. Thomas's Street, Southwark, S.E. *Trans.* 9.

1875 GOODHART, JAMES FREDERIC, M.D., Assistant Physician to, and Curator of the Museum at, Guy's Hospital; 25, Weymouth Street, Portland Place, W. (C. 1880–2.) *Trans.* 9, *C.S.* 1.

1869 GOODRIDGE, HENRY FREDERICK AUGUSTUS, M.D., Physician to the Bath Royal United Hospital; 10, Brock Street, Bath.

1882 GOODSALL, D. H., 17, Devonshire Place, Upper Wimpole Street, W.

1881 GORDON, HUGH ALEX., M.D., Holloway Prison, Camden Road, N.W.

1877 GOULD, A. PEARCE, M.S. (C.), Assistant Surgeon to the Middlesex Hospital; 16, Queen Anne Street, Cavendish Square, W. (C. 1885.) *Trans.* 6.

1871 GOVER, ROBERT M., M.B., 12, Hereford Gardens, W.

1875 GOWERS, WILLIAM RICHARD, M.D., Physician to University College Hospital; 50, Queen Anne Street, Cavendish Square, W. (C. 1881–2.) *Trans.* 4.

1868 GREEN, T. HENRY, M.D., Physician to, and Lecturer on Pathology at, the Charing Cross Hospital; Assistant Physician to the Hospital for Consumption, Brompton; 74, Wimpole Street, W. (C. 1877–9.) *Trans.* 2, *C.S.* 1.

1875 †GREENFIELD, WILLIAM SMITH, M.D., Professor of General Pathology in the University of Edinburgh; 7, Heriot Row, Edinburgh. (C. 1881.) *Trans.* 3.

Orig. Memb. GREENHOW, EDWARD HEADLAM, M.D., F.R.S., Consulting Physician to the Middlesex Hospital; Castle Lodge, Reigate. (T. 1867–78, P. 1879–80.) *Trans.* 24.

1883 GROSS, CHARLES, Medical Superintendent, St. Saviour's Infirmary, Westmoreland Road, Walworth, S.E.

ELECTED

1868 †GUENEAU DE MUSSY, HENRI, M.D., 15, Rue du Cirque, Paris.

Orig. Memb. GULL, Sir WILLIAM WITHEY, Bart., M.D., D.C.L., F.R.S., Physician Extraordinary to the Queen; Consulting Physician, Guy's Hospital; 74, Brook Street, W. (V.P. 1868–70, P. 1871–2.) *Trans.* 6.

Orig. Memb. HABERSHON, SAMUEL OSBORNE, M.D., 70, Brook Street, W. (C. 1873, V.P. 1878–9.) *Trans.* 6.

1882 HADDEN, WALTER BAUGH, M.D., Demonstrator of Morbid Anatomy at St. Thomas's Hospital; 21, Welbeck Street, Cavendish Square, W. *Trans.* 5, *C.S.* 1.

1875 HALE, C. D. B., 8, Sussex Gardens, Hyde Park, W.

1878 HALL, F. DE HAVILLAND, M.D. (C.), Assistant Physician to the Westminster Hospital; 46, Queen Anne Street, Cavendish Square, W. (C. 1885.) *Trans.* 4.

Orig. Memb. HARLEY, JOHN, M.D., F.L.S., Physician to, and Lecturer on General Anatomy and Physiology at, St. Thomas's Hospital; 39, Brook Street, Grosvenor Square, W. (C. 1875.) *Trans.* 1.

1872 HARRIS, HENRY, M.D., Trengweath, Redruth, Cornwall.

1881 HARRISON, CHARLES EDWARD, M.B., Grenadier Guards Hospital, Rochester Row, S.W.

Orig. Memb. HART, ERNEST, 38, Wimpole Street, Cavendish Square, W. (C. 1867–8.)

1869 HAWARD, J. WARRINGTON (C.), Surgeon to St. George's Hospital; 16, Savile Row, Burlington Gardens, W. (C. 1876–8, 1884–5, S. 1881–3.) *Trans.* 11.

Orig. Memb. HEATH, CHRISTOPHER (*Treasurer*), Surgeon to University College Hospital, and Holme Professor of Clinical Surgery in University College; 36, Cavendish Square, W. (C. 1867–71, V.P. 1876–8, T. 1879–85.) *Trans.* 18.

1879 HENDERSON, GEORGE COURTENAY, M.D., Kingston, Jamaica, West Indies.

1885 HENTY, SYDNEY H., 308, Camden Road, N.

1882 HERON, GEORGE ALLAN, M.D., Assistant Physician to the City of London Hospital for Diseases of the Chest, Victoria Park; Assistant Physician to the West London Hospital for the Paralysed and Epileptic; 57, Harley Street, Cavendish Square, W.

ELECTED

1884 HERRINGHAM, WILMOT PARKER, M.B., 22, Bedford Square, W.C.

Orig. Memb. HEWETT, SIR PRESCOTT GARDNER, Bart., F.R.S., Surgeon-Extraordinary to H.M. the Queen; Consulting Surgeon to St. George's Hospital; Chesnut Lodge, Horsham, Sussex. (V.P. 1869–71, P. 1873–4.) *Trans.* 3.

Orig. Memb. HEWITT, GRAILY, M.D., Professor of Midwifery in University College, and Obstetric Physician to University College Hospital; 36, Berkeley Square, W. (C. 1878–9.) *Trans.* 1.

Orig. Memb. HICKS, J. BRAXTON, M.D., F.R.S., F.L.S., Consulting Physician Accoucheur to Guy's Hospital; 24, George Street, Hanover Square, W. (C. 1875–7.)

1868 HILL, BERKELEY, M.B., Professor of Clinical Surgery in University College, London, Surgeon to University College Hospital, and Surgeon to the Lock Hospital; 66, Wimpole Street, W. (C. 1870–1.) *Trans.* 7, *O.S.* 1.

1874 HOLDERNESS, WILLIAM BROWN, 15, Park Street, Windsor.

1868 †HOLMAN, CONSTANTINE, M.D., Reigate, Surrey.

1868 HOLMAN, WILLIAM HENRY, M.B., 68, Adelaide Road, South Hampstead, N.W.

Orig. Memb. HOLMES, TIMOTHY, Surgeon to, and Lecturer on Surgery at, St. George's Hospital; 18, Great Cumberland Place, Hyde Park, W. (C. 1867–9, V.P. 1873–5.) *Trans.* 13.

Orig. Memb. HOLT, BARNARD WIGHT, Consulting Surgeon to, and Lecturer on Clinical Surgery at, the Westminster Hospital; Medical Officer of Health for Westminster; 14, Savile Row, W. *Trans.* 1.

Orig. Memb. HOLTHOUSE, CARSTEN. (C. 1870–2.) *Trans.* 8.

1878 HOOD, DONALD WILLIAM CHARLES, M.D., Assistant Physician to the West London Hospital; 43, Green Street, Park Lane, W. *Trans.* 1.

1873 HOPE, WILLIAM, M.D., Senior Physician to Queen Charlotte's Lying-in Hospital; 56, Curzon Street, Mayfair, W.

1883 HOPKINS, JOHN, Medical Superintendent, Central London Sick Asylum; Cleveland Street, W. *O.S.* 1.

ELECTED

1884 HORSLEY, VICTOR, M.B., Assistant Surgeon, University College Hospital; Assistant Professor of Pathological Anatomy, University College, London; Superintendent of the Brown Institution, Wandsworth Road; 80, Park Street, Grosvenor Square, W.

1878 HOUGHTON, WALTER B., M.D., late Assistant Physician to Charing Cross Hospital; Church Villa, Warrior Square, St. Leonard's-on-Sea.

1880 HOVELL, T. MARK, Junior Aural Surgeon to the London Hospital; 3, Mansfield Street, Portland Place, W.

1876 HOWSE, HENRY GREENWAY, M.S. Surgeon to, and Lecturer on Anatomy at, Guy's Hospital; Surgeon to the Evelina Hospital for Sick Children; 10, St. Thomas's Street, S.E. (C. 1881–3.) *Trans.* 3.

Orig. Memb. HULKE, JOHN WHITAKER, F.R.S., Surgeon to the Middlesex Hospital, and Surgeon to the Royal London Ophthalmic Hospital; 10, Old Burlington Street, W. (C. 1867–9, V.P. 1878–80.) *Trans.* 13.

Orig. Memb. HUMPHRY, GEORGE MURRAY, M.D., F.R.S., Professor of Surgery in the University of Cambridge, and Surgeon to Addenbrooke's Hospital, Cambridge. (V.P. 1867–70.)

Orig. Memb. HUTCHINSON, JONATHAN, F.R.S., Consulting Surgeon to the London Hospital; Surgeon to the Hospital for Diseases of the Skin, Blackfriars, and Surgeon to the Royal London Ophthalmic Hospital; 15, Cavendish Square, W. (C. 1867–8, V.P. 1875–6.) *Trans.* 9.

1879 INKSON, JAMES, M.D., Surgeon-Major, Army Medical Department.

1883 JACKSON, GEORGE HENRY, Lansdowne House, Tottenham.

Orig. Memb. JACKSON, J. HUGHLINGS, M.D., F.R.S., Physician to the London Hospital; Physician to the National Hospital for the Paralysed and Epileptic; 3, Manchester Square, W. (C. 1872–3.) *Trans.* 1.

1877 JACOBSON, WALTER HAMILTON ACLAND, M.B., Assistant Surgeon to Guy's Hospital; 41, Finsbury Square, E.C.

ELECTED

Orig. Memb. JENNER, Sir WILLIAM, Bart., M.D., K.C.B., D.C.L., LL.D., F.R.S., Physician-in-Ordinary to H.M. the Queen and to H.R.H. the Prince of Wales; Consulting Physician to University College Hospital; 63, Brook Street, W. (V.P. 1867–70, P. 1875–6.) *Trans.* 2.

1875 JESSETT, FREDERICK BOWREMAN, Surgeon to the Royal General Dispensary; 16, Upper Wimpole Street, W.

Orig. Memb. JOHNSON, GEORGE, M.D., F.R.S., Physician to King's College Hospital; 11, Savile Row, W. (V.P. 1874–6.) *Trans.* 5.

1878 JOHNSTON, WILLIAM, M.D., M.C., 16, Lonsdale Terrace, Upper Kent Street, Leicester.

Orig. Memb. JONES, SYDNEY, M.B., Surgeon to, and Lecturer on Surgery at, St. Thomas's Hospital; 16, George Street, Hanover Square, W. (C. 1867–8.) *Trans.* 1.

1872 JONES, THOMAS RIDGE, M.D., Physician to the Victoria Hospital for Children; 4, Chesham Place, Belgrave Square, S.W.

1876 JORDAN, FURNEAUX, Surgeon to the Queen's Hospital, Birmingham; 22, Colmore Row, Birmingham. *Trans.* 1.

1878 KEETLEY, CHARLES ROBERT BELL, Assistant Surgeon to the West London Hospital; 10, George Street, Hanover Square, W. *Trans.* 2.

Orig. Memb. KELLY, CHARLES, M.D., Medical Officer of Health for the West Sussex District; Worthing, Sussex.

1882 KESTEVEN, WILLIAM HENRY, 401, Holloway Road, N. *Trans.* 1.

1883 KIDD, PERCY, M.D., Assistant Physician to the Hospital for Consumption, Brompton; 60, Brook Street, Grosvenor Square, W. *Trans.* 1.

1878 LACEY, THOMAS WARNER, 196, Burrage Road, Plumstead, S.E.

1873 LACY, C. DE LACY, M.B., 31, Grosvenor Street, W.

1883 LANE, WILLIAM ARBUTHNOT, M.B., M.S., Assistant Surgeon to the Hospital for Sick Children; 14, St. Thomas's Street, Southwark. *Trans.* 1, *C.S.* 2.

Orig. Memb. LANGTON, JOHN, Surgeon to, and Lecturer on Anatomy at, St. Bartholomew's Hospital, and Surgeon to the City of London Truss Society; 2, Harley Street, W. (C. 1878–80.) *Trans.* 2.

ELECTED

1885 LARDER, HERBERT, St. Marylebone Infirmary, Notting Hill, W.

1883 LAWRENCE, HENRY CRIPPS, 49, Oxford Terrace, Hyde Park, W.

Orig. Memb. LAWSON, GEORGE, Surgeon to the Middlesex Hospital, and Surgeon to the Royal London Ophthalmic Hospital; 12, Harley Street, W. (S. 1871–3, C. 1874–6, V.P. 1881–3.) *Trans.* 16.

1877 LEDIARD, HENRY AMBROSE, M.D., Surgeon to the Cumberland Infirmary; 41, Lowther Street, Carlisle. *Trans.* 4.

Orig. Memb. LEE, HENRY, Consulting Surgeon to St. George's Hospital; 9, Savile Row, W. (V.P. 1870–2.) *Trans.* 7.

1882 LEEDS, THOMAS, Wanderers' Club, 9, Pall Mall, S.W.

1877 LEES, DAVID B., M.D. (C.), Physician (with charge of out-patients) to, and Lecturer on Materia Medica at, St. Mary's Hospital, and Assistant Physician to the Hospital for Sick Children; 2, Thurloe Houses, Thurloe Square, S.W. (C. 1885.) *Trans.* 2.

1879 LICHTENBERG, GEORGE, M.D., 47, Finsbury Square, E.C.

1878 LISTER, Sir JOSEPH, Bart., D.C.L., L.L.D, F.R.S., Professor of Clinical Surgery at King's College, and Surgeon to King's College Hospital; 12, Park Crescent, Regent's Park, W. (P. 1881–2.) *Trans.* 3.

1868 LITTLE, LOUIS STROMEYER, China.

1875 LIVEING, EDWARD, M.D., 52, Queen Anne Street, Cavendish Square, W.

1872 LIVEING, ROBERT, M.D., Lecturer on Dermatology, and Physician to the Skin Department at the Middlesex Hospital; 11, Manchester Square, W. (C. 1883–4.) *Trans.* 2.

1878 LLOYD, ROBERT HODGENS, M.D., Medical Superintendent Lambeth Infirmary, Brook Street, Kennington Road, S.E. *Trans.* 1.

1876 LONGHURST, ARTHUR EDWIN TEMPLE, M.D., 22, Wilton Street, Grosvenor Place, S.W.

1881 LUBBOCK, MONTAGU, M.D., Assistant Physician to Charing Cross Hospital; 19, Grosvenor Street, W.

ELECTED

1876 LUCAS, R. CLEMENT, M.B., B.S. (C.), Senior Assistant Surgeon to, and Demonstrator of Operative and Practical Surgery at, Guy's Hospital; 18, Finsbury Square, E.C. (C. 1883–5.) *Trans.* 6.

1879 LUNN, JOHN REUBEN, Resident Medical Officer, New Marylebone Infirmary, Rackham Street; Ladbroke Grove Road, Notting Hill, W. *Trans.* 3, *C.S.* 2.

1871 MACCORMAC, SIR WILLIAM, Surgeon to, and Lecturer on Surgery at, St. Thomas's Hospital; 13, Harley Street, W. (C. 1877–9.) *Trans.* 5.

1883 †MACFARLANE, ALEXANDER WILLIAM, M.D., Consulting Physician to the Kilmarnock Fever Hospital and Infirmary; Walmer, Kilmarnock, N.B.

1884 MCGILL, ARTHUR FERGUSSON, Professor of Anatomy, Yorkshire College; Surgeon to the Leeds General Infirmary; 23, Park Square, Leeds.

1881 MCHARDY, MALCOLM MACDONALD, Ophthalmic Surgeon to King's College Hospital; 5, Savile Row, W. *Trans.* 1.

1882 MACKENZIE, FREDERIC MORELL; 10, Hans Place, S.W.

Orig. Memb. MACKENZIE, MORELL, M.D., Physician to the Hospital for Diseases of the Throat; 19, Harley Street, Cavendish Square, W. *Trans.* 4.

1879 MACKENZIE, STEPHEN, M.D. (*Hon. Secretary*), Physician to, and Lecturer on Medicine at, the London Hospital; 26, Finsbury Square, E.C. (C. 1884, S. 1885.) *Trans.* 6, *C.S.* 5.

1884 MACKERN, JOHN, M.B., Assistant Physician, Chelsea Hospital for Women; 30, Cambridge Street, Hyde Park, W.

1879 MACLAGAN, THOMAS JOHN, M.D., 9, Cadogan Place, Belgrave Square, S.W.

1875 MACNAMARA, CHARLES, Surgeon to the Westminster Hospital, and to the Royal Westminster Ophthalmic Hospital; 13, Grosvenor Street, W. (C. 1879–81.) *C.S.* 1.

1879 MAGILL, JAMES, M.D., M.C., Surgeon, Coldstream Guards; Coldstream Guards Hospital, Vincent Square, Westminster, S.W.

1885 MAGUIRE, ROBERT, M.D., Assistant Physician to St. Mary's Hospital; St. Mary's Hospital College, 33, Westbourne Terrace, W.

ELECTED

1881 MAKINS, GEORGE HENRY, St. Thomas's Hospital, Albert Embankment, S.E.

Orig. Memb. MARCET, WILLIAM, M.D., F.R.S., 39, Grosvenor Street, W. (C. 1867–9.) *Trans.* 1.

1868 MARSH, F. HOWARD, Assistant Surgeon and Lecturer on Anatomy to St. Bartholomew's Hospital, and Surgeon to the Hospital for Sick Children; 36, Bruton Street, Berkeley Square, W. (C. 1876–7, 1881–83, S. 1878–80.) *Trans.* 9, *C.S.* 2.

1875 MARSHALL, F. J., Resident Medical Officer, St. George's Hospital, W.

1884 MAUDSLEY, HENRY, M.D., Resident Medical Officer, University College Hospital, Gower Street, W.C.

1868 †MAY, EDWARD HOOPER, M.D., High Cross, Tottenham, Middlesex, N.

1868 MEADOWS, ALFRED, M.D., Physician-Accoucheur to, and Lecturer on Midwifery at, St. Mary's Hospital; 27, George Street, Hanover Square, W. (C. 1871-4.) *Trans.* 1.

1876 MELLADEW, H. F. L., M.D., Surgeon-Major, Royal Horse Guards; Regent's Park Barracks, N.W.

1878 MEREDITH, WILLIAM APPLETON, M.B., C.M., Surgeon to the Samaritan Free Hospital for Women and Children; 6, Queen Anne Street, Cavendish Square, W.

1873 MICKLE, WILLIAM JULIUS, M.D., Physician Superintendent, Grove Hall Asylum, Bow, E.

1877 *MILNER, EDWARD, Surgeon to the Lock Hospital; 32, New Cavendish Street, Portland Place, W.

1882 MONEY, ANGEL, M.D., Assistant Physician to the City of London Hospital for Diseases of the Chest, Victoria Park, and to the Hospital for Sick Children, Great Ormond Street; 24, Harley Street, Cavendish Square, W. *Trans.* 1.

1874 MORGAN, JOHN HAMMOND (C.), Assistant Surgeon to the Charing Cross Hospital, and to the Hospital for Sick Children; 68, Grosvenor Street, W. (C. 1883–5.) *Trans.* 1, *C.S.* 2.

1877 MORRIS, HENRY, M.B. (C.), Surgeon to, and Lecturer on Surgery at, the Middlesex Hospital; 2, Mansfield Street, Portland Place, W. (C. 1884–5.) *Trans.* 6.

ELECTED

1877 MORRIS, MALCOLM ALEX., Lecturer on Skin Diseases at St. Mary's Hospital; 63, Montagu Square, W. *Trans.* 1.

1885 MOTT, FREDERICK WALKER, M.B., C.M., 55, Torrington Square, W.C.

1879 MOULLIN, CHARLES W. MANSELL, Assistant Surgeon to the London Hospital; 69, Wimpole Street, Cavendish Square, W. *Trans.* 1.

Orig. Memb. MOXON, WALTER, M.D., F.L.S., Physician to, and Lecturer on Medicine at, Guy's Hospital; 6, Finsbury Circus, E.C. (C. 1874–6.) *Trans.* 2.

1878 MUIR, J. C. POLLOCK, 44, Cornwall Road, Westbourne Park, W.

1875 MURPHY, SHIRLEY F., 158, Camden Road, N.W.

1885 MURRAY, ALEXANDER DALTON, M.B., Rickmansworth, Herts.

1883 MURRAY, HUBERT MONTAGUE, M.B., Assistant Physician to Charing Cross Hospital; 27, Savile Row, W.

1868 MYERS, ARTHUR BOWEN RICHARDS, Surgeon to 1st Battalion of the Coldstream Guards; Vincent Square, Westminster, S.W. (C. 1877–9.) *Trans.* 1.

1882 MYERS, A. T., M.D., 24, Clarges Street, Piccadilly, W. *C.S.* 1.

1873 MYRTLE, ANDREW S., M.D., 8, Park Parade, Harrogate.

1874 NANKIVELL, ARTHUR WOLCOT, Resident Surgeon, St. Bartholomew's Hospital, Chatham.

1875 NETTLESHIP, EDWARD, Ophthalmic Surgeon to, and Lecturer on Ophthalmology at, St. Thomas's Hospital; 5, Wimpole Street, Cavendish Square, W. (C. 1881–82.) *Trans.* 2.

Orig. Memb. NORTON, ARTHUR TREHERN, Surgeon to, and Lecturer on Surgery at, St. Mary's Hospital; 6, Wimpole Street, W. (C. 1874–6.) *Trans.* 6.

Orig. Memb. NUNN, THOMAS WILLIAM, Consulting Surgeon to the Middlesex Hospital; 8, Stratford Place, Oxford Street, W. (C. 1873–74.) *Trans.* 5.

1880 O'CONNOR, BERNARD, M.D., Physician to the North London Hospital for Consumption, and Physician to the Westminster General Dispensary; 17, St. James' Place, S.W. *Trans.* 1.

ELECTED

Orig. Memb. OGLE, JOHN WILLIAM, M.D. (V.P.), Consulting Physician to St. George's Hospital; 30, Cavendish Square, W. (C. 1867–8, V.P. 1884–5.) *Trans.* 6.

1868 †OGLE, WILLIAM, M.D., Physician to the Derbyshire General Infirmary; 98, Friar Gate, Derby.

1883 OLIVER, GEORGE, M.D., West End Park, Harrogate.

1868 OPPERT, FRANZ, M.D.; 128, Leipzigerstrasse, Germany. *Trans.* 1.

1877 ORD, WILLIAM MILLER, M.D., Physician to, and Lecturer on Medicine at, St. Thomas's Hospital; 7, Brook Street, Hanover Square, W. (C. 1882–4.) *Trans.* 5.

1884 ORMSBY, LAMBERT HEPENSTAL, M.D., Lecturer on Clinical and Operative Surgery at, and Surgeon to, the Meath Hospital and County Dublin Infirmary; Surgeon to the Children's Hospital, Dublin; 4, Merrion Square West, Dublin.

1883 ORTON, GEORGE HUNT, M.B., 1, Campden Hill Road, Kensington, W.

1877 OWEN, ISAMBARD, M.D., Assistant Physician to St. George's Hospital; 5, Hertford Street, Mayfair, W. *Trans.* 1.

1875 PAGE, HERBERT W., M.C., M.B., Surgeon to, and Joint-Lecturer on Surgery at, St. Mary's Hospital; 146, Harley Street, W. (C. 1882–4.) *Trans.* 1.

1884 PAGET, STEPHEN, 5, Wimpole Street, Cavendish Square, W.

1873 PARKER, ROBERT WILLIAM, Surgeon to the East London Hospital for Children; 8, Old Cavendish Street, W. (C. 1882–4.) *Trans.* 4, *C.S.* 4.

1881 PARKER, RUSHTON, M.B., Professor of Surgery in University College, Liverpool, and Assistant Surgeon Liverpool Royal Infirmary; 59, Rodney Street, Liverpool. *Trans.* 1, *C.S.* 1.

1881 PASTEUR, WILLIAM, M.D., Medical Registrar to the Middlesex Hospital; Physician to the North-Eastern Hospital for Children; 19, Queen Street, May Fair, W.

1883 PAUL, JOHN LISTON, M.D., 43, Queensborough Terrace, W.

Orig. Memb. PAVY, FREDERICK WILLIAM, M.D., F.R.S., Physician to Guy's Hospital; 35, Grosvenor Street, W. (C. 1869–71, V.P. 1882–4.) *Trans.* 3.

ELECTED

1879 PEEL, ROBERT, 130, Collins Street East, Melbourne, Victoria.

1882 PEPPER, AUGUSTUS JOSEPH, M.S., M.B., Surgeon to St. Mary's Hospital; 122, Gower Street, W.C. *Trans.* 1.

1874 PHILLIPS, CHARLES DOUGLAS F., M.D., 10, Henrietta Street, Cavendish Square, W.

1884 PHILLIPS, SIDNEY PHILIP, M.D., Physician to St. Mary's Hospital; 12, Radnor Place, Hyde Park, W. *Trans.* 1.

Orig. Memb. PICK, THOMAS PICKERING (V.P.), Surgeon to, and Lecturer on Surgery at, St. George's Hospital; Surgeon to the Belgrave Hospital for Children; 18, Portman Street, Portman Square, W. (S. 1874–7, C. 1878–80, V.P. 1885.) *Trans.* 3.

1885 PITT, GEORGE NEWTON, M.D., Assistant Physician to the East London Hospital for Children; 34, Ashburn Place, South Kensington.

1883 PITTS, BERNARD, M.A., M.C., Assistant Surgeon, St. Thomas's Hospital; 31, Harley Street, W. *Trans.* 1.

1871 †PLAYNE, ALFRED, M.B., Maidenhead.

1884 POLAND, JOHN, Demonstrator of Anatomy, Guy's Hospital; 16, St. Thomas's Street, Southwark, S.E.

1884 POLLARD, BILTON, Surgical Registrar to University College Hospital; 50, Torrington Square, W.

1868 POLLOCK, JAMES EDWARD, M.D., Consulting Physician to the Hospital for Consumption and Diseases of the Chest; 52, Upper Brook Street, Grosvenor Square, W. (C. 1878–80.)

1871 POORE, GEORGE VIVIAN, M.D., Professor of Medical Jurisprudence in University College, and Assistant Physician to University College Hospital; 30, Wimpole Street, W. (C. 1879–81.) *Trans.* 2.

1873 PORT, HEINRICH, M.D., Physician to the German Hospital; 48, Finsbury Square, E.C.

1881 POWELL, H. A., M.A., Elm Cottage, Beckenham, Kent.

Orig. Memb. POWELL, R. DOUGLAS, M.D., Physician to, and Lecturer on Practical Medicine at, the Middlesex Hospital; Physician to the Hospital for Consumption and Diseases of the Chest, Brompton; 62, Wimpole Street, Cavendish Square, W. (C. 1874–76.) *Trans.* 4.

1868 PRENTIS, CHARLES, Surgeon-Major, Bengal Medical Service; India.

ELECTED

1884 PRINGLE, JOHN JAMES, M.B., Assistant Physician to the Middlesex Hospital and to the Royal Hospital for Diseases of the Chest; 35, Bruton Street, Berkeley Square, W. *Trans.* 1.

1884 PYE-SMITH, PHILIP HENRY, M.D., Physician to, and Lecturer on Medicine at, Guy's Hospital; Examiner in Physiology at the University of London; 54, Harley Street, Cavendish Square, W.

Orig. Memb. QUAIN, RICHARD, M.D., F.R.S., Consulting Physician to the Hospital for Consumption and Diseases of the Chest; 67, Harley Street, W. (C. 1867–9.)

Orig. Memb. RAMSKILL, J. SPENCE, M.D., Consulting Physician to the London Hospital; Senior Physician to the National Hospital for the Paralysed and Epileptic; 5, St. Helen's Place, Bishopsgate Street, E.C.

1873 RANSFORD, GIFFORD, M.D. (C.), 27, Gloucester Place, Hyde Park, W. (C. 1884–5.)

1868 RASCH, ADOLPHUS A., M.D., Physician for Diseases of Women to the German Hospital; 7, South Street, Finsbury Square, E.C.

1877 RAYNER, HENRY, M.D., Lecturer on Mental Diseases at St. Thomas's Hospital; Middlesex County Lunatic Asylum, Hanwell, W.

1883 READ, THOMAS LAURENCE, 11, Petersham Terrace, Queen's Gate, W.

1874 REE, FREDERICK G., Royal India Asylum, Ealing, W.

Orig. Memb. REES, GEORGE OWEN, M.D., F.R.S., Consulting Physician to Guy's Hospital; 26, Albemarle Street, Piccadilly, W. (V.P. 1871–3.)

1868 REEVES, HENRY A., Assistant Surgeon to the London Hospital; 78, Grosvenor Street, W. *Trans.* 2.

Orig. Memb. REYNOLDS, JOHN RUSSELL, M.D., F.R.S., Consulting Physician to University College Hospital; 38, Grosvenor Street, W. (C. 1867–8.)

1868 RICE, MICHAEL W., M.D. (C. 1876–8.)

1883 RING, EDMUND CUTHBERT, Salisbury Club, 10, St. James' Square, S.W.

Orig. Memb. RINGER, SYDNEY, M.D., Professor of the Principles and Practice of Medicine in University College, and Physician to University College Hospital; 15, Cavendish Place, W. (C. 1871–2.)

1877 RIVINGTON, WALTER, M.S., M.B., Surgeon to, and Lecturer on Surgery at, the London Hospital; 22, Finsbury Square, E.C. *Trans.* 2.

ELECTED

1873 †ROBERTS, DAVID LLOYD, M.D., Physician to St. Mary's Hospital, Manchester; 23, St. John Street, Manchester.

1883 ROBERTS, FREDERICK THOMAS, M.D., Professor of Materia Medica and Therapeutics in University College, London, and Physician to University College Hospital; Physician to the Hospital for Consumption, Brompton; Examiner in Materia Medica in the University of London; 53, Harley Street, Cavendish Square, W.

1885 ROBSON, A. W. MAYO, Surgeon to the Leeds General Infirmary, Hillary Place, Leeds. *Trans.* 1.

1875 ROGERS, WILLIAM RICHARD, M.D., 56, Berners Street, Oxford Street, W.

1877 ROTH, BERNARD M. S., 48, Wimpole Street, W., and Rossmore, Preston Road, Brighton. *Trans.* 1, *C.S.* 2.

Orig. Memb. ROUSE, JAMES, Surgeon to St. George's Hospital, and to the Royal Ophthalmic Hospital, Charing Cross; 2, Wilton Street, Grosvenor Place, S.W. (C. 1875–7.) *Trans.* 2.

1874 ROWLAND, EDWARD R.

1882 SAINSBURY, HARRINGTON, M.D., 27, Gower Street, W.C.

1868 SANDERSON, HUGH JAMES, M.D., 26, Upper Berkeley Street, W.

Orig. Memb. SANDERSON, JOHN BURDON, M.D., LL.D., F.R.S., Waynflete Professor of Physiology in the University of Oxford; 50, Banbury Road, Oxford. (S. 1867–9, C. 1870, V.P. 1871–3.) *Trans.* 3.

1878 SANGSTER, ALFRED, M.B., Physician to the Skin Department and Lecturer on Skin Diseases at the Charing Cross Hospital; 6, Savile Row, W. *Trans.* 4.

1873 SAVAGE, GEORGE HENRY, M.D., Bethlem Royal Hospital, St. George's Road, S.E. (C. 1882–3.)

1885 SAWTELL, TOM HENRY, M.B., 14, Stapleton Hall Road, Stroud Green, N.

1877 SEATON, EDWARD, M.D., 35, George Street, Hanover Square, W.

1869 SEDGWICK, LEONARD WILLIAM, M.D., 2, Gloucester Terrace, Hyde Park, W. (C. 1879–81.)

ELECTED

1878 SEMON, FELIX, M.D. (C.), Assistant Physician for Diseases of the Throat to St. Thomas's Hospital; 59, Welbeck Street, Cavendish Square, W. (C. 1885.) *Trans.* 4.

1884 SHARKEY, SEYMOUR J., M.B., Assistant Physician and Lecturer on Pathology to St. Thomas's Hospital; 2, Portland Place, W.

1875 SHERWOOD, ARTHUR PAUL, 8, Seaside Road, Eastbourne.

Orig. Memb. SIBLEY, SEPTIMUS WILLIAM, 7, Harley Street, Cavendish Square, W. (C. 1871–4.)

1879 SKERRITT, EDWARD MARKHAM, M.D., Physician to the Bristol General Hospital, Lecturer on Medicine at the Bristol Medical School; Coburg Villa, Richmond Hill, Clifton, Bristol. *Trans.* 2.

1877 SKINNER, WILLIAM A., 45, Lower Belgrave Street, Eaton Square, S.W.

1872 SLIGHT, GEORGE, M.D., 3, Clifford Street, Bond Street, W.

1882 SMITH, E. NOBLE, Senior Surgeon, and Surgeon to the Orthopædic Department, of the Farringdon Dispensary; 24, Queen Anne Street, Cavendish Square, W. *Trans.* 1.

1868 SMITH, HEYWOOD, M.D., Physician to the Hospital for Women, and Physician to the British Lying-in Hospital; 18, Harley Street, Cavendish Square, W.

1868 SMITH, PROTHEROE, M.D., Physician to the Hospital for Women; 42, Park Street, Grosvenor Square, W.

1884 SMITH, R. PERCY, M.D., Assistant Medical Officer, Bethlem Royal Hospital, S.E.

Orig. Memb. SMITH, THOMAS, Surgeon to, and Lecturer on Clinical Surgery at, St. Bartholomew's Hospital, and Surgeon to the Hospital for Sick Children; 5, Stratford Place, Oxford Street, W. (C. 1869–71, V.P. 1880–82.) *Trans.* 13.

1875 SMITH, T. GILBART, M.A., M.D. (C.), Assistant Physician to the London Hospital; Physician to the Royal Hospital for Diseases of the Chest, City Road; 68, Harley Street, Cavendish Square, W. (C. 1883–5.)

1873 SMITH, WILLIAM JOHNSON, Surgeon to the Seamen's Hospital, Greenwich, S.E.

1873 SMITH, WILLIAM WILBERFORCE, M.D., 14, Stratford Place, Oxford Street, W.

ELECTED

1883 SMITH, WINCKWORTH TONGE, M.D., 129, Ladbroke Grove, W.

1868 SNOW, WILLIAM V., M.D., Richmond Gardens, Bournemouth.

Orig. Memb. SOUTHEY, REGINALD, M.D., Commissioner in Lunacy, 32, Grosvenor Road, Pimlico, W. (C. 1867–70, 1876–8, S. 1873–5, V.P. 1883–4.) *Trans.* 16.

1885 SPICER, FREDERICK, M.B., Royal Free Hospital, Gray's Inn Road, W.C.

1882 SPOONER, FREDERICK HENRY, M.D., Howard House, Clapton, E.

1876 SQUIRE, A. BALMANNO, M.B., 24, Weymouth Street, Portland Place, W. *Trans.* 5.

1879 STAPLES, FRANCIS PATRICK, late Assistant Professor of Military Surgery, Netley; Army Medical Department, Royal Victoria Hospital, Netley, and Junior Army and Navy Club, King Street, St. James', S.W. [Station Hospital, Gibraltar].

1871 STEWART, WILLIAM EDWARD, 16, Harley Street, Cavendish Square, W.

1874 †STIRLING, EDWARD C., M.D., late Assistant Surgeon to, and Lecturer on Physiology at, St. George's Hospital; Adelaide, South Australia. [care of T. GEMMELL, Esq., 11, Essex Street, Strand, W.C.]

1881 STOKES, HENRY FRASER, 2, Highbury Crescent, N.

1878 STOKES, WILLIAM, M.D., Professor of Surgery, Royal College of Surgeons, Ireland; Surgeon to the Richmond Surgical Hospital; 5, Merrion Square North, Dublin. *Trans.* 2.

1884 STONHAM, CHARLES, Curator of the Anatomical Museum at University College; Assistant Surgeon to the Cancer Hospital, Brompton; 109, Gower Street, W.C. *C.S.* 2.

1878 STRUGNELL, FREDERICK WILLIAM, 45, Highgate Road, Highgate, N. *C.S.* 1.

1878 †STURGE, WILLIAM ALLEN, M.D., late Assistant Physician to the Royal Free Hospital; 9, Rue Longchamp, Nice, Alpes Maritimes, France. *Trans.* 4.

1872 *SUTHERLAND, HENRY, M.D., Lecturer on Insanity, Westminster Hospital; 6, Richmond Terrace, Whitehall, S.W. *Trans.* 1.

1868 SUTRO, SIGISMUND, M.D., Senior Physician to the German Hospital; 37A, Finsbury Square, E.C.

ELECTED

Orig. Memb. SUTTON, HENRY GAWEN, M.B., Physician to, and Lecturer on Pathology at, the London Hospital; 9, Finsbury Square, E.C. (C. 1878.) *Trans.* 2.

1882 SYMONDS, CHARTERS JAMES, M.S., Assistant Surgeon to Guy's Hospital; 26, Weymouth Street, Portland Place, W. *Trans.* 4, *O.S.* 1.

1876 SYMONDS, HORATIO PERCY, Surgeon to the Radcliffe Infirmary, Oxford; 35, Beaumont Street, Oxford.

1868 TATHAM, JOHN, M.D., Physician to the Hospital for Consumption and Diseases of the Chest; 12, George Street, Hanover Square, W.

1878 TAYLER, FRANCIS THOMAS, B.A., M.B., 224, Lewisham High Road, S.E.

1875 TAYLOR, FREDERICK, M.D., Physician to, and Lecturer on Materia Medica at, Guy's Hospital; Physician to the Evelina Hospital for Sick Children; 11, St. Thomas's Street, Southwark, S.E. (S. 1879–81, C. 1882–4.) *Trans.* 9. *O.S.* 1.

1882 TAYLOR, SEYMOUR, M.D., Physician to the North London Hospital for Consumption; 22, Taviton Street, Gordon Square, W.C. *Trans.* 1.

1885 †TAYLOR, W. C. EVERLEY, 34, Queen Street, Scarborough.

Orig. Memb. TEEVAN, WILLIAM F., Mostyn Villa, Brockman Road, Folkestone, Kent. (C. 1880–2.) *Trans.* 8.

1882 THIN, GEORGE, M.D., 22, Queen Anne Street, Cavendish Square, W. *Trans.* 1.

Orig. Memb. THOMPSON, EDMUND SYMES, M.D., Physician to the Hospital for Consumption and Diseases of the Chest; Gresham Professor of Medicine; 33, Cavendish Square, W. (C. 1880–82.) *Trans.* 1.

Orig. Memb. THOMPSON, Sir HENRY, Knt., Surgeon-Extraordinary to H.M. the King of the Belgians; Emeritus Professor of Clinical Surgery in University College; 35, Wimpole Street, W. (C. 1867–8.) *Trans.* 1.

Orig. Memb. THOMPSON, HENRY, M.D., Fellow of St. John's College, Cambridge; Consulting Physician to the Middlesex Hospital; 53, Queen Anne Street, W. (V.P. 1875–7.) *Trans.* 4.

1872 THORNTON, WILLIAM PUGIN, Canterbury. *Trans.* 5.

ELECTED

1876 THRUPP, JAMES GODFREY, Fern House, Heston Hounslow.

1885 THURSFIELD, THOMAS WILLIAM, M.D., 26, The Parade, Leamington.

1877 TIBBITS, HERBERT, 68, Wimpole Street, Cavendish Square, W.

1874 TRAVERS, WILLIAM, M.D., 2, Phillimore Gardens, Kensington, W.

1884 TREVES, FREDERICK, Surgeon to, and Lecturer on Anatomy at, the London Hospital; 18, Gordon Square, W.C.

1882 TURNER, FRANCIS CHARLEWOOD, M.A., M.D., Physician to the London Hospital, and to the North-Eastern Hospital for Children; 15, Finsbury Square, E.C.

1882 TURNER, GEORGE ROBERTSON, Visiting Surgeon, Seamen's Hospital, Greenwich; Joint Lecturer on Practical Surgery, St. George's Hospital; 49, Green Street, Park Lane, W. *Trans.* 3.

1877 TWEEDY, JOHN, Professor of Ophthalmic Medicine and Surgery at University College, and Assistant Ophthalmic Surgeon to University College Hospital; Assistant Surgeon to the Royal London Ophthalmic Hospital; 100, Harley Street, Cavendish Square, W.

1878 TYSON, WILLIAM JOSEPH, M.D., 10, Langhorne Gardens, Folkestone. *Trans.* 4.

1881 UHTHOFF, JOHN CALDWELL, M.D., 46, Western Road, Hove, Brighton.

1868 VENNING, EDGCOMBE, 30, Cadogan Place, S.W. (C. 1876–8.) *Trans.* 2.

1868 WAGSTAFFE, WILLIAM WARWICK, Purleigh, St. John's Hill, Sevenoaks. (C. 1878.)

1885 WAKLEY, THOMAS, Jun., 96, Redcliffe Gardens, S.W.

1875 WALSHAM, WILLIAM J., Assistant Surgeon to, and Demonstrator of Practical and Orthopædic Surgery at, St. Bartholomew's Hospital; Surgeon to the Metropolitan Free Hospital and to the Royal Hospital for Diseases of the Chest; 27, Weymouth Street, Portland Place, W. (C. 1882–4.) *Trans.* 3.

1876 WATERS, JOHN H., M.D., 101, Jermyn Street, St. James's, S.W.

ELECTED

1868 WATKINS, EDWIN T., M.D., 61, Guilford Street, W.C. (C. 1881–83.)

Orig. Memb. WATSON, WILLIAM SPENCER, M.B., Surgeon to the Great Northern Hospital; Surgeon to the Royal South London Ophthalmic Hospital; 7, Henrietta Street, Cavendish Square, W. (C. 1880–82.) *Trans.* 10.

1879 WATTEVILLE, ARMAND DE, M.A., M.D., B.Sc., Medical Electrician to St Mary's Hospital; 30, Welbeck Street, W.

Orig. Memb. WEBER, HERMANN, M.D., Physician to the German Hospital; 10, Grosvenor Street, W. (C. 1867–71, V.P. 1873–5.) *Trans.* 9.

1876 WEIR, ARCHIBALD, M.D., St. Mungho's, Great Malvern.

1868 WELLS, SIR THOMAS SPENCER, Bart., Surgeon in Ordinary to H.M.'s Household; Surgeon to the Samaritan Free Hospital; 3, Upper Grosvenor Street, W. (C. 1873.)

1882 WEST, SAMUEL, M.D., Physician to the City of London Hospital for Diseases of the Chest, Victoria Park; Physician to the Royal Free Hospital; Medical Registrar and Medical Tutor at St. Bartholomew's Hospital; 15, Wimpole Street, Cavendish Square, W. *Trans.* 2, *C.S.* 1.

1874 WHEELHOUSE, CLAUDIUS GALEN, Senior Surgeon to the Leeds General Infirmary, and Lecturer on Surgery, Leeds Medical School; Hilary Place, Leeds. *Trans.* 1.

1868 WHIPHAM, THOMAS TILLYER, M.B., Physician to, and Lecturer on Clinical Medicine at, St. George's Hospital; 11, Grosvenor Street, Grosvenor Square, W. (C. 1878–80.) *Trans.* 10.

1874 WHISTLER, W. M., M.D., 28, Wimpole Street Cavendish Square, W.

1882 WHITE, EDWIN FRANCIS, St. Thomas's Hospital, and 7, Dealtry Road, Putney.

1883 WHITE, WILLIAM HALE, M.D., Assistant Physician to Guy's Hospital; 4, St. Thomas's Street, Southwark. *Trans.* 3, *S.C.* 2.

1883 WHITE, WILLIAM HENRY, M.D., Assistant Physician to the Royal Hospital for Diseases of the Chest; 43, Weymouth Street, W.

ELECTED

1882 WHITTLE, EDWARD GEORGE, M.D., 65, Dyke Road, Brighton.

1871 WIGHT, GEORGE, M.B., C.M.; 428, Liverpool Road, N.

1879 WILCOX, HENRY, M.B., Dorchester House, Herbert Road, Woolwich.

Orig. Memb. WILKS, SAMUEL, M.D., F.R.S., Consulting Physician to Guy's Hospital; 72, Grosvenor Street, W. (C. 1871–2.) *Trans.* 1.

1884 WILLCOCKS, FREDERICK, M.D., Assistant Physician to the Charing Cross Hospital; Physician in charge of Out-patients at the Evelina Hospital for Children; 14, Mandeville Place, W. *C.S.* 1.

Orig. Memb. WILLETT, ALFRED, Surgeon to St. Bartholomew's Hospital; 36, Wimpole Street, W. (C. 1872–5.) *C.S.* 1.

Orig. Memb. WILLIAMS, CHARLES THEODORE, M.D., Physician to the Hospital for Consumption and Diseases of the Chest; 47, Upper Brook Street, Grosvenor Square, W. (C. 1877–9.) *Trans.* 8.

1881 WILLIAMS, JOHN, M.D. (C.), Obstetric Physician to University College Hospital; 11, Queen Anne Street, Cavendish Square, W. (C. 1885.)

1870 WILLIAMS, WILLIAM RHYS, M.D., Commissioner in Lunacy; 19, Whitehall Place, S.W.

1876 WILLIAMSON, JAMES MANN, M.D.; Ventnor, Isle of Wight.

Orig. Memb. WILLIS, FRANCIS, M.D., Braceborough, Stamford.

1868 WILTSHIRE, ALFRED, M.D., Joint Lecturer on Obstetrics, and Assistant Physician-Accoucheur, St. Mary's Hospital; Torridon, Somers Road, Reigate. (C. 1880–82.) *Trans.* 1.

1880 WOOD, JOHN, F.R.S., Professor of Clinical Surgery in King's College, London, and Senior Surgeon to King's College Hospital; 61, Wimpole Street, Cavendish Square, W.

1883 WOODCOCK, JOHN ROSTRON, Hagley Road, Birmingham.

1879 WOODWARD, GEORGE P. M., M.D., Deputy Surgeon-General; Sydney, New South Wales.

1884 WORTS, EDWIN, 6, Trinity Street, Colchester.

1872 YEO, I. BURNEY, M.D., Physician to King's College Hospital; 44, Hertford Street, Mayfair, W. (C. 1881–3.) *Trans.* 5.

REPORT

OF THE

COUNCIL OF THE CLINICAL SOCIETY.

DECEMBER, 1884.

IN making the customary Annual Statement the Council is gratified to assure the members that during the past year the Society has continued to maintain its position and to justify its existence. The number and character of the communications, the quality of the debates, and the large attendances at the meetings have been evidence to its growth in importance and estimation.

The roll of members steadily increases; it comprises now 288 resident and 94 non-resident members. Thirty-one new members have been elected during the past year. Two have resigned membership, and three resident members have become non-resident.

Since the last Annual Meeting the Society has had to deplore the loss of three of its members by death, viz. Dr. G. Hall Davis and Dr. A. W. Barclay, who were both original members of the Society, and Dr. F. A. Mahomed, who at the time of his death was a member of the Council and was serving upon two of your Committees. By the death of Mr. Cæsar Hawkins the Society loses an honorary member, and in Professor S. Gross a foreign honorary member. The death in January last of Mr. B. R. Wheatley, the much-esteemed Librarian of the Royal Medical and Chirurgical Society, deprived the Secretaries of one whose assistance had always been highly valued. The Council marked its sense of Mr. Wheatley's long-continued and ungrudging service by voting

the sum of £25 to the Testimonial Fund raised on behalf of his sister and niece.

The balance-sheet presented herewith shows that financially the Society is in a most satisfactory condition. Grants of money have been made in aid of Committees; and the Society's balance at the bankers amounts to £112 11*s.* 2*d.*, exclusive of £600 invested in Consols.

The exhibition of living specimens at the meetings has been continued during the past session on an increasing scale; and it is felt that in the development of this practice the value of the Society is much enhanced. The records of cases so exhibited now form a distinctive feature of the *Transactions*, and the current volume contains illustrations of such cases. Arrangements have been recently made to facilitate this exhibition.

At the close of last year a large and influential Committee was nominated to investigate the remarkable affection known as myxœdema, in records of which the Society's *Transactions* are rich, including the earliest published cases. The Committee is actively pursuing its labours, and will, in due course, present a report, which cannot fail to throw much light upon the clinical history and pathology of the disease. In connection with this inquiry some important experimental researches were commenced on behalf of the Committee by Mr. Godlee, in conjunction with Professor Horsley, at the Brown Institution, and have been pursued by the latter gentleman, who dealt with the results so far obtained, in his course of lectures delivered at the University of London in December.

The Committee on spina bifida and its treatment by iodoglycerine injection has nearly completed its inquiry; and the report, which will comprise a very thorough investigation of the subject, will shortly be presented.

The Council cannot conclude this report without alluding with satisfaction to the recent valuable and exhaustive debate upon Charcot's disease of joints, which arose out of a paper by Mr. Morrant Baker, one of the Vice-Presidents. So large a number of illustrative living specimens and of morbid preparations, the latter including a valuable series kindly lent by Professor Charcot, has never hitherto been brought together in this country; and the thanks of the Society are due to all those gentlemen who thus contributed to the success of the meetings. The effect of this prolonged debate will be to enlighten the profession upon the nature and relations of the

arthropathy. In view of the interest it excited the Council has made arrangements to secure a verbatim report for publication in the *Transactions*, and has also nominated a Committee to report upon the cases brought forward.

The seventeenth volume of the Society's *Transactions*, recently published, exhibits a few slight but desirable improvements in style. It contains forty-eight communications, twenty records of living specimens, and twelve lithographic plates.

CLINICAL SOCIETY OF LONDON.—BALANCE SHEET, 1883–84.

1883		£	s.	d.	1884		£	s.	d.
	To balance from last account	160	11	2		By purchase of £50 Consols	50	13	9
	„ 271 Annual subscriptions	284	11	0		„ Cost of printing, paper, and binding Vol. XVII of *Transactions*	90	4	4½
	„ 2 Arrears	2	2	0		„ Cost of illustrating ditto	49	19	6
	„ 30 Admission fees (resident members)	63	0	0		„ „ delivery of ditto	7	15	7½
	„ 3 „ „ (non-resident members)	6	6	0		„ „ 500 copies of Rules	4	12	6
	„ 1 Composition fee for *Transactions*	3	3	0		„ „ 50 Lists of Members	1	9	6
	„ Sale of *Transactions* by Messrs. Longmans	17	14	3		„ Payment to Royal Medical and Chirurgical Society for use of rooms	78	15	0
	„ Dividend on £550 Consols	8	1	7		„ Refreshments and attendance	31	10	0
	„ „ £600 „	8	16	3		„ R. Coldrey for attendance	7	10	0
						„ Secretarial assistance	23	12	6
						„ Collection of subscriptions	13	6	0
						„ Petty cash	4	6	0
						„ Printing and stationery	8	7	6
						„ Payment to Wheatley Memorial Fund	25	0	0
						„ „ Committee on Spina Bifida	10	0	0
						„ „ „ Myxœdema	15	0	0
						„ „ Martindale for Committee on Urine	4	12	1
						„ Cost of advertising, and Messrs. Longmans' commission on sale of *Transactions*	4	19	9
						„ Balance at Society's bankers	122	11	2
		£554	5	3			£554	5	3

Amount of Stock standing in the names of the Trustees, January 1, 1885 . . . £600.

Examined and found correct { T. J. MACLAGAN, WALTER RIVINGTON, } *Auditors.*

CHRISTOPHER HEATH, *Treasurer.*

SIDNEY COUPLAND, *Hon. Sec.*

PRESIDENTIAL ADDRESS

DELIVERED AT THE

CLINICAL SOCIETY OF LONDON

ON FEBRUARY 13TH, 1885,

BY THOMAS BRYANT, F.R.C.S.,

PRESIDENT.

GENTLEMEN,—Inclination, no less than a sense of duty, urges me to thank you warmly for the distinguished honour you have conferred upon me by placing me in this chair. I know of no higher compliment than the one you have bestowed upon me which the working members of a Society such as this can pay to a working brother. I value it accordingly, and will do my best to justify your choice. Should I fail, however, I must throw some of the responsibility upon your shoulders, since it has been by your kind interpretation of my fitness for the position that I am placed here.

The work that the Society has already done has been very good, but it is yet allowable to hope that that achieved in the future may be still better. Our predecessors have laboured productively for our advantage in various ways. Let us endeavour to improve upon their work, aud so add to the sum total of attained results that the next generation may be able to say like things of us. Let us continue to walk—as they have walked—in the paths of patient observation, and be precise about our facts; upon these facts and observations let us think closely and consistently, and then without fear or hesitation carry our reasoning to its legitimate conclusions. Let us, however, in our facts beware of accepting the false for the true, and so escape false reasoning; and let us be sure that the words we use convey clear thoughts. Let us, moreover, in our anxiety to be discoverers, guard against announcing as a

novelty some thought or observation which, though new to us, may not be so fresh to others; remembering in the pursuit of knowledge that our own observations are so mixed up with those of others, that our thoughts are so often merely the outcome of others' thoughts; our ideas are so constantly no more than the assimilation of the thoughts and observations of other men, that to be original is almost impossible. With respect to the use of terms, for instance, can we say that, under all circumstances, we are as careful as we should be, and that we never deceive ourselves or others by using phrases which, from our not being clear as to their meaning, tend in reality either to confusion or perhaps to something worse? To illustrate my meaning, may I ask what is meant by "strumous" disease when that term is applied to a joint, bone, or other local affection? Do we all agree as to its meaning? When we apply it, do we feel that we are conveying any accurate idea of the case under discussion to our pupils or hearers? As a teacher I unhesitatingly say that by the term "strumous disease" applied to any local affection, whether of joint, bone, or gland, we do not convey any clear thought. Indeed, I may say that we do the reverse; we confuse instead of clearing ideas.

Let me ask the members of this learned Society what they understand by the terms "strumous disease of a joint," "strumous glands," &c. It would be interesting to have in writing the definitions of these phrases from the individual members. Would they all agree? Would they even be at all consistent? The answers to these questions would, I fear, be in the negative. With such an admission, can we therefore possibly say that this very common term is an intelligible one, or that it ought to be retained? If we mean, when we use the term, to convey the idea that the enlargement of the affected tissue is a special one caused by struma, may I ask what is struma? and how does it cause the local disease? Or, do we mean that it is an inflammatory affection of some form, in which the inflammatory process is modified by a condition of body which may or may not be associated with the deposition of tubercle? If the latter be the correct view, as I take it to be, why should we not say so, and why should we not call the local affection a chronic inflammation in a strumous subject, or a chronic strumous inflammation either of the bone, of the synovial membrane, or of both, the inflammatory process being modified by the constitutional tendency of the individual, in the same way as a chronic inflammation in a gouty or syphilitic subject may be

modified? It need hardly be pointed out that by this change of expression much good would accrue, since the alteration would tend to clear both the pathological and the clinical aspects of the case, and help our pupils instead of confusing them. At the present time, we hear of strumous, scrofulous, and tubercular disease of a joint or of bone, as if inflammation had nothing to do with the changes met with, and as if any one of the terms carried with it a precise thought.

Again, may I ask—and I do so with some amount of trepidation— what are we now to understand, when discussing the treatment of wounds, by the term "antiseptic precautions?" How, with the diversities of practice encountered, is the expression to be interpreted? Has it, indeed, at the present day any special significance? One surgeon, when asked the question as to its meaning, will answer, It means, of course, the spray and gauze system in one or other of its modes of application. A second, with equal decision, will apply it to one of the many forms of antiseptic irrigation, in which the antiseptic in solution is employed either during or after an operation, and some antiseptic gauze or other dressing later on. A third surgeon will use the term as applied to some antiseptic dry or moist dressing. Each one, at any rate, will apply it to his own system, and not to another, the former being in his own sight orthodox and all others heterodox. Is this confusion of meanings right, or even necessary? Is it not confusing to the seniors of the profession? And if so to them, how much more confusing it must be to our pupils. Why should we not, therefore, when we mean it, say the "aseptic" or "antiseptic spray and gauze system," or "antiseptic irrigation," or "antiseptic dry or other dressing?" Such terms would be perfectly intelligible to all, and would leave the special antiseptic drug employed as detail to be introduced in the description or not, as wished, and at the same time allow the term "antiseptic precautions" to lapse into a general expression as denoting the well-established and recognised principles of antiseptic surgery. At the present day, the phrase "antiseptic precautions," as applied to any single case, is absolutely unintelligible as indicative of any special form of practice.

Again, have we not in past times too generally mixed together cases of intestinal strangulation with those of intestinal obstruction?—having been led to do so by the fact that bowel obstruction is a common symptom of both classes of cases. And have we not, by so doing, obscured and rendered difficult of understanding cases concerning which it is very

requisite that we should entertain clear views? In past, pre-pathological ages this confusion of terms was possibly excusable, but with our present exact pathological and clinical knowledge are we not obliged to admit that strangulation of the bowel is one thing and obstruction another?—that in the former class of cases the symptoms are all due to the strangulation of the intestine, and but little, if at all to the obstruction? —whereas, in cases of the latter class, all the symptoms are in a general sense due to the obstruction and to the changes brought about by it!—these changes being experienced either at the seat of obstruction, or, when the obstruction is in the rectum or sigmoid flexure, found in the cæcum or ascending colon. Are we not therefore impelled, for the sake of a clear understanding of these two large subjects, to separate the cases and give to each its own proper place? Shall we not by so doing, gain clearer thoughts upon each, and thus be able, as teachers, to impart them to others?

Again, is it not most important that we should have very clear views on such general and elementary subjects as repair and inflammation? And yet, may I ask, are our thoughts upon the relations of these two pregnant processes sufficiently clear? Do we, or do we not, in our teaching, mix them up inextricably, and, by so doing, encourage, if not impart, erroneous views? Experience leads me, as an examiner of students, to believe that this confusion of thought is too general, and that "the healing of wounds is still supposed by some to be essentially an inflammatory process," and that even an anchylosis of a joint, the result of disorganising inflammatory changes, is "a formative termination of the inflammatory process itself." As if, indeed, repair and inflammation, from their both presenting in their respective courses certain histological changes which are allied, are on that account to be deemed identical; and as if the union of the articular ends of the bones following a disorganisation of a joint, the result of an acute or chronic inflammation, can be brought about by an inflammation, or by any other process than a reparative one, which does not begin until all inflammation has ceased, which is continued only so long as the inflammatory process is kept in abeyance, and which ends in the desirable result of anchylosis because the reparative process is allowed to go on undisturbed, without either the aid or hindrance of inflammatory action.

In the treatment of a wound is it not the surgeon's chief object to prevent inflammation, and is not this object, based upon the knowledge that when a wound is undergoing quick

repair by primary union, and becomes the seat of inflammation, the repair at once stops, and what may have taken place in the way of repair becomes disrepair—the exposed surface of the wound, if the inflammatory process continues, becoming the seat either of ulceration or of other destructive changes? When ulceration follows a local inflammation it continues so long as the inflammatory process lasts; when this stops repair begins by what is called granulation, and this formative reparative process continues and ends in cicatrisation, so long as no inflammation reappears to interfere with its progress.

When a granulating wound becomes the seat of inflammation the reparative process at once ceases, and what had been a granulating soon becomes an ulcerating surface. With these clinical facts before us, which are familiar enough to practical surgeons, there should be no difficulty in demonstrating that repair and inflammation are not only not identical, but that whilst the one is wholly formative the other is mostly, if not always, destructive; the one is physiological, the other pathological. Is it not therefore absolutely necessary that the teachers of students should have clear views upon these points, and not by such terms as "the formative termination of the inflammatory process," and other allied mixed expressions, put into students' minds a cloud of words to cover their own uncertain views? Is it not incumbent upon all teachers to enunciate that repair and inflammation are not only not identical but that they are incompatible?—that repair only begins in a tissue that has been inflamed when the process called inflammation has left it, and continues to complete its work as long as the inflammatory action is kept away?—that when inflammation attacks a wound in which repair is progressing the process is at once arrested; and that what was repair then becomes disrepair, if not ulceration? I think I can hear you—the members of this Society—say "yes" to my questions, and may calculate upon your working in the lines I have laid before you.

With these suggestions, which I trust you will not consider out of place, as they are truly clinical, allow me for a few minutes to call your attention to some practical points which require looking into, since it seems that in our general advance in medicine and surgery we sometimes in special subjects recede to the practice of our ancestors. This may be said to be true in the application of ligatures to arteries in their continuity; for some of our surgical brethren now prefer to tie in two places, and divide between the ligatures, an artery

that has to be occluded for any cause, thus following the practice of last century rather than that of more recent times. It is not my intention here to criticise this practice, which I look upon with favour, but it would be interesting if we could obtain during the present session full particulars of the results of this revised method, and thus be able to estimate from a practical point of view the relative advantages of the different methods employed.

Again, may I ask, is there any truth in the accusation, which in recent times has been raised against surgeons, that the great successes which they have all round achieved in surgical operations have to a degree encouraged them not only to do, and to do rightly, what they would never have thought of doing a few years ago, but also to undertake operative measures which may with some justice be looked upon as speculative, if not rash? Have our successes engendered an over-estimation of our own powers, and led us to attempt and perform operations which past experience has not supported, and which seem to be less founded on scientific probabilities of success than on the sanguine hopes of their performers? Are operations upon the pylorus, or stomach, for cancer to be placed in this last category? Are there other operations which should be so classed? I have no wish to answer definitely these questions, but I do feel that it is very necessary that the principles which have hitherto regulated operative surgery, and which have tended to suppress all experimental work, unless based on a scientific probability of success, should be carefully observed, and that we should avoid even the semblance of an experimental operation.

And here let me express for surgeons generally the satisfaction with which in our best new surgical work we greet the kind aid we are receiving from physicians. We are now working, more than we have ever before worked, hand in hand with them to make the diagnosis of disease of the brain, kidney, bladder, and abdomen more certain. And we are thus, with a clearer diagnosis, mutually helping to bring within the domain of scientific surgery large classes of disease which have hitherto been deemed to lie outside its pale, and have consequently been either allowed to drift or to pass into the surgeon's hands only when the time for effective action has passed or almost passed, and when operative measures can at the best be carried out for purposes of relief, but not of cure.

May I now ask for even more help in this direction, and urge our medical friends to seek surgical conference early, at

least in all abdominal cases in which symptoms of intestinal strangulation exist, as well as in all cases in which intestinal obstruction is present, in order that operative interference, in both classes of cases, may not be delayed longer than the scientific diagnosis of the case requires ; and that the subjects of these troubles may have a chance of relief from operative measures whilst there is still a reasonable hope of obtaining it. Let us remember that exploratory operations undertaken for diagnostic purposes, but which may be used for curative ends, when the exploratory proceeding shall have cleared up or established a diagnosis (which could not have been made by other means), are as scientific as any other operations, and often more satisfactory.

Let us therefore encourage our medical brethren to consider closely with us surgical problems, in order that we may have their efficient help in diagnostic questions as well as their valuable support when action, by way of operation, is called for; and let us employ the opportunity to convince them of the expediency of expediting action as soon as the necessity for action has arrived, and at the same time to demonstrate the evil effects of postponing operative interference when such is demanded for either diagnostic or curative ends. In surgery, as in so many conditions of life, action, to be effective, must be decisive and not dilatory; it should ever follow closely upon decision.

These remarks which I have thus brought before you I have been tempted to make under an impression that they will be generously received, and under the conviction that they have reference to subjects of grave clinical importance. Some of the subjects to which I have alluded are important in themselves; others are important on account of the principles embodied in them.

Should my observations appear to some too critical, let me say that they have not been made in any captious spirit, but with the feeling that it is always better for us to criticise our own work than to leave such criticism to others ; that as your President, it is as much my duty to point out what I believe to be defects in our work or in our mode of work, as it is to indicate the direction in which we should travel; and in the hope that, by so doing, I am likely to receive the full support of the members of this Society in what I believe to be the best for the "cultivation and promotion of practical medicine and surgery," objects for which this Society was formed, and which we all have so much at heart.

COMMUNICATIONS.

I.—*On Certain Nerve Symptoms in Rheumatic Affections.* By W. B. Hadden, M.D. *Read October* 10, 1884.

I WISH to bring before the Society a few cases illustrating certain nerve symptoms which are apt to supervene in the course of acute and chronic articular affections. One of these conditions, the wasting of muscles in relation to diseased joints, has been pointed out by several observers, some of whom have ascribed it to simple disuse of the part, whilst others have argued in favour of its central origin.

In the following cases it will be seen that not only muscular atrophy, but anæsthesia, rigidity, and cutaneous trophic lesions sometimes make their appearance.

Case 1.—The first case I shall mention is one of glossy skin, following on an acute joint affection.

The patient, who was a woman æt. 35, was admitted into St. Thomas's Hospital under Dr. Bristowe. A year before admission she had been laid up for six months with an attack of acute rheumatism, which affected the knees, left ankle, and hands, especially the right. She was jaundiced for six weeks after the attack. A month before admission she had rheumatic pains in the knees, back, and hands, and for the last fortnight had suffered from sickness, pain between the shoulders, and dyspnœa.

State on admission.—She complains of rheumatic pains in both shoulders and arms, but the joints are not swollen or tender. All the fingers of the right hand are extended. The fore and middle fingers are wasted, and sensation is a little impaired. She can only flex them very slightly. The ring and little fingers are more readily bent. She cannot separate the fingers of the right hand so well as those of the left.

The fingers and thumb of the right hand, and more especially the two distal joints of each finger, and the distal joint of the thumb, are wasted, smooth, and shiny. The nails are much longer and more filbert-shaped than those of the other hand.

The muscles of the upper forearm, especially on the outer side, are more full and plump on the left than on the right side. The antero-posterior thickness above the wrist seems also greater on the left side. The thenar and hypothenar eminences are also less full on the right side. All the muscles of the arm, forearm, and hand may be made to act by using twenty-five cells; the muscles on the ulnar side respond more rapidly and vigorously than those on the radial side.

The continuous current was used daily with great benefit. A week after admission she could bend the fingers of the right hand fairly well, and had a pretty good grasp. The skin on the back of the hand was much less tense, and the wrinkles reappeared over the joints. She was discharged in about five weeks, nearly cured.

In this case we find the co-existence of a well-marked cutaneous lesion, *i. e.* glossy skin, with slight anæsthesia, enfeeblement of muscular power, and muscular wasting.

In connection with this case I will mention briefly another instance of what appears to me to be a trophic lesion of the skin.

Case 2.—A young married woman, æt. 26, came to me as an out-patient. For a week or so before, her finger-joints and knees had become swollen and painful, particularly at night. The right fingers were most affected. She complained chiefly, however, of two small rounded superficial sores, one on the extensor surface of the second joint of the right little finger, the other on the extensor surface of the left middle finger, near the base of the nail. They appeared as little red specks about the time the joints became affected. They were very tender, and itched a good deal. On examination I did not find any marked tenderness or swelling of joints.

The patellar tendon reflex was normal. The catamenia, which had been previously regular, every four weeks, had for the last six months appeared at intervals of seven or eight weeks. They were preceded by leucorrhœa, but there was no dysmenorrhœa. I mention this fact because it seems to support the idea, put forward by Dr. Ord, that there is a relation between menstrual disorders and articular affections. I prescribed Liq. Arsenicalis ♏ iij t. d. s. In ten days the ulcers

were quite healed and the joints were only a little painful at night. She subsequently recovered perfectly.

I must add that I sent the patient to Dr. Ord, who agreed with me that the sores were probably trophic.

The next case is one which I venture to give somewhat in detail, because the patient exhibited quite a series of nerve disorders, including muscular atrophy, anæsthesia, rigidity of limbs, and increased tendon reflexes.

Case 3.—A man, æt. 57, came under my care as an out-patient, complaining of severe articular pains. In the course of examination I discovered the various nerve symptoms above mentioned, so I sent him to Dr. Ord, who kindly took him into his wards at St. Thomas's Hospital, and investigated his condition with great care.

The only point to note in his family history is that a sister suffered severely from gout.

As regards his personal history it must be mentioned that he had never had acute rheumatism or anything like an attack of gout. The man had been a soldier, and had passed fourteen years in foreign service. He ascribed his rheumatic affection to the exposure he underwent in the Crimea.

According to his account, he was perfectly well until nine months ago, when he was seized one day with a fit of giddiness and fell down. He did not lose consciousness, and got up again immediately without help, feeling perfectly well and afterwards doing a hard day's work. Soon afterwards he felt he was losing power in his left side. Six months before admission he began to lose power in his right arm and leg.

I do not think that the attacks he described were of a hemiplegic nature. The loss of power he mentioned seemed rather to depend on his joint affection.

For many years he has suffered from cramps in the legs, and seven months ago became an out-patient at the Middlesex Hospital. At that time he was passing a large quantity of urine, and his legs were much swollen. Possibly this attack was renal, but during his stay in St. Thomas's Hospital no indications of kidney mischief were discovered.

For the eight months preceding admission he had been subject to sudden pains in the hips, knees, shoulders, elbows, and wrists, accompanied by sudden flexion of these joints.

State on admission.—On examination the patient was found to be a spare man with a fixed expressionless face, holding himself stiffly, and with a marked forward inclina-

tion. His appearance indeed, when first I saw him, suggested paralysis agitans. He complained of articular pains.

The skin over both hands is shiny and smooth, especially over the phalangeal joints, where the usual wrinkles of the skin are wanting.

The metacarpo-phalangeal joints of the first two fingers of right hand are very large, the enlargement affecting more especially the head of the metacarpal bones. The corresponding joints of the ring and little fingers are not involved. The bases of the first phalanges of the first and second fingers are partially dislocated into the palm, and there is a similar partial dislocation of the second phalanges of the first three fingers, so that each of the first three fingers appears concave on the dorsum and convex on the palm. The ungual phalanx of the little finger is dislocated slightly in the direction of the ring finger. There is a distinct collar of bone at the terminal joints of all the four fingers. There is slight grating in the joint between the first and second phalanges of the index finger, but not elsewhere.

The condition of the fingers of left hand is very similar to that on the opposite side. The left thumb, however, is more affected than the right, having a distinct collar of bone at the end of the first phalanx.

The fingers generally are fixed in an extended position and have a nodulated appearance, due to the thickening of the ends of the bones. The interossei are much wasted. In both hands there is power of flexion and extension at the metacarpo-phalangeal articulations, but the patient cannot move any of the other joints. The right thumb, however, can be flexed and extended at both joints.

There is crackling of the right shoulder-joint, none of the left. There is some thickening of the tissues about the left knee-joint. The hips and right knee seem unaffected, except that movement is difficult on account of pain.

The toes are not involved. The prick of a pin is not felt over the back of the left hand, except over the terminal phalanges of the second and ring fingers. There is also loss of sensation over the ball of the thumb, the radial part of forearm behind, the whole anterior surface of forearm and all the upper arm, except near the elbow-joint. On the right side sensation is much impaired over the front and back of thumb, absolutely lost over the radial half of the front of forearm, and all around the shoulder-joint. Sensation is a little impaired about the left temporal region. There is no loss of feeling on

the trunk. The prick of a pin is not felt over the front and back of the left leg and dorsum of left foot, but elsewhere in the lower limbs there is no loss of sensation.

The muscles on the front and back of both legs can be seen vibrating through the skin.

In addition to the atrophy of the interossei mentioned above, there is evident wasting of the muscles on the backs of the forearms. All the muscles on the right side respond well to the interrupted current except the palmar interossei, which do not act quite readily. On the left side the extensors do not act so freely as on the right side. The extensor communis digitorum does not respond at all.

All the cutaneous reflexes are readily obtained, the plantar being especially brisk, particularly on the right side. The abdominal reflexes, on the other hand, are more marked on the left side. The tendon reflexes in the upper and lower limbs are exaggerated, and there is ankle clonus on both sides, but this is sometimes difficult to elicit on account of the rigidity.

The mouth seems a little drawn to the left, but there is no other sign of facial paralysis. The tongue is protruded straight and the ocular muscles are natural.

There is a myopic crescent in the right eye and some impairment of hearing on the left side. The special senses are otherwise natural. The gait of the patient is decidedly spastic, the knees being flexed little during progression.

There are two tender spots on the spine, one over the third dorsal vertebra, the other over the second lumbar.

The thoracic and abdominal organs appear healthy. The temperature was generally subnormal, the lowest being 96·4°. Usually it was between 97° and 98°.

What is the explanation of the various nerve disorders in this case? Is there an extension of the inflammation from the joints to the nerves? This assumption might account for the anæsthesia and muscular atrophy of the upper limbs, the small joints of which were profoundly affected. But the anæsthesia of the left leg and the rigidity of the lower extremities cannot be so explained. The knees and hip-joints were very slightly affected, and the small joints of the feet and toes not at all.

It seems to me probable that in this case there was a rheumatic neuritis as well as a rheumatic arthritis, and that the nerve disorders were neither concerned with the causation of the joint affection, nor dependent on it.

In the first two cases which I narrated, the trophic disorders occurred during the subsidence of the articular rheumatism, and might really be looked upon as a relapse affecting the nerves. Rather more than three years ago a female patient who had just recovered from an attack of acute rheumatism, chiefly involving the finger-joints, complained to me of numbness. On examination I found absolute anæsthesia strictly limited to the course of the ulnar nerve. This condition lasted only a day or two.

So far as I know, anæsthesia has not been previously noticed in connection with rheumatism, although its occurrence in chorea is far from uncommon.

The muscular atrophy in Case 1 was undoubtedly of nerve origin. In Case 3 the wasting seemed to me greater than could be accounted for by simple disuse, and, moreover, the extensors were much more affected than the flexors. This seems to be a law in all joint affections, whether rheumatic or not. We see examples in the wasting of the deltoid in shoulder-joint disease and of the gluteal muscles in hip disease. It has been urged that this depends on some selective action exerted by the spinal cord. But in lead palsy and alcoholic paralysis the lesion is essentially one of the peripheral nerves, and in these affections the paralysis of the extensors is the most prominent feature. In Case 3 the wasted extensors, with one exception, responded to the interrupted current, though less actively than normally. This is exactly the experience of Professor Charcot. The change in the electrical reactions is quantitative. It is interesting to note, however, that the left extensor communis digitorum had the reaction of degeneration —a qualitative alteration.

I have seen rigidity of the lower limbs and ankle clonus twice before in chronic articular rheumatism. One case was under my own care and the other was shown me by Professor Charcot. Such a condition is probably due to structural change in the spinal cord. Nevertheless, I have occasionally found ankle clonus in cases of pressure upon the lumbar plexus by new growth, and in one such instance there was no lesion of the spinal cord when examined microscopically. It is quite possible, therefore, that the rigidity and ankle clonus occasionally observed in these cases may depend on lesion of the peripheral nerves.

The occurrence of these nerve disorders in rheumatic affections cannot be referred either to the duration or intensity of the joint affection. From time to time I have examined in

our infirmaries cases of chronic rheumatoid arthritis of ten, fifteen, and twenty years' standing, but have found nothing which could be looked upon as a trophic disorder.

The cases which I have given neither support nor disprove the theory of the neurotic origin of articular rheumatism, so ably advocated recently by Dr. Ord and Dr. Dyce Duckworth. They merely show, I think, that certain symptoms, referable to the nerves and possibly in part to the spinal cord, occur in rheumatic subjects.

I beg to express my thanks to Dr. Ord, who not only first directed my attention to this subject, but who has also assisted me in obtaining the material which has illustrated the paper.

ERRATUM.

The stone referred to as Plate IX, fig. 3, on page 9, is the largest of those marked fig. 2 in Plate IX.

October 10, 1884.

THE specimens which I have the pleasure of exhibiting were given me by my friend, Mr. B. Corney, who is an emigration medical officer in Fiji. He met with these instances of preputial calculi in the course of his duties as an inspector of coolies, as his following notes will show.

"Case 1.—In September, 1881, R., æt. about 17, a native of the Rubiana group, in the Solomon Islands, came before me at Sura (Fiji) for physical examination before being allotted to his employer for field labour.

On taking hold of his penis I felt a curious grating sensation between my finger and thumb as of a bag of pebbles; I also heard the sound produced by their grating. He had phymosis of congenital origin, but of course no history of the pebbles could be extracted from R., there being no one present who understood his language. I thought at first that he had introduced them himself, and ordered him into hospital that I might remove them. This I did one by one with a very fine pair of bullet forceps, after which I decided to circumcise the boy to prevent a recurrence of the annoyance. It is easy to understand how the retention of some of the smegma preputii may afford nuclei for the beginning of these calculi when the degree of phymosis is so great that some urine gets left in the preputial folds each time of micturition. There were twenty-two stones in all. (Plate IX, fig. 2.)

Case 2.—B., æt. about 18, a native of Guadalcanar, one of the Solomon Islands, was brought to Sura (Fiji) in September, 1882, under an indenture to work for three years on a sugar estate. He came before me at the immigration depot for physical examination, prior to being allotted by the Government to his employer. My eye was attracted by what appeared to be an enormous glans penis. On taking hold of it, however, it felt too hard for that, and on closer examination I found an excessive degree of phymosis without inflammation. I was able with just a little difficulty to introduce the bulbous end

of an ordinary probe within the prepuce. Upon arriving there it struck a hard stony body, and having previously met with a similar case I readily perceived that this was a case of sub-preputial calculus.

The orifice of the prepuce was of so much less calibre than the urethra and meatus urinarius that during micturition the prepuce became distended like a bladder, and a small stream of urine spurted out from it in an odd jerky manner like water under pressure from a leak in an india-rubber tube.

This effect was the cause of much mirth amongst B.'s compatriots. I ordered him into hospital, and next morning slit up the prepuce on a director, when the stone rolled out. The director stretched the orifice of the prepuce to its utmost. The stone when removed, being wet, weighed 1 oz. 110 gr. (Plate IX, fig. 3.) The edges healed in a fortnight.

Nearly all the natives of the Solomon Islands have congenital phymosis. About 1½ per cent. of them have the orifice no larger than a pin's head. Only a few tribes remedy this condition by art. Instead of circumcising they slit it up generally with a sharp shell.

In the New Hebrides the foreskins are not usually so long, though phymosis is exceedingly common.

In the island of Malakula all the natives, except one tribe at the north-east corner, slit up the prepuce, and very neatly and effectually. This, too, is the only island where compression of the skull is practised (during infancy, as with some aborigines of North America), and the north-east tribe which does not slit the prepuce does not compress the skull.

As a result of this congenital phymosis, balanitis, which I believe to be of a non-specific nature, and to originate merely from want of cleanliness and consequent irritation, is very common amongst these simple people, whose prejudices and customs restrain them from universally adopting so simple and safe a precaution as that which is necessary.

The Fijians, who are a much superior race, all circumcise or slit."

Dr. Bernays, of St. Thomas's Hospital, was so kind as to examine these calculi, and he reported that they consisted of the triple phosphate and traces of urates.

In South's 'Chelius,' under heading of "Urinary Stones external to the Urinary Passage," p. 632, vol. ii, we find :—"In the College (Royal College of Surgeons) collection there is also a

very curious case of Vincent's (of St. Bartholomew's), numerous small calculi which, with about 200 others, were removed from between the prepuce and glans penis of a very old man. The patient had congenital phymosis, the orifice of the urethra scarcely admitting the introduction of a common probe. From the pressure of the calculi the prepuce was distended to the size of a large pullet's egg, and retention of urine was finally produced. On dividing the prepuce one of the calculi was found completely blocking up the orifice of the urethra. The glans penis was in a state of ulceration, and a large portion of its substance had been absorbed. The patient had during many years occasionally experienced great pain and difficulty in making water, and latterly he had a constant stillicidium.

The calculi are composed principally of the fusible compound. Most of them have a small nucleus of uric acid; their external surface is varnished over with urate of ammonia. From the composition of the nucleus there can be no doubt but that the greater number of these calculi had passed from the urethra into the sac of the prepuce, and their irregular form and close adaptation to each other proves that in this situation they had increased considerably in size by the deposition of the earthy phosphates."

These calculi are of some interest beyond their extreme rarity in this country. They show how little the form of the bladder is concerned in modelling the concretions. Conditions similar to those which determine concretions in the bladder determine concretions in the phymotic prepuce. This condition is a sac which possesses a relatively small inlet and still smaller outlet.

Although it does not appear to be a matter of any importance to decide whether the nuclei of these concretions were formed in the bladder or within the phymotic prepuce, this is a subject of interest as it bears upon the mode and seat of commencement of urinary calculi generally. It appears to be most probable that the nuclei of these concretions, large and small, were ejected from the bladder and urethra under the influence of the compensatory strengthening of their walls, but failed to escape through the minute orifice of the prepuce, and, having become lodged there, increased in bulk by molecular coalescence in the ordinary manner of vesical calculi. All the calculi of which sections were made present central cavities lined by crystals, but examination of these fails to discover any obvious nuclei of organic matter.

I think I am justified in pointing to the facts mentioned in Mr. Corney's notes and remarks as emphasising the propriety of early operations for congenital phymosis. On this account, and because of the clinical and ethnological interest, and on account of the great rarity of such calculi, I have ventured to place these specimens before the Society.

III.—*A Case of Urticaria Pigmentosa, or Xanthelasmoidea.* By H. Radcliffe Crocker, M.D. *Read October* 10, 1884.

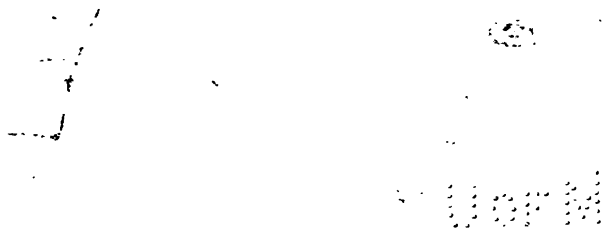
LAVINIA N., æt. 4½ months, came to University College Hospital on September 22, 1883, with the following history:

A day or two after birth, a blister was noticed in one groin, and two or three days later some came in the axilla and on the neck. The mother saw no more for two or three weeks, when the present eruption began, first on the neck, and then all over the body.

It commences as tubercles, singly, or more often in groups of three or four, about the size of a small split pea, distinctly raised above the level of the skin, of a yellowish-red colour with a narrow pink areola (Plate I); on some of the tubercles a vesicle about a quarter of an inch or more in diameter forms with clear contents which get absorbed in a few days, the tubercle still remaining, but covered with a thin crust of dried epidermis. Many of the tubercles increase in size subsequently, perhaps by coalescence where there was a group, and as they get older become of a distinctly yellowish colour, and thus there are lesions varying in size from a hempseed to a good-sized bean, and in colour from a brownish red to a pale fawn, but most of them are of the yellow shade. They are somewhat firmer than the normal skin, and at first did not itch, but latterly their evolution has been attended with some irritation, but there is none after they are fully formed; the eruption is still coming out at intervals of a few days, but there are never any transitory wheals of the ordinary kind.

The tubercles are all over the head and face except the vertex, very thick on the neck, not so numerous on the arms, but are on both backs and palms of the hands. There were not many about the genitals, and the anus and its immediate neighbourhood were quite free for some time, but now all these parts are pretty thickly covered, as well as the loins and backs of the thighs. There are a few on the soles, but they are less abundant, on the whole, below the knee.

The child was a fine baby at birth, but has lost flesh to some extent, but is still fairly nourished for a London baby,



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Dr Crocker's Case of Urticaria Pigmentosa

but rather pale. She is suckled entirely, and seems well on the whole, and the eruption does not trouble her in any way except at the period of evolution. There is no evidence, either in herself or in the other children, of congenital syphilis. Three of the children had rickets, but are now well, and the father and mother are apparently healthy. In deference to the opinion of others, who thought the eruption must be syphilitic, hydrarg. cum creta gr. j three times a day, alternating with mercurial inunctions, was given for two months, but without any effect upon the eruption. The child has now been under observation for six months, during which period fresh lesions have appeared at irregular intervals of a few days, and others have undergone partial involution, getting wrinkled and less prominent, but very few have completely disappeared, leaving only pigmented patches on their former site. Rubbing does not appear to produce exacerbations of the older wheal-like tubercles, but on the other hand fresh vesicles have sometimes formed on the old tubercles. Factitious urticaria could never be produced.

This is the twentieth published case of this rare affection, which was first described by Mr. Nettleship in the *British Medical Journal* for September 8, 1869, but which did not attract attention until Mr. Morrant Baker and Dr Tilbury Fox showed cases to this Society, which are published in vol. viii of the *Transactions*. From these twenty cases the following general account may be given.

In nineteen cases the sex is mentioned, and there were fourteen boys to five girls. It begins in the first six months of life, the earliest age being three days. This very early commencement points to a congenital defect as the primary cause of the affection. The first lesion is usually a wheal-like tubercle of rapid evolution but great persistence, often coming out in the night, about the size of a pea, yellowish white at the apex, with a red areola; less frequently vesicles have appeared, though probably, as in my case, they were preceded by erythematous, if not urticarial, elevations. In many cases where the mothers speak of blisters, no doubt wheals are intended, as that is a common expression for them.

No part of the body is exempt from the eruption, though the lesions are most abundant on the trunk and neck, next on the limbs, and only occasionally on the palms and soles. The palatal and buccal mucous membranes were affected in two cases. Itching is generally present during the evolution of the tubercles, but it may be slight or quite absent; when this

symptom is prominent, ordinary urticarial wheals usually make their appearance from time to time, and factitious urticaria is often easily producible.

The disease is little if at all influenced by treatment, but tends to get well of itself. After some years fresh lesions cease to appear, and the old tubercles are gradually absorbed by the time puberty is reached. This is only an approach to the truth, for in Lewinski's case fresh lesions were still making their appearance at eighteen years of age.

Microscopical examination of the tubercles has been made by Dr. Thin from a case of Mr. Morrant Baker's, by Pick, of Prague, and by Dr. Colcott Fox from Dr. Tilbury Fox's most severe case. Thin came to the conclusion that the structure was indistinguishable from that of lupus. Pick found hæmorrhages surrounded by small-celled infiltration, while Colcott Fox described the structure as that of a typical wheal plus some cell infiltration and small hæmorrhages. These observations are probably less conflicting than they appear at first, and seem to me to represent a lesion affecting the upper part of the corium, the result of hyperæmia, œdema, small hæmorrhages and a variable amount of cell infiltration, the last very great in Dr. Thin's observation, slight in the other two. This might well represent a wheal made permanent by cell and blood exudation. That the disease is in any way related to lupus no one who has observed its clinical aspect and course could believe.

With regard to the urticarial nature of it, were they all like Dr. Tilbury Fox's first case and my own, the difficulty of regarding the disease as a modified urticaria would be considerable, as, except in the shape of the tubercle and the sudden evolution of the initial lesion, they have no other feature in common. With no itching, persistent lesions, bullæ, and great pigmentation, no wonder Tilbury Fox could not recognise urticaria in this condition, especially as many of the intermediate links had not then been demonstrated. Our difficulties diminish, however, when we consider the gradations produced by other cases and also certain occasional features of ordinary urticaria. Thus there are cases of recognised urticaria where bullæ are a pronounced feature; pigmentation sometimes follows ordinary wheals, as in a case of Dr. Liveing's shown at the Congress in 1881; persistence of the wheals occurs in urticaria perstans; cell exudation is present in the papules following the wheals in the urticaria of children, and hæmorrhage into wheals is not unknown. The peculiarity

of this disease is that all these features, which individually are rather rare in the course of ordinary urticaria, are combined in these cases of urticaria pigmentosa, a name suggested by Dr. Sangster, which, though not altogether satisfactory, has met with general acceptance, displacing the uncouth xanthelasmoidea of Dr. Tilbury Fox, which was only applicable to the most aberrant cases, such as his first and worst, and perhaps to some extent to the one of which the drawing is shown to-night.

I have not discussed some of the points so fully as I might have done, as Dr. Colcott Fox has recently given an elaborate *résumé* of the subject in the last volume of the *Medico-Chirurgical Transactions*, with an abstract of all the cases published up to that time, and I should therefore only have traversed recently trodden ground.

Since this paper was sent in, last session, the child has unfortunately died of whooping-cough and bronchitis on May 6, but I did not hear of the death until some days afterwards. I saw the child a few weeks before its end and the skin had improved considerably; some of the tubercles had undergone partial involution so that the skin over them was wrinkled, while others had completely flattened down, leaving only purplish brown stains.

I am not aware that any other cases have been published recently, but Dr. Wallace Beatty, of Dublin, has brought forward two cases, an abstract of which is given in the *British Medical Journal* of April 26, which he thought deserved the name of urticaria pigmentosa, but the cases were so unlike in many important respects that unless connecting links are discovered it would not be justifiable to include them in the same category as the cases that we have just been considering.

IV.—*A Case of Perforation of the Vermiform Appendix with Peritoneal Abscesses. Death after a long interval from Pyæmia.* By David W. Finlay, M.D. *Read October* 10, 1884.

JOHN L., æt. 24, a baker, came under my care in the Middlesex Hospital on January 4, 1884.

On admission he was described as a spare, dark-complexioned man of muscular development, complaining of severe pain over the whole of the abdomen. His pulse was 96, compressible; temperature 99·6°; respirations 24, and entirely thoracic. The tongue was dry and coated with a brownish strip down the centre; the cheeks were flushed, and the lips dry and cracked The abdomen was moderately distended, everywhere tender, and apparently tympanitic, palpation or percussion, however, causing him so much pain that they had to be sparingly employed. The skin of the abdomen was very red from the previous application of mustard poultices, and the tenderness was no doubt partly due to this cause. The areas of liver and splenic dulness were normal.

In the chest the percussion resonance over both fronts was fair and equal. The breath-sounds were also fair, although accompanied sometimes by a faint sibilant sound. Over the backs the resonance was not very good, but breath- and voice-sounds were nearly normal.

The heart's maximum impulse was seen and felt in the fifth interspace a little outside of the nipple line, and its sounds were normal.

The urine was turbid with lithates, very acid, of a specific gravity of 1034, and free from albumen or sugar.

The patient stated that his illness had commenced suddenly a week before admission, namely, on the evening of Saturday, December 29, 1883, with pain of a griping character across the lower part of the belly. This pain continued all night, and on Sunday morning he began to vomit, and continued to do so at intervals of one and a half or two hours all day. The vomiting had continued more or less every day up to the date of admission. He had also suffered from headache at first, and had had some diarrhœa on the Tuesday, Wednesday, and Thursday.

He had had a similar attack three years previously which consisted of abdominal pain with sickness and feverishness. With this exception his health had been generally good. There was nothing suggestive in his family history.

As regards treatment, he was ordered a subcutaneous injection of one fourth of a grain of morphia at once, to be followed by half a grain of extract of opium in pill every four hours, a lead and opium lotion to the abdomen, and a diet consisting of milk and beef tea.

Next morning (January 5) he was sick and vomited, bringing up about two ounces of clear yellowish fluid. There was still marked abdominal distension and tympanitic resonance, but less tenderness; no spots were observed. He said that he felt much better although he had slept badly. His morning temperature was 99·8°, that of the evening 100°.

On the following day (January 6) his tongue was cleaner, and he was rather better. The morning temperature was 100·2°, evening temperature 100°.

On January 7 the note states that he expressed himself as feeling better. During the night, however, he had vomited five or six times and had been much troubled with hiccough. The tongue was very dry and brownish in the centre but not much coated; the bowels not open; respirations still thoracic; pulse 92. He complained of no pain, and the general abdominal tenderness had disappeared, but there was a spot midway between the costal margin and iliac crest in the left flank where tenderness remained, and where the percussion note was dull. The evening temperature was 100°.

On the evening of the 8th he was suddenly attacked by pain in both parotid regions; and during the night his bowels were opened three times, the motions being loose, and light in colour.

On the morning of the 9th the tongue was cleaner and less dry. There was marked swelling and tenderness of both parotids with inability to open the mouth to any extent. During the day the bowels acted four times. The following night he was restless, and slept badly.

Next day (January 10) the parotid swelling had increased, and he was found to be sweating profusely. His pains were relieved by poppy-head fomentations. The urine was acid, sp. gr. 1032, free from albumen.

His condition did not vary for a couple of days, but on January 14 a sense of fluctuation was detected in the parotid swelling on the left side accompanied by a slight discharge

from the ear, and at my request Mr. Lawson saw him and made an opening into it, evacuating about two drachms of pus.

During the following night he was restless and delirious. There was a profuse discharge of pus from the opening in the parotid to which poultices had been applied. Eggs were now added to his diet, and he was ordered ammonia and bark with brandy.

On the morning of January 16 his temp. was 101·2°. Pulse 128, weak, but regular, resp. 44. Tongue dry and glazed. He had slept fairly well and had no pain. He looked apathetic and was somewhat emaciated. A purpuric eruption of small reddish-purple spots, not completely fading on pressure, was noticed over a space about the size of the palm of the hand on the surface of the abdomen above the umbilicus, and another patch over the præcordia. A smaller area in the right mammary region was also occupied by a similar crop; there was none elsewhere.

No adventitious sounds were heard over the chest, but the breath and voice sounds were feeble.

The swelling in both parotid regions had diminished, but there was some discharge from the right ear, and accordingly about 3 P.M. Mr. Lawson incised the right parotid swelling, but no pus appeared. At half-past 5 o'clock I saw him again, and found that he had been sweating profusely for the last half hour; and now the pulse was feeble and irregular both in force and rhythm. The temperature was 103°, and he was delirious and trying to tear off his bandage. At 6.30 his temperature rose to 104·8°, and in two hours he was dead.

At the post-mortem examination, which was made by Dr. Fowler eighteen hours after death, the great omentum was found spread out upon the surface of the intestines, and firmly adherent to the parietal peritoneum in each iliac region. In the adhesions here formed there were abscess cavities, each about the size of a walnut, containing creamy yellow pus. Their position was about the internal inguinal ring.

A very large sac was found in the right lumbar region which contained about a pint of brownish pus. It was bounded above by the under surface of the liver and the gall-bladder, below by the cæcum, in front by some coils of small intestine; on its outer side by ascending colon and small intestine, and behind by the abdominal wall covered by peritoneum. The cavity was lined by a thick pyogenic membrane which was pigmented and of a yellowish-brown colour.

On the left side of the abdomen another similar but smaller cavity appeared enclosed by firm peritoneal and omental adhesions. This was bounded above by the spleen, behind by the kidney and descending colon, and elsewhere by coils of small intestine. It contained about eighteen ounces of pus; its lining membrane was rough and deeply pigmented.

In the pelvis there was a third abscess cavity, formed by the pelvic walls, roofed in by peritoneal adhesions, full of brownish coloured pus.

There was also a small sac, containing thick yellow pus, in the mesentery of a coil of bowel lying in the right iliac region.

Over the whole peritoneal surface there were old fibrous bands and greasy-looking flakes uniting the coils of intestine together, and also some recent injection, but no recent lymph.

On examining the intestines after removal, the vermiform appendix was seen to be ulcerated through about halfway from its attachment, the opening communicating with the sac of the pelvic abscess. A small mass of fæcal matter plugged the interior of the appendix on the cæcal side of the perforation.

The liver, kidneys, and heart were normal, the spleen congested, the lungs œdematous. The left pleural sac contained a quantity of clear serous fluid, the right was obliterated by adhesions.

Since these notes were written I have ascertained that the man was employed at a large hotel, and had been there for a year and eight months, during which he had shown no sign of illness until the last, and had always been fit for his work. I have also learned from his previous medical attendant that he formerly suffered from attacks of obstinate constipation, and that on the occasion referred to by the man himself, three years before I saw him, his temperature rose to over 103°: also that he always appeared to recover perfectly from his attacks.

The case seems to me interesting as suggesting how long serious disease may remain latent without showing any immediate tendency towards death. I would explain it by supposing that the attack of abdominal pain and vomiting from which the man suffered three years before coming under my notice, was due to the perforation then of his vermiform appendix, adhesions being formed which prevented for the time a fatal result; and although this may seem to do violence to probability, such a supposition has its analogy in other varieties of disease. For instance, a small empyema or its

caseous remains may exist for years before it becomes the immediate focus which determines an outbreak of tuberculosis in the lungs or the meninges of the brain. It is impossible, at all events, that the condition found post mortem should date only from the time when his last illness commenced, and if so we must suppose one of two things, either that the perforation of the appendix took place without giving rise to any symptom at all, or that many months at least elapsed between its occurrence and death. Another alternative may perhaps occur to some, viz. that the abscess around the cæcum was first in point of time and helped in some way towards the perforation, which may then be supposed to have taken place just before I saw him; but in that case it is all the more difficult to account for the presence of the abscess.

In any case it is certain that the abscess-sacs were of very considerable age, and it is a striking fact that the man should have been able to go about his daily work, apparently in good health, with such a condition of his abdominal cavity.

V.—*A Case of Myxœdema.* By James Anderson, M.D.

Read October 24, 1884.

THE patient, Jessie A., an unmarried woman, æt. 40, came to Moorfields Eye Hospital in February of this year as an out-patient under the care of Mr. Gunn, to whom I am indebted for permission to bring the case before this Society. She complained of swelling of her eyelids, especially the upper, which she had noticed for the past nine years in gradually increasing amount.

The patient is one of a family of nine, none of whom have shown any symptoms resembling hers, her four sisters, all older than herself, being in perfect health, one of them who was seen contrasting markedly with the patient. Her father died at seventy-four of "ulceration of the bowels," having previously been a very healthy man. Her mother died at the age of fifty-seven from "hæmorrhage," having had "coffee-ground vomiting" and oozing of blood from the mouth. Of her grandparents she knows nothing. There is no history of gout or hæmophilia either in near or distant relatives, so far as the patient is aware.

Up to the age of twenty the patient lived at home in Liverpool. Thereafter she was for twelve years nurse and maid in a family in Liverpool, and for the last eight years she has held a similar position in a family living in Kendal. She has always had a good home, with plenty of fresh air and exercise, and freedom from anxiety or exposure. For the first twenty-three years of her life she enjoyed excellent health, when, seventeen years ago, she had two teeth extracted, the bleeding from which lasted twenty-four hours and was very copious. She says she has never been well since, and to this she attributes all her trouble. She believes that when she pricks or cuts herself she loses more blood than others, but of this she did not complain spontaneously. Her gums are extremely vascular and hypertrophied. They bleed freely, and she sometimes awakes in the morning with her mouth full of blood. She began to menstruate at the age of fourteen and has menstruated regularly since. The amount has always been copious, but sometimes after the discharge

has ceased it commences again, and may last for three weeks, leaving her very exhausted. She has never suffered from epistaxis or hæmorrhoids, has had no swellings of the joints or dark coloured urine. I may state here that in June of this year, after leaving London, she was under the care of Dr. Symington, of Wolverton, for severe menorrhagia.

The patient presents the characteristic *facies* of myxœdema as described by Dr. Ord, the defined scarlet flush of cheek and nose contrasting markedly with the waxy pallor round eyes and mouth. I need hardly describe her loose, baggy, almost translucent lids, dry, brawny cheeks, and thick blue lips, her swollen, red, "expressionless" hands, with their stunted fingers, and her characteristically deliberate speech and action. Her sister says she noticed the difference in her twelve years ago, and that it has become steadily more and more marked. The patient is a woman of considerable intelligence. She says she always spoke slowly and deliberately, but she is conscious of sometimes feeling heavy and listless, at others bright and active.

Her digestive system is somewhat defective, but her appetite is fairly good except for breakfast. Her front teeth are good, but the molars have crumbled away bit by bit, and she has the unhealthy condition of gums noted above. The heart's impulse is in the usual position and the sounds normal. The pulse in the peripheral arteries is regular, but very feeble. She is occasionally troubled with palpitation and dyspnœa on exertion. The lungs are normal. The liver, spleen, and other abdominal organs are apparently healthy. Her urine varies much in quantity, from scanty to copious; but scanty or copious, the total amount of urea excreted daily is invariably deficient, only rising to half the normal amount, and frequently being as low as one fourth. The specific gravity varies from 1015 to 1025, and neither sugar nor albumen has on any occasion been detected. There is no pitting on pressure in any part of her body, but the whole body is tolerably uniformly affected with the solid œdema peculiar to these cases. The mucous membrane of the mouth is tumid and dirty grey in colour. All her skin is dry and scaly, and she never perspires even on exertion. Her extremities are always cold, and her axillary temperature always subnormal. She has a small mole on her right cheek. The supraclavicular regions are full and elastic to touch; there is no definite tumour. It is difficult to make out the precise condition of the thyroid gland. It is certainly not hypertrophied,

it may be atrophied. She gives a history of fulness of the throat with thickness of speech, and a "choking feeling," when she had the severe hæmorrhage seventeen years ago.

On the side of the nervous system she has the characteristic slowness of speech and action, from which we perhaps too hastily infer a corresponding slowness of sensation, thought, and volition. United with this apparent hebetude is the constant recurrence of a nervous restlessness which she terms "fidgets," so marked and troublesome that she sometimes dreads night coming on as she cannot lie still. The motor system is intact, and sensation as regards touch, pain, and temperature is normal and not appreciably delayed. The knee-jerks are present. Taste, smell, and hearing are up to the usual acuteness. She is slightly hypermetropic, but with the correcting lenses has perfect vision. Ocular movements, pupils, and colour vision are normal. Under atropine there is seen to be slight peripheral opacity of the left lens, otherwise the media are normal. In neither eye, however, according to the observation of Mr. Gunn and Mr. Nettleship, is the retina of normal transparency, a haze surrounding the vessels, especially in the neighbourhood of the disc. The appearances differ entirely from those of a past neuritis or retinitis, and, as has been said, do not interfere with perfect vision.

As regards treatment the usual tonics seemed to have no effect whatever. Dr. Stephen Mackenzie kindly admitted her under his care at the London Hospital, and to him I am indebted for detailed observation during her stay there. A fortnight after her admission she was put upon half-drachm doses of Tinct. Jaborandi, which she continued till she left the hospital three weeks later. Of objective improvement there was none or almost none, but she expressed herself as feeling greatly better within a few days after commencing the jaborandi. This subjective improvement was greatest when the skin was somewhat moist, and was accompanied by a steady and marked increase in the daily amount of urea excreted, which doubled itself during the period, and was at its maximum on her discharge from the hospital. Fallacy from variation in diet was guarded against as carefully as possible. Since leaving London she has been under the care of Dr. Symington, of Wolverton, who has kindly written me regarding her. She appears to remain in very much the same condition.

The points of interest in the case seem to be—(1) the history of commencement from a severe hæmorrhage combined with the

present hæmorrhagic tendency of the patient; (2) the occasional state of nervous restlessness so apparently incompatible with the general character of the disease; (3) the retinal condition, which is peculiar, whether characteristic of the disease or not; and lastly, (4) the effect of jaborandi on the subjective condition of the patient, and on the amount of urea excreted by her.

VI.—*A Case of Paralytic Dislocation of the Patella.*

By C. Hilton Golding-Bird, M.B. *Read October 24, 1884.*

Emily M., æt. 11, came to the out-patient department Guy's Hospital on July 17, 1883, with this history:

Six months ago, whilst running, her left knee gave way under her and she fell; in getting up again she felt it click. There was no bruising, pain, or swelling, and she went about as usual.

A week later, and at intervals during the next six months, the same circumstance recurred, but at last, the knee getting painful, she came for advice.

The note then made was, "Capsule of left knee very lax; on flexion, the patella rides on to the external condyle but returns to the middle line with a click on extension; this causes no pain. The patella is very moveable laterally when the knee is bent to a right angle; there is a slight tendency to genu valgum."

From July to November the knee was encased in plaster of Paris. On its removal this note was made: "The patella on flexion still rides on to the external condyle, and goes back to the intercondyloid notch on straightening, with an audible click. This occurs both with active and passive movement. The patient now walks with the knee stiffer than on the former visit."

Nothing more was done for her till February, 1884, when she was admitted as in-patient.

The same conditions were noticed except that on flexion the patella now lay completely on the outer side of the external condyle.

The following facts were now observed in addition. Both lower extremities were of the same length, and from the knees downwards equally developed, but the thighs were of unequal girth. The posterior half circumference of the thighs was the same at all levels, but the anterior half circumference of the left thigh was everywhere three quarters to one inch smaller than on the right side, due to wasting of the quadriceps extensor. Palpation showed the left extensors to be markedly wasted, though they were with the right extensors

equally irritable to the interrupted constant electric current. When lying flat in bed the patient could lift both legs up, but the loss of power on the left side was very evident.

When, during flexion, the patella was on the outer side of the external condyle, the ligamentum patellæ as a rigid cord was seen passing obliquely from the tip of the patella to its tibial attachments.

The displacement being clearly due—as will be directly mentioned—to structural shortening of the extensors, I divided the ligamentum patellæ subcutaneously, and replacing the patella in the middle line and strapping it down, put the knee in a splint in a semiflexed position.

When examined a week later it was found that whilst on flexion, active or passive, the patella still rode over the external condyle, yet that by slight pressure with the finger this could be prevented. No amount of pressure prevented the displacement on flexion prior to the operation.

The child was sent out ten days after operation wearing a plaster-of-Paris splint, which was removed four weeks later. The note on its removal (in May, 1884) was, "The only perceptible difference now is that whilst the patella can be displaced outwards as before, yet that when it returns to the middle line it does so by a gliding movement and without the 'click.' The child walks as when first seen, *i.e.* with the knee rather stiff and the foot somewhat everted. There is no pain or loss of power."

Remarks.—The wasting of the extensors seems to have been due to infantile paralysis although its existence was not known before. The growth of these muscles not keeping pace with that of the femur, a time came when, in the flexed position, it was a shorter route for the tendon and muscle from origin to insertion to pass first over and then outside the external condyle. I do not think that this displacement occurred suddenly at the time of the fall; but that it was the first time the "click" was noticed and hence attention was drawn to the deformity.

The operation performed was founded upon the explanation given, in the hope that by adding to the length of the tendon the dislocation might be cured. Though this did not result, yet the absence of the click proved that some tension had been removed.

The condition described and the explanation of the case given are in accord with the statements of Hueter, though it seems to me that the name "paralytic" dislocation of the patella

better expresses the real state of affairs than "congenital" dislocation. The reason that I attempted operative measures for a condition in which it is known patients get along very comfortably through life, and in which this case certainly will have to remain, was, that pain was complained of, though in a less degree than the annoyance of the "click." The former was early relieved by the plaster splint, the latter by operation.

VII.—*A Case of Skin Eruption due to Bromism.* By R. E. Carrington, M.D. *Read October* 24, 1884.

I AM able to bring the case before the Society by the kindness of Dr. Boyd, of Victoria Park, under whose care the patient came for the skin affection. The subject of the sketch is a male child, aged one year. There is phthisis in the father's family, but none in the mother's. Both the parents are healthy and well. The mother has not lost any children, nor had any miscarriages. She has another child, a female aged three years, who has never been ill in any way except from an attack of impetigo capitis, from which she completely recovered by the use of tar ointment. This child is now plump, healthy, and rosy. I first saw the child, the subject of the present communication, on March 8, 1884, and received the following history. The boy was quite healthy when born, and remained so for nine months, except that after vaccination, when three months old, he was affected by a red rash, according to the mother's description resembling measles. This remained out for a week and then subsided. He never suffered from "snuffles," or any other evidence of congenital syphilis.

Three months before I saw the patient the mother noticed that the child was unable to hold up his head, which fell backwards when unsupported. At this time he was able to say a few words and to walk a little with assistance, but he has been unable to do either since the present illness. Six weeks before I saw him the child became convulsed and unconscious, and medical advice was sought. Dr. Bothamley, of South Hackney, under whose care the patient came, has been so kind as to furnish me with the following note of the case at that time. He wrote to me as follows: "It was a case apparently of cerebro-spinal meningitis with very severe convulsions, the movements being constant for hours at a stretch. I started with one scruple of bromide of potassium in an ounce and a half mixture of which one drachm was administered every four hours. I pushed it for a time to one drachm every three hours, afterwards diminishing it again to the four hours. He went on without change, to as nearly as I can remember seven weeks. For a time I substituted the ammonium for the potassium salt. The rash came out very gradually, and as soon as I saw the

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Mintern Bros. Chromo Lith.

Dr Carrington's Case of Bromide Rash.

papules I stopped the bromide, but in spite of that they increased until they arrived at the severe looking eruption you saw."

Under this treatment the child, after a month, recovered from the unconsciousness to a considerable degree, but was still liable to convulsions from time to time, and indeed had been so affected on the day I saw him; there had been progressive loss of flesh since the onset of the illness. Four weeks previously to my first visit the child had had a discharge from the eyes and nose which had got quite well in a week and was but slight at any time. The eruption had first appeared three weeks before I saw the patient. The mother, who is an intelligent, observant woman, said that it came out in the form of minute red spots, which in the course of three days increased to the size of split peas, and that then in a few hours many of them enlarged to the size of penny pieces. At first each spot was surrounded by a narrow deep red halo. They first appeared on the right side of the body, and three days elapsed before any were seen on the left. The child always lay on the left side. Fresh spots appeared from time to time, and some became smaller, none, however, had disappeared up to the time I saw him. At my first visit, on March 8, the boy was lying on the left side, for the most part quiet, but crying when disturbed. He was unobservant, more or less unconscious. There was considerable wasting but it was not extreme. There were no signs of rickets nor of congenital syphilis. Only the two upper and the two lower central incisor teeth were cut. The anterior fontanelle was rather widely open, but not depressed then, though the mother had noticed that it became so from time to time.

The parts affected by the eruption were the buttocks, legs, and thighs; there were a few spots on the scalp and face, the front of the abdomen, one on the chest, one or two on the left arm, none on the right. The back of the trunk and the hands and feet were free. There appeared to be no itching, the child lying quiet unless disturbed; this conclusion was also confirmed by the mother. The spots varied greatly in size from that of a pin's head to that of a penny piece. They were for the most part discrete, but here and there two or three had run together. They had never been moist from the beginning. The small spots at first were of a bright crimson colour, subsequently they became of the dull brownish hue most of them presented at the time I saw him. In only one spot did I see a halo present, and in this case it was bright red and about one eighth of an inch in diameter. The small spots were hemi-

spherical and smooth, the larger ones were flattened, circular, or elliptical in shape, with an elevated, sharply defined edge at least one eighth of an inch in thickness (Plate II). Their surfaces were corrugated and wrinkled. A thin film could be removed by means of a lancet, without pain, and the masses appeared porous and spongy, without the least trace of serum or of pus in the interior. The chest, with the exceptions I have indicated, appeared healthy, and I could discover no physical signs of visceral disease. Dr. Boyd had been treating the child with half a grain of iodide of potassium and one minim of Fowler's solution three times a day, and this was maintained throughout.

I again saw the child on March 22. There was now very great improvement in every respect. He was now quite conscious and observant. The nutrition was also greatly improved and his colour was good. He had had some convulsions during the preceding two days, but save these none since I last saw him. All the spots now were much altered. Some of these, formerly of the largest dimensions, were represented only by a pinkish discoloration, others were of a dark coppery red, much shrunken and nearly level with the skin, and evidently in process of disappearance. There had never been any moisture, and no new spots had appeared since my previous visit. All the spots that had disappeared had been on the right side, none had completely gone from the left; so that those which came out first disappeared first. Several of the small spots had died away without any increase in size. The child was still being suckled by the mother.

I have little further to add, for the child made an uninterrupted recovery from all his troubles. I saw him again on May 20. He was then plump and healthy looking. Dr. Boyd's medicine had been discontinued for six weeks. All the spots were gone except one or two on the scalp and one on the face, and these appeared as dry scabs. No new ones had come out since I last saw him. It seems worthy of note that though the eruption was apparently due, without doubt, to bromism, it subsided completely during the continuous administration of iodide of potassium.

VIII.—*A Case of Tumour of the Right Kidney in an Infant successfully removed by an Abdominal Incision. Recurrence. Death.* By Rickman J. Godlee, M.S., M.B. *Read October* 24, 1884.

I HAVE ventured to submit an isolated case of removal of the kidney to the attention of the Society, because it is, I hope, a not unimportant one in connection with the treatment of a disease of children not very uncommon, which if left alone is necessarily fatal, but which, if diagnosed tolerably early, it will be seen can be dealt with easily and safely by the surgeon; and even though this child did not prove to be permanently cured, I think the result of the operation should encourage us to endeavour to make a diagnosis and to interfere surgically at a much earlier period. This is I am aware more easily said than done, because it is well known that such tumours of the kidney very commonly cause no symptoms, and thus the existence of a tumour is most likely not suspected by the mother until it has reached a very considerable size.

J. I. K., æt. 1 year 10 months, was admitted under my care at the North-Eastern Hospital for Children, on August 3, 1883, on account of a very obvious swelling in the right lumbar region. It was brought to the out-patient room some weeks previously, but it was impossible to admit it at that time because it had suffered from measles in the early part of July, as a result of which, it was when first seen pale and sickly looking. The mother first noticed that the child's abdomen was swelled in the month of June, and when I saw it there was a very distinct tumour, very easily to be felt, and which it was scarcely possible to mistake for anything but an enlarged kidney. It was of a rounded or ovoidal shape, the long axis being vertical or nearly so, smooth on the surface or but slightly irregular, very elastic but without giving a sense of fluctuation. The upper end of the mass reached the middle line about two inches above the umbilicus; the lower end was situated further backwards. No bowel passed over the surface of the tumour, though some was present over the lower extremity. It reached upwards towards the liver but obviously was not continuous with it; downwards it did not extend much below the level of the umbilicus, and behind it

so that it was out of the question to attempt any operation for the removal of the recurrence. The persistence of the chest symptoms, moreover, and the irregularity of their course, made me suspect the presence of secondary deposits in the thorax, though there were not at any time definite physical signs pointing to such a condition. After some weeks, while the general condition became worse, the tumour extended with great rapidity, filling up the iliac fossa, and extending downwards through the inguinal canal along the scrotum, where it formed a hard pyriform swelling. It was last seen about the end of February, after which it was not brought to the hospital, but I am informed by Mr. Ritchie Norton, of Tottenham, who afterwards attended the case, that it died in the course of a few weeks, and that he was unable to obtain permission to examine the body.

The tumour, which, I am sorry to say, was accidentally destroyed a few weeks ago, weighed about a pound; it was of a pretty uniform ovoidal shape, but somewhat knobby on the surface; it was completely enclosed in a very definite and quite firm capsule, and when cut into was found to be excessively soft, having a whitish, brain-like appearance. The growth appeared to involve the whole of the kidney, as no normal kidney substance was to be discovered, and the ureter simply passed out of the lower part of the mass. I am not able to say whether the suprarenal capsule was involved, but I imagine that it was not, and that it was not interfered with by the operation. I have placed some microscopical preparations upon the table which will show that its structure is that of a sarcoma, the cells being of moderate size. The nuclei are for the most part irregularly round or oval, but a considerable number are oat-shaped, corresponding to strands of spindle cells which traverse the tumour, dividing the rounder cells in parts into larger or smaller irregularly shaped masses. The tumour is excessively vascular, the vessels being thin walled and of moderate size. No trace of the proper kidney substance was seen in any of the sections examined, nor were any striped muscular fibres discovered.

A certain number of cases of removal of the kidney for tumour in infants have been published, and there are no doubt several others which have not been recorded.

Mr. Jessop removed a kidney affected with "encephaloid" disease, in 1877, from a boy æt. 2½, by means of the lumbar incision; and although there was considerable hæmorrhage and the operation was not performed antiseptically, the child

made a good recovery, but died eight months afterwards of a recurrence (*Lancet*, 1877, vol. i).

Kocher, of Bern, had a similar case, "adenosarcoma," which he removed from a boy æt. 2½, in 1878, by the abdominal incision. Here again there was considerable hæmorrhage, and the child died of septic peritonitis (*Deutsche Zeitschrift für Chirurgie*, Bd. xix, 1878).

Czerny has the youngest case on record, a girl æt. 11 months. The tumour, which affected the left kidney, was removed through a median abdominal incision. There was but little bleeding, but the child died three days after the operation of septic peritonitis (*Deutsche Med. Wochenshrift*, 1881).

Hüter operated on a girl of 4 years by an abdominal incision over the tumour in 1876, but death occurred during the operation from hæmorrhage.

These are the four cases mentioned in Czerny's table appended to the paper read by him at the Medical Congress in London in 1881.

Hicquet records a case which perhaps hardly comes under the present category. The patient was a girl æt. 6 years, and the tumour which when first observed in February, 1880, was no larger than a hen's egg, by August had filled the greater part of the abdominal cavity. It was removed, however, apparently without much difficulty, by the median abdominal incision. A drainage-tube was employed, and the child made a good recovery in thirty-six days. In five months no recurrence had taken place, but we have no late note of the case.

There is also a case, the only other on record of which I have been able to find, by Bókai, junr., of Orvosi. The age is not given; the tumour, which was on the left side, was a medullary sarcoma weighing 4 kg., about a quarter of the weight of the child. It was removed by an abdominal incision. The child died on the third day of septic peritonitis. There were no secondary deposits.

I can add, however, another not yet recorded, which was under the care of Mr. Heath at University College Hospital. It was in a little girl and the tumour was of large size. I helped Mr. Heath with the operation, which was one of great difficulty; in fact it was not possible to remove the whole of the growth which had involved the surrounding parts extensively. The child died.

It may be said that these are not very encouraging statistics

on which to argue in favour of surgical interference in these cases. Eight children in all, five of whom died of the immediate effects of the operation, two who recovered and remained well for some months and then died of a recurrence, and one who recovered and was well when last heard of. But, on the other hand, it is a condition which, if left alone, is so absolutely hopeless and leads to a result so miserable, both for the child and its parents, that I think we are justified in recommending an operation, at all events until a certain number of small tumours—I mean smaller than mine—have been dealt with. If after the removal of such small tumours, recurrence after a certain number of months should be shown to be the invariable result, the question would have to be reconsidered.

I am far from advocating the attempted removal of those enormous masses of growth that we not infrequently meet with. The mere size of the tumour I believe materially increases the shock of the operation, and although, as was said above, it is not invariably the case, there is very great probability that secondary deposits or the infiltration of surrounding structures may have taken place; under which circumstances the chance of cure is of course absolutely *nil*, while the surgeon will very likely have to endure the painful reflection that he has performed a useless operation which has been immediately followed by a fatal result.

As to the minute structure of these growths, there is not a very large amount of evidence forthcoming. Some of them are described as cancers, but when an accurate description of them is given it has usually proved to be (as far as my investigations have gone) some form of sarcoma, as it was in the present case.

There is a very interesting class of cases, some of which were brought to the Pathological Society in 1882 by Mr. Eve and Dr. Dawson Williams, in which a large part of the growth was composed of striped muscular fibre. These tumours are thought to be congenital; they may reach an enormous size, and have in more than one instance affected both kidneys. This last peculiarity would obviously make them, if diagnosable, particularly unsuitable for surgical interference.

IX.—*On the Treatment of Chronic Dysentery by Voluminous Enemata of Nitrate of Silver.* By Stephen Mackenzie, M.D. *Read November* 14, 1884.

IN 1882 I brought before another Society* a series of cases of chronic dysentery, or dysenteric diarrhœa, treated by voluminous enemata of nitrate of silver. This plan of treatment of an admittedly most rebellious disease had yielded, in my hands, most satisfactory results. Further experience has strengthened my belief in its efficacy, and I now feel that we have in it a safe and sure remedy that will arrest the disease in most cases. The mode of procedure I adopt is as follows: The quantity of nitrate of silver to be used is dissolved in three pints of tepid water in a Leiter's irrigating funnel. This is connected by india-rubber tubing with an œsophageal tube with lateral opening or openings. A stopcock in the india-rubber tubing renders the apparatus more managable but is not essential. The patient is brought to the edge of the bed, and made to lie on his left side with his hips well raised by a hard pillow. The terminal tube of the apparatus being well oiled is then gently passed eight or ten inches along the rectum, and the Leiter's funnel raised, or if previously raised to a suitable height, the stopcock turned, when the solution of nitrate of silver gradually, but irresistibly, forces its way along the colon until the whole is injected. Sometimes no pain or discomfort is experienced, but occasionally the injection gives rise to some pain, rarely of any severity. The bulk of the enema causing contact with the whole or nearly the whole of the colon usually promptly excites peristaltic contraction of the bowel and causes the prompt evacuation of the injected fluid. Usually it is not retained for more than five minutes, often less, occasionally it is retained for a quarter of an hour or longer. It has been thought advisable in the latter case to inject a solution of chloride of sodium for fear of absorption of the silver salt. The injection of the latter has given rise to more discomfort than the enema of nitrate of silver, but it is perhaps advisable when the fluid, as occasionally but rarely happens, is long retained. Usually little difficulty is experienced in injecting the whole of the three pints. I have tried

* Medical Society, March 20, 1882. *Lancet*, 1882, vol. i, p. 640.

various strengths of the nitrate of silver, from thirty to ninety grains to the three pints, according to the severity of the case and the vigour of the patient. One drachm of nitrate of silver to three pints of water has been the most usual strength employed.

The treatment is based on the view that whatever the nature of dysentery, whether constitutional or local in the first instance, its later effects, when chronic, are due to ulceration or chronic inflammation of the colon, and that this local disease of the bowel is best treated, as ulceration or chronic inflammation elsewhere, by topical applications. The large bulk of the enema insures probably the whole of the colon being bathed with the medicament employed. I have once or twice tried perchloride of iron instead of the silver salt, but the results have not been so satisfactory.

The immediate effect of the enema has been in some cases to temporarily increase the looseness of the bowels, but only for a day or so. In the majority of cases a marked but rather gradual recession of the diarrhœa takes place, and in several the injection has required to be repeated before the case was cured. In one case I have published, as many as twelve injections were used, and in another seven injections. In no other cases have more than two injections been required. In some cases a single injection immediately arrests the flux, and the stools become solid.

In some of the cases all other treatment has been suspended when the injections were employed; in some the Dover's powder, which the patient had been previously taking without restraining the looseness of the bowels, has been continued; and in some, small doses of perchloride of iron have been continued or subsequently administered.

In the earlier cases various plans of treatment were previously employed, and it was only when these were found wanting that the large injections were had recourse to. In this way the fallacy that other recognised plans of treatment would have been equally successful was avoided. During the period over which these cases have been distributed I have successfully treated many other slighter cases of chronic dysentery or dysenteric diarrhœa without injections. But I have become so convinced of the saving of time and suffering to the patient in severe cases by the large enemata of nitrate of silver, that in the last two cases I did not waste time by preliminary treatment.

The plan of treatment has no claim of novelty. Enemata of nitrate of silver have been recommended and used; volu-

minous enemata of different kinds have been used for years by some physicians; and voluminous enemata of nitrate of silver have been employed by a few. But it is a fact that large enemata of nitrate of silver are not in general use, and are not in use by those who have ample opportunity of treating the disease, as I showed in my former communication.

In no case in which I have employed this treatment has it failed, and I now bring forward six additional cases, comprising all I have treated in this manner since my former series. All the cases have been under my care in the London Hospital, and the following brief abstracts of them have been made for me by my house physician, Mr. Alfred Peskett.

Case 1.—H. P., æt. 38, a painter, was admitted into the London Hospital on January 1, 1881. Twelve years ago, when at Mauritius, he had an attack of dysentery which lasted five weeks. Six months after this, when on his way to England, he was attacked again; this time it lasted a few days. He then enjoyed good health for two or three years, when he was again attacked, and each year since then he has suffered from diarrhœa, the attack every year becoming more severe, the present illness being a continuation of one of these attacks.

On admission.—He was wasted and anæmic; he complained of sleeplessness, a feeling of fulness after food, and diarrhœa, the bowels acting five or six times a day. He was treated with compound ipecacuanha powder in gr. x doses and rest in bed; this was continued till the 15th January with no benefit, so on the evening of that day an enema of one drachm of nitrate of silver to three pints of tepid water was given. All other treatment was suspended.

January 16.—Bowels acted four times. January 17.—Five times. January 18.—Four times. January 19.—*Injection repeated* as before. January 20.—Four times. January 21.—Three times.

From this date till February 8 his bowels were opened from twice to three times a day, but the motions have been formed, and have been passed without tenesmus or any trace of blood.

February 18.—Passed two loose motions during last twenty-four hours. Has been on fish diet for two days; compound ipecacuanha powder ordered, in gr. v. doses, three times a day.

February 28.—Motions again well formed; and from this time until his discharge on March 22, his bowels generally acted once, and occasionally twice a day.

Case 2.—H. S., æt. 25 years, a sailor, a native of Bombay, was admitted on December 13, 1881.

He has had dysentery once before, and assigned the present attack to the cold weather.

On admission.—He was well nourished. He complained of great pain in his abdomen and burning pain in his rectum, with diarrhœa; the bowels acting ten times in twenty-four hours, T. 98°. He was treated with Pulv. Ipecac. Co. gr. v, 4tis horis, and an enema of starch and opium (ɱ xxv of tincture).

He rapidly improved under this treatment until on the 18th December he passed only one motion during the twenty-four hours. He continued to improve till the 26th December, when his bowels again became relaxed and were opened four times; he gradually got worse, so on 6th January, 1882, an enema of one drachm of nitrate of silver to three pints of tepid water was given. The injection returned in a quarter of an hour.

January 7.—Bowels acted four times, temp. 104·5.° January 8.—Three times, temp. 101·8.° January 9.—Five times, temp. 99·5.° January 10.—Twice, temp. normal. January 11.—Five times.

The motions were fluid, of a yellowish-brown colour, with no blood or mucus. *Enema repeated.*

From that time till 28th January, the bowels acted, on an average, three times. January 29.—*Enema repeated.*

February 10.—Very little improvement since last note. *Enema repeated.*

February 11.—One motion. From this date till May 20th, when he was discharged, he passed only one formed motion a day.

Case 3.—F. F., æt. 52, a sailor, was admitted on October 27, 1882. Two months before, when on shipboard in the Indian Ocean, he was attacked with diarrhœa, which was somewhat relieved by treatment, but just before arriving home he became worse and passed a little blood.

On admission.—He was a well-nourished man with a sallow complexion. He complained of pain in his stomach and diarrhœa, the bowels acting eight times in the day. Temperature normal.

October 28.—Bowels acted six times. October 29.—Five times.

October 30.—He passed eight motions, so an enema of

forty grains of nitrate of silver to three pints of tepid water was given.

October 31.—Bowels acted six times. November 1.—Five times. *Injection repeated.* November 2.—Three times. November 3.—Once. November 4.—Twice, motions formed, with a slight streak of blood in them.

From that date till November 24 his motions remained formed, with occasionally a streak of blood in them. Discharged cured on that date.

Case 4.—W. P., æt. 61, a labourer, was admitted on December 19, 1883. At the age of 21 when in India he had a severe attack of dysentery which lasted eighteen months, but from that time until five years ago he had always enjoyed good health, when he was again suddenly attacked, the onset being ushered in by severe abdominal pain and diarrhœa, and passing of blood and mucus in his motions; ever since then the symptoms have persisted, varying in degree from time to time.

On admission.—He was fairly well nourished, but stated he had lately got much thinner, and complained of flatulence and diarrhœa, the bowels acting from six to eight times a day; the motions consisted of yellow slimy blood-stained fluid. He was treated with compound ipecacuanha powder in gr. v doses three times a day and rest in bed; this was continued for a month with no benefit, the bowels acting from nine to five times a day, so on February 4, an enema of one drachm of nitrate of silver to three pints of tepid water was given.

February 2.—Bowels acted twice. February 3.—Once.

From this date till February 27, when he was discharged, he averaged two motions a day, which were semi-solid, of a dark colour, but no blood was passed.

During his stay in hospital he gained 13lbs. in weight.

Case 5.—W. R., æt. 51, ship smith, admitted January 1, 1884. Enjoyed exceptionally good health until four years ago, when he had rheumatic fever, which left his health much impaired, and he was unable to resume his employment for two years. Eighteen months ago was attacked with looseness of the bowels, which has continued up to admission. For the last six weeks this has been unusually severe, the bowels acting as many as sixteen times in the twenty-four hours on some occasions, the motions being generally of a pale yellow colour and containing mucus and streaks of blood. He lost control

seemed to extend about as far as the mid-axillary line. It was freely moveable when grasped between the hands, and manipulation caused the child no inconvenience, nor did it give rise to the appearance of blood in the urine. The long axis of the tumour was about four inches, the short axis about two inches.

The child was naturally fair, but was somewhat paler and thinner than was consistent with good health; it was slightly rickety and suffered from cough last winter. It never had any urinary symptoms; the water, I regret to say, was not examined as the child passed it usually into a napkin.

No facts of importance were elicited from inquiring into the family history. The mother had had five children, the eldest of which died with convulsions.

I had been on the look-out for a case of this kind for some time, because from the appearances observed at a certain number of post-mortem examinations on cases presumably of a similar nature, I had been struck with the fact that even after these sarcomas of the kidney have obtained the enormous size which is frequently observed in children, they often infiltrate or involve surrounding structures, comparatively speaking, to a slight extent. It seemed, therefore, not improbable that, while the tumour was small, it would be found tolerably free from adhesions and its removal would be easy. The event proved that this was the case.

On August 10, while the child was under the influence of chloroform, an incision was made for six inches near the outer edge of the rectus over the most prominent part of the tumour. The rectus was unexpectedly wide, and the sheath had to be dissected off for some little distance before the edge of the muscle became visible. When this was reached the peritoneal cavity was opened and the smooth surface of the tumour was at once seen, covered by peritoneum, with the cæcum and ascending colon at the lower and inner part. The peritoneum was divided over the outer part of the tumour and stripped forwards, so as to separate the mass from the colon, which was then covered with a sponge placed in the lower angle of the wound and was not again seen during the operation. A very little separation with the finger served to free the posterior part of the tumour, and a little traction then drew the mass out of the wound on to the surface of the abdomen, the cellular tissue round it being quite soft and the growth perfectly circumscribed. At this stage the second part of the duodenum was seen, and separated carefully from the front of the mass. A large vein which followed a curved course across the hilus was then tied

in two places with catgut (sulphurous acid and chromic acid), and then the pedicle was isolated and transfixed with a blunt aneurysm needle armed with a stouter piece of the same catgut and firmly tied, the ends being cut as short as possible. The pedicle was then cut well beyond the tumour. No other vessels required ligature. No hæmorrhage of any consequence occurred during the operation. The sutures were applied as in a case of ovariotomy, and the wound was dressed with carbolic-acid gauze secured by a roller. The child was protected by a sheet of thin mackintosh during the operation from the spray, which was purposely made as fine as seemed consistent with its efficiency. The operation was completed in about half an hour and caused very little shock.

There is really nothing to say about the progress of the case, because the child did not suffer from a symptom of any kind. The temperature reached 99° the day after the operation and then kept always at, or a little below, normal. It was given three drachms of brandy during the first twenty-four hours and none afterwards. It did not pass water till the next morning, and then, and always afterwards, in good quantity and without trouble. It took its food well from the first, and instead of being a very fretful child it became at once quiet and happy. The dressing was changed on the third day because it had become soaked with urine; it was changed again a week after the operation and the stitches were all removed, the wound being completely cicatrised. Another dressing was applied and kept on for another week. The child was sent home on August 26, sixteen days after the operation.

I saw the child frequently after its discharge; it grew very fast and looked rather pale and delicate; it also had a little internal strabismus which I had not noticed before, but my attention was not directed to its presence or absence. It became cross and fidgety when it was cutting its teeth, but at other times seemed well and happy.

I examined the abdomen carefully many times but failed to detect any return of the tumour for several months. At last, however, in January, 1884, while the child was suffering from a severe cough, which had apparently originated in an attack of whooping-cough, it was clear that there was a tumour in the right iliac fossa considerably below the position of the original mass.

At this time the child was very ill from the effects of its cough, and it developed well-marked symptoms of pneumonia,

over the sphincter ani. With these symptoms he has had much abdominal pain and at times cramps in the lower extremities. Nine months before admission he experienced great thirst and frequent micturition, and his clothes were spotted with a sort of powder where the urine fell on them. He lost about four stones in weight in nine months.

On admission.—The patient was wasted and worn; his tongue was red. There were no abnormal signs in the chest. He passed about 3500 to 4000 cc. of urine with about 5 per cent. of sugar and ·9 per cent. of urea. On the first two days the bowels acted five and six times respectively. Feeling that with this double drain upon him it was imperative that the diarrhœa should be promptly arrested, I ordered an enema of forty grains of nitrate of silver to three pints of water. It was retained for a considerable time and caused slight pain. He improved greatly with this, the motions becoming nearly solid, and from two to three in the twenty-four hours. On January 8 a second enema of one drachm of nitrate of silver to three pints of water was ordered. The motions after this were formed and free from blood, he regained power over the sphincter, and his general condition improved, the diabetes continuing. The patient remained in the hospital until April 23, under treatment for the diabetes, which was greatly lessened but was not cured. During this time he passed large, bulky, solid, pale motions, generally two, sometimes three and occasionally four in the twenty-four hours. The motions were never fluid, and they never contained blood or mucus. He gained one stone in weight during his stay in the hospital.

Case 6.—W. B., æt. 17, a ship's steward, admitted August 6, 1884. Fourteen months before admission, when at Hankow, he was attacked with diarrhœa, which in three weeks became dysenteric and continued up to the present time.

On admission.—Fairly nourished, tongue dry, red, and furred at back. Sleep disturbed by action of bowels. Motions liquid, of a dark-green colour, with some scybala, very offensive. Temperature 100° F. He was at first treated with the following mixture: Ex. Catechu ʒss, Sp. Chloroform. ♏xv, Ext. Belæ liquid. ʒj, Aquam ad ℥j, ter die sumenda.

On August 9 he had passed three motions in the night and two in the day. There were four motions the night before. He was ordered an enema of forty-five grains of nitrate of silver to three pints of water. It was retained about four

minutes. He passed a very liquid motion with a trace of blood the same evening after the injection.

August 10.—Two motions, liquid, with a trace of blood.

August 11.—One motion, liquid.

August 12.—One liquid motion.

August 15.—One motion, with scybalous masses and a few streaks of blood.

August 16.—One motion more formed, no blood.

August 20.—Two motions, liquid, with a trace of blood.

August 27.—Temperature rose to 101·4°, only one motion.

September 5.—Four motions yesterday, of dark colour, and partly formed. No blood. Temperature normal.

September 8.—Passed a perfectly formed motion, but yesterday there was a trace of blood.

September 11.—Motions perfectly formed and contained no blood.

September 13.—Stools well formed, with a little mucus and blood. The bowels act regularly once a day. Has been up two days and is on a fish diet.

September 16.—Stools well formed. Bowels act every other day. Allowed to go into garden.

October 1.—Discharged cured. The patient gained 1 st. 1 lb. whilst in the hospital.

To summarise these cases:

Case.	Duration of disease previous to treatment.	No. of injections employed.	Duration of treatment.	Result.
1	Several years on and off	2	6 weeks	Cure.
2	Uncertain; 2nd attack	4	5 „	„
3	2 months	2	3½ „	„
4	5 years	1	3 „	„
5	18 months	2	—	„
6	14 „	1	7 weeks	„

I hope the narration of these cases will lead any members who have employed this plan of treatment to contribute their experience, and that it may induce others who have not tried it to test it in suitable cases.

X.—*Three Cases of Joint Disease in connection with Locomotor Ataxy.* By W. Morrant Baker. *Read November* 14, 1884.

CASE 1.—(For the following notes I am indebted to Mr. Francis and Mr. Aldous, surgical dressers.) A woman, E. M., æt. 54, was admitted into St. Bartholomew's Hospital under the care of Mr. Morrant Baker, October 18, 1883, suffering from disease of the right elbow-joint and of both hip-joints, and with symptoms of advanced locomotor ataxy.

History.—She had always been in somewhat delicate health. Was married at twenty-nine years of age, and has had two children who are alive and well. She has suffered from rheumatic fever, but has never had chorea or heart disease. Her father and mother are still alive and well. A brother and sister died of "consumption."

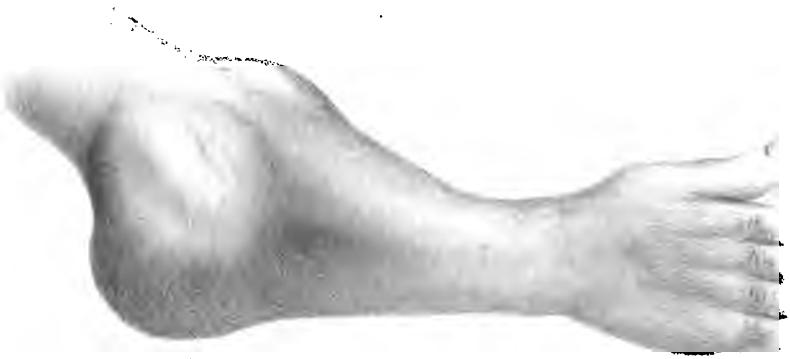
The present illness began about twenty years ago, when she had lightning pains for the first time. They were principally lancinating and confined at first to the legs. These have persisted to the present time, and are now more constant and more severe.

Soon afterwards *boring* pains commenced, as if a "red-hot skewer were being thrust into the flesh." At first these were confined to the neighbourhood of the hip- and knee-joints, but now extend all over the legs, arms, and occasionally on the body, and are very severe.

At about the same time she began to have a sensation as if "the skin were very tightly stretched all round her waist." She had had similar constrictive sensations in the legs and thighs, at first only occasional, but now almost constant.

Many years ago she began to suffer from diplopia, and of late her vision has been very defective and glasses do not help her. She has been especially troubled with *muscæ*, and objects always appear very misty.

Ataxic symptoms began about eighteen years ago. Her feet felt as if "wrapped up in something soft." The movements were tremulous and rapidly became worse, so that she could only with difficulty walk across the road or any such short distance, and the legs in walking felt "hardly separated



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from one another if she did not look at them." She was unable to stand or walk in the dark. At present she cannot lie down unless a light is burning in the room.

Seventeen years ago she was admitted to Guy's Hospital, and was there off and on for more than a year, under the care of Dr. Wilks and Dr. Habershon, and was said to be suffering from paraplegia. She had loss of power and of sensation in both limbs. She has partially recovered from this, but for eight years could not use her legs in the least. Before she was admitted, and while at Guy's Hospital, she was troubled with severe vomiting, but with no violent pain in the abdomen.

Present condition.—The patient is anæmic, thin, and debilitated. The pupils are dilated and do not respond to light, but contract on looking at near objects. There is no colour blindness. There is no affection of the facial muscles. She speaks fluently and without fatigue. There is no ear affection. At times she suffers from violent headaches and "neuralgic" pains shooting to the lower jaw and temporal region.

Her skin is smooth and glossy, and appears thin as if from atrophy. This, she says, was remarked when she was at Guy's Hospital. She suffers continually from pain in the epigastric region, occasionally becoming extremely acute and at times with vomiting, the "crisis" lasting several days. She suffers also from boring and lancinating and constrictive pains in the abdomen.

She has scarcely any power of movement in the lower extremities. The muscles are much wasted. Tactile sensation is much impaired. Two pin-points, seven inches apart, are felt in the leg as one. As she lies in bed she can raise the thighs to about an angle of 45°, but cannot lift the feet.

There is no patellar tendon reflex or ankle clonus perceptible.

Sometimes she suffers from involuntary micturition. Sometimes she has difficulty in expelling the contents of the bladder and rectum. During micturition there are generally pains in the region of the sacrum, and occasionally there are bearing-down pains in the bladder and rectum. The bowels are seldom moved without the aid of medicine.

Joints.—The right elbow-joint is much enlarged (Plate III, fig. 1), measuring twelve inches in circumference against eight and a half in the opposite limb, and has a roughly globular outline. It is tense and in parts elastic, this condition seeming on examination more due to gelatinous synovial membrane than to the presence of fluid. On flexing the arm there is much grating

perceptible, and the arm admits of abnormal movement in a lateral as well as antero-posterior direction. Hard nodules can be felt forming part of the enlargement, especially on the inner aspect. The joint seems as if scarcely held together at all by ligaments, but the patient is able to bend and extend it almost perfectly, although with creaking and grating. Now and then there is a hitch for a moment, and then suddenly the joint surfaces again slip. The superficial veins are enlarged.

Sensation is defective in the little finger and on the ulnar side of the ring-finger. The disease of the elbow-joint began about a twelvemonth ago, after a slight injury produced by falling off a sofa.

On examination of the hip-joints they are found to present the same loose and flail-like condition that has been mentioned with respect to the right elbow-joint. The trochanters are about one inch and a half above the level of the anterior superior spines of the ilium, but they can be brought down to their proper level by making traction on the legs, the abnormal position being again assumed when the traction is discontinued. On flexing and extending the thighs there is some creaking at the hip-joints, and on flexing and adducting the limb in such a way as to throw out the great trochanters the head of the femur cannot be recognised. It seems as if the trochanter formed the upper end of the bone with no head or neck attached to it. There is no feeling as of gelatinous synovial membrane or of nodular deposits of bone.

The hip-joints have been affected for many years. It is difficult to assign an accurate date for the commencement, on account of the disablement caused by the paralysis.

Metatarso-phalangeal joint, great toe (right).—The great toe of the right foot is shorter by an inch than that of the left. The metatarso-phalangeal joint is freely movable, and there is well-marked grating when the articular surfaces are rubbed together. There is no evidence of nodular bony deposits; there appears to be some fluid in the joint. On the base of the first phalanx three scars are visible, one on the dorsum, one on the plantar surface, and one in the cleft between it and the second toe. These, the patient states, are the scars of three "perforating ulcers" which healed two years ago under stimulating ointment, after they had existed for many months as little sinuses discharging matter. She knew of no exciting cause of these; they did not begin in the site of corns.

No material change in her condition occurred during her stay in the hospital, and she was discharged on December 8th.

Mr. Francis was so good as to find out her condition as it was three months afterwards, and reports that she has been getting worse. She has had four severe gastric crises. The lightning pains have been more severe and the sight has been worse. At times the *left* elbow-joint is very painful and enlarged, resembling the condition of the *right* when it first became affected. She has not injured it. The right elbow-joint is in about the same condition as when she was in the hospital. There is no apparent change in the condition of the hips. The knees swell nearly every night, but with little pain; they become smaller towards the morning. There is no œdema of the legs. The great toe-joint (right) is in about the same condition as before described. No other joints are affected. The patient complains of being very cold, in spite of abundance of fire and blankets. She is living, and has lived, for several years in a very damp and draughty cottage, built in a damp garden, without any foundations.

At the present time (October, 1884) she is reported by Dr. Deeping, of Southend, to be in about the same condition, "but the gastric crises recur at longer intervals. The joint condition does not grow materially worse."

Case 2.—(For the details of the following notes I am indebted to Mr. R. Cross, surgical dresser.) A man, W. E., æt. 56, a driver, was admitted into St. Bartholomew's Hospital under the care of Mr. Morrant Baker in July, 1884, on account of disease of the right knee-joint.

History.—The patient is said to have enjoyed excellent health all his life until about two and a half years ago, when, in helping to carry a heavy piece of furniture, he gave his knee a severe twist. He heard it crack distinctly, but he did not fall. The joint at once began to swell and he walked with great difficulty. After keeping his bed for three weeks the knee remained swollen, but he could walk and bend the knee without much pain. Three months after the accident he went to a bone-setter and was under his treatment for five months. The knee was moved and painted with iodine about once a fortnight. He next became an out-patient at the London Hospital, where rest was advised and an india-rubber bandage applied. For some little time he wore a splint, which gave him much support. About five months ago a horse trod on his right foot, and for this injury he has been a patient at the German Hospital to within the last month. Possibly as the result of this injury the middle toe is contracted and

drawn up above the level of the rest. Corresponding to it on the plantar surface is a scar about an inch and a half long. He has never suffered from syphilis and has not been specially exposed to cold or damp. He has never suffered from any gastric troubles.

Present condition.—The *right* knee-joint is much swollen and distorted (Plate III, fig. 2), measuring in circumference at the level of the patella three inches and a half more than the *left*. The *internal* condyle of the femur, although preserving its normal shape, gives the idea on examination of being enlarged and of projecting downwards and inwards, not resting at all on the inner half of the head of the tibia, which has apparently been absorbed. The *external* condyle has almost disappeared, and in its place can be felt a semi-detached, rounded nodule of bone, freely movable, of about the size of a walnut.

The *outer* half of the head of the tibia seems to take the place of the wasted external condyle of the femur, projecting as it does on a higher level, by about four inches, than the lower surface of the *internal* condyle of the femur.

The joint is very loose and flail-like, allowing hyper-extension of the tibia on the femur. The leg can be also readily bent outwards and inwards as well as in an antero-posterior direction. On grasping the knee the bones can be felt grating against each other when the patient bends and extends the limb. The leg cannot be flexed beyond a right angle. The superficial veins over the joint are somewhat dilated. The patient suffers no pain in the joint. On the plantar aspect of the great toe of the same foot is a small perforating ulcer. A probe enters it for about an inch in a direction towards the sole of the foot. The skin of the toe is swollen and somewhat inflamed. There is slight œdema of the ankle.

In both legs sensation is impaired. There has been a feeling of numbness in the right leg ever since the accident, but in the left leg only during the last six weeks. There is no patellar tendon reflex and no ankle clonus. At times the patient has suffered from what he terms lightning-pains in all parts of his body. There is loss of sexual desire. Micturition is normal.

The pupils are much contracted and do not react to light. They contract during accommodation for near objects. There is no colour blindness. The feet are said to be always damp from sweat.

About ten days after the patient's admission into the

hospital, a small abscess which had formed on the right great toe was punctured; but no communication between it and the perforating ulcer could be found. Dead bone was felt on probing the latter. The patient complained of a good deal of pain extending up the calf of the leg, and of aching pain in the stomach and hypogastric region.

On the 23rd August one of my junior colleagues, under whose care the patient was during my absence from town, judged it best to amputate the great toe, which seemed to be the source of most of his trouble. The second joint was found much diseased, the phalanx being necrosed throughout.

On the following day the wound looked well, but on the day afterwards there was a good deal of unhealthy discharge from it. On the 27th of August the patient had a severe rigor lasting for half an hour.

On the 29th August the temperature was normal in the morning, but the patient was suffering from abdominal pain, with vomiting and diarrhœa. The motions came away involuntarily. On the 30th the diarrhœa had ceased and there was less vomiting; and from this date to the 1st September there was some slight improvement in the symptoms, although the patient seemed to steadily get weaker. The temperature varied from about 100° to 103°; the urine was of sp. gr. 1013 with a trace of albumen.

On September 3rd the diarrhœa returned, and on the following day the patient died.

The right thigh and leg had assumed during the last two days a yellowish colour, as if from incipient decomposition, a large bulla forming on the inner side of the right knee. The odour of the limb was very offensive.

From the symptoms, which need not be further detailed, it may be concluded that the patient died from acute septicæmia.

Post-mortem examination.—Head.—Permission could not be obtained to examine the head.

Thorax.—Old adhesions in pleuræ. Fibroid and cretaceous nodules at apices of both lungs.

Abdomen.—Liver normal; spleen soft and engorged. Kidneys slightly granular.

The *spinal cord* and posterior tibial nerve were removed for future examination. Mr. Bowlby has since examined them and has kindly given me the following account:

"Transverse sections of the spinal cord were made on its removal from the body. To the naked eye the posterior

median columns presented a more greyish hue than the remainder of the white matter. (Portions of the cord were then preserved in Müller's fluid for several weeks, and after being kept a short time in spirit were cut with a freezing microtome and stained in picro-carmine and in osmic acid.)

"Microscopically examined, the columns of Goll were found to be degenerated through the entire length of the cord. There was in them an excess of connective tissue, a marked disappearance of the nerve-fibres, and a good deal of granular *débris*. In some parts of the field but few normal fibres were to be seen.

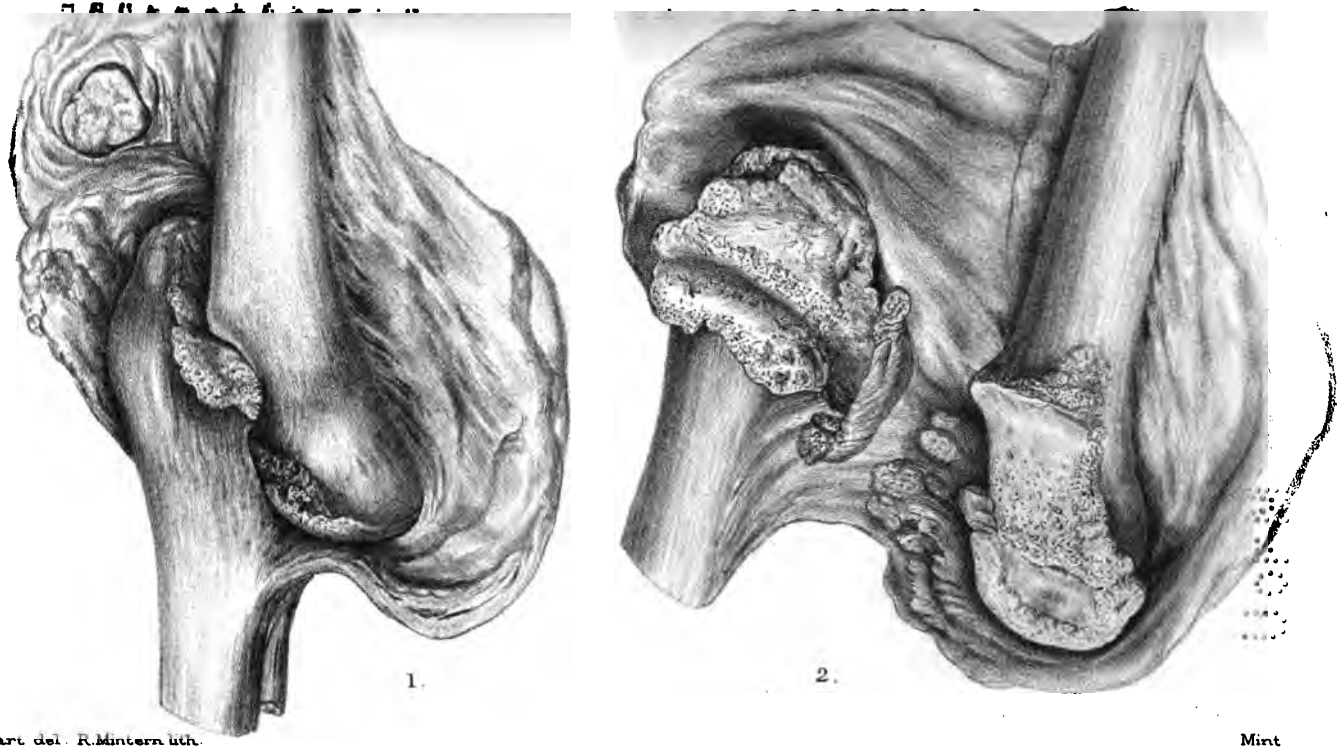
"In the dorsal and lumbar regions that part of the postero-lateral tract which was contiguous to the columns of Goll presented similar degenerative changes. The blood-vessels also were unusually large and numerous in this region, and in places their walls appeared to be thickened.

"Microscopic examination of the posterior tibial nerve did not show any definite lesion. It could not be certainly stated that there was any atrophy of the nerve-fibres."

Right knee-joint.—The right knee-joint, which was found distended with thin and foul purulent matter, was removed and is exhibited this evening to the Society. (No pus was found in any other joint.)

On examination the joint was found much enlarged; the enlargement being due to thickening and development of the various folds and processes of the synovial membrane and to alterations in the shape of the bones. The lower end of the femur and upper end of the tibia have undergone remarkable alterations in shape (Plate IV).

The external condyle of the femur has almost disappeared, its place having been taken by two irregular nodules of bone, together of about the size of a horse chestnut, which lie embedded in the thickened synovial membrane. The internal condyle appears remarkably enlarged by contrast, but the appearance is deceptive, and is produced partly by the almost complete absence of the fellow condyle, and partly by a compensatory alteration in the shape of the tibia to be immediately noticed. The internal condyle seems much flattened from side to side, and near its inner and upper surface is a marked projection or ridge which overhangs a groove produced by friction on the opposed surface of the head of the tibia. The shape of the lower end of the femur, indeed, resembles that of an enormously enlarged external malleolus. At the back of the internal condyle is a large nodulated mass



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Mr Morrant Baker's cases of Charcot's joint disease.

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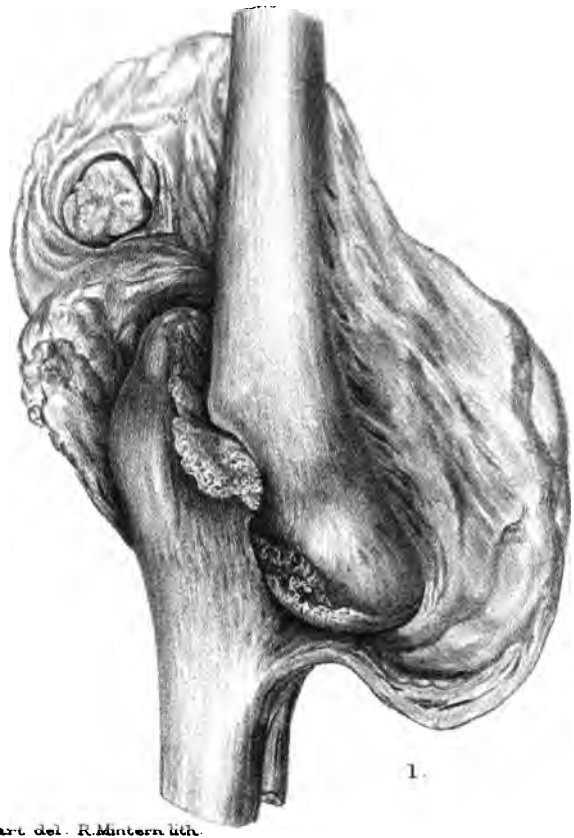

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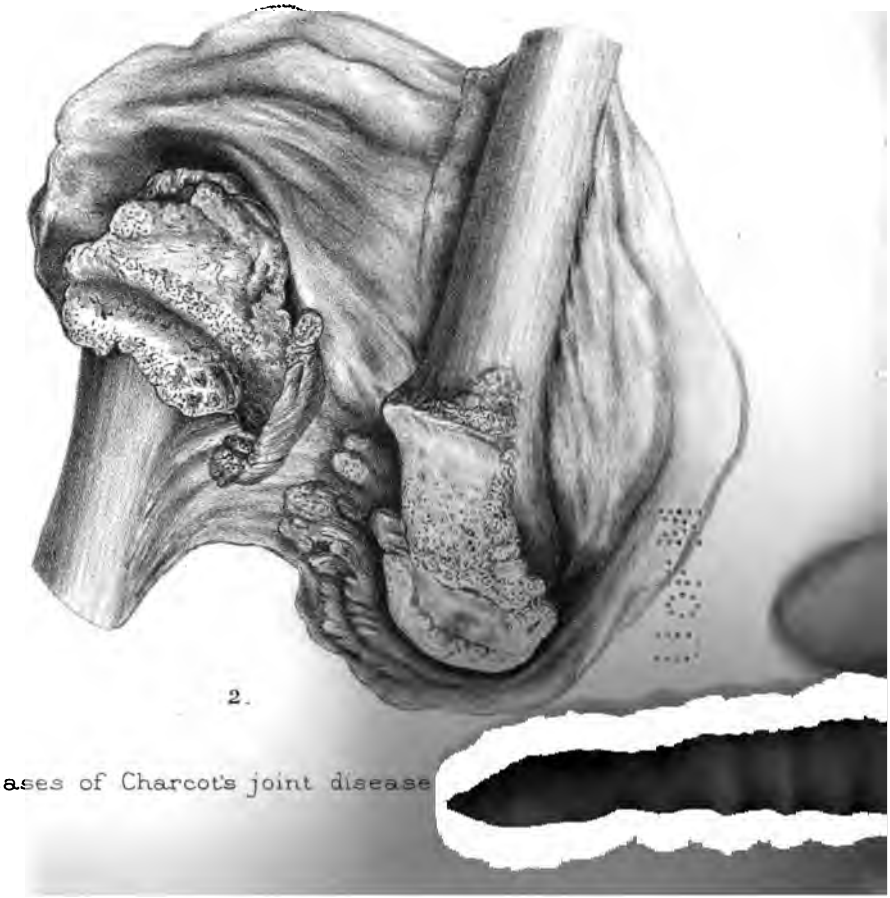

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Mr Morrant Baker's cases of Charcot's joint disease

of bone, which fits into a corresponding cup-shaped depression on the posterior surface of the tibia.

The articular surface of the head of the tibia has undergone a compensatory alteration. The inner part seems to have been completely worn away by the internal condyle of the femur, while the outer side, on the other hand, takes the place of the absent external condyle. To such an extent has this alteration occurred that the line of the tibio-femoral articulation, instead of being horizontal, is almost vertical; while the only part of the bones which could serve as a support in standing or walking is the narrow ridge on the femur just referred to, and the corresponding narrow surface of the head of the tibia (Plate IV, fig. 1).

The bone which covers the articulating surfaces of the femur and tibia is smooth and hard, and forms a continuous layer, but in other parts this smooth lamella has disappeared and the cancellous tissue is exposed as in superficial caries, the bone being pitted and irregular. The cartilage has almost entirely disappeared, but here and there a patch has escaped the grinding process. These patches have undergone advanced fibrous degeneration. (Mr. D'Arcy Power has kindly prepared sections of the cartilage and exhibits them this evening to the Society.)

The patella has undergone less alteration than the other bones, but it seems thickened and irregular. Its articular surface is covered by cartilage in an advanced stage of degeneration, and the bone is on this aspect irregular and pitted.

The inner surface of the synovial membrane has developed in many parts villous outgrowths, some of which contain calcareous matter, while others are still soft.

The development of osteophytes or of calcareous matter in the soft tissues which surround the joint has taken place to a remarkable extent. Nodules are, so to speak, infiltrated in the tissues around the ligamentum patellæ, and in various parts of the synovial membrane. They are especially well marked in the tissues which cover and protect the lower edge of the internal condyle. The edge of the head of the tibia is covered by overhanging and irregular ridges of bone, exactly resembling those seen in museum specimens of osteo-arthritis. The shaft of the femur, four inches above the condyles, and that of the tibia at about the same distance below its head, appear to be in all respects normal.

Case 3.—(For the details of the following notes I am

indebted to Mr. J. Close.) A man, J. G., æt. 46, a French polisher, was admitted under the care of Mr. Morrant Baker into St. Bartholomew's Hospital September 22nd, 1884, on account of disease of the right knee-joint.

History.—For about two years the patient has suffered occasionally from shooting pains in the right leg, which he thought rheumatic, as he had been often exposed to cold and wet. He had been quite well in health, however, and was not at all lame until about the end of December of last year (1883), when, in going upstairs he fell, and then found on getting up that the right knee was very painful and that he could only with difficulty walk across the room. He believes there was no swelling of the knee; and in two or three days he was able to go about his work as usual, only occasionally limping a little.

Three months or thereabouts before his admission into the hospital while walking he heard and felt his knee crack, and immediately found that he could scarcely get along, not on account of pain but because he felt he had lost nearly all power in the limb. He managed, however, to get home, but found it quite useless to attempt to get about, and the knee began to gradually increase in size. From that time to the present he has been quite unable to walk without support. He has had no shooting pains since the knee became swollen until the night before his admission into the hospital, when they were very severe for about two hours.

When a young man he was somewhat intemperate and dissolute. He suffered from gonorrhœa but never had syphilis. He has had smallpox. Twelve years ago he hurt his back in a fall from a cart and two abscesses formed in the lumbar region. He was laid up at this time for two months, but, so far as he knew, quite recovered from all effects of the accident.

His father and mother both lived to a good old age and were always healthy. There is no history of epilepsy, insanity, or phthisis in the family. He has never suffered from special gastric troubles, nor from headache or neuralgia.

Present condition.—The right leg (measuring from the anterior-superior spine of the ilium to the internal malleolus) is about two inches shorter than the left, obviously on account of disease of the knee-joint, which is much deformed and enlarged, measuring in circumference about two and a half inches more than the left.

On examination of the affected joint, it seems as if the *external* condyle were enlarged and projecting downwards and

backwards, overlapping the head of the tibia and not at any point resting on its upper surface. The apparent enlargement is probably illusory, and is produced by the wasting of the *internal* condyle, the latter having disappeared, its place being taken by the internal part of the head of the tibia, which seems to extend upwards and backwards. The patella can be felt, but seems higher up the thigh than normal. The ligaments of the joint seem much weakened or even destroyed. The leg can be flexed to an angle of about 135° and then is suddenly stopped. It can be hyper-extended, and the joint admits of a very abnormal amount of rotation and of lateral movement both inwards and outwards. There is no sensation communicated to the hand as of grating when the patient moves the joint. The superficial veins over the joint are somewhat enlarged, and the skin is slightly glossy.

The patient suffers no pain in the joint. The *left* knee is also somewhat abnormal. There is slight genu valgum; and the articular ends of both the femur and the tibia are irregular at their edges and nodulated. The synovial membrane seems lax but thickened; and there is much crackling or grating on flexing and extending the limb. The bursa beneath the semi-membranosus tendon is enlarged. The muscular power of the legs seems not much impaired, and the patient says that he has never noticed any defect of sensation. He has had no tingling or numbness in the feet, or any other part. When the soles of the feet are tickled, however, the left seems more sensitive than the right.

The pupils are moderately contracted, they do not react to light, but contract on accommodation for near objects. There is no diplopia, amblyopia, or strabismus; and no colour blindness.

The patient suffers occasionally from nocturnal incontinence of urine, and the fæces occasionally pass involuntarily. He dates the latter from his accident twelve years ago, when he fell from a cart. He has not lost sexual desire.

The urine is slightly acid and apparently quite normal. The body temperature is rather variable, being for the most part subnormal.

Little or no change in the patient's condition occurred during his stay in the hospital. A leather splint was applied to the right knee-joint, and from this he obtained considerable relief. He left the hospital at the beginning of November, 1884.

He is exhibited this evening to the Society.

Postscript, June, 1885.—This patient died at his home within a few months after exhibition to the Society. I am indebted to my late house surgeons, Mr. J. N. Vogan and Mr. Alfred Hind, for the considerable trouble they took in obtaining the knee-joints which are now in the Museum of St. Bartholomew's Hospital.

On examination of the *right* knee-joint, the synovial membrane was found everywhere thickened and pulpy, and in some parts papillated. The cartilage of the condyles was ulcerated, the ulceration being best marked on the inner side. On the bones are small pearly concretions like sago-grains. The articular surface of the patella is completely covered by thickened synovial membrane.

The whole of the posterior surface of the upper end of the tibia, for a depth of three inches, is worn away in such a manner as to allow of the dislocation of the bone forwards upon the femur, and a new articulating surface has been formed on the eroded surface (Plate V, fig. 1), partly by a moulding of this surface, and partly by osteophytic processes. A portion of the *lower* surface of this new articulating cavity is formed by the posterior part of the original articulating surface of the head of the tibia, which, having been apparently undermined, seems to have slipped down bodily, letting the femur fall, so to speak, and carrying with it a part of the external semilunar cartilage. This part of the original joint surface, however, assumes now a nearly vertical instead of a horizontal direction.

The posterior crucial ligament, with a portion of the external semilunar cartilage, remains attached to the femur.

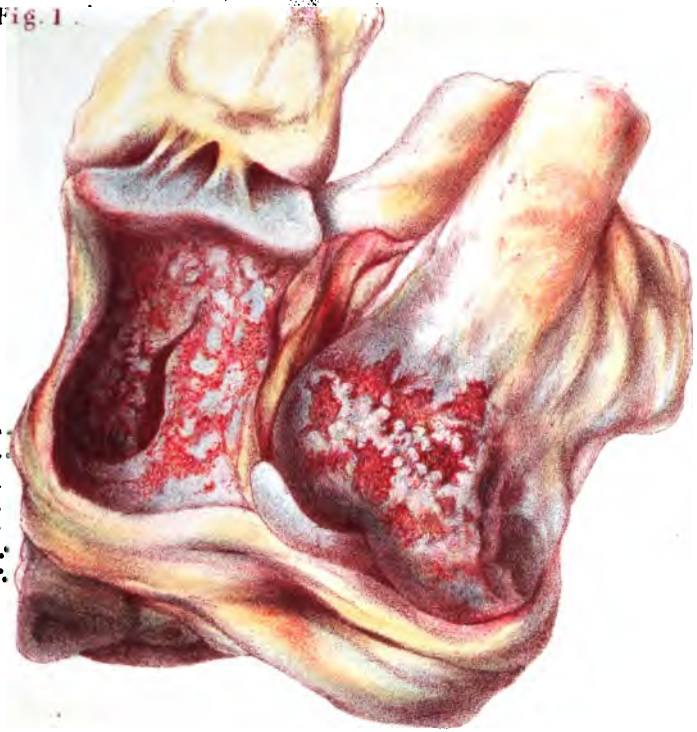
The anterior crucial ligament retains its normal attachment to the tibia, but it has lost its attachment to the femur, and is connected above with the thickened tissue surrounding the patella.

The margins of the articulating surfaces of the femur and tibia are "lipped" by slightly projecting outgrowths.

The synovial membrane of the *left* knee-joint (Plate V, fig. 2) was found vascular and papillated, the portion below the patella being pulpy. The cartilage covering the internal condyle is ulcerated at one spot. It is thickened, and clearly undergoing fibrous degeneration over its whole extent, although it still retains its polished surface.

The cartilage covering the external condyle is thickened, except at one spot where it is worn away, leaving dense eburnated bone.

Fig. 1.



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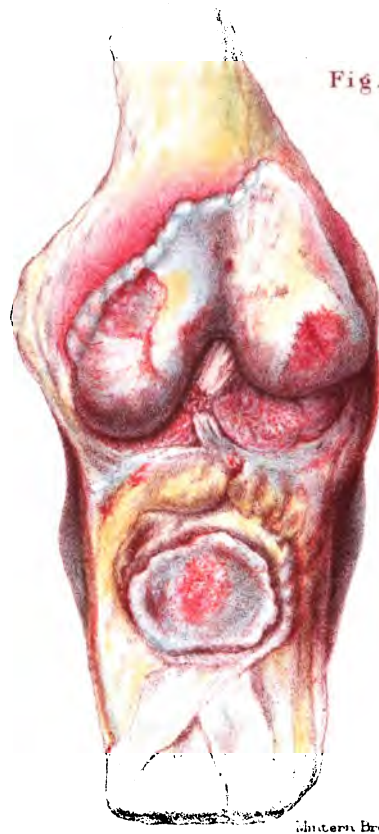

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Mr Morrant Baker's cases of Charcot's joint disease.

The edges of the articulating surfaces of the condyles are "lipped."

The semilunar cartilages are intact.

The anterior crucial ligament is pulpy and in part eroded; the posterior is also softened. The patella is "lipped;" it is covered by cartilage undergoing pulpy degeneration. It is not overgrown by thickened synovial membrane. The articulating surfaces of the head of the tibia are bare of cartilage. The external surface is undergoing erosion at the point at which it is opposed by the eroded surface of the external condyle of the femur. All the soft tissues are more or less thickened, pulpy, and degenerated.

Remarks.—If one tries to formulate the conditions found on examination of this remarkable disease of the joints, one might say that the fault lies not so much in active destructive processes as in *incapability of repair*. There is decay without re-construction; or the attempts at repair are inefficient and disorderly. Patients are able, however, in a wonderful manner to use joints which appear completely and hopelessly spoiled. They can bend and extend an arm, or bear considerable weight on a leg; in the latter case causing, one might say, some alarm to one who watches the movement at the knee, when this joint is affected, lest the limb should break in two. But the effect of using the limb is to wear out the joint, as if it were a mere mechanical hinged apparatus, made of bad materials, with no more power of repair in the bone-surfaces than if they were bad mill-stones. The osteophytes, if not remnants of the old bone which have escaped the grinding process, are only examples of degeneration, not of true development; and the surface of the bones presents the appearance not of active destruction or breaking down by some pathological process within, but rather that of having been merely rubbed down as by a grindstone. The tissues are equally incapable of resenting injuries and of repairing them.

Is not this "incapability of repair" expressed by the pathological changes of other parts in tabes? We see it at a glance in the bones, because in the performance of their mechanical functions they are worn away. But in tissues not subject, like the bones, to mechanical attrition, the diseased condition may be perhaps equally well expressed as depending on incapability of the physiological reconstruction which, for health, must always accompany physiological decay.

It is clearly impossible within the time at my disposal to

do more than refer to the different theories which may be adopted with regard to the causation of Charcot's disease of the joints, but I would venture to suggest for discussion the following questions.

(1) Is this disease of the joints a new disease, or is it one long known, but which seems new on account of its connection with other diseased conditions only lately recognised? For myself, I cannot resist the belief that the disease is only, in an exaggerated form, what has been long familiar to us under the names chronic rheumatic arthritis or osteo-arthritis. Apart from the *a priori* improbability of the sudden evolution of a new disease, I think we must come to this conclusion on comparing the pathological appearances with those presented by the specimens of chronic rheumatic arthritis preserved in our museums. One is struck by the apparent identity in the two cases. There is the same kind of deformity of joint surfaces, the same overgrowth or apparent overgrowth at one point with erosion at another, the same eburnation, the same thickening of tissues with infiltration of bony or calcareous deposits, the same dendritic outgrowths of synovial membrane, the same fibrous degeneration of cartilage.

(2) If the two diseases are identical, the next question is "What is the connection between the arthritic disease and locomotor ataxy? Is the connection a mere coincidence or, in other words, a mere accidental occurrence of rheumatic arthritis in a patient the subject of tabes? This question I am disposed to answer in the negative. Since attention has been drawn to the subject, one has been in the habit of looking for symptoms of locomotor ataxy in all severe cases of joint disease similar to those which have been related, and in almost all the cases which have come under my notice, it has been possible to guess and to guess rightly that the patient was the subject also of tabes dorsalis. And although one may allow that exactly similar conditions may exist without the usual signs of tabes, the concurrence of these pathological conditions is too frequent to permit the notion that their relationship is a coincidence only.

(3) But if this be so—if this form of rheumatoid arthritis be closely connected pathologically with tabes—how can we explain the fact that rheumatoid arthritis is not always accompanied by symptoms of tabes dorsalis?

(*a*) One way out of the difficulty is to assume that all cases of osteo-arthritis, whether accompanied by signs of tabes or not, have a neurotic origin, and that their frequent association with tabes indicates to us their pathological origin in

cases in which the usual signs of tabes do not co-exist. In other words, if a certain group of symptoms are unquestionably the result, in some cases, of a nerve-lesion, it is fair to assume as probable, to say the least, that the same group of symptoms in other cases have a direct relationship to a nerve-lesion, even when the signs of the latter are, for some reason or other, undiscoverable. If, for example, that peculiar condition of glossy fingers, of deformed nails, of low vitality of all the tissues with tendency to ulceration or even gangrene, with which all surgeons are familiar, be undoubtedly the result, in many cases, of injury to one or more nerves of the forearm, it would be a fair inference, if a case presented itself with exactly similar symptoms, but with no history of injury, that there was a lesion somewhere in the corresponding nerves or nerve-centres, even if the *effects* only of such a lesion were, at the time, discoverable. So with the disease of the joints which has been long termed osteo-arthritis. Does not its frequent association with locomotor ataxy form a strong reason for believing that its alliance with disease of the nervous system is a close one, even when the symptoms of the latter are not sufficiently pronounced to be demonstrable?

(*b*) Another theory, however, may be adopted which seems to me to explain matters equally well or better. Even if we assume the pathological relationship between locomotor ataxy and osteo-arthritis to be a close one, we are not compelled to assume that the two diseases stand in the relationship, one to another, of cause and effect; nor indeed that osteo-arthritis has an immediate neurotic origin. It seems to me conceivable that the ataxy and the joint disease may be due to some pre-existing depraved condition common to both of them, of which the effects are seen most clearly, sometimes in disease of the nervous system, with its direct consequences—paralysis, &c.—sometimes in diseased conditions of the joints, sometimes in both. That one organ or tissue should be the chief point of attack in one case of a given disease, and that another organ or tissue should chiefly suffer in another case of the same disease, is only what happens unquestionably in many affections of known origin. Syphilis and tuberculosis may be quoted as examples. Why should not tabes dorsalis and chronic rheumatic arthritis be examples of the same general law? Does not the Protean character of tabes (to which Dr. Wilks has referred so acutely of late) suggest that, as at present described, it is not a distinct pathological entity, but only a small bit of a large disease, of which we see the effects,

sometimes in one organ, sometimes in another; a disease of which we cannot understand the true size and proportions until we become better acquainted with its effects on other organs than those of the nervous system.

Mr. Barker referred to a case now exhibited to the Society. It was of a porter æt. 38, who came first under his notice in 1881 for treatment of some urinary affection attended with difficult micturition. Some years previously the patient suffered from dyspepsia and vomiting, with nausea, distension of the stomach, &c. He complained of numbness in his feet, shooting pains in the lower limbs, and a sore place on one foot. Later, he became an in-patient, and was examined by Dr. Gowers, who could find in him none of the ordinary symptoms of locomotor ataxy. He left the hospital, but subsequently returned with a second ulcer of the foot, which healed under treatment; but last year he once more returned, the sores having broken out afresh. The gastric disturbance was worse also. Dr. Barlow then examined him in vain for the usual ataxic symptoms. The patient improved, but the ulcers later on were a source of trouble, and in January, 1884, Mr. Barker had him for the fourth time under observation. The pains, weakness, and gastric disturbance were all aggravated, the left thigh being the seat of shooting pains. On the day preceding the meeting of the Society the same man presented himself yet again, and this time with complete disorganisation of the left knee-joint. This was increased in size, but could be moved without pain; it was partially dislocated inwards and backwards. He had had no evidence of structural change in this knee until the previous July, when without any assignable cause it suddenly swelled up, and, so far as the patient's power of observation went, at once assumed its present condition, which has not since varied. His pupils are now normal. The knee jerk is still present on both sides; the erect position is easily maintained when both eyes are closed; the gastric trouble and pain are both less in amount. Swelling of the foot has recurred, and a red papular rash lately erupted during the patient's residence in the hospital.

Dr. Dyce Duckworth congratulated Mr. Baker on being one of the few surgeons who had formed the opinion that there was something more to be studied in cases of so-called Charcot's disease than the characters of ordinary chronic rheumatic

arthritis. He thought it was a fitting task for the Clinical Society to undertake to clear the ground for a comprehensive discussion of the whole subject involved in Mr. Baker's communication. Although M. Charcot's position was now completely accepted in France, and perhaps in Germany, there remained sceptics in this country who refused to see anything specific and very remarkable about these cases of joint disease. It seemed at the outset very unlikely that M. Charcot, who had himself so well and minutely studied the features of chronic rheumatic arthritis, and taught the profession so lucidly on the subject twenty years since, should be mistaken in adopting a different view of the malady now under consideration. His exceptional opportunities at the Salpêtrière afford him the largest scope for a study of the whole matter. First, with respect to the specimens brought forward, it was undoubtedly true that many of them were quite indistinguishable from joints affected with chronic rheumatic arthritis. But it was also true that certain characters pertained to the bones, to the form and degree of the arthritis in these cases, which were never met with in the rheumatic disease. Now, it was to the existence of these peculiarities and the clinical features associated with them that heed must be paid in discussing this question. The essential point of difference in the morbid specimens was the extreme wasting and wearing away of the bones. Attempts at repair and bony outgrowths were met with, but in many cases the atrophic changes predominated. Putting aside this feature, it was difficult, if not impossible, after death to tell what form of arthritis had led up to the appearances, because both rheumatism and gout will disturb both cartilaginous and bony nutrition. The clinical features related especially to somewhat sudden and rioteus inflammatory action, more injury being suffered in a joint in a period of three weeks than would accrue in thirty years of chronic rheumatism. Surely there was something specific and very significant in this. It was difficult to believe that the disorder was a new one. He believed that many museums contained specimens of so-called Charcot's disease labelled as those of ordinary chronic rheumatic arthritis. Still it was remarkable that no museums had hitherto contained specimens of the wasted, porous, and worn-down bones which were so characteristic and were now being everywhere collected. He had hitherto believed in the special features of this disease, and accepted M. Charcot's teaching as to its neurotrophic origin. We were now bidden to regard all these joint affections as dependent on one common

or general cause. If such should be the opinion ultimately arrived at, he, for one, should feel gratified, since he had come to believe that not only the disorder under consideration, but also rheumatic and gouty arthritis, were largely the result of neurosis. Let it be remembered, however, that M. Charcot had distinctly declared that in cases of locomotor ataxia, chronic rheumatic arthritis may supervene and present none but its ordinary characters.

Dr. Hale White said he thought the hypothesis that Charcot's disease was an arthropathy peculiar to locomotor ataxy was hardly tenable, for many English observers of large experience had no knowledge of the train of symptoms described by Charcot; thus whilst he (Charcot) found them in five out of fifty patients examined, Dr. Moxon had never seen them in thirty cases he observed. It was pointed out that there was not a single symptom in Charcot's description of his arthropathy that was not also a symptom of ordinary arthritis. Dr. Hale White had seen six cases of joint lesion in locomotor ataxy, all of which were considered to be examples of Charcot's disease by some one or another who had seen them, but not one of them was universally allowed to be an example of the disease; on the other hand, he had never seen a case which was universally allowed to be rheumatoid arthritis in an ataxic patient. He also believed that an examination of the specimens after death would show that there were no real points of distinction between Charcot's disease and other forms of arthritis; certainly this seemed true of the specimens existing in England. Thus there is one in the College of Surgeons' Museum which is said in the catalogue to present just the features of Charcot's joint disease, but still to remain one of general rheumatism. There was the possibility that some of the cases of Charcot's disease might be due to nervous lesion, but then they would belong to the same class as those diseases of joints sometimes produced after lesions of nerves, diseases of the cord and hemiplegia, and would not be peculiar to locomotor ataxy; probably, however, the majority of cases of Charcot's disease were examples of rheumatoid arthritis occurring in patients with locomotor ataxy; or in those patients in whom the arthritis came on late in the course of the ataxy it might be due to injury from flinging the legs about or due to the prolonged inactivity of the limbs in bed. Putting these two causes aside, however, the connecting links, both clinically and pathologically, between rheumatoid arthritis and Charcot's disease

are so numerous that in the present state of our knowledge it seems rash to think that the latter disease is a distinct one peculiar to locomotor ataxy. Even if there be found cases which cannot be included either in rheumatoid arthritis, the traumatic, or the prolonged rest group, it is probable that they belong to the same group as other nervous arthropathies.

Dr. BUZZARD concurred in the remarks made by Dr. Duckworth, and was strongly of opinion, with the latter, that Charcot's joint disease was not a new disease, but that our recognition of it was new, and arose out of the refinements introduced into methods of examination during the last few years. No absolute proof of this could be adduced, but at least there was evidence that tabes itself was not a new disease. He had found in Dr. Graves's classical work on clinical medicine an account of a patient affected with typical gastric crises dating from the year 1823. The case was evidently one of tabes. It ought to be remembered also that, as compared with the state of things existing at the time named, the profession of to-day enjoyed multifold opportunities of observation. Not only had an enormous increase of population occurred, but until within the past fifty years or less there was no system of seeing out-patients at our hospitals, so that it was not strange that we should come across many more cases in proportion than would have been met with many years ago. It should be remembered that the disease under discussion is a disease of bone. The question of the connection of joint disease with tabes was introduced by Charcot in 1868, and five years later he brought forward the case of a woman with several joints typically affected and some of her long bones spontaneously fractured. Charcot deduced from that and similar examples that the osseous tissue was primordially involved in these cases, the joint disease being a secondary affection. Lionville investigated the subject by comparing a case of arthropathy, associated with tabes dorsalis, with a case of spontaneous fracture of the bones occurring in another patient in the same disease. He found that the same changes obtained in each instance. There was enormous dilatation of the Haversian canals, the osseous substance being thinned and eroded, a condition of what was called rarefying osteitis. Blancard found that the lesion started by a disappearance of calcareous salts, the erosion of the Haversian system being a secondary feature. Chemical analysis, according to Regnard, shows that in the osseous affection of tabes we have to do with a true trophic lesion of

bone, a fatty degeneration with disappearance of the mineral substance. The phosphates he found, in a certain case, reduced from 48 parts to 11 in the 100, and the proportion of fat had risen from 5 to 37 parts in 100. In these circumstances Dr. Buzzard suggested that surgeons, when the opportunity occurred, should measure the specific gravity of the bones entering into the composition of the joints affected both in tabes and in rheumatoid arthritis, when probably a considerable contrast would be observed.* In Dr. Adams's classical work on rheumatoid arthritis he found no mention made of such a chemical alteration of bone as that described, nor did he find any record of spontaneous fracture of a long bone in any of the cases detailed in that work. It was to the surgeons that cases of joint disease naturally went, and that was the reason why although he (Dr. Buzzard) had probably seen two or three hundred cases of tabes yet he could count upon the fingers of his two hands the number of patients with joint disease who had consulted him amongst the sufferers from tabes. Referring to Dr. Hale White's difficulty as regards the contrasted experience of Dr. Moxon and Dr. Charcot, he reminded that gentleman that Dr. Charcot's opportunities at the Salpêtrière were very different from those of a physician to a London hospital. His field was a vast one, and included patients such as are found in our workhouse infirmaries, and who remained for years under observation. On the other hand, in our hospitals a process of sifting took place ere admission to the wards, and patients presenting such visible lesions as would be suggested by enlargement of a joint would be naturally referred to a surgical member of the staff. A large number of persons—all sensible people, in fact—would be anxious to consult his friend, Dr. Moxon, on account of almost any departure from health, but probably the last thing about which they would think of seeking his opinion would be the sudden occurrence of an enormous and painless swelling of a joint! Having had to do for many years with numerous cases of nervous disease in the out-patients as well as in the in-patients of a hospital devoted to diseases of the nervous system, it was not surprising that he (Dr. Buzzard) should have met with several examples of associated joint disease, yet in every instance in which the joint affection presented the peculiar characters of that which was under discussion, the disease was invariably tabes dorsalis. He had never seen it in association with cases of hemiplegia, paraplegia, epilepsy, lateral or dissemi-

* It will be necessary to compare fresh wet preparations.

nated sclerosis, or other of the numerous forms presented by disease of the nervous system. This was surely a sufficient answer to those who thought that there was but a fortuitous connection between tabes and the joint affection, and who looked upon it as the accidental occurrence of rheumatoid arthritis in a patient affected with tabes. On the other hand, cases of typical rheumatoid arthritis might be watched for years and no development of symptoms of tabes would be seen to occur. At least, he had never seen or heard of such a case, although this subject had engaged his attention for the last eleven years. Now, if we had to do here with a simple alliance of rheumatoid arthritis with tabes this particular chronological sequence would surely not *always* obtain. We should at least *sometimes* find the symptoms of rheumatoid arthritis occurring antecedently to those of tabes. He had investigated a large number of typical cases of rheumatoid arthritis in our workhouse infirmaries, and except where the mechanical conditions interfered with the test he had found the knee phenomenon always present, the pupillary condition normal, and an utter absence of other symptoms pointing to tabes. Charcot's joint disease contrasted strongly in its clinical aspects with rheumatoid arthritis. Without going further into detail he believed the disease to be due to a trophic change in the osseous tissues incident upon a lesion in the medulla oblongata. Further observation had tended to confirm this view, which he had submitted to the Pathological Society in February, 1880. He thought that in the bulb there was something in the nature of a centre concerned in the nutrition of the osseous skeleton.

Mr. Barwell felt himself unable to follow Mr. Baker in his excellent paper concerning the neuropathy, the neural origin, of joint disease. It was a question of which but little was known, and, as all the members were well aware, many of the multiarticular forms of joint disease had been ascribed to neuropathy, such as acute rheumatism, and many forms of chronic rheumatism, some even of gonorrhœal rheumatism, &c. Mr. Barwell felt that he could not agree with Mr. Baker in this further than to say that there was great evidence to show that arthritis deformans originated in some nervous pathological injury or lesion. But was it, therefore, necessary to suppose that this nervous injury should be the same as the nervous injury in locomotor ataxy? He ventured to think that, though these two joint diseases, which he believed to be separate, might both be due to neuropathy, they nevertheless

were not the same, and were not due to the same neuropathic affection. The subject might well be divided into two, namely, the anatomical appearances and the clinical phenomena. The anatomical appearances were really more different than appeared at first sight. Taking an elbow-joint (ataxic), in which the form of the elbow was pretty well lost, it was seen that one bone, the radius, had its head and its neck almost entirely worn away. Very much the same thing was seen with regard to the ulna, and to the internal condyle of the humerus. The repair which had taken place was merely by a very slight ossification of one of the ligaments, and was not the same sort of thing as was found constantly in such specimens of chronic rheumatic arthritis as the speaker had brought for exhibition. If, again, this same joint were examined, it looked very unlike a joint affected with chronic rheumatic arthritis. The bone was affected almost half way down its length, and large pieces thrown out; it reminded Mr. Barwell very much of a case which Dr. Buzzard described some time ago, but occurring in the hip. On close inspection there would be seen none of the porcellanous deposit characteristic of arthritis deformans, as described some years ago by Mr. Quekett, an absolute deposit into the material of the bone, like the wax with which a French polisher filled up porous wood before giving it the final polish. On yet closer inspection, a great many of these tabetic bones were found to be filled up by an almost tufa-like growth, which was not such as in rheumatic arthritis. Mr. Barwell showed that in typical arthritis deformans, hyperplasia, especially marginal hyperplasia, very much exceeded any wearing away. The bone was evidently much more solid and much denser than in any such specimens of tabetic disease as were exhibited that evening. In arthritis, there were polished surfaces of a very considerable extent raised above the level of the other parts of the joint, while the marginal hyperplasia had gone on to such an extent as to embed and almost involve the whole of the neck; the wearing away being, on the contrary, very slightly marked indeed, such as had never been found in the same condition in the tabetic joint disease. Mr. Barwell showed a cast which was another example of marginal hyperplasia, nearly always so strongly marked in chronic rheumatic arthritis. The enlargement of the head of that radius was unlike what was seen in locomotor ataxy. Enlargements of bone were almost constant in that disease, but such conditions of hyperplasia were never seen as in this cast. Then, in these

specimens (rheumatic arthritis), the bone was perforated, like a wormeaten piece of mahogany, by large holes, evidently the wearing away of the Haversian systems; what he ventured to call interstitial atrophy, in contradistinction to the marginal hyperplasia which was also found in the arthritic specimens which he exhibited. Mr. Barwell was not aware that porcellanous deposits and very highly polished surfaces ever occurred in locomotor ataxy; but rough surfaces were of common occurrence. Then, again, in locomotor ataxy, false bodies were not common. In arthritis deformans, false bodies were not only common but the rule, and they occurred in very large numbers. Mr. Barwell did not think that the filling of a joint with dendritic synovial outgrowths ever took place in locomotor ataxy. He was rather surprised to hear Mr. Baker speak of fibrous degeneration of cartilages occurring in locomotor ataxy. The fibrous change of cartilage in rheumatic arthritis was very peculiar; and Mr. Barwell had never seen it in any other disease. It might possibly occur in locomotor ataxy, but must be proved to do so by a careful microscopic examination. Mr. Barwell thought that the clinical differences were very great indeed. As a rule, the arthritic troubles of ataxy commenced in a perfectly painless manner. The whole limb one morning was found swollen; and after a little time, a day or two, this swelling subsided, leaving only a loosened joint. Arthritis deformans never commenced thus, but by pain, followed by gradual hydrarthrotic swelling of the joint, not of the whole limb. In locomotor ataxy, this hydrarthrosis frequently returned and remained very long. In a number of the patients then present, the joints were full of fluid, and there were various forms of displacement of bone; but in arthritis deformans the hydrarthrosis was a comparatively short and painful stage; then, when the fluid was absorbed, the grating and the immobility appeared again. In all the cases of ataxy which Mr. Barwell had seen, excessive normal and false movement in the joint predominated greatly over the limitation of movement. It was precisely the contrary in arthritis deformans; there might be some false movement, but that was generally pretty limited, and the natural movement was very limited. Then, in ataxy, as he had said, the first attack, as well as the rest of the malady, was usually painless, even when deformity was extreme. In arthritis deformans, pain was usually the commencement, and was continuous. After the patient had taken exercise, on getting up in the morning, the pain might diminish, or perhaps dis-

appear; but, throughout the disease, the first movements of the joints, after rest, were always painful, especially certain movements, as of rotation of the thigh outwards, or abduction. That appeared never to be the case in the ataxic disease. Tabetic conditions never appeared to affect small joints, but always large ones, like the knee. Mr. Barwell hardly thought he could find it in a smaller joint than the elbow; but arthritis deformans was especially liable to affect small joints, such as the fingers, as well as large ones. Whenever several large joints are affected by arthritis, the small ones never remain unaffected. Even though not nodose, the fingers are peculiarly adducted in a manner never seen in ataxy; they exhibit that peculiar slope towards the ulnar side which was always found in multiarticular arthritis deformans. Even though the patient himself might be unconscious of any trouble about the hands, the phalanges would be seen to slope away towards the ulnar side, and the toes towards the fibular side; they sloped considerably at the metacarpo-phalangeal joints.

Sir JAMES PAGET was disposed to say that he agreed with Mr. Baker very nearly, though not perfectly, on almost all the points upon which he had spoken. He would avail himself of Mr. Baker's suggestions of points for discussion. The first was, whether Charcot's disease might be regarded as a new malady. In speaking of a new disease the great difficulty lay in the fact that one had to establish, not a positive conclusion, but a negative one; for, when proving that a thing was new, the real thing to prove was that it was not old, and to do that was a much greater difficulty in regard to most diseases than at first sight appeared. Sir James Paget knew that enormous tracts of knowledge had, in past times, been overlooked. A man could not study his own career, or observe, as science made progress, the number of things that had been constantly within his sight, of which he remained totally unconscious, without feeling sure that many minds must have been in somewhat the same condition, and that it was hardly possible to reckon the full extent of the facts which were overlooked. Yet it seemed nearly sure that the older observers of cases of diseases of bones and joints had really not seen such conditions as had now become familiar in the changes of joints in locomotor ataxy. It had been said that these were cases that presented themselves chiefly in the out-patient room and to surgeons at workhouses. But John Hunter collected specimens

by the hundred in workhouses. That great collector of specimens, George Langstaff, was a Poor-Law medical officer, and the greater part of his practice was in workhouses. Cruveilhier's great pathological work was in the Salpêtrière, and among exactly the same class of patients; and, to speak of but one more, closely within Sir James Paget's own knowledge, there was scarcely a workhouse in London which did not once supply Mr. Stanley with every specimen of diseased bone of any apparent value that could be found in them. Now, this extent of work began more than a century ago, and ended about forty years ago. But these sixty or eighty years of work in collecting diseased bones had, he believed, left nothing in any book or museum to show that any of these watchers ever found such a specimen as was now a well-known thing in all museums. If any one of the poorhouse patients, who were now in the next room, had existed in a workhouse in London during any part of the thirty or forty years in question, they would certainly have been dissected, and their bones preserved. Thus, considering the fact that, in the collections of those great collectors, there did not now remain a single specimen characteristic of Charcot's disease, there was as nearly as possible sufficient evidence of the negative that the disease did not exist, at least in anything approaching its present frequency. Sir James Paget had not only had much experience himself in museums, but had sought information from others; yet he could not find a specimen of old date in London. So, then, Mr. Baker's first question might be answered: Yes. In general terms this was a new disease; in more especial terms it was a new compound of diseases. In this belief the chief direction of inquiry must be followed. In all the discussions that Sir James Paget had heard the question had been asked: "Is this a neurosis? Is it rheumatic arthritis? Is it this or that?" He thought that it was neither this nor that, but rather some of these or some of those—a disease made up of several different constituents, and appearing, therefore, only at a time when, we knew not by what external conditions, all these constituents of disease met in the same person. Sir James Paget was conscious that in speaking of this he was referring to subjects which were altogether obscure, and very uncertain; but if an apology were needed, he would say that we were talking of "Charcot's" disease, and he thought it would be in the observation of all that, so long as a disease was called by the name of any person, or any nation, it might be concluded that its pathology was very imperfectly ascertained. The old

terms morbus Anglicus and morbus Gallicus had been left off, now that something was known about rickets and syphilis. The term Bright's disease was fast vanishing as the pathology of the disease was better known. Thus, the very name of the disease under consideration implied that its pathology was altogether obscure, and he held that it was really to be studied not by endeavouring exactly to define what was its type, or whether it was to be called by this name or by that, but by trying to ascertain of what diseases, and in what proportion of each, it was compounded. And there was a fair illustration of such compound diseases in what surgeons had consented, apparently, to call chronic rheumatic arthritis. There was no much better practical name for it than rheumatic gout, and, speaking generally, there might fairly be an impression that there was in one person more or less of gout, and in another more or less of rheumatism, and that in some way or other there came to be a compound of these two things. Thus whether "Charcot's" disease should be called rheumatic gout, or be regarded as an example of rheumatic arthritis, might be answered by holding that it was a method of rheumatic arthritis, altered from its ordinary fashion by the intervention of the locomotor ataxy. What Mr. Baker had said of the general characteristics of locomotor ataxy was quite enough to explain the contrasts between the ordinary typical cases of rheumatic arthritis, such as Mr. Barwell introduced, and the mere wasting of a part, such as would follow deficient nervous nutrition. Mr. Baker was doubtless right in saying that the character of the disease, as distinguished from rheumatic arthritis, was, that it was wasting without repair. The characteristic of those specimens which Mr. Barwell had produced was, that they showed disease with wasting, but with coincident attempts at the reparative process. But though rheumatic arthritis and the joint-disease of locomotor ataxy could be broadly distinguished from one another, it was not fair to assume that there was in locomotor ataxy no measure whatever of rheumatic arthritis. It might well be a rheumatic arthritis modified by its coincidence with a disease of the spinal marrow, which hindered the ordinary, however ill directed, processes of repair found in the ordinary rheumatic arthritis. The general feature of the most marked cases of locomotor ataxy was wasting; but all the cases of partial wasting, with some new production of bone about the articular borders, brought it so near to the characteristics of some of the cases of rheumatic arthritis, that one could not doubt that there was a

certain relation between the two. And when it was said that the ordinary course of the diseases was very different, it could be answered that this belief was founded only on having observed the characters of each in typical examples. Sir James Paget would agree with what Mr. Barwell had said, as to the general progress of an ordinary case of rheumatic arthritis; but it was no very rare thing to see a person with his knee-joint distended, and the tibia displaced and worn away and gone altogether from its natural holding, in that which might be fairly called rheumatic arthritis, although there were no locomotor ataxy connected with it. If one took, on the one side, the whole range of joint-affections in locomotor ataxy, and, on the other, the whole range of joint-affections in rheumatic arthritis, it was impossible not to see that cases could be found in which it would be hard to say, when one looked at the pathological specimens, to which class of cases each belonged. And, as they thus ran into the border, and became confused by the extreme cases on each side, it might justly be suspected that the diseased joints in locomotor ataxy were really examples of chronic rheumatic arthritis so-called, occurring in persons with a special tendency to disease of the spinal marrow. Another element, syphilis, might be suspected in many cases; but Sir James Paget had not found a distinct reference to an examination on this point in many of the cases which had been published. If the existence of three such diseases together could be assumed, a very wide range of characteristics might no doubt be explained. Syphilis was just one of those diseases in which one could most distinctly discern the various complications into which it might enter. In ordinary practice, at least, it could be clearly seen that syphilis implanted in a person with distinctly gouty inheritance was, to all external appearance in its tertiary state, a very different thing from syphilis implanted in a person of tubercular or scrofulous tendency. It pursued two courses, which were as unlike as almost any two so-called typical diseases could produce. There was here no difficulty in studying the combination of diseases; it was only by a fair extension of the same method of inquiry that it might justly be believed that three or four or more things might enter together into the composition of such a disease as was in locomotor ataxy. It seemed vain to hope that any of these diseases should ever be reduced to one typical form, so that one should be able to say it was this or that. The younger men whom Sir James Paget saw round him had rather to study their pathology after the manner, if

possible, of minute analysis, to determine the several constituents of each case and the proportion of each constituent in the general composition of each. Instead of trying to reduce pathology into a system similar to that of natural history, in which one could use Latin and Greek words to express exactly what one meant, one should rather study it after the fashion of organic chemistry, and find, if possible, what were the constituents of which each disease was composed, and even in what proportion each disease, or each morbid condition, entered into the composition of that of which the whole was seen.

Dr. Ord had seen cases of the disease under the care of other physicians; he had never had a case of his own under constant observation, and so, with regard to the relation of the joint affection now being dealt with to tabes dorsalis, or locomotor ataxy, he would say very little. Of course, he had had many opportunities of seeing the preparations of the bones and joints; and when he considered Mr. Baker's statement that there was a general agreement between the morbid anatomy of Charcot's disease of joints and chronic rheumatoid arthritis, although he agreed with him in his general conclusions very thoroughly, he was compelled to recognise more than Mr. Baker had recognised in the matter of waste. Looking at the various specimens exhibited that night, and looking, before this, with greater leisure at others, in some of the specimens exhibited, Dr. Ord recognised what Dr. Buzzard had pointed out, the actual wasting of the bone substance, generally quite independently of its wearing down at the points of weakness. And, on the other hand, it did not seem accurate to state that there was no attempt at repair. In very few, if any, cases were the outgrowths that Mr. Barwell had very eloquently described totally absent; and, in some cases, certainly, he had seen something like eburnation; in many cases, he had found an excessive development of osteophytes. There was a specimen in the other room, exhibited by Professor Humphry, which presented these in an excessive degree. Dr. Ord, after many years' deliberation, was inclined to place this among the conditions which were included under the head of what Sir James Paget had been speaking of, chronic rheumatic arthritis. Sir James Paget's first remarks on this subject implied that this disease, as being called rheumatic gout, included both rheumatism and gout; and this was, to a certain extent, true. His later remarks brought out what was the real

outcome of such a consideration as engaged the Society to-night; that was, the absolute necessity of analysing fully and completely all the individual cases that might be included under presumably common heads; not to deal here, as in comparative anatomy, with typical cases, but to qualify, as one had to deal with individual men, the idea of the typical case by its application to the particular person, to the particular exponent with whom one was dealing. In the case of rheumatic arthritis—a term which Dr. Ord did not think a very good one, the more neutral term, osteo-arthritis, appearing preferable, as it fettered the mind less if one considered the whole question of osteo-arthritis—one could recognise lesions having the same essential details, or at all events what appeared to be the same essential details, as those which were being discussed. Lesions consisting in waste of cartilage, waste of bone, and, at the same time, in outgrowth around the articulation, were to be seen in relation to gout, were to be seen in relation to chronic rheumatism, and, again, were to be seen in a number of cases in which the presence neither of gout nor of rheumatism, as it was at present understood, could be recognised. In those cases one sometimes called in the explanation of blood-poisoning, and in others a neurotic explanation. Dr. Ord then described three cases (women) under his care, at St. Thomas's Hospital, the general lesions of which were, to all appearances, those of chronic osteo-arthritis. In the first, a young married woman, of 34, there were irregular lesions of the joints of the fingers, with well-marked wasting, and outgrowths of many joints, but complete ankylosis of both wrists, and of one elbow, and partial ankylosis of one hip. There was no trace of gout that one could make out. In another case, with much less marked joint lesions, and with no trace of nervous disorder at all, the woman had a very well-marked nodosity of the joints, an affection chiefly of the wrists and hands, without any ankylosis, some looseness of the joints and that well-marked deviation of the digits to the ulnar side to which Mr. Barwell had very properly directed attention. In this case the woman, having no nervous affection, gave a history of nine attacks of acute rheumatism, in each of which the joints were affected. That the disease had been probably acute rheumatism was evident, from her having had a slight attack since she had been under Dr. Ord's observation, and from her having extensive affection of the valves of the heart. In the third case the woman had, in its typical form, such a lesion as Mr. Barwell had described, enormous thickening of

the joints, looseness of the joints, deviation to the ulnar side of the fingers, in fact, all that belonged to a subluxation. With this, although the woman was generally in her whole body well nourished, there were great wasting of the skin of the fingers, a glossy or satiny condition of the skin, particularly towards the ends of the digits, wasting of the nails, wasting of the muscles, quite in excess, as far as one could see, of the disease; and with this the presence of those interesting phenomena, subcutaneous nodules. These were three very different cases, and yet they were all such as would be included roughly under the head of osteo-arthritis, or rheumatic arthritis. For some years Dr. Ord had urged the necessity of analysing this very difficult and obscure change in joints; he was very grateful to Mr. Baker that he should have brought the question forward in a way which enabled him once more to emphasise these observations. He did not wish to push forward the idea of a neurotic process as being necessarily present in all cases of rheumatoid arthritis; but he did recognise that, in the cases before the meeting, and in many others, there was strong evidence of a neurotic influence, direct or reflex, being the main, if not the actual, agent. At the same time Dr. Ord did not forget the many other causes that might give rise to chronic osteo-arthritis more or less related to that which was under discussion.

Professor Humphry agreed with the remarks which fell from Sir James Paget, that this was a disease with which surgeons were not familiar in olden time. Till lately he had never seen such rockings of joints and such deformities of articular surfaces taking place with the rapidity now observed, and we did not formerly find these specimens so marked in museums as we now do. He also quite agreed with Sir James Paget in the view of its being a compound affection between the disease called rheumatic arthritis and tabes dorsalis; that it was a combination of the wearing away with the nervous affection. In the old affections, however, it was rather a wearing away, a rubbing and wearing away, proceeding altogether from the articular surfaces, and a resultant, as it always appeared to him, of a low inflammatory affection occurring in the synovial membrane, altering the synovial fluid and causing a slow change in the articular cartilages, and a subsequent slow change in the osseous surfaces, associated with a certain amount of osteophytic growth from the marginal parts, as described by Mr. Barwell. In this present affection there

was a rapid decay, as it were, a rapid wasting, a rapid removal of the bone; and in the specimen which had been alluded to by Dr. Ord, which Professor Humphry had brought from Cambridge, this was most marked. In this specimen, in which tabes dorsalis in a woman had been going on only about three months, the whole of the head of the tibia was absolutely gone, and the rough upper end of the shaft was exposed and lying in contact with the articular surface of the femur. And not only so, but the cartilaginous surfaces of the tibia were in part preserved, and had been broken away from their surroundings, and one of them was applied quite against the outer surface of the femur; so that there had not here been that usual wearing away commencing with the articular surface, but a decaying of the bone beneath the articular surfaces, the bone giving way and allowing the articular surfaces to be applied against the side of the femur, in this respect differing considerably from the familiar disease called rheumatic arthritis. Professor Humphry did not quite agree with Sir James Paget and Mr. Baker in the view that there was an entire absence of reparative process. In some instances, as in the specimen he produced, there was a very marked and large amount of what was called reparative process, that is to say, a very large amount of osteophytic growth—a very large amount of new bone-formation taking place in the immediate neighbourhood of very large bone-destruction. This was very curious, and very remarkable. The only thing which he could at all compare with it in that respect was the intracapsular fracture of the neck of the thigh-bone, where there was, in many instances, rapid removal of the neck of the femur between the fracture and the trochanters, associated with very considerable outgrowth at the base of the trochanters. In that instance, too, rapid bone-absorption was associated with rapid bone-formation. The speaker had recently made a good many observations about old people, and had published, not long ago, a short paper on the quick repair of bones in old persons; and he remarked here that although bone-absorption from atrophic condition was taking place throughout the skeleton, yet the work of bone-repair was capable of being rapidly called into existence. These examples of the association of atrophic bone-absorption with abundant new bone-formation, and others of a like kind might be adduced, were of interest in connection with the specimen he showed, in which the effects of the two processes were so marked.

Mr. HUTCHINSON said that he had taken much interest in this subject from the time that Professor Charcot first published his views, and had made several visits to Paris to see his cases. In a course of lectures on the connection between the nervous system and lesions of nutrition delivered at the College of Surgeons about seven years ago, although we were indebted to Dr. Buzzard for the first cases observed in England, he had, he believed, been himself the first to discuss the subject in detail and to illustrate it by producing a collection of specimens. The views which he should express to-night were much the same as those which he brought forward in the lectures referred to. In the first instance he had been so struck with the peculiarities of some well-marked cases that he had been inclined to accept Charcot's view that the conditions were almost *sui generis* and were in essential connection with ataxy. He had at the same time been inclined to admit that their occurrence did illustrate and prove the direct trophic influence of the nervous system. The more, however, he worked at the subject the further he had got from this opinion, and the conclusions which he finally expressed in his lectures were by no means exactly those with which he had commenced their preparation. He had come to believe that many causes might contribute to the production of the peculiar condition in question, such, for instance, as injuries, rheumatism, and gout, but above all the senile state, whether general or local. The changes of ataxy might in some sense be said to produce a sort of tumultuous and irregular senility in which some parts of the body got old before the others. The loss of normal reflexes was a condition common both to old age and the ataxic state, and on this disturbance of reflex sensibility and partial abolition of the sense of pain, many peculiarities in modes of nutrition and disease might be held indirectly to depend. It was a noteworthy fact that if ever we found the conditions of the ataxic joint simulated in a patient not the subject of ataxia it was almost always in association with senile rheumatism. In this relation he had been much interested in the observations just made by Prof. Humphry. The contracted pupils, the weak bladder, the sluggish bowels and dulness of general sensation, but especially of pain, were all features in which the ataxic and the senile state approach each other. In both there was the liability at once to almost painless inflammatory changes, resulting in atrophy and outgrowth at the same time. It would not be asserted that senile rheumatism, as seen in the morbus coxæ senilis, was always painless, but all must have been struck by

the observation with what extensive local changes, such as absorption of the head of the bone, and growth of osteophytes, tolerably free use of the limb was still compatible. In no other diseased states did persons continue to use their joints freely after the cartilages had been removed than in senile rheumatism or locomotor ataxy. His impression was that the question might be much simplified by bearing rather strongly upon the line of argument which he had just suggested. In the ataxic patient many of the special sensations were dull, extremely dull, and hence the attacks of painless retention of urine which were so characteristic of the disease. The bladder might be distended until it reached the umbilicus, and the patient yet avow that, excepting a little feeling of fulness, he had no discomfort. The distress which attends retention of urine in the healthy is wholly absent in the ataxic. Then again we have the perforating ulcer of the foot with its very remarkable features, all of them, he believed, due to the obtunded sensation in the sole of the foot. This ulcer, common to ataxy and to leprosy, is in both diseases a "pressure sore." The patient, unable to feel well in the skin of his sole, is apt to stand too long, and to make injurious pressure on one particular part. The success which attends treatment by appliances which exempt the sole from pressure fully prove this assertion. There is no need whatever for assuming any trophic influence of the nervous system in this instance, although very probably the loss of perfect reflex functions may by its influence on dilatation and contraction of blood-vessels much modify the process of inflammation set going by undue pressure. In applying this suggestion to the explanation of the phenomena of the joint changes in ataxia, he would fully admit that no abrupt line of demarcation could be drawn between them and those met with in some cases of rheumatic arthritis; especially in the early stages of the two diseases it was impossible to say whether the changes were likely to advance in the special directions of the ataxic joint or not. Admitting, however, that there were many cases which it was impossible definitely to diagnose, he still joined with what Mr. Barwell had just asserted, and with the original proposition of Charcot, that there were many cases in which the final results were characteristic. Certainly he had never witnessed in connection with rheumatic arthritis any approach to the extreme cases which he had now seen in ataxy,—cases in which a patient might have one, two, or more joints so much disorganised that their bones might be pushed out of place

and pulled back again almost at will, and without giving the patient much pain or inconvenience. The changes in rheumatic arthritis, however great, were usually of a nature to prevent dislocation, whilst in ataxy they often favoured it. In no other disease than in ataxy had he ever seen any approach to what is witnessed in the typical examples of joint disorganization in that malady. Mr. Baker had suggested, and Sir James Paget had seemed to support the suggestion, that the ataxic joint was an example of rheumatoid arthritis assuming peculiarities from the fact that it occurred in an ataxic patient. To some extent, and in some cases, he (Mr. Hutchinson) was quite inclined to agree with them, but if the proposition were made universal then he must with diffidence say that he could not accept it. He was not at all prepared to admit that all those that became the subjects of ataxic arthropathy were also the subjects of the rheumatic-gout diathesis, whether from inheritance or otherwise. The clinical evidence on this point would, he felt sure, fail in a certain proportion of cases. So deficient, indeed, would it appear to have been found by some observers that the assertion has gained credence that the subjects of ataxic changes are never the subjects of rheumatism. Charcot himself, and other observers, have described rheumatoid arthritis occurring in ataxic subjects and still keeping its own features. He (Mr. Hutchinson) well remembered a case in which a patient who had for some time been known to be the subject of ataxy, came under his care with effusion into one knee-joint. He was put to bed in the hospital and treated as if for rheumatism, and his joint got perfectly well in a comparatively short time. Such a fact did not, to his mind, prove that ataxic arthritis and rheumatoid arthritis were wholly different, but that arthritis in an ataxic subject, if treated by rest, might pass away. It affirmed the proposition for which he had been contending, that the peculiarities of the affection in advanced cases were due to the fact that the patient did not experience pain from motion, and consequently did not rest. In some cases of ataxia it was quite true that the joint disorganization advanced even after the patient had been confined to bed; these were, however, exceptions, and they were so rare that he had himself no personal experience of them. As a rule it appeared certainly to be the fact that, in both rheumatoid and ataxic arthritis, it was the patients who used their joints most who got the most peculiar changes. The less the pain, the more the use, and the more the disorganization. Such, he said, was the general statement of his

creed. In rheumatic arthritis if a patient took to his bed joint changes of a certain kind would, it was quite true, occur. Ankylosis might happen, extensive erosion of cartilage might occur, but the tendency to osteophytes, loose bodies, alterations in the shape of the bone, and eburnation of the articular surfaces were very rarely seen except in those who continued to walk about. Thus then he would submit that after all, neither in rheumatism nor in ataxy did the state of the nervous system take a direct share in the production of the arthritis. In each it was probably true that the peculiarities assumed were due to nerve changes; that the share taken was passive or permissive rather than active or initiative. He found more difficulty, he must admit, in explaining brittle bones and spontaneous fracture in ataxy. But these after all were rare, much more rare than the joint affections. And they had their parallels in other cases in which the nutrition of the osseous system generally, and possibly of other tissues as well, became disturbed in connection with nerve disorder. It was not unlikely that the general failure in nerve power might exert some influence on assimilation and nutrition. Thus even for the brittle bones he was indisposed to call in aid so large an hypothesis as trophic nerves and of special centres. Before resorting to such speculations he preferred to see how far more simple explanations would go, and amongst these he felt sure that the obtunded sensation of the ataxic limb was one of the most important elements in causing peculiarities when its joints became inflamed.

The PRESIDENT observed that Sir James Paget said that he considered this Charcot's affection somewhat in the light of an arthritis associated with a definite disease of the spinal cord. It would be important for us to know whether by the expression "definite" he meant the pathological changes associated with tabes, as obviously Mr. Hutchinson meant, or whether he meant other pathological changes akin to them, or whether he meant, in the last place, pathological changes quite independent of these with which he associated tabes or any like affection.

Sir JAMES PAGET replied that he was not disposed to say that the changes that took place in the arthritic diseases of locomotor ataxy were dependent merely on what Mr. Hutchinson referred to as the passive condition of the nervous system, nor merely that they were such changes as would take place

in any person who had lost sensibility. He thought rather that the nervous system had in itself, by whatever morbid conditions might be found in it, a power of actually determining disease; not merely of permitting disease to go on unchecked when such disease arose from other causes, but that it had in itself a definite power of producing disease in this part or that. For example, taking a most typical instance of it, the nervous system showed a power of producing disease in what was called herpes zoster; there, after attacks of definite, and often exceedingly acute, neuralgia, an eruption occurred along the lines of the diseased nerve, and that neuralgia often continued, as was well known, after the eruption had ceased. But he would not refer the changes either in locomotor ataxy, or in any other disease to which a neurotic origin might be assigned, wholly to the condition of the nervous system. He thought that nervous disturbance could determine the occurrence of disease, but he did not believe that the method of a disease and its morbid changes could be determined by any condition of the nervous system alone. In the case of the herpes zoster, a disease was produced by an error of nervous force; but surely it could not be held that the nervous system had a determining power to make that disease a vesicular eruption, and not a scaly one, or not an ulcerative one. He would hold that there was in none of these instances a simple disease, or a morbid process determined solely by an error or deflection of nervous force, but in each case a disease determined by disorder of the nervous system, and having its method or manner of external appearance determined by other conditions. And so in cases of locomotor ataxy there were, he believed, many morbid conditions, rheumatic gout, syphilis, gout, rheumatism, scrofula—or any of these combined; but the occurrence of that disease was determined by the nervous system.

Mr. Hulke felt quite unable to agree with those who looked upon Charcot's affection of the joint as a distinct and separate thing from that affection which had been known for a very great number of years past either as malum senile or arthritis, rheumatoid arthritis, or arthritis deformans. In using the term rheumatoid arthritis, he did not wish to tie himself down to be understood to mean that he thought it was necessarily the expression of rheumatic diathesis. He simply used it to label, to describe, a particular joint affection more or less familiar to all. In considering the essential distinc-

tions or identity of these two affections, neither in their morbid anatomy nor in their clinical history, taking a large number of cases, could Mr. Hulke see any essential difference; and he might point, perhaps, to one of the specimens which Mr. Barwell first of all exhibited—the bones of the arm. The humerus was excessively wasted, as could be seen in a large number of instances which might be considered typical of Charcot's joint disease. Most persons would agree that this was not at all a bad specimen of Charcot's joint disease. There was considerable wasting of the articular end of that bone, considerable bony new growth in relation to the ligaments and the synovial structures. But Mr. Hulke showed the knee of the same individual. There was no great wasting of the ends of the bones; on the contrary, there were those expansions, those sorts of stalactitic masses and outgrowths round the margins of the articular surfaces which many would consider to be eminently typical of chronic rheumatic arthritis, so that in the same individual there was in the upper limb, as was alluded to at the former meeting, a condition which would be agreed to by most to be typical of the morbid anatomy of Charcot's malady, and here in the lower limb was another specimen typical of chronic rheumatic arthritis. Now, either it must be supposed that they were essentially the same disease with rather different expressions in the upper and lower limbs, from different circumstances to which he might presently allude, or that there were concurrent in the same individual two essentially distinct disorders. Mr. Barwell also stated that in Charcot's joint disease there was no eburnation. Now, here was a thigh-bone, or what remained of the thigh-bone, and a portion of the pelvis of the same individual that yielded this knee and the elbow-joint. There was certainly no neck of the femur. All that had gone, and a good piece of the upper portion of the inner surface of the shaft had gone. This man had the extreme misfortune to have fracture of the neck of the thigh-bone some time before his death. There was here an instance in which one would be puzzled to distinguish between the eburnated surfaces and this condition. It looked exceedingly like eburnation; so that there were in the same individual these specimens, some essentially those of Charcot's malady, some essentially those of chronic rheumatoid arthritis. Therefore Mr. Hulke was quite unable, not merely from the examination of these two, but from the examination of a large number of specimens, to satisfy himself that there was an essential difference in their

morbid anatomy. His own strong feeling was in favour of their being essentially the same; the morbid anatomy of the two seemed to present no essential differences. It had been said by Mr. Barwell that in Charcot's malady the lesser joints were not affected. There was a specimen sent by Professor Charcot himself, where almost every joint of the tarsus and of the phalanges and of the metatarsus was affected with this malady. Many looking at it would say that it was a typical example of rheumatoid arthritis. Not long since, he had, in the Middlesex Hospital, a Frenchwoman who had been an actress and a dancer in a theatre. She had had for a long time darting pains in her lower limbs, and darting pains in her back, and she had been unable to follow her occupation for some months in consequence of a gradual failure of her knees. She had considerable enlargement of all the joints of both the lower limbs, and particularly of the left, but with a stick she had been able to hobble about until a few days before coming into the hospital; then, while she was walking, she was suddenly seized (to use her own words) with excruciating pains in her left knee, immediately followed by swelling. She came into the hospital with as typical an attack of synovitis as one could wish to see. Thus, every now and then in Charcot's disease, cases occurred which were not perfectly sluggish, nor perfectly painless, so far as these clinical manifestations were concerned. There could not be a shadow of a doubt that a considerable number of tabetic cases occurred without any articular affections whatever. Again, in a large number of articular affections there was no tabes whatever. Then there was a third class where the two were concurrent; in such, he thought there would not be any difference in the morbid anatomy, nor did he believe that it would be found that there were in the clinical circumstances essential differences. He therefore looked upon the joint affections of Charcot's disease as a chronic rheumatoid arthritis. But then the nature of the association had to be considered. Mr. Baker asked, was this an accidental, or was it a causative connection, or did they both stand in relation to some third as a cause? Mr. Hulke's impression was in favour of the latter; he was particularly pleased with Sir James Paget's suggestion of syphilis, because that had been a long time passing through his mind. There could be no doubt that a very considerable proportion of tabetic patients were males who had had syphilis. He would not wish to state positively that he was convinced that [illegible] at the bottom of both; it might be, or it might

not; but he thought this was a hint which it would be well for all to follow up, and endeavour to elucidate as far as possible.

Dr. DYCE DUCKWORTH read abstracts of two letters he had received from Professor Charcot. M. Charcot regrets that his winter course of lectures prevents his coming to London at this time to take part in this debate. He desires to take part, however, *indirectly*, by sending some of his preparations and casts illustrating the disease in question. One of the most important of these is the preparation of a foot illustrating his *first* case of *pied tabétique*, and of which Mr. Page showed an example in a patient during the London Congress in 1881. Models illustrating this affection accompany the specimen. He sends also a pelvis from an ataxic patient with the upper extremities of the femora—specimens indicating very plainly, he thinks, that the disorder is not ordinary rheumatic arthritis, but a very different thing. The femora show the wearing away to be of mechanical nature. M. Charcot cares little what term is applied to the disease, or whether it be considered rheumatic arthritis, so long as it is acknowledged *to be truly a spinal one*, both clinically and nosographically. He believes that the disease is not met with, nor anything at all comparable to it, apart from cases of locomotor ataxia. He thinks that rheumatic arthritis may certainly supervene in the subjects of ataxia, but that is a different matter. *The condition of the bones is as remarkable as that of the joints in cases of arthropathy. The disease affecting the diaphysis as well as the epiphysis has nothing in common with that met with in rheumatic arthritis.* (The porosity and fragility are here referred to.—D. D.) He avows his surprise that in London the profession should still be in doubt about a question that in France is considered definitely resolved by anatomists, by surgeons, and by pathologists. He is very pleased, however, that the Clinical Society should again discuss the whole matter, not only for illustration of the pathology of tabes, but for the benefits that will accrue both to clinical surgery and the whole field of pathology.

The PRESIDENT said that Professor Charcot had contributed in a very large degree to the interest of the debate, and he suggested that someone should propose that the thanks of the Society be given to him. Although they were so deeply indebted to him, they were also indebted to Dr. Duckworth, who had put

himself immediately in communication with Professor Charcot, and used his influence with him to induce him to send his specimens, and to write the important letters which had been read.

Dr. Moxon proposed a vote of thanks to Professor Charcot, for whose powers of imagination and observation he expressed great admiration. It might be accepted as certain that a great fact had been put before the Society, evidently a very suggestive fact. But when he asked what it was that this fact suggested, he felt an uncomfortably indefinite state of mind. At the last meeting, the President had asked Sir James Paget to make more clear what it was that he meant. He (Dr. Moxon) felt a certain degree of disappointment at the answer which Sir James Paget gave. He was particularly sorry to find that he dragged in *herpes zoster*. He would be very thankful to any member who would explain in what possible way herpes zoster threw any light on this so-called Charcot's disease. Physicians know a little about herpes zoster. Only last week an old lady, capable of much suffering, both in mind and body, the wife of a clergyman, obtained his services with reference to a disorder which, she told him, had been diagnosed three days before, by her very competent medical man, as a combination of gout, neuralgia, and rheumatism; and she said that, after three days of agony, pimples appeared in the place where the pain had tormented her. Dr. Moxon did not know whether there had been any gout, or neuralgia, or rheumatism; but unquestionably the lady had herpes zoster of the neck when he saw her. He was able to promise her that her disease would run a definite course; that it might end, perhaps, in some painful after-effects; but that, even supposing ulcers should occur, there would be sound healing; and that it would never come again. He took it that those were the characters of herpes zoster, and he would like any member of the Society to show him what a disorder so characterised had to do with so-called Charcot's disease; for Charcot's disease commenced in a condition extremely like common chronic rheumatic inflammation of the joint affected, only that there was no pain in the joint, nor any nervous symptoms. Very little indeed that was definite had been put before the Society about this so-called new disease; but a good deal of what was definite came from Mr. Hulke, who showed that in the initial stages of this so-called peculiar disease of the joint there was nothing peculiar at all. It was only after at first simple rheumatism (or whatever it might be) had domiciled itself in the particular

joint that it began to show those peculiarities which made up Charcot's disease. He would ask everybody who believed in Charcot's disease to show him any parallel between herpes zoster, which was definite in its origin, exact in its course, complete in its termination, and this other disorder, which at first showed no definiteness at all, which was absolutely, hopelessly indefinite in its course, and which was peculiar amongst rheumatisms in having no nervous phenomena at all, whereas herpes zoster was full of nerve from the beginning. Such things failed absolutely to fit. In one line only did they touch each other—the line of indefiniteness—the line in which nothing was known about them. If, only on the line where ignorance was, these coincided, might it not be through ignorance that they coincided or appeared to coincide at all? Having so far spoken negatively, he would advance a positive contribution. Locomotor ataxy had branches; and it had a branch which was scarcely a grafted branch, yet which seemed naturalised in another quite distinct affection. Seven or eight years ago, Professor Westphal showed that, in what was called general paralysis of the insane, the conditions of locomotor ataxy were present to a large extent. That statement interested him much, and he visited St. Luke's Hospital, taking with him a galvanic battery, and several Guy's men. By the kindness of the resident physician he was able to make a careful examination of the general paralytics there; and he came to the conclusion that about one third of all those unhappy sufferers had the physical conditions objectively characteristic of locomotor ataxy. Dr. Savage had told him that, in his experience, about one-third of all general paralytics in Bethlem showed locomotor ataxy; but he had never met with an example of Charcot's disease in the hospital. If, in truth, the conditions of ataxy were present in a paralysing disorder, and if this peculiar joint disease, supposed to belong to ataxy, did not appear in the ataxy that accompanied paralysis, then might not one look for some traceable effect of paralysis to explain the absence of the joint affection? Sir James Paget had very well expressed the peculiarities of Charcot's joint disease, as summed up in the presence of the destructive effects of inflammation and the absence of repair. Now, if that combination never occurred in certain ataxics, who, if ataxic, were also paralysed, had the paralysis anything to do with the absence of it? Was it not a fair suggestion that if a man were paralysed, and therefore did not go about upon his ataxic joint, and therefore did not keep up a continual irritation, this might

be the reason why he did not make an originally chronic simple rheumatism into an aggravated, unrepairing, irritative, severe, therefore strange and peculiar disease of the joint? He had a specimen from Guy's Museum—the shoulder-joint of a cabman who was thrown from his cab and hurt his shoulder a year before his death; but, in order to support his wife and family, he continued driving his cab. A year's cab-driving with an injured shoulder-joint would not be very far different from a year's unsteady stumbling about on a knee-joint. Now, this cabman's shoulder-joint was a Charcot's joint, or something nearly approaching it. Dr. Moxon had also had a normal humerus brought with it for comparison. In the cabman's shoulder-joint, half the head of the humerus was entirely gone, and yet there was no production of new bone about it. Those were, he considered, the most striking characters of the so-called Charcot's disease, as defined and summarised by Sir James Paget; so that a half Charcot's joint would be made by the irritation connected with the unguarded and continued use of a joint suffering rheumatically or from any injury. He would ask whether, if due allowance were made for those conditions of joint which arose from the disablement that could be traced to ataxy, all the phenomena of Charcot's disease must not be expected to supervene upon any form of rheumatism casually occurring. When he was demonstrating anatomy at Guy's Hospital, he always tried to show his class some reason why that which was evident should be expected to exist. Amongst the most interesting general facts of anatomy first observed, and to the larger extent, by Mr. Hilton, was this: Mr. Hilton said that whenever a nerve supplied a muscle, it supplied the skin over that muscle. That was not entirely true, nor was it so suggestive as it should be. The proposition which observation taught Dr. Moxon to lay down was this: whenever a nerve supplied a muscle, it supplied the skin of the part moved on the side towards which motion was effected, and it likewise supplied the joint which that muscle moved, and generally on the side towards which motion took place. If that were true, and he believed it to be so largely true as to be a general principle, there must be some meaning in it. It could not be accident, and the meaning lay easily to hand. The meaning must be that there was some underlying necessity that the part moved should be sensitive to the effect of motion; given that anything harmful was occurring through a movement, the moving agency might be immediately and duly checked. If that were

a requisite of joint-life—and it was probably necessary—take away the sensibility of a joint, make the muscles spasmodic, and ask what ill-result might afterwards be traceable. Must there not be found some standard to show what the probable result of it all must be, before saying that, when a joint, deprived of its sensibility and subjected to spasm, showed serious signs of chronic inflammation, there must be something mystical and marvellous in the nervous system to explain it? But this so-called Charcot's disease began as a common rheumatism, as Mr. Hulke showed, and ended in extensive disorganisation, only after long experience of spasmodic irregularity of action in the presence of a wholly broken-down condition of the protective system. Given the cabman's shoulder, and added thereto an insensitive state of his joint, *plus* a spasmodic state of his muscles, and Dr. Moxon would advance the proposition that the half-Charcot joint of the cabman would be a whole Charcot joint. To what extent was security in disease really due to those protective efforts of muscle to which he was now alluding? He had in his mind a most painful example. He was in the clinical ward when a man, æt. 19, came in for perityphlitis. This man had the usual condition of board-like hardness of the muscles in the lower and right part of the abdomen, and this hardness appeared to be, and was proved to be, protective of an inflammatory condition beneath. Dr. Moxon had him put under chloroform, so that he might examine the condition of the intestines apart from the spasm. He was profoundly anæsthetised, and Dr. Moxon himself, and two or three members of his class, certainly not coarsely nor carelessly, examined the part whilst the muscular protective spasm was thus taken off. The unfortunate youth came out of his chloroform narcosis in intense agony, and died of peritonitis from rupture of the intestines within nineteen hours. Painful as that was, it taught him a lesson. It made him ever since very respectful to, very recognitive of, the value of the sense of pain in mechanical disturbance of inflamed parts calling upon the protective agency of the muscles to prevent injurious movements. Something parallel undoubtedly existed in the protection of the inflamed joints of healthy persons, by the pain which they would otherwise suffer in the severe and coarse action which a large joint had to undergo. He asked the Society, before deciding upon anything obscure in the matter, to make it clear to what extent it was probable that the later stages of a joint inflammation would be aggravated by insensi-

bility and spasm, even when that inflammation commenced as common rheumatism. Would it not become an exaggerated, irritative, severe, perhaps strange-looking disorder? It was no new thing in the history of the medical profession, that a mysterious origin should be claimed for a local manifestation. About fifteen or twenty, or more years ago, strong attention was drawn to the occurrence in lunatic asylums of a remarkable condition of the external ear, in which it swelled up, and, indeed, became full of blood, so that the disease went by the name of hæmatoma auris or auricular hæmatoma, and papers were written to show how it was that the strange condition of the nervous system which was peculiar to highly maniacal lunatics should so govern the nutrition of the external ear, that, under certain conditions remarkably limited to asylums, the external ear should grow into a bleb of blood. Those who were not content to ignore a conclusion so improbable were compelled to put up with it until recently, when a parallel was instituted. The present secretary of the Football Association had a hæmatoma auris. One of the most distinguished football players in Guy's Hospital admitted, almost with pride, that in the very height of a scrimmage he was distinctly maniacal. Those whose imagination in scientific subjects was equal to such a scope, might think that there was in a football scrimmage an instantaneous discharge into the ear of the peculiar kind formerly supposed to belong only to inmates of lunatic asylums, and which was thought to produce hæmatoma; but Dr. Savage had told him that a gentleman, going round Bethlem Hospital with him, being shown one of those hæmatomata on one of the patients, said that in his travels he had seen a statue of a Greek boxer, upon whose sculptured ear was represented a shrunken hæmatoma auris. He trusted that the Society would not think that there was anything frivolous or trivial in the parallel he had endeavoured to draw. The general proposition common to both positions was this: that in the absence of proven direct causes, there was a tendency in the profession to appeal to the nervous system. There used, once, to be some degree of moderation in that invocation, until what was called the trophic nervous system was invented, which had charge of the special supplies of the several textures, such as the bones and the joints, having its seat in the medulla oblongata, and being, in fact, a sort of secretary of state for the joint department. He thought he had a right to ask of those who supposed that there was a special nervous provision for the nutrition of the joints, why it was

that one joint especially should become a victim of some frightful, tenacious, unsparing catastrophe? He would admit that there was some physical sense in it, and not mere metaphysical nonsense, if those who believed that the knee-joint in Charcot's disease was affected by the nerves would show that the nerves themselves were affected. Danielssen and Boeck showed that, in anæsthetic leprosy, the anæsthesia was due to a disease of the nerves; but they showed that the nerves were diseased. Who had shown that, when any special joints were attacked with Charcot's disease, which we were asked to attribute to the nerves, the nerves were in fault? Was there an exostosis pressing on the nerves? Was there any particular degeneration, any structural disease, anything that would induce that which was assumed to be a consequence of a thing, the existence of which was neither shown nor attempted to be shown?

The PRESIDENT believed that two of Professor Charcot's pupils had demonstrated the existence of nervous disease in the neighbourhood of the joint.

Dr. MOXON would stop the rest of his remarks until he had read the contributions of those gentlemen. Nevertheless there was a very large opening here for further contributions, and it was not enough to show that the nerve was pink, or yellowish, or of any colour whatever, or a little swollen. He had examined nerves—for instance, cervical ganglia—to try to find out something exact in their pathology. When a little pinkness, or a little yellowness, or a little swelling, or a little shrinking, or a little excess of fibre, was observed, one was often tempted to find pathology where there really was not any; and, unless M. Charcot's pupils made plain, not only a diseased condition of the nerves, but one which was itself both destructive and irritative, he must still hold that the nervous condition which gave rise to a tenacious, continuous, and absolutely destructive influence on a joint ought not to be set down to some mere change of colour, or mere alteration of fibre. Not merely hæmatoma auris, but a certain very fragile state of the bones had been asserted to characterise some inmates of lunatic asylums. The theory was, that the bones of the insane were softer and more fragile than other people's bones. But a part of his demonstation at Guy's Hospital, in morbid anatomy, was to take the ribs of a somewhat elderly person, and to show the students that, with two fingers of either hand, he could easily fracture the ribs without any conspicuous or

evident effort. In fact, fragility of bone was a thing which existed to an extent that became startling only when the conditions were mystic. There was a tendency to bring in the vague and the general, and to avoid measuring the forces of the direct and measurable causations; and this was a tendency which, he thought, ought to be severely repressed. M. Charcot had brought forward, as evidence of some very mysterious influence of the trophic nerves, the very strikingly significant occurrence of one-sided bedsore in persons paralysed on one side only; and Dr. Moxon was very much impressed by the picture in Charcot's book, so clean on the one side, and with a hideous patch on the other. He had seen similar cases. He saw a lady with profound hemiplegia, and, within a fortnight, he was astounded at the degree in which the buttock sloughed on the paralysed side. A large part of it mortified and proceeded to separate, so that the chasm looked almost as if a spade had been thrust in and a spadeful turned out. But he was not ready to admit that it was necessary to here suppose any vague provision of trophic nerves to explain such an occurrence. There were two points which he could advance with reference to the hemiplegic bedsore. He had observed that, whenever a person was severely hemiplegically paralysed,the tendency always was for the body to roll round and lie upon the paralysed side. That seemed to be due to the continued action of the still active limbs of the sound side, and especially of the muscles of accessory respiration. This happened in a way that could easily be conceived, and even watched; the activity of the limbs that were still active tended to drag under and roll round the paralysed side. There was another point which was a little more abstract. When he was demonstrator of anatomy he used to point this out to the students. Whenever an artery was passing by a bone to enter a muscle, it ran under a tunnel of tendinous fibre, and this tunnel of tendinous fibre had muscular fibre arising from the outside of the arch of the tunnel, in such a way that the contraction of the muscle held the tunnel open, and so kept free the course of the artery from pressure by the mass of contracting muscle. The meaning was obvious. In fact, it evidently was the duty of the muscle, in acting, to pull open the channels of its own arterial supply. A contrivance of the kind could not be traced further into the muscle, though he believed that, if it were necessary to prevent an artery from being compressed against a bone by the muscle swollen in contraction it must be equally necessary to prevent the arteries from being

compressed within the muscle itself. If this were true, then an absolutely paralysed muscle was deprived of a system whereby it was able to secure its own nourishing supplies. If the tone were taken away from a muscle by a total paralysis which reduced its previously vital mass to the condition of inanimate clay, and if it were then heavily compressed, its nutrition was prejudiced, so that mortification was likely to occur, not only of the muscle, but of any texture which received its blood-supply by arteries sent through the muscle. The skin of the buttock was fed by arteries coming through the gluteus maximus; and with the whole weight of the body, through the roll of the patient, resting on the gluteal mass of the one buttock, unprotected by the tone of the muscle, what was the wonder that the muscle and the textures over it should perish exactly in proportion to the pressure? In drawing towards a conclusion, he must remember that, indeed, in point of form, he was not speaking as to Professor Charcot's joint disease at all, but only proposing a vote of thanks to him, which he did in the highest admiration of the great services which he had rendered to medicine. But this was not, if he remembered correctly, the first occasion on which a somewhat mystic light had been thrown by Professor Charcot on an obscure subject. About eight or ten years ago, Professor Charcot was very prominently before the profession throughout Europe in association with the marvellous. Some years ago, when he was in the clinical ward, a young professional friend of his, who was now called a gynæcologist, came home from Paris, and his advent threw the ward into a very singular condition, which to this day he (Dr. Moxon) had never quite grasped. He remembered that this gentleman had a splendid case of what people called "hysteria." In short, he had as patient in one of the beds a young woman who was extremely anæsthetic on one side. She could not feel anything whatever on one half of her face, arm, leg, side, &c., yet she was quick enough on the other. Well, this gynæcological colleague was hard at work with sovereigns and shillings, and he said it was all according to Charcot; he was putting a sovereign on one arm, or leg, or side, and a shilling on the other, and he did not know what happened, but it was something very mystical. There was a transfer of total want of sense from place to place, from time to time, and a pricking went on. Shortly afterwards, Dr. Moxon and others, who were not gynæcologists, did the same thing with circular bits of potato-parings, and all happened as if under gold and silver. At

the same time, there was no doubt that, on that occasion, the marvellous was brought upon the ordinary in a very suggestive manner. And now, thanking Professor Charcot profoundly, he asked the meeting to pass him a vote of thanks.

Mr. MORRANT BAKER seconded the vote of thanks to Professor Charcot, which was carried by acclamation.

Mr. HENRY MORRIS related the histories of two cases.

CASE 1. *So-called Charcot's disease of the left knee-joint. Recurrent rodent ulcer.*—On March 20, 1877, Robt. M., æt. 53, a collier, was admitted into the Middlesex Hospital with what had been supposed to be recurrent cancer of the face, and malignant disease of his left knee-joint. It was expected that amputation of the thigh would be required. Five operations had been performed at different times upon his right upper lip and cheek. On admission a rounded, flattened, hard mass occupied the scar and extended upwards and outwards upon the malar bone. I removed the diseased tissues, which under the microscope proved to be rodent ulcer. He recovered quickly from this operation. The knee-joint affection was looked upon by us at the hospital as "chronic rheumatic arthritis," but of most exceptionally severe form. The leg was like a flail. The knee was considerably enlarged, the enlargement being very unsymmetrical and most marked on the inner side of the joint. The measurement around the left knee was nineteen inches; around the right fourteen inches. The tibia was bowed inwards from the knee, so that when he attempted to stand the femur formed an angle with the tibia, the convexity of which was on the outer side of the knee. The synovial membrane was distended unequally with fluid. There was more bulging on the inner than on the outer side. The ligaments were all relaxed, and there was considerable lateral movement of the leg upon the thigh. In the internal lateral ligament, concealing the tuberosity of the tibia, was a thin plate of bone. The head of the fibula was not enlarged, and no outgrowths from the femur could be felt. Voluntary and passive movements were equally painless. Flexion was only possible as far as a right angle, but extension was complete. The tibia slipped backwards and forwards on the femur with a most distinct creak. The man could stand and walk in spite of the flail-like condition of the leg and the outward bowing at the knee. The disease of the knee began three years (or six years?) before his admission, after he had been working in water in a mine, and it set in with, as he called it, "rheumatic

pain." The pain was variable, according to the state of the weather, and after it had continued twelve months the joint began to swell. The swelling was first noticed on the inner side, but it gradually increased until there was bulging on each side of the quadriceps tendon. Then followed thickening and enlargement and lateral increase of the head of the tibia. He had noticed the joint give under him and bow outwards for twelve months, and during this period the swelling of the knee and the occasional pains had increased. No other joint was affected. He was fitted up in a Thomas's knee-splint and discharged from the hospital. Twelve months later he was heard of as being in good health and following his usual occupation. On November 26, 1884, I heard from Mr. Court, of Staveley, to this effect:—"Robert M., died one and a half years since, æt. 60. Six years before death you removed a cancer from his face, and applied a splint for the joint disease. He wore the splint and worked regularly up to eight weeks before his death. The joint got smaller, so that at the time of his death the size was not much greater than that of the other knee. He could move it in every direction. His last illness began eight weeks before death with hoarseness and complete loss of voice, but no pain anywhere. Gradually the weakness increased until he could not sit up, and he kept his bed for six weeks before death. He could not bear the least draught of air upon his face, and therefore kept his face, head, and mouth closely muffled up. He lost strength and appetite, gradually. Night sweats were profuse. Two days before death a quantity of blood came from his mouth, and he bled again the next day. The blood seemed to come from his throat." No post-mortem examination was obtained.

Case 2. *Old-standing, painless deformity of ankle-joint (Charcot's disease) and elephantiasis of the leg and foot of the same side. Death from intestinal obstruction due to volvulus.*—Thomas C., æt. 53, formerly a draper, but for the last fifteen years a clerk in a large tea warehouse, was sent into the Middlesex Hospital at 8 A.M. on Friday, March 14, 1884. The patient had a large irreducible inguino-scrotal hernia on the right side, and when admitted was suffering from stercoraceous vomiting and other symptoms of intestinal obstruction. His abdomen was much distended, very tense and hard, encroaching on the thorax and causing dyspnœa. He had been ruptured for many years, but had not worn a truss. Under ether an oblique incision over the neck of the tumour was made and the sac

opened. The intestine was greatly distended with flatus; deeply congested, and of a dark greenish purple colour. It was large intestine, and was contained in a distinct hernial sac from which a quantity of blood-stained serum having a fæcal odour escaped. No obstruction existed at the inguinal rings or neck of the sac, yet the bowel could not be returned on account of its enormous size and distension. An aspiratory needle was introduced, and some gas escaped, but reduction being still impossible the bowel was opened and stitched to the skin. About two pints of blood-stained fluid fæces flowed away. The patient, however, died at 12.30 P.M. When on the operating-table it was observed that his *right* lower limb from his knee downwards was greatly deformed. It looked like an immense elephantiasis; but, in addition, the angle of this foot to the leg was very unnatural, the foot being displaced externally and having a very loose connection with the leg. It could be moved and rotated freely and painlessly, but with a peculiar scranching noise. Movement outwards around the antero-posterior axis of the foot was specially free, and the patient walked upon the inner edge of the foot. There were scars and sinuses on either side of his ankle. The toes did not share in the elephantiasis. The great toe was shorter than usual, in proportion to the second toe. There was also a perforating ulcer of the great toe of the left foot. I ascertained the following facts from his wife:—He was of a quiet, cheerful disposition and very active, and had continued so till his last illness. He had always been very thin, but had gained in flesh slightly during the last three years. His eyesight was good, and though for the last two years he had worn glasses, his wife was quite sure there was "nothing unusual about his sight." He only used his glasses while reading, not for office work. He did not suffer from headache, pains in his head, nor had he any twitchings. Of late he had been subject to great coldness in his hands, which often "went quite pallid." Before his marriage, twenty-five years ago, and throughout his subsequent life, he used occasionally to suffer most acute pains in his "stomach." These came on chiefly after meals and did not last long, but they had been more severe during the last year or two of life. He had all the "virile" power natural to a man of his age. There was no loss of muscular power in any part of him except in his right leg; and on the Saturday before his death he had spent the afternoon (after leaving his regular work) in fitting up a window-garden.

When remonstrated with by his wife for carrying both arms full of flower-pots across the street and up to the second floor of his house, he answered, "Why not? It does me no harm, and if I had but a rod to steady my foot, I could walk without the least inconvenience." "Water" used to pour off his leg at night; in fact, the night perspiration of the limb became so bad that his wife provided mackintosh for his limb to rest upon.

Twenty-one years ago he had a bunion on the toe of the right foot and pieces of bone had worked out and been taken away from time to time from this toe. For more than fifteen years he had had a sore on the great toe of the left foot. Ten years ago at least the right ankle began "to swell and in other ways to be deformed, but it was a matter of surprise to him that with such a bad foot there was no pain." Up till about seven years ago he used to cut his right boot in one way and another so as to make it fit; but for the last seven years at least he has never bought a pair of boots, as the right one always required to be specially made and not always in the same way. The deformity of the ankle and the great increase in size of the foot and leg had been increasing more rapidly during the last three or four years. Four years ago a box fell on the right foot, but did not hurt him sufficiently to cause him to lay up. Since then offensive pus had been discharged from the sinuses which had formed first on one side and then on the other of the ankle. Still he continued his occupation uninterruptedly, carrying with him a second sock and pieces of linen to apply during the business hours of the day, to keep himself free of the smell. The patient was the father of six children, one of whom, the eldest (daughter), died from peritonitis and obstruction of the bowels after eating a quantity of unripe fruit when three and a half years old. His own father died from cause unknown when about fifty-five years, his mother lived to be seventy-eight, and was always very healthy and died of old age. One brother died of "consumption of the throat" when forty-three years old; another at fifty-five still lives; his only two sisters are also living, aged sixty and fifty-six. The third brother died suddenly a few days after patient's funeral from hæmorrhage from a cancer of the stomach.

The right leg was amputated after death, and on dissection Mr. Sutton found the posterior tibial nerve enormously enlarged. It is seen in the specimen * to be the size of the

* The specimen is in the Museum of the Middlesex Hospital. Catalogue, No. 7, under "Elephantiasis."

little finger or larger. Both tibia and fibula were altere shape, and the edge and angles of the bones were irreg and unduly prominent. The fibula was much enlarged, at the lower end it had been fractured obliquely; the fr ments had united and were overlapping. The outer r leolus is represented by a large oval articular surface, so what resembling one of the condyles of the femur; fitted into a large, shallow, irregular, saucer-shaped surf on the upper aspect of the os calcis. The upper surface the astragalus is roughened and eroded in places, and also is the head of this bone, which moves freely from side side on the os scaphoides. New bone has been formed at margins of the articular surfaces, and the ligaments of ankle had been in great part destroyed. There was a p forating ulcer of the great toe of the left foot, and the posterior tibial nerve was somewhat enlarged. Mr. Leop Hudson has made careful preparations of parts of th nerves, and gives the following account of their microscop appearances:

"I. *Horizontal section of upper third of right posterior ti nerve.*—(*a*) There is very great increase in the epineural c nective tissue, and this increase is more marked in the cer than at the periphery (Plate VI, fig. 1). Large w bundles of white fibrous tissue, taking principally a vertica oblique direction, are everywhere found separating the ne fasciculi. This tissue is fully developed, contains few nuc and is abundantly supplied with vessels, a considerable amo of adipose tissue surrounding the latter in many places. H and there are groups of axis cylinders, devoid of medull sheath and tightly bound together with fibrous tiss evidently the atrophied remains of nerve fasciculi. (*b*) T perineurium is not abnormally developed nor is there appreciable change in its intimate structure. (*c*) Within fasciculi the endoneurium is seen to be very considera increased in amount, its constituent fibres running vertic and appearing in section as minute red points closely surrou ing the ultimate nerve-fibres (Fig. 2). The nerve-fibres the selves are greatly diminished in number, in many places, inde being only sparsely scattered through the fasciculus. Th are seen to consist almost entirely of *large* nerve-tubes, such are found to preponderate in purely motor nerves (Fig. The smaller fibrils which are now held to be sensory or trop and which are relatively abundant in normal post-tibial ner have almost completely disappeared, their place being ta

DESCRIPTION OF PLATE VI.

Figs. 1, 2, 3.—To illustrate Mr. Morris's case of Charcot's Joint Disease. P. 94.

Fig. 1.—Part of upper third of right posterior tibial nerve. (× 16.)

a a a. Dilated veins of epineurium.

b. Vascular adipose tissue (to the left of *b*, lying in the epineurium, are the remains of an atrophied fasciculus).

Fig. 2.—A single fasciculus surrounded by its perineurium. (× 65.)

a a. Corpora amylacea.

b b. Nuclei of endoneurium.

Fig. 3.—Part of preceding fasciculus. (× 260.)

a. Vertical.

b. Horizontal fibres of perineurium.

c. Intra-fascicular lymph-space (the endoneurium has somewhat shrunken in hardening).

d. Greatly hypertrophied endoneurium.

e. Large white nerve-tubes.

Fig. 4.—To illustrate Mr. Anderson's case of Papilloma of the Bladder. P. 315.

A. General view under a low power.

B. One of the papillæ highly magnified.

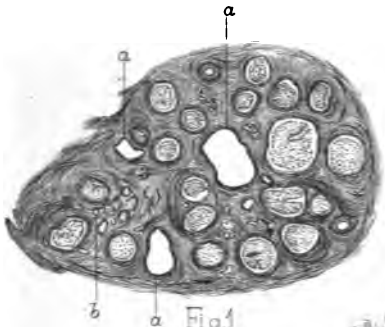


Fig. 1.

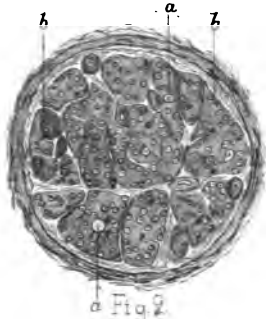
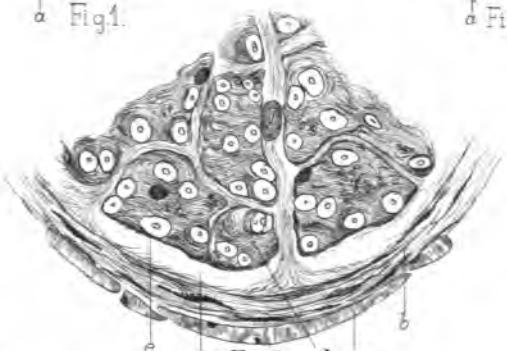
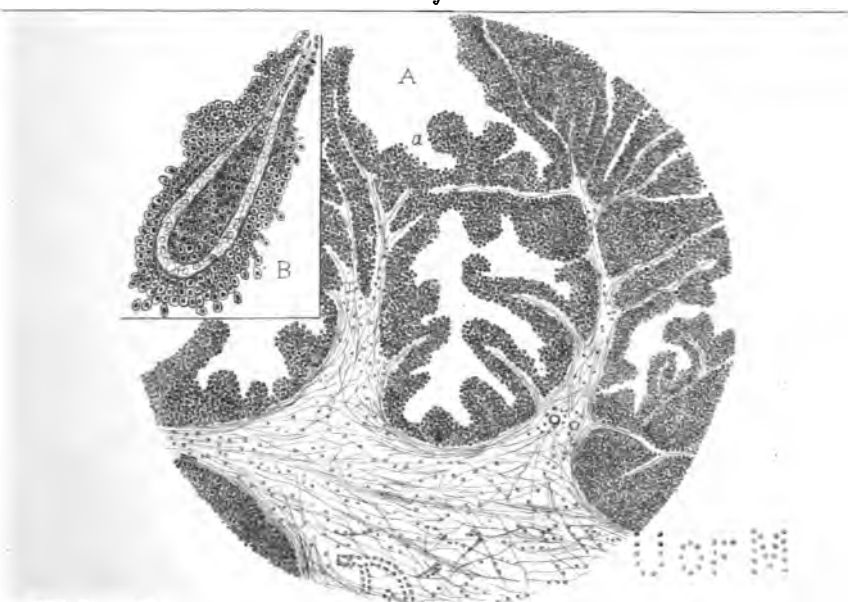


Fig. 2.



L. Hudson delt.

Fig. 3.



W. Anderson, delt.

Fig. 4.

Danielsson & Co, lith.

by the hypertrophied endoneural tissue. This intrafascicular change is most marked near the periphery of the nerve-trunk. Numerous small 'corpora amylacea' are found in those fasciculi in which the degenerative change is most advanced."

"II. *Horizontal section of middle of left posterior tibial nerve.* —(*a*) There is some overgrowth of the epineurium, but to far less a degree than on the opposite side. (*b*) The perineurium is normal. (*c*) Changes precisely similar in kind, but less in extent, to those of the right side are seen within the nerve fasciculi. Almost complete absence of the smaller fibrils with corresponding increase of endoneurium. The fibrous tissue in this nerve stains more deeply and appears to be of more recent growth."

At the post-mortem inspection there was found by Dr. Fowler to be an enormous volvulus of the lower five feet six inches of the small intestine, cæcum, and part of the ascending colon. These parts of the large bowel had each a very large mesentery, and thus a portion of the ileum which was twisted on its own mesentery had become folded beneath the cæcum and ascending colon in the right iliac fossa. The enormously dilated cæcum and colon were flaccid and contained very little fæcal matter, but there were three linear rents in their peritoneal coat, the effects of over-distension. The cæcum was attached to the wound in the right groin. At the hepatic flexure of the colon, at a point corresponding to the lower end of the volvulus, the great omentum had been gathered up into the form of a purse eight inches long, with a narrow neck. It had evidently lain for some time in the sac of the hernia. The mesentery of the cæcum and colon was scarred and ribbed with raised ridges, evidence of old chronic peritonitis, the probable result of stretching, dragging, and twisting. The mucous membrane of the dark portions of the cæcum and colon was intensely congested but not gangrenous. The transverse and descending parts of the colon were contracted. The post-mortem examination thus afforded ample explanation of the acute abdominal pains so often felt during life.

Remarks.—The first case presents the changes in a single joint which are typical of those described by Charcot. The points noticeable about it, are: (1) the long duration of the joint affection without any ataxic symptoms supervening; (2) the disappearance of the swelling in the joint, under the condition of rest provided by the splint; (3) the co-existence of malignant disease of the face and mouth from a recurrence of which, in all probability, his death is to be

attributed. The last point was worth noting, especially in connection with the family history of Case 2, one of the brothers of whom died of cancer, because it may possibly be that malignant disease is one of the constituent elements which go to make up the composition of this joint affection, and more extended observations will show what proportion, if any, it bears to the whole number of constituent conditions which lead to it. Cancer, as is well known, leads to marked changes in bones, softening their structure and predisposing them to spontaneous fractures, which fractures, however, readily unite. It has sometimes been associated with osteitis deformans; possibly it may be found to have some determining influence on joint changes. The improvement which occurred under the rest secured by a Thomas's splint (notwithstanding that the man kept at his work during the time he wore the splint) lends support to the view of Mr. Hutchinson and others, who regard the changes in the joints as being due to the rubbing and pressure effects from continued use of the joint, and that these changes would cease if rest be maintained. The commencement of the affection after long exposure to wet, and the rheumatic pains which so long preceded the structural changes in the joint, go to support the view of Mr. Hulke and others, who regard the affection as rheumatic arthritis. The noticeable points of the second case are (1) the perforating ulcers which had existed for from fifteen to twenty-one years; (2) the painlessness, the necrosis, the profuse perspiration of the foot and leg, and the pathological changes in the nerves of the leg which are characteristic of perforating ulcer; (3) the enormous hypertrophy of the cellular tissue of the limb which resembled the ordinary Barbadoes leg, (4) the changes in the ankle-joint, which in respect of the wearing away of bony surfaces, and the great laxity and swelling of the misshapen joint are seen in the joint affection described by Charcot; (5) the great thickening and irregularity of the bones of the leg, such as have been described as occurring to a less degree in some cases of perforating ulcer; and lastly, complete absence of ataxic symptoms. Whether this is to be regarded as primarily a nerve disease or not, the case shows the occasional association of perforating ulcer of the foot with an affection of the ankle, like that of so-called Charcot's disease; and at the same time with a condition of the soft tissues like that of elephantiasis Arabum.

Mr. Herbert Page called attention to a case of tabes dorsalis brought by him before the Society early in last year. It was the case of a man who for some years had been the subject of gastric crises, attacks of vomiting to an extreme degree, and who also had lightning pain of great severity. The man came under observation for an affection of the tarsal bones of one foot, and, while he was under treatment, there arose the same sort of condition in the other foot. The tarsal bones of both feet underwent a change which obviously consisted in extensive destruction; but, under the influence of rest, ankylosis ultimately took place, leaving the feet "extremely misshapen." So far, the history of the case, it might be said, was nothing but rheumatoid arthritis attacking the bones of the feet, possibly promoted by certain nerve-changes. The later history of the case made the appearance of things somewhat different. All the various symptoms subsided. The man was able to go about his business as he had done before. There were few or none of the symptoms of ataxy in the case. He was at work, in what was to him perfect health, till about the end of August, when he again came into St. Mary's Hospital, with a recurrence of the attack in one foot. He had lately been in the hospital, and most extensive changes had taken place in one ankle and one series of tarsal bones; but the other foot had been wholly unaffected. It seemed to Mr. Page inconceivable that, if this condition of bone-changes were due to rheumatoid arthritis, and this attack were an exacerbation of the rheumatic affection, it should not have at the same time attacked both feet, as on the first occasion. The fact that it only attacked one foot also seemed to point to some other cause than a general condition such as rheumatoid arthritis. In the debate, speakers had referred to pathological changes, and very little indeed had been said of the clinical features of the disease. Various speakers had referred to the gradual wearing away of ends of bone in this condition; and doubtless one did meet with, in the course of months, a change induced by the gradual wearing away, which very much simulated the ordinary appearance in specimens which unquestionably were only met with in tabes dorsalis. But that was not the ordinary condition which the clinical history of these cases showed; here the wearing away was extremely rapid, and he thought there must be some other cause than the rheumatoid change, or the friction of bones one against another, to account for that condition. Charcot himself had pointed out numbers of cases in which a rapid wearing away had taken place in association

with exacerbation of other symptoms of the disease, gastric crises, and other phenomena of the same kind. There was no reason to believe, in a case such as that which he (Mr. Page) had related, that friction of the bone could have given rise to the condition; because, in the later stage, only one joint had been affected, both being subjected to precisely the same influence. Then, putting aside this rheumatoid arthritic change, might not the nervous system itself have some share in the production of this rapid destruction of bone? Certain irritative lesions of nerve-trunks did give rise to certain definite effects. In a nerve which had been divided, and in which the distal end was irritated by inflammation, or by being bound down in cicatricial tissue, if the distal end of the nerve were released by operation from the cicatrix, the changes due thereto ceased, but the condition of nerve-inactivity remained for some time till the nerve-functions had been restored by the union of the nerve. They might or might not be trophic changes, but they were essentially due to some effect upon the nerve-trunk at the part where the division had taken place. If such a change should take place there, the same sort of change might be due to lesions in more central parts of the nervous system. But recent observations had shown that nerve-trunks themselves underwent very extensive and serious changes in this disease, which were degenerative changes of the nerves going to the limbs and to the various parts affected; and even in the case of bedsores, observation had shown quite recently that extensive changes were found in the nerve-trunks themselves going to the seat of the acute bedsores. And it was quite possible that that might really be the thing underlying this condition, determined by the central lesion in the spinal cord. Mr. Page had also had a case, which he published in *Brain*, very like that which Mr. Morris had related, where there had been a history of perforating ulcer with anæsthesia, and so on, and where after some years, when the perforating ulcer had healed up, there came changes in the joints, and doubtless in that case changes in the nerve, because there was considerable anæsthesia combined with the change in the joint. Dr. Moxon had referred to the bedsores met with in hemiplegia; and Mr. Page believed that in some of the cases described by Professor Charcot, where paralysis was induced by injury, not only was bedsore met with on the opposite side where there was anæsthesia, but actual changes were met with also in the joints on the same side as the lesion, where there was motor paralysis. Hence possibly, if the conditions on one

side were due to changes in the nerve, the same cause might exist on the other side also. He believed that the disease under consideration was a distinctive disease of joints, induced by some change in the nervous system, and that Professor Charcot himself was not far wrong when he originally decided that it was a distinctive disease having special clinical characteristics of its own. The various matters mooted in the course of the debate with reference to rheumatic arthritis and so on had all been considered by Professor Charcot in his various papers; and, with his numerous opportunities of observation, he still adhered to the view he originally put forward.

Dr. MOXON asked Mr. Page whether his patient with this tarsal disease, as he described it, had or had not locomotor ataxy definitely; and, secondly, whether Mr. Page would say in what way locomotor ataxy was a less general disease of both feet than rheumatoid arthritis.

Mr. PAGE said there was certainly no ataxy in the ordinary sense of the word. There was no ataxy in locomotion, and there was no flinging about of the limbs, such as to cause unusual rubbing of the bones against each other. The man walked perfectly well, even with his eyes shut. Then, with reference to the second question, in the course of this year, the patient had no changes whatever in one foot, although he had extensive changes in the other; and that seemed to him a strong point against the existence of rheumatoid arthritis being the cause of the condition, because he could hardly conceive of rheumatoid arthritis being absolutely quiescent in one limb, previously affected to so extreme a degree, and yet active in the other.

Dr. PYE-SMITH said the *first point* brought before the Society by Sir James Paget was whether Charcot's disease was a new disease. His opinion was that there was no evidence of any really new disease, certainly not of this. Cholera, diphtheria, and cerebro-spinal insular sclerosis, had all been only lately recognised; but none believed that these diseases did not exist previously. The *second point* was the relation of so-called Charcot's disease to osteo-arthritis; and, on the whole, it seemed to him that there was no adequate evidence for separating them. No doubt, in somewhat extreme cases of the two affections, there was a great excess of fresh bone in osteo-arthritis, and there was a great diminution in Charcot's

joints. But, in almost every joint with osteo-arthritis, one could discern, along with the osteophytes and hypertrophy, much wasting, and loosening, and shrinking of tissue, not only of the cartilages and soft tissues, but also of the bone itself. And, as had been well pointed out by Mr. Hulke, in most cases of Charcot's disease one could, where there were several joints to look at, discern in some of them an attempt, more or less successful, to produce new bone. The difference also, which no doubt was a real one, between the one affection being chiefly confined to a joint, and the other affecting the shafts of the bones, was, he thought, not absolute, and was affected by conditions of age and duration of disease. If one admitted it as belonging to the somewhat large group of osteo-arthritis, one must see, also, that even in that well-recognised group of affections, certain varieties could be distinguished. There was apparently but little similarity between the early stage of osteo-arthritis, as it affected the girl and the young woman—where it affected very symmetrically the small joints in the fingers, with considerable pain, and a little febrile reaction perhaps, and a very little distortion—and a disease affecting one hip-joint of an old man, with, perhaps, hardly another joint in the body, very slow, very local, very traumatic, very little constitutional. Therefore, he did not think that the mere fact of a well-marked typical case of Charcot's disease differing from an ordinary case of osteo-arthritis, would lead one entirely to separate them. Moreover, he would suggest that, beside the anatomical changes in the joints, the clinical characters of the disease must also be considered; and he thought all would agree that it was quite possible for any long-continued chronic inflammation of the joint, not suppurative, not of an irritative character, to produce some of the changes which were recognised in osteo-arthritis. He had seen a joint with all the clinical features of gout, in which the edges of bone round the articular surfaces presented characters which if one dissolved away the urate of soda, would have left the joint very much in the condition of osteo-arthritis. This had been also observed by Professor Virchow; but no one would say that gout and osteo-arthritis were the same disease. Acute rheumatism he believed most thoroughly to be an absolutely distinct disease, entirely separate from osteo-arthritis, or the affections included under "chronic rheumatism," "rheumatic gout," or other vague terms. At the same time, deformity and some changes occasionally resulted from acute rheumatism, which were far more frequently seen in osteo-

arthritis. Two cases had lately come under his care. One was that of a young man with acute rheumatism and valvular disease, with the deformity of the wrist and knuckles which was quite characteristic. Another was the case of a young lad who had also been subject to acute rheumatism several times, with valvular disease. He had a decided beginning of thickening of the phalangeal joints. All must have seen like cases occasionally, and no doubt, after the death of such patients, changes were found in the joints not unlike those of osteo-arthritis. *Thirdly*, had this osteo-arthritis or Charcot's disease, taking the two together, any definite relation to rheumatism, to gout, to trophic disease, to nervous disease, or to tabes? With regard to trophic nerves, he could not agree with Dr. Moxon. The existence of trophic nerves had been placed beyond the possibility of reasonable doubt; and when once that had been done, then the many clinical and physiological facts which before one had endeavoured to explain upon other hypotheses, without calling in these obscure trophic nerves, became not only clear, but added much weight to what one knew before. He therefore admitted the existence of trophic nerves, but he could not admit that they were proved to have much to do with Charcot's disease; because, in the first place, there was no reason to suppose that trophic nerves had any connection with the posterior columns of the spinal cord, and there was reason to believe that the trophic nerves passed out through the anterior roots, which, so far as was known, were unaffected in tabes. Then, with respect to gout, was there the slightest connection with gout of any sort? There was an entire absence of urate of soda from the true cases. Again, the distribution was different. Whence was it that most in the early history of osteo-arthritis had been learned? It was to the Irish surgeons that we owed most of our knowledge respecting it, and yet in Ireland gout was almost unknown. Again, osteo-arthritis was extremely common in the dead-houses, not only in Paris, but also in Vienna, where gout was so uncommon that he once found a joint with urate of soda, and the assistants of one of the professors were quite ignorant of what it was. Rokitansky recognised it, but he said he had never seen another such case. The extreme rarity of gout in certain parts of the world, and the extremely common diffusion of the other disease, was surely enough to show that there was no connection between them. Again, the same disease was common among animals. He had brought a specimen of the fetlock of a horse, which showed a good

example of osteo-arthritis. Mr. Busk once exhibited at the Pathological Society some bones of bears from a bone-cave at Gibraltar, which, in the glacial period, had been affected by something which would now be called osteo-arthritis. In the Zoological Society last year, Professor Flower showed some cervical vertebræ of a porpoise exhibiting exquisite examples of osteo-arthritis. But no one certainly had traced gout in horses, nor in prehistoric bears nor in porpoises. Had syphilis anything in the world to do with either of the affections? Surely the distinctions between syphilitic and gouty disease were as broad and essential as could be. *Finally*, with regard to the connection of Charcot's disease with tabes, he felt in some difficulty, because he had never seen a case of joint affection supervening in the course of this disease. No doubt the explanation was that which Dr. Buzzard gave—that physicians generally did not, in medical practice, see these cases. They knew them from the ataxic side, and the ignorance which physicians consequently had on this subject stood in striking contrast to the extensive and accurate familiarity of surgeons with rheumatism and gout. His knowledge of the connection of these two forms of disease was almost or entirely derived from the cases of medical and surgical friends which he had had the opportunity of seeing, from museum specimens, and descriptions. But with regard to the connection between tabes and Charcot's disease, tabes itself was not at all such an absolutely fixed and definite entity that one could hang a still more doubtful disease on it. He thought it had suffered very much from want of accurate definition; there had been too much tendency to include under the wide category of tabes a number of cases which were wanting in many of its essential features; and, to gain better knowledge, it was not philosophical views as to its origin and connection with other forms of disease that were wanted, but more well-observed cases of joint affection in persons with locomotor ataxy, and more well-prepared sections of spinal cords in persons who had had that disease. In studying Charcot's disease or any allied questions, it was not by generalisations or theories, or by referring to occult causes, or by invoking dyscrasiæ, diatheses, blood-diseases, neuroses, and such vague and undetermined causes that any further knowledge of them would be gained. The very desire for explanation—for finding a cause—seemed to have retarded the progress of medicine.

The PRESIDENT said from some remarks made in a previous

part of the discussion he suspected that the exact position of the illustrious French physician to this question was not fully understood; and he thought that in one, or at most, two, sentences, he could put the matter with sufficient clearness and accuracy to prevent any further mistake. Charcot distinctly admitted that arthropathies might occur independently of any special influence from any special nervous disorder; nay, he even went further than that, he said that an ordinary osteo-arthritis might occur in the course of tabes dorsalis, and have no specific characters; but he contended that there were injuries and diseases, acute and chronic, of the nerves of the spinal cord and of the brain, which produced arthropathies of various kinds; and he furthermore contended that, amongst this class of arthropathies, there was one which, by its anatomical characters, and by the assemblage and progression of its clinical symptoms, possessed such an individuality as to deserve, and, indeed, demand, a specific name; and it was this form of joint disease occurring in tabes, and, as he alleged, peculiar to it, which the Society had been discussing.

Mr. MACNAMARA said it seemed hardly possible that those who had cases of Charcot's disease under their care, or who had taken the trouble to examine the remarkable series of patients brought before the Society, could have any doubts as to the existence of the affection. As a proof that the disease was not so rare as some people supposed, it was a fact that, through the kindness of Mr. Lunn and Dr. Larder, there had been no fewer than five typical cases of this affection brought to the rooms of the Society from the wards of a London Infirmary. The essential point of the suggestive questions put by Mr. Morrant Baker was, not as to the existence of Charcot's disease, but whether it depended upon lesions of the nervous system. He thought that joints should be regarded as interruptions in the continuity of bones. Embryology taught that such was their origin; histology enforced the same lesson, demonstrating the fact that the synovial membrane, like the periosteum, contained numerous osteoblasts, especially where it was reflected from the bone. Disease confirmed this idea; for in non-suppurative chronic affections of joints, osteophytes were built up from osteoblasts in the synovial membrane, in the same way as they were formed from the periosteum round the extremities of an ununited fracture, and in various forms of disease. Consequently, he failed to appreciate Mr. Hulke's argument that, because

osteophytes were a prominent feature in cases of rheumatic arthritis, and were also found in Charcot's disease, therefore, these affections were identical. The dried bones were very unreliable landmarks at the best of times, but utterly unsafe guides as indicating the nature of the abnormal action which during life had caused osteophytes to form on their surface. Moreover, as Dr. Buzzard remarked eleven years ago, there could be no reason why a patient suffering from Charcot's disease of the joints should not be affected by rheumatic arthritis, synovitis, or any of the other maladies to which human beings were liable. If one turned from the pathological to the clinical side of this question, we could not have a better illustration of the disease than that presented by the patient whom Mr. Barker had brought to the rooms of the Society. This man had for some years been suffering from symptoms indicating lesions of the nervous system, among others, perforating ulcers on the right foot. One morning, about two years ago, he went to his work as usual, but at breakfast-time, finding his right foot hot and uncomfortable, he took his boot off, and then discovered that the foot was so much swollen that he could not put his boot on again; by dinner-time the right leg and thigh were greatly swollen, but there was no pain in the limb. The patient was, however, obliged to return home in consequence of severe retching, lightning-pains, and in truth, a violent nerve-cyclone, out of which he emerged at the expiration of three days. His leg, however, remained swollen for two months, when it gradually resumed its normal size, and at present the limb, and its joint, were quite sound. In July last, this man was suddenly seized with rapid painless swelling of his left thigh and leg. This had not passed off as yet, and now his left knee was totally disorganised; the leg hung like a flail on the thigh and could be moved in any direction without pain. This patient had never had any symptom of rheumatism, gout, or syphilis. In Charcot's disease, therefore, there were invariably well-marked antecedent symptoms of lesions of the nervous system; there was sudden serous effusion into the affected limb, as well as into the joint; the joints were rapidly disorganised, without pain; the bones were easily fractured; there was seldom any fever; ankylosis did not occur; recovery was not unfrequent; the affected bones underwent a rapid rarefying osteitis. In cases of rheumatoid arthritis there is always some amount of fever, with marked synovitis, and long-continued rheumatic pains, and a progress from bad to worse, ending in stiffness

and often in ankylosis of the affected joints. The lesions and the symptoms of Charcot's disease and of rheumatoid arthritis were, therefore, dissimilar. Rheumatoid arthritis was characterised by well-marked symptoms and lesions, and it was fair to infer that a disease having entirely different characters was not induced by rheumatism. And, further, as Charcot's disease was never met with unless among persons suffering from a remarkable train of symptoms referable to disorders of the nervous system, he would not pretend to say if the neurosis was due to changes in the spinal cord, or, as Dr. Buzzard had suggested, to disorder of a nerve-centre controlling the nutrition of the diseased structures.

The PRESIDENT asked if he understood Mr. Macnamara aright, that there was always pain in chronic rheumatic arthritis, and that there was never condensation or eburnation in Charcot's disease?

Mr. MACNAMARA replied that, in chronic rheumatic arthritis, there was always rheumatic pain, and pain in the affected joint, tendons, and bursæ. In Charcot's disease, the pathological changes were destruction of the epiphyses of the affected bones, and sometimes the shaft of a bone by means of a rarefying osteitis, with osteophites round joints and along tendons, if long-continued motion of the joint had existed after the disease had set in. The osteophites, as in rheumatoid arthritis, were due to the irritation and pressure of the diseased ends of the bones against one another; the same condition was found in chronic traumatic synovitis as in the case referred to by Dr. Moxon, and was seen to perfection in ununited fractures.

Dr. BROADBENT remarked that there was nothing in the extent of his experience, and nothing in any new views that he had to present to the Society, which justified him in taking part in this discussion earlier, but the protraction of the debate might perhaps leave time for the few remarks he should make. It seemed to him that one of the most important points which had come out in the discussion was the conclusion by Sir James Paget that the disease in question, the articular affection arising from tabes, as described by Charcot, was a new disease. He scarcely thought that the effect and bearing of that conclusion had been rightly appreciated, because it seemed to leave absolutely no room for any identification of

this disease with chronic rheumatic arthritis. While Sir James Paget's authority was accepted as very great on any subject on which he spoke, on this subject his authority he (Dr. Broadbent) supposed was unrivalled. It was evident, from the place which chronic rheumatic arthritis took in Sir James Paget's work on *Surgical Pathology*, that it occupied his attention early, and it was certain from his late declarations that he had continued to interest himself in that and like subjects, so that his conclusion might be taken as one of special value; it seemed to Dr. Broadbent to reduce to a level of mere cavil the attempts which had been made to connect the two diseases by intermediate links, especially when those links were based on dried specimens of bones. But even were the interpretation of morbid specimens by Sir James Paget less equivocal, he agreed with Mr. Macnamara, Mr. Barwell, and others, that the clinical features of the two diseases established a complete distinction between them. In the history of disease, vital processes were of greater importance than morbid anatomy, and the life-history must be allowed a determining influence in the opinion to be formed as to the nature of any disease. He thought that at least four different modes could be traced in which joints became deformed in a way which gave results similar to those described as belonging to osteo-arthritis, or chronic rheumatic arthritis. For example, two distinct diseases were included under that term, the early disease of which examples were seen in young females, and the late disease of morbus coxæ senilis; those seemed to him, although included under the same name, to be really two distinct clinical diseases. Then, besides chronic rheumatic arthritis, there were the effects of tabes to be considered; and he thought there were very similar results merely from neglected chronic synovitis, of which an illustration was quoted by Mr. Morris. But, as he had said, he thought the clinical history, which had been minutely described by Mr. Barwell and Mr. Macnamara, and had been referred to by Mr. Page, of itself established this great distinction. There was then a new disease, and the important point in this inquiry was what was the new morbid factor. Sir James Paget, in one part of his remarks, he thought had coincided very much with Professor Charcot's conclusion; but in other parts he had sent them to search for the causation of this particular result as a sort of a resultant of possible different morbid processes, and had left them to determine between rheumatism, gout, rheumatic arthritis, and syphilis, as the dominating influence

which gave rise to this particular condition. It seemed to him that research in this direction would bring results of no value. With regard to syphilis, indeed, if they admitted (as he certainly would) the relation between tabetic arthropathy and tabes; and if they admitted (as he thought they must) the extraordinary predominance which syphilis had among the antecedents of tabes, then there seemed some sort of remote connection between syphilis and this particular disease, and perhaps between the greater frequency of tabes, and the greater frequency, as he thought, which there had been of late years of syphilitic disease of the brain and the spinal cord. He thought all these might be, to some extent, traceable to the slipshod treatment of syphilis which prevailed for a great many years when antimercurial ideas exercised considerable influence, and led to neglect of radical treatment of early syphilis. At any rate, in his own experience, both in regard to tabes and to syphilitic diseases of the nervous system, it was comparatively rarely that he found that a patient had undergone anything like adequate treatment for the primary syphilis. This was a mere parenthesis. It seemed to him that no result of any particular value would be reached, if it were determined that it was through a rheumatic arthritis, or through gout, or through any other tendency, that tabetic disease acted in the production of this joint disease. But it seemed to him that it would be quite another thing if, for example, Charcot's disease were regarded not as chronic rheumatic arthritis intensified by nerve-disorder, but if the relation between chronic rheumatic arthritis and Charcot's disease were looked on as a relation established by like causation, that both were effects of the derangement of nutrition through the influence of the nervous system. In one, in chronic rheumatic arthritis, it was reflex; in the other, it was the effect of the persistent irritative lesion in the spinal cord. If that were the relation established, a like causation in this way, then it seemed to him that an important step had been gained; but it would be an explanation of ordinary chronic rheumatic arthritis through tabes, and not an explanation of the tabetic disease through chronic arthritis. This was very much the position which had been taken up by Dr. Ord, and argued for some considerable time. It would, perhaps, be premature to say that that view was established, but certainly the weight of evidence tended strongly in that direction. There seemed to him to be no explanation possible of this joint disease, except a disturbing influence through nervous

agency. Dr. Moxon had found fault with the illustration of herpes zoster, and, of course, there was a contrast between the definite duration and spontaneous cure of herpes zoster as compared with lesions that came in the course of tabes. But Dr. Moxon had left out of sight the fact that herpes zoster left cicatrices; that herpes attacking the region of the fifth nerve would damage an eye permanently; that, from time to time, cases occurred in which the entire area of skin supplied by the fifth nerve became atrophied, and thinned, and shiny after an attack of herpes ophthalmicus. He thought that, with certain qualifications, the comparison held good, and was a fair illustration. Mr. Hutchinson had suggested that the explanation of these tabetic lesions might be the use of a diseased joint, which was permitted by the loss of sensation, or the loss of sensibility to pain, and Dr. Moxon had maintained the same view. But surely a view of this kind admitted of illustration by facts. Was it in accord with experience that those cases were the ones to suffer from this disease, in which there was the most exaggerated flinging about of the limbs, and in which there existed the most striking anæsthesia? He thought that the reverse was the case; and while it was not in every case that there were the gastric crises, to the association of which with this disease Dr. Buzzard had called attention; while that association had not been, in his own experience, absolute, yet that was the rule; and those were cases in which the violence of the movements and the degree of anæsthesia were not particularly marked. Cases were going about with impunity in which these exaggerated movements were carried to their greatest extreme; and there were others in which the ataxy had actually to be discovered after the occurrence of these tabetic phenomena. He therefore had no doubt whatever in admitting the accuracy, the exactness, of Professor Charcot's views, as stated by the President at the commencement of the meeting. In conclusion, he thought he might be permitted to express before the Society his regret that, in the vote of thanks to Professor Charcot for sending his specimens, which had been moved by Dr. Moxon, there was a tone of sarcasm. Whilst all would be sorry to miss Dr. Moxon from their meetings and from the periodicals, while he thought that the loss of his criticism would be a loss to medical science, yet he did think that, on this occasion, it would have been better omitted. Of course, if he had been speaking otherwise than as moving this vote of thanks, it would not, so to speak, have committed the Society, but it had, in a way, almost compromised the

Society that this tone should have been employed in moving this vote of thanks. He further deprecated the bringing in of an allusion to the cases of hysterical hemi-anæsthesia—in order to throw discredit on M. Charcot's work in connection with this particular disease—burlesqued as they were in the amusing scene which Dr. Moxon related with regard to his gynæcological pupil or colleague. He thought it only fair that there should be some statement made in this Society of the part which Professor Charcot took in relation to the subject. It was the honour of the Society which had moved him to make these remarks; and he would only further say that Professor Charcot's attitude was throughout that of a scientific observer, who was as far from partaking in any extravagances which might have attached to this idea as Dr. Moxon himself. He was quite sure that Dr. Moxon, seeing, as probably he would after what had been said, that the occasion was of a special kind, would be the first to regret that his remarks should be understood as a slight upon Professor Charcot.

The PRESIDENT said that, in Dr. Broadbent's reference to the speech of Sir James Paget bearing upon the antecedent causation of arthropathy and tabes, he thought Dr. Broadbent had omitted to mention that Sir James spoke not only of antecedent processes, but most particularly and specially of the combinations of antecedent processes, such as the combination of gout and syphilis.

Mr. CLEMENT LUCAS thought that many would regret, especially those who, like himself, held the more advanced views concerning this disease, that the weight of authority in the discussion had been thrown almost wholly on the side of conservatism. He attributed this in great measure to the lead given by Sir James Paget in his brilliant address at a former meeting. When he (Mr. Lucas) listened to that oration he felt himself fairly carried away by its eloquence, but when he reflected on what had been said, he found himself scarcely able to agree with any of Sir James's conclusions. He quite wished that someone would rise at once and overthrow what he believed to be the erroneous doctrines of that speech. But the distinguished surgeons who followed—Mr. Hutchinson, Professor Humphry, and Mr. Hulke—seemed to be under the same influence. They used very similar arguments and arrived at very similar conclusions. Mr. Hutchinson and Professor Humphry, it was true, saw very great distinctions

between the pathological results characteristic of Charcot's joint disease and those characteristic of osteo-arthritis, but they could not disentangle themselves from the idea of osteo-arthritis—that this disease must be osteo-arthritis modified by some other disease added to it. Mr. Hulke seemed to take firmer ground; and he used an argument which at first sight appeared to be a powerful one, for he said: "I take here a joint which is from the upper extremity of one who is ataxic, and it shows atrophy, wearing away, characteristic of Charcot's joint disease; and I take here another joint from the lower extremity of the very same patient, and here are outgrowths of bone similar to what you have in osteo-arthritis; therefore, these are one and the same disease." But these joints were in different stages of inflammation, and he would ask, Was it not true that all groups of diseases were very much alike at their commencement? Was it not true, for instance, that fevers at their commencement were undistinguishable until the rashes became developed? Inasmuch as there were only certain anatomical structures in the joint which could undergo inflammation, must not joint diseases of necessity have certain similarities, and, *a fortiori*, he would say, Was it not certainly true that osteo-arthritis and Charcot's joint disease, being both of them chronic joint diseases, must therefore, of necessity, present certain similarities? To his mind, it would be very extraordinary if it were not so. If any further argument were required in opposition to what Mr. Hulke had said, he would take it from the speech of Dr. Moxon. It was well known that Dr. Moxon disagreed with everything Professor Charcot had described. He did not believe that this was a distinct disease; and to disprove it he brought a joint which was from a case of traumatic arthritis. He said, "Look at my case of traumatic arthritis, and see the wearing away of the humerus, similar to what you get in Charcot's joint disease." But those who examined that specimen further would have seen that, arising from the scapula, were outgrowths of bone and pieces of bone attached by fibrous tissue, not unlike the outgrowths in osteo-arthritis. Here, then, was shown a connection between three diseases, viz.: traumatic arthritis, Charcot's joint disease, and osteo-arthritis. Why? Simply because they were all chronic joint diseases. The question had been asked, Was this a new disease? Sir James Paget gave a double answer; he said, "Yes, in a general sense it is a new disease, but in an especial sense it is the result of a combination of diseases." To Mr. Lucas's mind that was a combination of errors. In one sense

Sir James declared this as a new disease. Why? Because no old specimen could be found in any of the museums. He thought that was a very fallacious argument, for collectors of specimens collected what they believed to be typical specimens; and this disease had hitherto been confused with osteo-arthritis. All the museums in the world would not hold the specimens of osteo-arthritis which might be collected; therefore only typical cases were preserved. These cases of Charcot's joint disease were not typical of osteo-arthritis; they were different, yet similar, and so rejected. Moreover, curators of museums annually or periodically visited their shelves, and threw away the specimens which they did not consider typical, or not good for teaching purposes, or not complete as to their histories. He perfectly remembered that when Dr. Moxon was curator of the Guy's Hospital Museum he threw away a great many specimens that he considered useless. How, then, was it known that he did not throw away all the cases of Charcot's joint disease? In his present state of scepticism he might still be inclined to throw away these specimens. As to Sir James Paget's second conclusion, that this was a result of a combination of diseases, that, he thought, was a most erroneous doctrine. Sir James said that there might be three or four or or more diseases acting to cause this disease. If one had to search for three, or four, or a dozen diseases before determining what was the matter with a patient, he would be dead and buried before one knew what was the matter with him. Sir James Paget had said that syphilis ran a different course in a gouty person and in a strumous person. That was true enough; but did not all diseases run different courses in different persons? Yet syphilis was syphilis all the world over. It gave rise to a number of secondary and tertiary lesions, but it was the same disease all through, and to those who knew all the different lesions, and knew them well, they were each and every one of them characteristic; therefore he thought Sir James Paget's illustration most unfortunate. Then, if the pathological specimens were not sufficient to distinguish one disease from the other, there were the clinical signs, and he thought it was unfortunate for the Society that more attention had not been paid to the clinical distinctions. He was happy to hear Mr. Macnamara referring to the clinical signs, and he thought sufficient credit had not been given to Mr. Barwell for what he said about them. These were chiefly the sudden onset, great swelling, the rapid disorganisation which often took place, the peculiar joints affected, and the

very peculiar painlessness of those joints. He should like to refer, in illustration, to the case he had brought before the Society last year, when they first had Dr. Moxon's experience of the disease, which was to the effect that after seeing many cases of locomotor ataxy, Dr. Moxon had never seen a single case of Charcot's joint disease. That case was of interest in this respect, that the symptoms of ataxy were so little developed that they had not attracted the attention either of the patient or of his medical advisers. He had come among Mr. Lucas's out-patients suffering from an inflamed foot. The speaker had studied this disease with great care, and for some years had worked at the cases which he had seen under Dr. Buzzard and Dr. Hughlings Jackson, as well as others which were brought to the Hunterian Society. These were all cases in advanced stages, and with the disease in this form he was familiar; but when this case came to him among his out-patients he saw something that was new. Here was a man with a great swollen foot, red, puffy, but not œdematous, extending from the malleoli up to the bases of the metacarpal bones. Was it gout? No; it was not intensely painful like gout. The great toe had never been affected; there was no enlargement of the veins such as accompanied gout. Was it osteo-arthritis? He did not recognise osteo-arthritis suddenly coming on as this had done in the tarsal joints, the others being unaffected. Here the foot suddenly inflamed, and the man was laid up for three or four days; and then he came walking about upon it. He asked, "Was there any other joint affected?" The man said, "I have something the matter with my elbow, but it does not trouble me very much." Upon his arm being stripped, there was seen a most extraordinary elbow-joint, a great bossy swollen joint, with which one would have said it was impossible for a man to work, and yet he had been working for two years with it. When it first became inflamed he came amongst the out-patients at Guy's Hospital, and the joint had been strapped. Then he ceased to attend and had worked with it for two years. Now, neither this joint nor foot, in the speaker's opinion, could be classified under either osteo-arthritis or gout. He was pressed by the students for a diagnosis but could not give one. He had Charcot's joint disease in his mind and looked at the man's pupils, but found they were not contracted. He asked if there were lightning- or girdle-pains, but the man denied them. He then let the man go, and wrote on his letter, "Inflamed foot and disease of the elbow," because he could not give a better diagnosis;

and he claimed some credit for recognising that the case was not one of ordinary osteo-arthritis, gout, or rheumatism. On the man's next attendance he went more thoroughly into his case. When he was told to shut his eyes he was found to be unsteady; he had the Argyll-Robertson pupils; and his knee-jerks were quite absent, yet he was himself unconscious of anything being the matter except these joint affections. In the speaker's opinion this was a newly recognised disease—one that had been unravelled from a confusion of diseases—and he believed a few years would settle it absolutely, and that people would look back to this discussion and wonder that those great men who had spoken in doubt had made such great mistakes. Here was a new disease, let there be but the courage to recognise it; here was a new field for more exact clinical inquiry, let it be pursued without prejudice; and, lastly, let there be but common honesty to give the credit of the discovery to that great man to whom the credit was due.

Dr. MACLAGAN desired to make a few remarks from a physician's point of view. He would simply condense all that he would say on the relation which it was possible that Charcot's disease bore to rheumatism, and gout, and rheumatoid arthritis, by saying that he would entirely homologate all that Dr. Pye-Smith had said. He thought the disease was perfectly distinct from all three. But, dismissing that point, he would proceed to discuss the pathogenesis of Charcot's disease. He did not see how this disease could be divorced from tabes dorsalis; there was no evidence that it had ever been found unassociated with that disease; though it occurred in only a very small percentage of cases in tabes dorsalis that did not show that there was no causal relationship between the two. It was not said, for instance, when a parturient woman had a white leg, that it had no connection with her recent confinement, simply because the immense majority of parturient women had no white leg; nor because only one leg was affected was it said that the local condition of the limb had nothing to do with the general condition. In the same way with Charcot's disease, simply because it was an occasional thing, and did not affect all the joints and all the limbs, it could not be said that it had no connection with tabes. The question was, What was that connection? The general characteristics of a tabetic limb were, that its general vitality was lowered, there was a loss of sensibility, and a tendency to spontaneous fracture. That was an important element in the pathogenesis of the

disease, which had been lost sight of in the discussion. But all these characteristics it had only in common with other forms of paralysis—general paralysis, for instance, as Dr. Moxon showed. The special peculiarity of a tabetic limb was the loss of the power of co-ordinate movement. Attention must not be concentrated solely on the posterior spinal cord, or on the muscles. The posterior columns of the cord were no doubt the seat of disease; the muscles must have lost their co-ordinating power; the bones were also affected, as were the ligaments—the fibrous, ligamentous, and tendinous parts of the joints. He would confine his observations to the knee-joint, though his remarks applied to all the others. The absence of patellar reflex was looked on as a characteristic sign of the disease. He would simply direct special attention to this phenomenon as evidence of the loss of sensibility and diminished vitality of the ligamentum patellæ; and what was known to occur in the ligamentum patellæ might reasonably be assumed to occur also in other tendinous and ligamentous attachments of the knee-joints. Ligaments were absolutely as essential as the muscles to co-ordinate movement; the muscles could not produce co-ordinate movement unless the ligaments bound the bones together. This he thought had a most important bearing on the pathogenesis of Charcot's disease; for one could not read the accounts of the early stages of that disease as given by Professor Charcot, Dr. Buzzard, and others—one could not look on the casts of limbs in the next room, without seeing that it was the ligaments that had given way; the heads of the tibia and fibula had lost their relationship to each other and to the patella; the swelling of the limb which had taken place was not an ordinary œdema, but it was such a swelling as he presumed would result in a limb with diminished vitality and in which all the soft parts were being strained by the absence of ordinary ligamentous support, such ligamentous support being absent at a time when the muscles retained a great deal of the power of movement, and were not in the condition of a paralysed limb at all. That seemed to be the condition that obtained in the early part of Charcot's disease. The ligaments gave way first, just as in later stages the bones gave way by spontaneous fracture, and they gave way without pain simply because of the loss of sensibility. In the same way, if the ligaments gave way, by-and-by, in the more advanced stages, the bones would be rubbing against one another, and with the diminished vitality the bones would waste and wear away, and in time there would be produced

the pathological change which was characteristic of the advanced stage of Charcot's disease. The limb, in short, of such a man suffering from tabes was, so far as vitality was concerned, the limb of a middle aged man. In connection with that, he would point out that Charcot said that this condition always occurred at first in the lower limbs, and when it occurred later and in the upper extremities it was a very advanced disease. It must be borne in mind that Charcot's observations were all made in the Salpêtrière; and the people there were in the habit of walking and pottering about on their legs but never using their arms; and naturally enough, their legs went wrong. There was a case to which Mr. Lucas referred, which had been exhibited there by Dr. Duckworth at the last meeting, in which the elbows were affected. But that man was a cooper, and used his arms a good deal more than his legs. That was a point to which he wished to direct attention, that they were apt to lose sight of among the attendant phenomena. The fibrous tissues of the joint affected were absolutely essential to co-ordinate movement, and there was a good deal of evidence to show that they primarily suffered in Charcot's disease. With regard to Professor Charcot, he entirely homologated all that Dr. Broadbent had said; his regret was that Dr. Moxon did not go a little further back in Professor Charcot's career. Seventeen or eighteen years ago, Charcot had first pointed out the existence of the minute aneurysms which were now associated with cerebral hæmorrhage; he threw more light than any other man on that subject, and had done excellent work in connection with it.

The PRESIDENT asked the speaker if he adopted Dr. Pye-Smith's assertion that a new disease was impossible.

Dr. MACLAGAN said that Charcot's joint disease had no connection, clinically or pathologically, with rheumatism, gout, or rheumatoid arthritis.

The PRESIDENT said that that was not exactly the question. It was stated, in reply to Sir James Paget, that a new disease could not arise, that a new disease was impossible. That statement was made by Dr. Pye-Smith; did Dr. Maclagan adopt it?

Dr. MACLAGAN did not say that a new disease was impossible, but there was not sufficient evidence that this was one.

Dr. BASTIAN said that his own experience of this particular joint affection was extremely limited. He had seen several well marked cases in the practice of others; but, though he had been on the look-out for the disease since 1868, when Charcot's description first appeared, he had never yet had a single marked case under his care, although he had seen a large number of cases of locomotor ataxy. He was compelled to believe, therefore, that this joint disease was not a necessary appanage of the ordinary lesions of locomotor ataxy, and that, he believed, was precisely M. Charcot's own position—that this joint affection, when it occurred, was to be looked on as the result of some altogether unusual extension or incidence of the ordinary pathological processes. M. Charcot's first view, at all events, was that it was the result of an extension of a pathological process, from the posterior nerve-roots and posterior columns into the grey matter of the anterior cornua. Whether that was the right view to adopt, at present, would seem perhaps open to doubt. He thought it necessary to call attention to this point, because, in the remarks both of Dr Moxon and of Dr Pye-Smith, it seemed to have been lost sight of; they argued as if the affection were assumed to be a result of the ordinary lesions of locomotor ataxy in the posterior columns. Then, again, it seemed to him that, if locomotor ataxy were such a very common thing as it was known to be, comparatively speaking, and this joint affection were so rare, these facts of themselves must tend to throw a considerable shadow of doubt across the views of some speakers, who had laid stress upon the fact of the loss of sensibility in this disease, combined with exaggerated movements, being of themselves adequate to produce Charcot's joint affection of this kind. A further reason in the same direction tending to discredit that view was to be found, as it seemed to him, in the fact that, in a great number of these cases the joint affection in question was said to manifest itself at quite early stages of locomotor ataxy. In several of the recorded cases it had manifested itself quite early, and in others at irregular periods of the disease. It seemed to him that in inquiring into the connection of this disease with neural disturbances the question ought to be looked at in the light thrown upon it by other simpler joint affections, which were more clearly related to certain disturbed neural conditions. He would refer specially to two sets of such cases. First, there was the fact that, in certain cases of hemiplegia, there were joint affections in the form of a simple arthritis, occurring in the paralysed limbs only, not on the

opposite side, and that in those cases there was often the coexistence of tenderness over the nerve-trunks and sometimes atrophy of the paralysed muscles. He believed that, in nearly all such cases, there was evidence of a sclerosis in the lateral columns of the cord, and he knew that in some of these cases it had been actually ascertained by M. Charcot himself, that there was an extension from the sclerotic region in the lateral column of the cord into the contiguous anterior cornu; so that, in this case, there might be a relation between the joint affection and changes in the grey matter, or the coincident changes in the nerves of the limb. There was another set of cases in which there was the association of simple arthritic inflammations with the onset of nerve affections. He alluded especially to progressive muscular atrophy. This had been observed not unfrequently of late years, especially since attention had been called to it. About two years ago he had seen a very remarkable case, in which the progressive muscular atrophy was of unusually rapid onset, and in which the joint affection, pain, tenderness, and inflammation about the joint was so marked as to have caused this disease to be regarded, at first, by the practitioner under whose care the patient came, as one of rheumatic fever. There could be no doubt that, in a certain proportion of these progressive muscular atrophy cases, a simple joint affection declares itself. Here, again, there was disease in the anterior cornua of the spinal cord, and, possibly, changes in the nerves in connection with those anterior cornua. It seemed to be impossible to define more closely the pathogenic relation between these simple arthritic affections and the two nervous diseases with which they are associated. In regard to Charcot's disease there might be extensions of the morbid process from the posterior columns into the grey matter, and, also, there was a reasonable probability that there might be implication of the peripheral nerves in relation with the affected joints. He thought it very important not to lose sight of the fact that, during the last two years there had been cases of locomotor ataxy of an ordinary kind, so far as their clinical characters were concerned, but in which post-mortem examination had shown not central changes in the spinal cord, but peripheral lesions only in the nerves. Several of these cases had been recorded. Of course, further information about them was wanted; but it seemed to him quite possible that, if symptoms of locomotor ataxy could be produced in this way by diseases of peripheral nerves, it was also possible that, in the ordinary cases of locomotor ataxy, cases in

which there were centric changes, there might also be some changes in the peripheral nerves, and that some of the changes might be specially related to the production of Charcot's joint affection. This was a subject which future investigators would have to decide. Then, even if it were supposed that there was a causal connection between the occurrence of these joint affections and these lesions in the nerves, it did not seem to him at all necessary that the existence of special trophic nerves should, therefore, be postulated. Some of the best marked trophic lesions seem to occur from the cutting off of ordinary nerve impressions from parts which were accustomed habitually to receive such impressions. He would mention, under this head, the secondary degenerations that occurred in the brain, those in the spinal cord, and also the degenerations that occurred when a motor nerve was cut across, and, again, the extremely well-marked atrophies that occurred in muscle under those conditions. Here, when there was a cutting across of a motor path in any of these situations, there was, as a result, a disturbance of nutrition below; and the part so affected either underwent fatty degeneration or a process of atrophy. At least half of the trophic lesions seemed to be of that kind; so that it seemed to him as if the mere cutting off of the neural excitations which were accustomed to pass along motor tracks might of itself produce one important set of these trophic changes in physiologically related tissues. He believed that at least a large proportion of the remaining known trophic lesions might be produced by unnatural influences passing along sensory nerves, on account of pathological changes of an irritative nature occurring either in their track or in the nuclei of the spinal cord and medulla at the root of such sensory nerves. In this way the trophic changes occurring in the skin might be explained likewise, that was to say, the various eruptions of the skin that occurred, some in the course of herpes zoster, some in the course of locomotor ataxy itself, over the course of painful nerves; the trophic ulcerations that occurred in the skin, and the ulcerations of the cornea, which had always attracted much attention; as well as those peculiar atrophies of the skin to which attention was first called by Sir James Paget, the so-called glossy skin, which he showed to be due to nerve-irritation. Although, as a rule, sensory nerves transmit impressions towards the centre, still, if the nerve were irritated in its course, or if its nucleus were irritated, there seemed no reason why impressions might not be sent to the periphery from which these nerves came. It had

been shown by physiologists that a nerve might transmit impressions in both directions. It seemed to him to be quite possible that this occurred, and that, therefore, if there were an irritating lesion in the course of a sensory nerve, or an irritation affecting the nerve-cells at the nuclei of one of these nerves, there might arise, in that way, a constant flow of unnatural nerve influences going to the tissues which might suffice to disturb and upset the balance of their nutrition. It seemed to him possible that, just as the skin affections were produced in this way, so the joint affections might also be the results of lesions occurring in the sensory nerves or the sensory nuclei rather than in the motor nerves or their nuclei. He threw that out merely as a suggestion, because he thought at present the point was one which could not be proved, and it must rather remain for future investigation to decide the question as to the exact pathogenesis of these joint changes. Whilst he thought it quite possible, therefore, that some one or other of the changes occasionally met with in locomotor ataxy, either some of the changes in the grey matter or some of the changes in the peripheral nerves, might act in this way as irritants and set up an inflammatory or perverted nutritive condition in the joint such as is met with in Charcot's disease, it was impossible to go further, and to say precisely how the morbid condition in the joint is produced. It seemed to him that if the disease were initiated in some such way as he had indicated, it might progress to a certain extent, and that then there might also come into play those causes whose influence had been insisted upon by many, that is to say, the continued use and movement of the anæsthetic joint which might tend greatly to aggravate the previous condition. With regard to the notion that a causative connection exists between the processes that may occur as part of the phenomena of locomotor ataxy and this particular joint affection, he thought there was at present good evidence for believing that that was so, and therefore that M. Charcot's position generally was one which must be fully conceded; but that, when one came to the details, and to ask exactly what was the precise pathogenesis, present knowledge would not enable an opinion to be given. In reference to the question whether this was a new disease or not, he said that it seemed to him that those who contended that this was a new disease drew a conclusion which was diametrically opposed to the conclusions of M. Charcot. He would submit that our nervous systems had always been submitted to much the same kind and range of influences during

periods of civilisation at all events; and it would be extremely difficult, therefore, to understand how, if this disease were one really due to the disturbing influences of certain not infrequently recurring pathological conditions of the nervous system, it should be new. On the other hand, it was quite possible to explain in ways which had been already hinted at, how it was that the joint conditions in question had not previously been recognised as an occasional appanage of locomotor ataxy. He thought that a distinct relation between this disease and locomotor ataxy should be admitted, although the exact pathogenesis could not be precisely defined.

Mr. Howard Marsh referred to the interest which Sir James Paget had introduced into the debate by his remark on the subject of the evolution of disease. From the fact that no specimen of Charcot's disease was to be found in the collections either of Hunter, Langstaff, and Stanley, Sir James believed that the affection did not exist, at least in anything like its present frequency, in their day. Had it existed it seemed nearly impossible that they could all have failed to preserve some examples of it. He therefore held that the disease was new, and that it had arisen out of the mingling of various constitutional affections, the chief among which we might justly suspect had been syphilis, rheumatic gout, and a special tendency to disease of the spinal cord. While recognising, in common with all those who were competent judges, Sir James Paget's great authority on such a subject he knew that Sir James would be the first to wish that his views should be freely discussed. Stanley worked at diseases of the bones down to about 1850. So that by Sir James's argument, the disease had become prevalent only within the last thirty or forty years. But, though Sir James had passed himself over, we could not forget what his labours in surgical pathology had been, nor that they extended down at least to 1865. So that if it were safe to conclude that the disease did not exist, or was very rare, down to 1850, because Hunter and Stanley had not recorded it, we, holding that it was fully as unlikely that Paget could have failed to notice it or that Stanley had done so, must believe that it had been developed since 1865, a conclusion which it was difficult to accept. Even since 1865, though probably no one would expect his claim to an equality with Hunter, a very large number of able pathologists and clinical observers had been at work, yet they had failed, till within the last few years—that is, for some ten years after

Charcot had described the disease in 1868—to bring forward any considerable number of examples, though it could not be doubted that numerous instances of it were in existence. Now, he thought that if in these more recent periods in which pathology and clinical observation had made such rapid advances, these cases had been so generally overlooked they might also have passed unrecorded by Stanley, and by Hunter, who, it must be remembered, was a student not so much of pathology as of biology and natural history. Three difficulties seemed to present themselves in respect to the view that this disease had been the product of evolution within the period named by Sir James Paget. In the first place, it did not appear out of what new elements or combinations it could have arisen, for syphilis and osteo-arthritis had been mingled with each other, and, as we must believe, with diseases of the nervous system long before even the time of Hunter. Secondly though all must own the strong probability that many diseases must undergo some modification by admixture with other forms, as time went on, these changes seemed to be very slowly effected, and the thirty or forty years assigned by Sir James Paget, and which did not cover the natural period even of a single life, or include even two generations, appeared too short for the evolution of a new disease presenting such remarkable features as those observed in the affection under discussion. Thirdly, in any process of evolution the original form gradually faded out, and the new form took its place; but, so far as we knew, syphilis, for example, except that it had assumed a milder type, was very much the same now as it was a hundred years ago. In endeavouring, therefore, to estimate the probabilities of the case, Mr. Marsh was inclined to believe not that the affection now termed Charcot's disease was really new, but that it had been till recently overlooked. The relationship of Charcot's disease to osteo-arthritis he believed was a more difficult question to solve than many appeared to think. Without venturing to assert their full identity, he must confess that he was unable to see any proof that the two affections were absolutely distinct from each other. In his opinion, the points of resemblance outweighed those of clear difference. Dr. Duckworth had allowed, and Mr. Hulke had conclusively shown, that in its morbid anatomy Charcot's disease was often not to be distinguished from osteo-arthritis, and the same must be said of its clinical features. No doubt by taking extreme cases very marked difference, both pathological and clinical, could be observed; but this was equally true of other

diseases, for instance, of syphilis; but these differences became far less obvious when intermediate examples were taken into account. He was aware that the argument that because two diseases could be connected by a number of intermediate links there was no essential difference between them, must not be carried too far; for this was a form of argument by which it was easy to show that there was no difference between a saint and a sinner, or between black and white. Still, it was entitled to considerable weight when the cases that lay between the two affections were carefully selected. He had himself recently seen a case in which an affection of the hip which begun ten years ago as a well-marked and apparently ordinary example of osteo-arthritis, had very gradually acquired the characters of a typical Charcot's joint; the limb had become three inches short, and completely everted; the head of the femur was gone, and the upper end of the bone could be made to slide upwards on the pelvis; large heaps of loose bone could be felt around the joint, and there was a large collection of fluid in Scarpa's triangle. The patient had no pain and could walk long distances on the limb. But although this joint now presented features which many asserted were never met with except in association with tabes, the patient showed no indication whatever of disease of the nervous system. But if, failing to discover any crucial differences between these affections in regard either to their pathological anatomy or their clinical history, we turned to the question of their essential nature, we were at once met with the difficulty that we were ignorant of the true nature of osteo-arthritis. Until we knew what osteo-arthritis really was, whether a disease, as many held, of nervous origin, or as others suspected, himself among the number, a common name for several distinct affections not yet distinguished from each other, we were not in a position to say how, if at all, Charcot's disease was related to it. It seemed wiser at present rather to continue our studies of these diseases than to venture upon the expression of strong opinions respecting them. Looking merely at the morbid anatomy of Charcot's disease, the affection seemed, as Dr. Buzzard has remarked, to be located primarily in the bones, and there was evidence to show that the condition was no mere failure of repair, but an active and destructive disease. No bones that were suffering merely from deficient repair would present the features observed in these cases, but whether the change was due to the direct action of the nervous system or not, it seemed impossible at

present to determine. Sir James Paget had observed that the nervous system had a power of actually producing disease, as, for example, in the case of herpes zoster. Some might remember that a few years ago Mr. Hutchinson had shown a skull and lower jaw in which, corresponding to the distribution of the fifth nerve, on one side, the several bones were intensely hard, greatly thickened, and deformed by irregular nodular swellings. In this case, the only thing common to the affected bones was their nerve-supply. Such examples were both rare and obscure, but, as far as we could read them, they seemed to point to some abnormal influence of the nervous system. As far as mere naked-eye appearances went, the change in the bone in Charcot's disease somewhat resembled that observed in some cases of mollities ossium, in which the bones became rarified and so brittle and soft that they bent, and broke very easily. A change somewhat similar in its effect was also observed in cases of fragilitas ossium, some of which were of a very remarkable kind. The origin and true nature of these affections of bone, however, were, at present, altogether obscure, and the same might be said of the disease which Sir James Paget had lately so fully described under the name osteitis deformans. It was by carefully studying this whole group, and taking not a narrow view of Charcot's disease, but by tracing out all its general and local characters, and its association with other affections, that we should at length ascertain its true position in pathology and clinical medicine.

Dr. Barlow said that, so far as he had followed the discussion, those who had been opposed to the specific origin of Charcot's disease had maintained that it was indistinguishable from rheumatoid arthritis. He should like to ask whether rheumatoid arthritis was itself so definite a clinical entity that this really added anything to the knowledge of the subject. The anatomical outcome of rheumatoid arthritis, thanks to the labours of Adams and others, was perfectly well known; but members should consider the very many different ways in which rheumatoid arthritis might begin. How would any pathologist, who had examined the bones of old people, distinguish many cases of senile degeneration of cartilage and bone from slight cases of rheumatoid arthritis? He differed from Mr. Macnamara with respect to the question of pain. It seemed to him that, in many cases, old people might have most extensive changes at the ends of the bone without any

pain at all. There was another point, to which Dr. Pye-Smith referred. Although he agreed that acute rheumatism was sharply defined from Adams's disease, rheumatic gout, yet there were unquestionably cases of young subjects who had begun with attacks of acute rheumatism, which had relapsed, and which had ultimately developed rheumatoid arthritis. Again, there was the relation of gout. There were cases like that to which Dr. Pye-Smith had referred typical cases of rheumatoid arthritis during life, in which, nevertheless, post-mortem deposits of urate of soda were found in connection with outgrowths of bone. A number of other cases had also been seen; so that it was clear, whatever the relation might be, that one did find outgrowths of bone, and so forth, in connection with unquestionable gouty deposits. Further, rheumatoid arthritis, as he had himself seen, might supervene upon acute specific disease. He remembered seeing an old lady, about eighty, who had typical malum coxæ senile, with the knee-disorder, and characteristic affection of the joints of the fingers, in whom the disease commenced with a virulent attack of scarlatina. During this attack, the knee and hip were affected; that was the starting-point of her rheumatoid arthritis. He could also quote a case of measles in a child, which led to a typical attack of rheumatoid arthritis supervening immediately upon it. Then there was the moot point of gonorrhœal rheumatism. Mr. Hutchinson and others, whose authority was undisputed, admitted that gonorrhœal rheumatism could not be distinguished anatomically from rheumatoid arthritis. He remembered two cases of spondylitis deformans, both in men aged about forty. In one case it had begun with an attack of acute rheumatism, and in the other with an attack of gonorrhœa; the latter not only had spondylitis deformans, but he had the shoulder and elbows and one knee affected, and the characteristic deformity of fingers. In every respect, it was a typical example of rheumatoid arthritis. Lastly, there were cases, to which Dr. Ord had drawn attention, where rheumatoid arthritis began in connection with dysmenorrhœa; as the dysmenorrhœa diminished, the rheumatic manifestations subsided. The outcome of what he had to say was, that rheumatoid arthritis was merely an anatomical term; it was a description of a morbid anatomical product, which could be brought about by many different processes. Was it reasonable to say that all these processes were identically the same, because the anatomical result was the same? Even granting that some of the cases of Charcot's

disease were indistinguishable anatomically from rheumatoid arthritis, he submitted that this added nothing whatever to knowledge in that category; it was the life-history which must come into consideration. He would refer, on the other hand, to the affection of the joints, and structures round joints, in connection with disease of the spinal cord. There were not only the hemiplegic cases, and cases of muscular atrophy, of which he had seen one notable example, exactly like what Dr. Bastian had described, but also that very common disease, infantile paralysis. If anyone would examine a case of old infantile paralysis he would find a very remarkable condition of joint. He alluded especially to the hip-joint. In some respects, the hip-joint in old cases was not very unlike a joint in locomotor ataxy. The mobility of the joint was remarkable, and also the looseness of the ligaments. Furthermore, the upper end of the femur was quite atrophied; all the bony prominences were bevelled down; and altogether there was an extremely atrophic condition. But if exception were taken to this, he would refer to a condition sometimes found during the acute stage of infantile paralysis, so that it was distinctly related to the acute onset of the disease. He had seen two cases in children, and one in an adult, similar to the case to which Dr. Stephen Mackenzie alluded a few weeks ago, wherein there was a condition of swelling around the joint-structures, redness, extreme tenderness, and some slight heat of skin, lasting a week or ten days, or more, within the first two or three weeks of an attack of infantile paralysis. That condition was more like the appearance of gouty swelling than anything to which he could compare it. But, of course, it would be absurd to suppose that in a child a few months old, within the circle of the febrile disturbance at the onset of infantile paralysis, such a condition as gout should be set up. He had had, also, under his observation for more than a year, a case of myelitis in a young lady whose initial symptoms suggested spinal apoplexy. There was complete paralysis of sensation and of movement of the lower limbs, the sphincters also being paralysed; and he had seen in her, no fewer than three times, a swelling coming on in the knee-joint, with painless effusion, without any obvious cause, lasting a time and then subsiding. As to how this was brought about he could not offer an opinion, but it was clear that affections of joints did occur in connection with disease of the spinal cord; and that being so, with the fact that rheumatoid arthritis was a mere anatomical name for conditions brought about by many

different processes, it seemed to him by no means absurd to suppose that these curious joint diseases in locomotor ataxy had a real association with the nerve condition.

Dr. B. O'Connor said that the impression left on his mind, as the result of the discussion on so-called Charcot's disease, was, that some speakers seemed to think the disease was not this, and some thought it might be that; and he believed there were some who regarded the disease as non-existent. It would be interesting to bear in mind that the views of Dr. Buzzard, Sir James Paget, Professor Humphry, and Mr. Jonathan Hutchinson, to a certain extent agreed. They were very similar on many points, notwithstanding the fact that it was believed, and very often stated, that patients advanced in life, presenting the symptoms of chronic rheumatic arthritis, were particularly and singularly free from ataxic symptoms. Professor Charcot himself, with reference to his typical cases at the London Congress, had referred to the ataxic symptoms; and, if he recollected rightly, the only thing which he said of a definite character respecting the joint affections was, that no ordinary pathological condition was found which would coincide with dry arthritis. Regarding it for a moment as an affection other than rheumatoid arthritis, the question was, what was the disease? It had been said by several speakers that there were symptoms in locomotor ataxy which came on very suddenly, which lasted for a time, which might remain permanently, or which might disappear. There were certain conditions which had been referred to by M. Charcot himself, affecting certain joints, which remained permanently; and he took it that those permanent elastic œdematous swellings which occurred in joints in cases of ataxy were the cases to which some persons now-a-days referred as Charcot's disease; but he could not say on what good grounds this was done. He could imagine an author writing an elaborate treatise on some of the permanent nerve paralyses in cases of ataxy, and another writing a book on amaurosis, and another saying that some ataxic patients were unable to walk backwards; but he failed to see grounds upon which one would say that they were dealing with three new diseases. At the Richmond Hospital in Dublin, fifteen or sixteen years ago, he certainly had seen a great number of joints and bones indistinguishable, at all events by himself, from many of those specimens which had been here regarded as very unusual, if not unique. With respect to the probable origin of it, it seemed to him that an hypothesis might readily

be put forward; and, after all, it seemed to him that many were working on an hypothesis, which was this—that a certain nerve lesion existed, and this nerve lesion, whatever it might be, showed itself in certain ways. On the one hand, there might be cases with symptoms of rheumatic arthritis; and, on the other hand, with ataxic symptoms; and these ataxic cases might themselves be divisible into two classes—those without and those with permanent joint trouble.

Dr. HADDEN thought there was very little doubt that Charcot's was a distinct disease, and the arguments he should have used, if they had not already been put forward, were chiefly drawn from clinical and pathological facts. Still, as to Sir James Paget's question, Was this a new disease? he remembered that, in the *St. Bartholomew's Hospital Reports*, three years ago, Mr. Eve called attention to a case of Mr. Stanley's which, he should think, was clearly a case of Charcot's disease. He gave the clinical symptoms—impairment of vision, incontinence of urine, and anæsthesia; and Mr. Stanley went on to remark that, unless the patient saw his legs, he could not tell their direction; but, on looking at them so as to know their position, he could readily move them. Then the condition of the joints was described, and he thought they were singularly typical of Charcot's joint disease. As far as he had understood Dr. Bastian, he intimated that Charcot's idea that the disease really lay in a lesion of the motor cells was a hypothesis. But, as a matter of fact, Charcot figured the condition of the anterior horns in two cases—one, a case of diseased shoulder-joint, in which there was atrophy of the anterior horns in the cervical region. The other was the case of a knee-joint, with atrophy just above the lumbar region.

Dr. BASTIAN said he was aware of that. The only question was whether that was the change which related to the joint disease.

Dr. HADDEN said that possibly it might be a coincidence; at any rate, it was highly suggestive. It had also been noted, in cases of arthropathy, that there was a rapid atrophy of muscles in the neighbourhood of the joint. He should think the lesion was an anterior poliomyelitis affecting certain multipolar cells.

Mr. HOPKINS said it appeared to him that there was a

traumatic element in the case of these joints, which would account for the manner in which they were affected asymmetrically. There were in tabes anæsthesia of the skin, delayed sensation, perverted sensation and anæsthesia of the ligaments; this condition of limb, it seemed to him, was sufficient to account for the joint affection. A limb in that condition might be readily injured. A very slight strain was sufficient to cause considerable injury when the ligaments were soft. A patient might have no knowledge of the accident, though effusion had resulted. He would briefly allude to a case that came under his care. A man the subject of tabes, whilst turning in bed, fractured the shaft of his femur; there followed a considerable effusion into the thigh, which extended from the iliac crest to the knee; there was also abundant effusion into the knee-joint. The force was not wholly expended upon the fracture of the femur; it was also sufficient to lacerate the ligaments of the knee-joint and cause effusion into it. It might be said that the fluid in the joint was due to the fracture. In that case injury to a limb at a distance from a joint could cause effusion into the joint. In any case, it showed how readily one might have effusion into a joint, from injury in an ataxic subject, and the effusion might be the starting point of the disease under discussion.

The PRESIDENT thought that the Society was greatly to be congratulated on having initiated, continued, and, so far as it was possible in the present state of knowledge, completed, an interesting and important discussion. The occasion had given the opportunity of bringing together more distinguished speakers on a subject of this kind than it was their privilege commonly to hear now-a-days. In the next place, it had afforded a pronouncement of English opinion, which had been much desired and needed, upon this question, which had received much attention abroad, and little public attention in England. In the third place, it would be the means of communicating that which was much needed—accurate, extensive, and important information—to the great body of the profession upon this subject. Lastly, certainly not least, it had given an opportunity of doing justice to the distinguished foreign physician, to whom was due a very great advancement in our common knowledge. It might have been considered interesting, even instructive, to have summed up the opinions which had been expressed concerning this subject in the course of the discussion; but it had already been done so well in one

of the journals, that, even if there had been time (which there was not), it had been rendered unnecessary. It would have been almost equally interesting, and perhaps a little more instructive, to have summed up, not the speeches which had been delivered, but the ideas which had been evolved in the course of the discussion; but, as he saw that Mr. Morrant Baker had been taking very close notes, he had no doubt he would himself unfold and deal with those ideas with the same lucidity and ability with which he had started the discussion.

Mr. MORRANT BAKER, in reply, expressed his gratitude to the Society for the very kind and altogether unexpected manner in which they had dealt with his paper. He would not attempt to enter into every detail that had been raised; for, if he dealt with each speaker in succession, he should be repeating many things that had been said before, and he feared, that if he did that at any length, he should at that late hour carry out that process which was known as emptying the church down to the sexton. He thought he had been a little misunderstood as to his views; and, although he had no pretensions to be an authority on the matter, he should be glad, as he had read the paper, to state briefly what his view was with regard to Charcot's disease. He believed that the disease was identical with what was known as rheumatoid arthritis. At the same time, one ought to be as clear as possible as to what was meant by rheumatoid arthritis. He meant by it neither rheumatism nor gout, nor anything that could be fairly called either the one or the other. He had been taught and had taught others, that there was a disease called rheumatoid arthritis, which was neither rheumatism nor gout. That disease might best be called arthritis deformans or osteo-arthritis. This arthritis deformans was a fairly definite disease; it was characterised by the same anatomical and pathological lesions which were undoubtedly met with in Charcot's disease—eburnation, osteophytes, fibrous degeneration of cartilage, wearing away of bone, and so forth. It was not fair to take an out-of-the-way case, which could hardly be called osteo-arthritis, and to say that it was unlike Charcot's disease. To be fair, one must take a typical case of each; and, if that were done, it seemed to him that it must be acknowledged that, from the pathological point of view, it was impossible to find any real distinction. In reading carefully one at least of Professor Charcot's descriptions of the disease, it seemed to him that he had failed to find any pathological difference, as to which it

could be said: "This constitutes a radical distinction between the one disease and the other." Yet, at the same time, the association with locomotor ataxy was not an accidental combination of two different diseases. He believed there was a most close pathological alliance between this form of arthritis deformans and locomotor ataxy; and the only point on which he differed from Dr. Duckworth and Dr. Buzzard was this, that, instead of looking upon the two diseases as being cause and effect, he should be inclined, from present evidence, to look upon them as dependent upon some common cause—that is to say, a disease which in one case fell especially on the nervous system, in another case on the joints, and in another on both. And supposing a case to exist in which the disease fell upon joint and upon the spinal cord, it could readily be imagined that the concurrence of the two things would make the disease different; in other words, if the patient had tabes and disease of the joints, one could not expect the symptoms in the joint disease to be exactly like those in a patient who had disease of the joint, but was not troubled by any disease of the nervous system. The difficulty in his mind in believing that it was merely a case of cause and effect was that he had seen cases which, as far as he could tell, clinically and pathologically, were identical with Charcot's disease, but in which there were no symptoms of tabes. There was a case that he had seen only a few days ago, under the care of Mr. Thomas Smith, of St. Bartholomew's Hospital, which he hoped Mr. Smith would bring before the Society. It was a case of a man, the condition of whose hip-joint seemed absolutely indistinguishable from Charcot's disease, as shown in the specimens; yet he was able to continue his work as a plasterer, and he had no symptoms whatsoever of locomotor ataxy. The man declared that he never had stomach-ache, so that he could not be accused of gastric crises. So long as cases of that kind occurred, it was difficult to say that this disease was due to, and always associated with, tabes; therefore they should withhold their opinion for a time, and try to obtain more facts. There was one other thing to which he would refer. In building up the knowledge of arthritis deformans, and saying that this disease was identical with it, they had been learning from cases that occurred during the years in which no questions were asked regarding the presence of locomotor ataxy, and they did not know, therefore, how many of these cases were really associated with tabes. He had seen these cases cropping up occasionally during the last few years, but only

within the last year or two had he asked a patient whether he had any symptoms of tabes. Therefore, in saying that this disease was identical with arthritis deformans, he was willing to allow that he might, in some cases, be merely saying that Charcot's disease was identical with Charcot's disease. He felt sure that there were many cases that would have shown symptoms of tabes also; but, so long as there were cases in which there were no such symptoms, there was a difficulty in believing that locomotor ataxy must always be expected when this joint disease was found. With regard to what Dr Buzzard said about the specific gravity of bones, he did not think there would be any radical distinction in the two cases, but it would be well worth observing. Dr. White had anticipated him in what he should have said with regard to the apparent difference of opinion between Professor Charcot and Dr. Moxon. It was obviously, he thought, that they had been working in a different field of observation. Dr. Andrew, Dr. Ord, and Dr. Bastian, had each said, "I have never had under my own care a case of Charcot's disease." He would quote Professor Charcot, who, if he were not mistaken, said that his proportion of cases was six out of fifty. If he might compare small things with great, his own experience as a surgeon was this, that out of the cases of locomotor ataxy he had had under his own care within the last year, 100 per cent. had also disease of the joints; so that one might look upon it that the explanation given by Dr. Buzzard was correct, that these cases naturally came to the surgeon. Since the debate began, he had been consulted about a case which he was told was a disease of the knee-joint. He had looked at the joint, and found it was a typical case of Charcot's disease. Mr. Barwell had had a very large experience in diseases of the joints, but he differed from him in thinking that one could set up a clear pathological distinction between osteo-arthritis and Charcot's disease. He thought that there was eburnation in both cases, that there was fibrous degeneration, and he would refer to the microscopic specimens put up for him by Mr. D'Arcy Power, at two or three of the meetings, showing apparently the fibrous degeneration in Charcot's disease had been long known in association with osteo-arthritis. He believed that the small joints were affected; indeed, in three out of four cases of Charcot's disease that had been under his care, some small joint or other had been affected as well as the larger ones; and with regard to clinical symptoms, at present he had not

been able to find any such group of symptoms as would make one say that they separated Charcot's disease from arthritis deformans. In the last case of this affection he had seen, the joint had been painless; but the patient said that his sensation was defective in the whole limb. The swelling had been gradual, and so had the symptoms. There was not that sudden onset of all the symptoms which occurred in a typical case. He could not agree with Sir James Paget with regard to what he said about this being a new disease. Sir James Paget had said that this must be a new disease, because there were no specimens in the museums; and, if it had a separate existence, how was it that it had been overlooked? With regard to the first point, he thought that he had brought specimens from the museum of St. Bartholomew's Hospital which were not to be distinguished from specimens acknowledged by all to be those of Charcot's disease; and he thought, if these bones could be clothed again with flesh, and live, and if there could be the opportunity of asking questions as to this, that, and the other, it would be found that many of those old bones had been taken from cases of locomotor ataxy. Of course, that could be only told from their present appearance; but he should imagine that that was the case. He had seen other bones besides those which he had brought, which appeared to be old specimens; and he should gather that some of these, at all events, were cases that had been enshrined in museums on account of something observed at the time as worthy of being recorded. They were, of course, labelled as osteo-arthritis, or chronic rheumatic arthritis. But he did not think with Sir James Paget that the differences between typical cases were so startling as he believed. He had referred to the case that he had seen with Mr. Thomas Smith, where apparently the symptoms were indistinguishable. With regard to the question, "How had the disease been overlooked?" he ventured to say, because it was so common. His (Mr. Baker's) argument would be this; that, as a rule, a museum was built up by carefully preserving anything which was out of the common. He believed that the reason why these specimens were not more numerous, was because they were looked upon as being common, and were not distinguished from other severe cases of osteo-arthritis. He also agreed with what Mr. Marsh had said. These cases were being overlooked five or ten years ago; and if they were overlooked then, why should they not have been overlooked fifty years ago? He had been much struck by a remark made to

him by a surgeon of a large infirmary in London after the first meeting. He had asked Mr. Baker whether he would like, not to see *one* case of Charcot's disease in his infirmary, but to see a waggon-load. It was not to be supposed that this large number of cases had occurred in an infirmary within the last few months. Some of them must have been there for years, and if this large number within five or ten years had been overlooked, did it not seem almost certain that cases for many years had been overlooked, because they had not been dissociated from cases of osteo-arthritis? Therefore, until Professor Charcot threw light upon them by showing the connection between them and tabes, specimens had not been accumulated in museums. Again, with the greatest deference to Sir James Paget's authority, he thought it unfortunate that the term rheumatic arthritis should find a strong friend, as it had, in Sir James Paget, and he wished all could agree to discard it altogether, and only use the term osteo-arthritis or arthritis deformans. With reference to the remarks of Dr. Ord, he had rather accused the speaker of saying that there was no attempt at repair. He (Mr. Baker) had hardly said that. He said, "either no attempt, or that it was inefficient and disorderly." He had not meant to imply that there was no attempt; that there was waste without repair at all; that would be, of course, rather a definition of death than of disease. He had meant to imply that the great characteristic of the disease was a wearing away, it might be at a natural rate, and that the repair was not at a natural rate, and was deficient, and that the tendency of the repair was disorderly. He was glad that Dr. Ord was on his side in preferring the term osteo-arthritis or arthritis deformans to that of rheumatic arthritis; and also that he had supported him in believing that there was a close pathological connection between osteo-arthritis and the nervous system. He was a little doubtful whether he might look upon Mr. Hutchinson as a friend or as an opponent. He agreed with him in what he said about premature senility. He thought that would express fairly in some respects the condition which there was in the joints, and he had attempted to express that, by saying that he thought the great feature of the disease was waste without repair. He would not suggest any term to express this, because words, which we intended, when we coined them, to be our servants, often became our masters; but still he hoped that someone with a greater knowledge of etymology would, in time to come, invent a word which would express

that idea. As to the theory about standing and walking, and that being the cause of this wearing away of the joints, he would quote the case of an old woman with Charcot's disease of the joint's who had been paralysed for seventeen years, in which both the hip-joints had almost disappeared as joints; that is, the heads of the femora had disappeared. The upper end of the femur was drawn up above the level of the anterior superior spine of the ilium. That old woman could not possibly have been the subject of much friction of the joint-surfaces by walking, because she was more or less paralysed for the whole of the time. It would not be difficult to find facts that would oppose a theory like that of Mr. Hutchinson's on the subject; and there was no doubt about the diagnosis of the case of this old woman, because she happened to be in Guy's Hospital, under the care of Dr. Habershon and Dr. Wilks, at the beginning of her illness, and her case was diagnosed there as one of paraplegia. He agreed with what Mr. Hulke had said with one exception, namely, with regard to the connection of Charcot's disease with syphilis. He could not think that syphilis had anything to do with it. So far as his own observation went, he could not trace any connection of syphilis with locomotor ataxy, or with osteo-arthritis, or with this joint disease. He could not think that Charcot's disease was indefinite in the sense in which Dr. Moxon put it, and he felt sure that, if the latter had a case presented to him, as it had been presented to himself on several occasions, he would be converted. When one saw a case, and one was only told it was a diseased knee-joint, and upon looking at the joint and examining it for two or three seconds, one could say that the joint was almost certain to be associated in the patient with symptoms of locomotor ataxy, and then, upon asking a few questions, one found that it was so—when that happened over and over again, one could not help feeling absolutely certain that it could not be a mere coincidence. Therefore, he hoped, when Dr. Moxon had the opportunity of seeing more of these cases, he would think differently on that point. With regard to the ex-cabman to whom Dr. Moxon had referred, he must doubt the appositeness of the illustration. He could not think that Dr. Moxon could seriously wish the members to believe that when a man had damaged his shoulder-joint, any amount of movement could wear down the head of the bone when the joint was more or less disabled from the injury. If that were the case with this unfortunate cabman, the movement must have been of a very forcible description. The cases

brought forward by Mr. Barker and by Mr. Henry Morris were cases of much interest, but he would not deal with them in detail, nor with those of Mr. Herbert Page. With regard to the remarks made, so many of them were in answer to previous speakers that, if he dealt with each in detail, he should be going over very much the same ground. Mr. Hopkins said that many of these cases might be determined by traumatic causes. He was inclined to agree with him. Certainly that would explain the want of symmetry in many. In more than one of his own cases the disease had apparently been started by an injury; in the case of one man by the wrenching of the knee; in the case of an old woman by falling off the sofa on her elbow. He thought it would be found, on closely examining into the history of many of these cases that, whatever their origin, whatever the connection with tabes, yet some slight injury started the disease. He had no wish to dogmatise with regard to this question; he had no authority to dogmatise on the subject. His anxiety had been to bring forward certain facts. He was anxious to add to the facts, and in raising the discussion he ventured to state, as clearly as possible, what theories might be adopted on the subject; but he felt that one could not be, at the present moment, sure as to the right one. He had his opinion. He thought Charcot's disease was identical with a disease long known; that the two diseases, tabes dorsalis and this form of osteo-arthritis, were not in the relation of cause and effect, but were due to one and the same cause; the disease of the spinal cord reacting unfavorably on the joint disease, when both were present; but, should fresh facts arise to show that one could put on one side cases of Charcot's disease with certain symptoms, and fail to find those symptoms in osteo-arthritis, he would be glad to acknowledge that such was the fact. But he felt that, in order to get these new facts and opinions from others, he could not do better than bring his cases before the Clinical Society, and he felt especially happy in bringing them forward during the presidency of Sir Andrew Clark.

XI.—*On Lesions of the Frontal Lobe.* By W. Hale White, M.D. *Read January* 9, 1885.

ALICE H., æt. 26, was admitted under the care of Dr. Wilks, June 14, 1884, for severe pain in the forehead and vertex. Family history of consumption. She never had any illness before the present, and had not been liable to headache or loss of sight. Eight years ago she fell down, striking her head on the left side, and was unconscious for some time. She has been ill a month with severe pain over both eyes, shooting up into the top of the head. She has also become very near-sighted; frequently when walking about she has lost the power of sight altogether for a few seconds without loss of consciousness. She is sometimes giddy when eating and has been sick twice since the beginning of the illness, but not severely. After her death I went to see her brother, with whom she lived, and questioned him closely but could obtain no further history from him; none of her friends thought her very ill, and they expected her to be well shortly. The doctor who saw her does not seem to have thought the case serious. The only mental symptom noticed was that she was a little "mysterious." She would often use the phrase "I am thinking," and when her brother suggested that she should tell what she was thinking about she declined to do so. He does not think that her memory, reasoning, or emotional faculties were at all impaired. She was engaged to be married and would take long walks with her *fiancé*, remaining constant to him till the end.

On admission.—No paralysis or anæsthesia; in short, the only sign to be detected was marked double optic neuritis. She complained of nothing but intense pain over the eyes.

June 22.—Was up for a short time to-day. Went to bed early because of the pain in the head. She had one sixth of a grain of morphia subcutaneously; a little while after this she vomited and then became quiet.

At 2 a.m., on June 23, she was found dead in bed.

Post-mortem examination.—Brain: Membranes and vessels healthy. The convolutions were flattened, especially those of the left frontal lobe, which was much more prominent than the right, projecting forwards beyond it considerably. In the left frontal lobe a hardness could be felt. On making hori-

zontal sections it was found to contain a very light-coloured greyish new formation, which at the front and outer part contained a cyst full of fluid which might possibly have been old blood. It was difficult to say whether this new formation was inflammatory or gliomatous, probably the latter. The lateral ventricle, caudate, and lenticular nuclei, internal and external capsules, and claustrum were none of them implicated. The island of Reil and ascending frontal convolutions were also free, but the new formation extended into the front part of the gyrus fornicatus, the marginal convolution, the first, second, and third frontal convolutions, and the orbital convolutions, but there was no bulging on the orbital surface of the frontal lobe. The lateral ventricles were dilated. The rest of the brain was absolutely healthy; no descending degeneration. Intense optic neuritis in both eyes, the discs being swollen and blurred. Every other structure in the body was quite healthy. Microscopic examination showed the tumour to be a glioma.

Case 2.—Alice A., æt. 31, was admitted into Guy's Hospital June 20, 1884. No illness before the present one, which she first noticed twelve months before admission. No history of scarlet fever, rheumatic fever, or fits. Work light. Occasional difficulty of late in her breathing. First consulted the doctor a month ago for stiffness confined to the right leg; this, together with swelling of the leg, has troubled her on and off till admission, five days before which she noticed in the morning a stupid feeling in her head and she could not speak plainly nor use her left hand efficiently; by the next day her left leg was similarly affected, and on the day after that she was unable to rise without assistance; her foot subsequently turned cold and she lost sensation from the mammary line to the foot.

On admission.—Healthy looking; lies in bed without any distress; complains of vertical and occipital headache; left arm and leg fall lifeless when lifted, and she does not appear able to move them. On tickling the sole of the left foot there is more movement than in right. Complains of a strange sensation running all down the left side and leading to twitching movements. Says she has had similar symptoms for some years. Finger ends much clubbed and bulbous. Cardiac impulse visible over whole of left mammary region and also in neck. Loud whiffing systolic bruit audible at apex and carried round to the back. To and fro basic murmur carried up the vessels

in the neck and also down the sternum. Pulse 84, water-hammer. Sphygmographic tracing shows it to be a characteristically splashing pulse. Lungs normal. Liver dulness an inch below the ribs. Spleen not to be felt. Urine normal.

The power in the leg began to improve directly after admission. Thus on the second day of her stay the nurse complained of the patient because she kicked off the bedclothes, and after she had been in five weeks she was walking about daily and using the left arm to work.

On August 18 she was heard to fall whilst in the water-closet; she was found in epileptiform convulsions, cyanosed, frothing at the mouth, insensible, and with Cheyne-Stokes respiration. On coming to, she vomited excessively, her pulse was found to be 150, and she was very collapsed. This condition of prostration lasted a few days, diminishing in intensity each day, till soon she returned to her former condition.

On September 7 she had an attack of inability to speak which lasted a quarter of an hour and was accompanied by sweating and a feeling of coldness.

Towards the end of October her breathing became worse; she gradually sank and died November 15.

Post-mortem examination.—Heart 22½ oz. All the cavities dilated considerably; thickness of muscle normal in all parts. No pericarditis; muscular tissue had undergone some fatty change. The segment of the aortic valve that has no coronary artery behind it was covered with a large irregular mass of vegetations with much fibrin on them; this mass had come into contact with the ventricular surface of the anterior division of the mitral valve and had here given rise to a mass of vegetations about the size of small sago grains; these had caused a roughness on the other surface of the valve, which rough part coming into contact with the posterior surface of the auricle above the posterior valve had there caused some vegetations to appear. All the orifices were dilated. *Lungs:* Some œdema at bases; white patch, probably small infarct, in right, at anterior edge. *Liver* fatty and nutmeg. *Kidneys* fatty; several small puckerings on the surface due to minute infarcts. *Spleen* contained three infarcts, one very large. *Stomach* congested. *Brain:* Vessels and membranes healthy. The posterior part of the right frontal lobe seemed perhaps slightly distended, and felt a little softer than the corresponding part of the opposite side. On slicing through the brain at the level of the corpus callosum one came on a brick-coloured clot about the size of a filbert nut, situated in the white matter of the brain in the

right frontal lobe, encroaching slightly upon the gyrus fornicatus just where it bends round the corpus callosum. This clot lay on the roof of the anterior cornu of the right lateral ventricle, but was separated from it by a layer of healthy brain substance, about a quarter of an inch thick. The clot was triangular, with the apex directed downwards, and, passing in front of the anterior cornu, it terminated at about the level of the floor of the cornu. The brain substance around the cornu was unaffected. The rest of the brain and spinal cord were absolutely healthy.

I have ventured to bring these cases before the notice of the members of this Society because of the many points of interest attached to them. Although, as is well known, tumours of the brain, especially those of the frontal lobe, may produce no symptoms in the earlier stage, it is rare for none to occur during the final stages of the illness. In the first case for a whole month absolutely the only symptom was headache, and although at the end of the illness this symptom became of the character indicative of cerebral mischief, yet earlier not any of the friends or the doctor thought it of any importance; the vomiting was very infrequent and very slight, the alteration in sight was unimportant, a history of it only being elicited when particularly asked for. After her death I questioned her brother, who was very intelligent, most carefully, and all I could find out from him was that the girl had had a headache, but that nobody thought anything of it, and that she went about her occupations as usual. On her admission the only thing discovered beyond this was optic neuritis. She was well enough to be about the ward twelve hours before she was found dead in bed.

In addition to the interest which the fewness of symptoms gives to the case, and the consequent liability, except for the optic neuritis, to confound it with a case of gastric derangement, we have also the peculiar mode of death, which can hardly be attributed to so small a dose of morphia as one sixth of a grain. Why did she die? Surely it is strange that she should do so when well enough to be up and about the ward a few hours beforehand, more especially as the growth was not situated in a part of the brain concerned with vital functions, nor was it likely that it affected other parts by pressure, because a few hours before death there was no evidence of such pressure. Tumours of the brain no doubt have frequently a sudden termination, but generally there are at the

same time some important symptoms such as coma, &c., present.

Then, again, in addition to the clinical interest which the case derives from the fewness of symptoms and the suddenness of death, it possesses great interest to the physiological psychologist owing to the large destruction of brain substance without either loss of motion, sensation, or intellectual faculties.

The second case is also very instructive. Dealing only with the nervous symptoms we may, I think, dismiss the phenomena of loss of motion in the left leg and arm as not due to any coarse lesion in the brain; for not only was there an entire absence of such lesion at the post-mortem examination, but the facts that the loss of power occurred first in the right then in the left, that it was so gradual in its onset, that it was accompanied by "queer sensations," and recovery was so rapid, are all against the supposition that it was due to embolism; probably it was functional; anyhow it could not have been the result of the hæmorrhage discovered post mortem. The only symptoms one can connect this with are those of the attack which came on in the water-closet, all of which, except perhaps the convulsion, are easily explicable by it. But the reason why this case is brought forward is that we have here a large destruction of the frontal lobe without any destruction of motor or sensory faculties or impairment of intellectual power. Cases of damage to the frontal lobe without any very marked symptoms are to be found scattered through medical literature; still they are sufficiently rare for it to be very unusual to meet with two in such a short space of time. It is pretty generally allowed that the front part of the frontal lobes have to do with the intellectual functions, for it is a matter of common observation that races of low intellectual calibre have receding foreheads. How is it then that there is in cases of injury of the frontal lobe such slight, if any, intellectual impairment? The reason is, I think, because the intellectual powers are the latest acquisitions both in the life of the individual and also in the animal series. Early formed functions, such as movements of the arms and legs, have fixed centres, whilst the later formed movements, such as those of the face, are not so well fixed. This is well seen in gestures; all the lower animals and children can move their arms and legs, but it is not till the child has advanced some way in life that it acquires the power of such gesticulation as elevating the eyebrows in surprise, contracting them in anger, &c. Now,

stimulation of one face centre causes movement of both sides, and the face is the least affected and the first to recover in hemiplegia. Both these facts bear out the hypothesis that impressions arriving at or proceeding from the face centre are not so definitely impressed as those from the movements of the arms and legs, and that the centre of the opposite side, on the Broadbent hypothesis, is quickly capable of taking up the work of the damaged one, just as Broca's right convolution is capable of quickly taking up the function of the destroyed left one, because here also the power of connecting objects with definite sounds is acquired late both in the life of the individual and in the history of the animal series. Expressed in the terms of the view of Mr. Horsley we should say that, in the later formed functions, the second of the bilateral associated centres is more quickly brought into play than in the earlier formed functions. Now, on this law, that the recently acquired functions are the less firmly fixed to one particular part of the brain, and that the rapidity with which they are capable of being taken up by some uninjured part either on the same side or the opposite is in proportion to the lateness, in the animal series, of their acquisition, it is not difficult to understand how, in cases of disease of the frontal lobe, which presides over the very latest formed powers, the uninjured should be capable of very quickly taking on the function of the diseased parts. It matters not on this hypothesis whether we generally in our intellectual operations use only one side or both, because in the latter case it would only be necessary to suppose the uninjured side capable of quickly taking on double work. In generations to come, what are now recently acquired intellectual functions, will become so fixed that when they are lost by injury of one frontal lobe, no other part will be able to take them up, whilst the still higher functions, as yet unimaginable, will be rapidly taken up by the sound side. In those cases in which, after injury to the frontal lobe, there has been intellectual impairment, it is because the lesion is so extensive, and so many intellectual functions are impaired, that the remaining parts of the brain can only take up some of them.

Without entering into the respective merits of the Broadbent hypothesis and that recently urged by Mr. Horsley, of bilaterally acting cortical centres to account for recovery of motion after cerebral lesion, I would point out that the great extent of the damage in my first case, with complete absence of any symptoms, drives one to the conclusion that in the case

of intellectual faculties the sound side of the brain is capable of taking on the work of the diseased side. I suspect that any newly acquired capability is at first represented on both sides of the brain, both thus acquiring a new but similar function; after a long time of transmission through many generations, one of these gets gradually more used than the other, until, if such a capability has existed for a multitude of generations, as the power of connecting ideas with certain movements of vocalization, it takes some months to educate the usually unused side, whilst if the functions belonging to the injured part be recently formed, the uninjured side can take them on at once. Of course if both frontal lobes are destroyed this is impossible; hence the idiocy of children without the front part of the frontal lobes.

XII.—*On a Case of Recurrent Hæmatemesis with Urticaria.* By J. J. Pringle, M.B. *Read January* 9, 1885.

THE subject of the curious affection I am about to describe is a gentleman, æt. 51, retired from the army with the rank of lieutenant-colonel, with a history of no illness previous to the present, except one slight attack of dysentery and one or two mild agues during a seven years' stay in India, from 1858 to 1865. He had always been most abstemious as regards alcohol, and is a tall, muscular, and robust-looking man, of active mental and bodily habits.

There is no family history of gout, hæmophilia, or of any decided diathetic tendency to throw light upon the present case, unless the facts that his father died of diabetes at the age of seventy, that a brother is subject to curiously capricious asthmatic attacks, and that several members of his family are liable to "biliousness," may be considered to do so. The elder of patient's two sons, æt. 3, is peculiarly subject to copious and troublesome nettlerash. None of his five daughters are so affected.

The history of the present ailment may be dated from 1872, when the patient had two severe attacks of ordinary nettlerash, attributed to indiscretions of diet. He remained in perfect health till September, 1878, when he began to suffer from repeated attacks of nettlerash with an unusual amount of prostration and malaise. During 1879 the nettlerash recurred with increasing frequency and severity, the tongue and mucous membrane of the mouth and fauces being involved in the more severe attacks. In the latter part of the same year vomiting of clear watery or glairy fluid with intense retching, and subsequently of altered blood in the shape of "coffee-ground matter" first showed itself. During 1880 the patient may be said never to have been entirely free from nettlerash, which appeared on the most trivial exposure to cold, or from such subjective symptoms as "itching," "twingings," "irritability," "heats and chills" of the skin. At intervals of about two months occurred attacks of great severity, during which vomiting often lasted for twenty-four hours at a time, and large quantities of pure red blood were ejected in addition to altered

blood. In 1881 the attacks were less frequent, an immunity for eight months being enjoyed, but those which did occur were of an even more alarming nature than the previous year. In 1882 he had one mild attack in May with vomiting only of altered blood, and in November occurred the most severe attack he has ever had. It was attributed to exposure to cold whilst fishing; the initial skin rash was very abundant, the affection of the mouth and fauces intense, and the hæmatemesis so copious that the patient's wife says "the blood poured out like water from a jug, filling two handbasins and saturating the bed." The condition was so critical that for forty-eight hours the patient could not be undressed, and his medical attendant—Mr. Hill, of Crickhowel—writes me, that "he could not allow his friends to entertain any hope of his recovery." I had the opportunity of witnessing the end of his next attack on April 15, 1883, kindly accompanied by Dr. Wilks, who was acquainted with the history of the case, and by Dr. Hurd-Wood of Leatherhead, who had watched it from its earliest commencement. The attack was a severe one, the first warning being a copious eruption of nettlerash over the whole body forty-eight hours before our visit, followed in due course by swelling of the tongue and fauces, and by vomiting of blood which filled a large handbasin. Bright pink urticarial wheals, showing in places a crescentic arrangement, were studded over the trunk and extremities; nowhere were they purpuric. The intervening body surface was of statuesque pallor, the face and ears, however, flushed; the temperature not raised; the skin moist; the pulse soft, beating 80 to the minute; the stomach apparently somewhat dilated to light percussion; the spleen not swollen; the voice a mere whisper; the pupils contracted; and the patient in such a condition as may be inferred from the amount of blood lost and the free use of morphia subcutaneously, which had been found the only remedy efficacious in allaying vomiting. A relapse of all the main symptoms occurred two days later without obvious cause, after which recovery was, as usual, rapid and apparently complete.

The treatment of the attacks had hitherto comprised all the various measures generally resorted to in cases of gastric hæmorrhage, with free purgation by salines on the earliest appearance of symptoms, and feeding exclusively by the bowel during and for some days after an attack. Hypodermic injections of morphia and ergotin and rectal injections of Battley's liquid extract of ergot had been employed, but only after hæmatemesis had already set in. Shortly after the above I suggested

that as soon as nettlerash appeared and nausea was complained of, hypodermic injections of ergotin (one third of a grain) and morphia (one sixth of a grain) should be at once administered with a view to controlling hæmorrhage on the one hand and vomiting on the other. An opportunity for employing this method of treatment presented itself four months later, *i. e.* in August, 1883, when the patient was resident in Bournemouth, under the observation of Mr. Scott, but during this interval an interesting and important new feature of the case presented itself in the form of two unequivocal attacks of acute gout, the first affecting the right foot, the second the left hand. In August the early symptoms of an attack set in with severity and vomiting of "bilious" matter ensued, but not of blood. After two injections vomiting ceased, and the attack was apparently arrested although the patient felt ill and was unable to get about for three days, at the end of which he passed a copious, dark, tarry motion, with complete relief to all his symptoms. On only one previous occasion early in the history of the case had anything in the motions suggestive of blood been observed. For nearly a year perfect health was enjoyed, but in July, 1884, he had a fresh attack with no new feature except persistent sneezing as an early symptom. A little altered blood was vomited before the injections were used, but after their employment all vomiting ceased and there was no subsequent melæna although fresh crops of nettlerash continued to appear for three days. His last attack was in December last; the earlier symptoms were severe, and "bilious" vomiting set in, but after employing the remedies prescribed the attack passed off. In addition to the more striking features of the case it has been noticed that before many of the attacks the patient has been out of sorts, with impaired appetite, furred tongue, constipated bowels, headache, and a yellowish tint of conjunctiva, but these symptoms have by no means been constant or in any relation to the subsequent severity of the attack. Since 1878 the diet has been carefully regulated, and for the last two years the remedies usually employed for the restoration of suppressed or disordered hepatic function have been conscientiously taken, the patient himself expressing a decided preference for podophyllin. Exposure to cold, to which the patient was formerly very susceptible, has in many instances been the undoubted immediate determining cause of an attack, the two last, for instance, having ensued upon a game of lawn tennis on a cold day, and upon attendance at a heated political meeting with subsequent exposure to chilly night air. The patient recuperates with

amazing rapidity after the attacks and in the intervals his digestive powers are in every respect excellent in so far as they are put to the test; he is absolutely devoid of pain or uneasiness in connection with food and does not wince under forcible abdominal palpation. His liver is of normal size; his spleen is not enlarged. I regret that I am unable to give details as to the state of the urine during the attacks; it is certain, however, that it is of high colour and concentrated, but that it has never contained blood-corpuscles or blood-pigment. The average amount of urine passed in twenty-four hours has, unfortunately, never been estimated, but for more than a year the patient has had to rise about 4 a.m. regularly to pass water. The specific gravity of the urine fluctuates between 1020 and 1025; it is of a pale sherry colour, sometimes with a deposit of pink urates, and all the specimens I have examined have been intensely acid and have deposited the various crystalline forms of uric acid in great abundance. I have had no opportunity of estimating the average amount of urea excreted. Neither albumen, sugar, nor casts have ever been present.

The heart is not obviously hypertrophied, its impulse is weak, the sounds free from bruit, but the second over the aortic area is sharply accentuated and occasionally reduplicated. The radial pulse is rather small, but hard and slow, sphygmographic tracings from it being flat topped with very gradual descent. The fundus in both eyes is normal, the arteries of fair size.

The blood has never been examined during an attack; in the intervals it has presented no abnormal microscopical characters and is of average corpuscular richness. Within the last six months, what I take to be minute tophi have made their appearance in the pinna of the right ear.

Remarks.—The main point of interest being the occurrence of coincident attacks of urticaria and hæmatemesis, I purpose to consider the relationship, if any such there be, between these two chief features of the case. The absence of symptoms of hepatic cirrhosis or other conditions producing portal congestion and of disordered blood states, such as scurvy or purpura, precludes the possibility of hæmatemesis from such causes. It is obviously impossible, in view of clinical and pathological experience, to *deny* the existence of a gastric ulcer, but on the other hand, there are none of the symptoms usually associated with such a lesion, the patient's digestion

being, as I have mentioned, unaccompanied by any subjective indication of gastric dyspepsia. Nor would the presence of a gastric ulcer account for any of the phenomena except the copious vomiting of blood.

I am forced then to the conclusion that the hæmorrhage from the stomach is due to capillary rupture, occurring when the mucous coat of that organ is in a state analogous to the urticarial condition of the skin, and in support of this view, bizarre as it may appear, I would submit the following considerations: (1) that on no occasion has the patient suffered from gastric disturbance or hæmatemesis without the previous occurrence of copious nettlerash upon the skin and in the mouth and fauces; (2) that in less severe attacks, only vomiting of mucous or "bilious" matter has ensued, indicating a less marked or more transitory dilatation of the gastric capillaries, whilst in attacks of intermediate severity the small quantities of altered blood vomited have indicated slight and gradual capillary hæmorrhage; (3) that the apparent success of the treatment in arresting the hæmorrhage renders its dependence upon gastric ulcer, or any condition involving the erosion of large vessels, extremely improbable.

Presuming then that such a condition is possible in the stomach, and bearing in mind that urticaria of the fauces shows itself as diffuse redness and swelling, not in the form of circumscribed wheals, we have, I think, some indication of the condition which obtains in the mucous membrane of the stomach during these attacks. The fact that the capillaries of the gastric mucosa form an extremely fine and extensive network, supported and separated from the cavity of the organ only by a delicate basement membrane, and a single layer of caducous, columnar epithelial cells fully accounts for the occurrence of hæmorrhage and for its amount in the more severe attacks described.

I have been unable to find any account of similar cases. Those most nearly approaching it are found in Graves's *Clinical Lectures.* He narrates* the case of a gentleman of very gouty habit who, after labouring for some time under languor and weakness accompanied by spasms, pains, and sense of weight in the stomach developed swellings which became as large as a pigeon's egg, were accompanied by a sensation like the bite of a gnat, were generally about the face and sometimes inside the mouth and about the palate and uvula; their duration lasted

* *Clinical Lectures*, 1848, vol. i, p. 462, *et seq.*

only a few hours. The patient was under the impression that similar swellings affected his stomach also.

Again, Graves* describes two cases which he calls "exanthema hæmorrhagicum." The first is that of a robust man æt. 29 admitted into hospital with febrile symptoms, vomiting dark fluid, passing blood in his urine and mingled with his fæces; subsequently blood oozed from his gums and mouth and he had hæmoptysis. On the seventh day from the commencement of bleeding from the intestines an eruption of rather large elevated red spots appeared on the arms and thighs, which never became hæmorrhagic and completely faded after five days. There is no mention of subjective symptoms in connection with these spots. The patient died on the twenty-ninth day of his illness from repeated bleedings, and at the autopsy no lesion beyond minute red spots dispersed over the surface of the mucous membranes was found.

Graves's third case is that of a labourer, æt. 34, robust, but with an alcoholic history and subject for years to bleedings from the nose, preceded by malaise. Fourteen days before coming under observation he took some cold water whilst in a state of perspiration, and was immediately attacked by rigors, nausea, and lassitude, soon followed by bleedings from the nose, mouth, and bowels, which recurred at intervals till his admission to hospital, and which seem not to have been mitigated by treatment there. On the twentieth day of his illness an eruption, ushered in by a tingling sensation resembling the sting of nettles made its appearance over the surface of the body, the spots having the same characters as in the preceding case. Vomiting of "coffee-ground" matter ensued and the patient died on the twenty-seventh day of his illness, the eruption having totally disappeared after existing for five days. There was no autopsy.

Murchison† mentions the case of a boy, æt. 9, who suffered from urticaria tuberosa and purpura urticans with hæmorrhage from the bowels, kidneys, and urinary passages, and with the discharge of much lithic acid in the urine. No further details are given regarding the case, which seems to have many points of resemblance to mine.

Dr. Sieveking‡ records a case of hæmatemesis without obvious cause in a girl, immediately relieved on the appearance of an erythema nodosum.

* *Ibid.*, vol. ii, p. 362, *et seq.*
† *Lancet*, 1874, vol. i, p. 581.
‡ *Ibid.*, 1868, vol. i, p. 12.

Mr. Milton* describes several cases of "giant urticaria" affecting the nostrils, fauces, glans penis, and probably the urethra, some of them in decidedly gouty subjects; and Leube† mentions the occurrence of temporary albuminuria in connection with eruptions of nettlerash.

The relation between urticaria and bronchial asthma, in many cases at least, is too well attested to need anything beyond mention here.

It would also be beyond the object of the present paper to enter into any discussion upon the complex subject of the relationship between the gouty condition, hepatic derangements, and skin eruptions, all of which form component parts of this case. It is rather curious, however, to note that very many writers upon dermatology neglect to enumerate the gouty diathesis among the causes of urticaria, and that among writers upon gout it should be a French physician—Dr. Lecorché, of Paris—who should most strongly insist upon the association.‡

Fanciful as it may appear to some, I cannot but think that the marked improvement which has occurred in the case cannot entirely be attributed to the greater attention the patient has latterly paid to the rules laid down by his medical attendants, but, in some measure at least, is due to the frank development of his arthritic affection.

Finally, I desire to express my thanks to Dr. Hurd-Wood of Leatherhead, Mr. Hill of Crickhowel, and Mr. T. B. Scott of Bournemouth, who have kindly supplied me with many of the facts of the case, and very especially to Dr. Talfourd Jones of Brecon, who first, in 1881, recognised its true nature, and with almost prophetic penetration laid down a line of treatment from which no tangible departure has yet had to be made.

Since the preceding paper was read before the Society the patient has been totally free from urticaria, with one exception.

In the last week of April he awoke one morning with the subjective warnings of an imminent attack. During the course of the day a copious crop of nettlerash developed and there was considerable retching and actual vomiting. At the same time the right wrist became much swollen, hot, and

* *Edin. Med. Journ.*, December, 1876.
† Kaposi, *Hautkrankheiten*, 1880, p. 300.
‡ *Traité de la Goutte*, 1884, p. 376.

somewhat painful, "as if it were sprained," but all symptoms disappeared in the course of twenty-four hours under the treatment already described.

This rapid and evanescent effusion into a joint confirms the view of the truly gouty nature of the case, and is explicable only on the theory of a trophoneurosis.

May 26, 1885.

XIII.—*Case of Hemianæsthesia from Congenital Brain Disease.* By Julius Althaus, M.D. *Read January* 23, 1885.

E. E., a lively girl, æt. 11, was admitted under my care into the hospital on May 15th last with the following history:

She is the eldest of four children, and the three others are quite well. There appears to be no neurotic tendency on the part of either of her parents, but she was delivered with forceps. Immediately after birth she had a succession of convulsive attacks, and her left side appeared to be paralysed. Ever since, the left side has been different from the right, and the arm more useless than the leg, for while she can run about as well as other healthy children, she has great trouble in using her left hand, and can only just hold her fork at table. She began to walk and talk at about the same age as other children, but continued having slight fits at intervals all along. About two years ago she had a much more severe attack than she had ever had before, having been violently convulsed for nearly an hour; and she has since then had one or two such strong attacks at long intervals, with continuous lesser seizures. Of the latter she has occasionally eight or ten in a single day, and then none for a week or ten days. With all this her general health is excellent; she can run about and swing, play battledore and shuttlecock, knit, and do a little reading and writing when she is inclined to, not otherwise. She is, however, subject to "violent moods," in which she kicks, bites, and smashes crockery; indeed, there is nothing too bad to expect of her. At other times she is anxious to please everybody. Her perseverance is great, an instance of this being that she knitted twenty-three pairs of cuffs for children at the Jenny Lind Hospital in Norwich last winter, besides a great many others for other people.

Present state.—The patient is a well-grown and muscularly well-developed child. Her physical health is good. There are no peculiarities in the formation of her skull. Her conversation is impetuous and occasionally silly; she answers questions readily and impulsively. On examining the side said to to be paralysed I found that there was no paralysis of

motion anywhere, but that she was subject to complete hemianæsthesia of the entire left side of the body from the vertex down to the toes. On the right side of the body, on the contrary, sensibility was unusually keen, and the line of demarcation between the sensitive and the anæsthetic zone was sharply defined. In order to show me that she felt nothing in the left hand she bit and scratched it, ran a pin right into the flesh of the hand and the forearm, and then expressed her astonishment that she should not feel anything of it; she added that as long as she could remember she had never felt anything at all in the whole of that side. Indeed, neither contact, nor pricking, nor pinching, are perceived, and there is therefore not only hemianæsthesia but also hemianalgesia. The sense of temperature is likewise absent, as the girl cannot distinguish between two test-tubes, one of which contains hot and the other cold water. The same is the case with the sense of pressure, for she is unable to distinguish between the weight of a sixpenny-piece and half-a-crown. The anæsthesia affects not only the skin, but also the mucous membranes of the eyes, nose, and mouth. The superficial reflexes were everywhere lost, while the deep or tendon reflexes could be elicited. The left pupil was large, although not different in size from the right, and it did not respond to the influence of light, while the right pupil contracted visibly when a lighted match was held near it.

In addition to all this there was anæsthesia of all the nerves of special sense. The patient saw nothing with the left eye. The ophthalmoscopic examination of the fundus of the eye, which was made by my colleague, Dr. Laidlaw Purves, showed "the left optic disc of pearly-white lustre, circular; calibre of vessels small." Smell was entirely lost. I tested the patient with camphor, assafœtida, eau de Cologne, and opoponax, none of which she perceived with the left, but all very keenly with the right nostril. She did not taste quinine, vinegar, tablesalt, and sugar on the left side of the tongue, but most plainly on the right side of it. Here also the line of demarcation between the sensitive and the anæsthetic zone was most sharply defined. The patient was deaf in the left ear, as she did not perceive the ticking of a watch which was pressed on that ear, while she heard the ticking quite plainly when the same watch was held at a distance of four inches from the right ear. Indeed, sensibility was everywhere on the unaffected side so unusually keen that the condition almost amounted to hyperæsthesia.

The muscular force of the affected limbs, as measured by the dynamometer, appeared to be normal. The left hand, however, was clumsy and awkward in its movements. The patient could play on the piano a little with the right, but not with the left hand. This awkwardness of the hand, however, partook of the nature of ataxy, and not of paralysis, and was evidently owing to the loss of sensation. There was ischæmia in the left side, as punctures did not bleed.

From these symptoms and the history of the case I drew the conclusion that the patient had, through the pressure of one of the blades of the forceps, during delivery, suffered from the effects of squeezing of the right hemisphere, and that this had probably led to rupture of a blood-vessel, and hæmorrhage limited to that part which is generally designated as the posterior third of the posterior segment of the white internal capsule. Flechsig has shown that the internal capsule consists of three distinct portions, viz. 1st, the anterior segment, which is situated between the nucleus caudatus and the anterior extremity of the nucleus lenticularis; 2ndly, a central segment, which he has called the knee of the capsule; and 3rdly, of the posterior segment, which is situated between the thalamus opticus and the posterior extremity of the nucleus lenticularis. This posterior segment has again to be divided into a larger anterior and a smaller posterior portion. The two anterior thirds of it contain the pyramidal strands, that is to say, the paths which conduct the power of motion that is generated in the central convolutions of Rolando and the corpus striatum, to the opposite side of the body; and Charcot has shown that the posterior third of the same segment contains the paths for the conduction of all forms of sensation from one side of the body to the centres of sensation in the cortex of the brain. Indeed, we find in permanent hemiplegia with late muscular rigidity, destruction of the two anterior thirds of the posterior segment of the capsule; while in hemianæsthesia of cerebral origin, the lesion has been discovered in the posterior third of that segment. As there was no trace of paralysis in the present case, it would appear that the clot had spared the anterior portion of the third segment of the capsule, but had compressed the posterior end of it, thus preventing the transmission of any form of sensation to the cineritious substance of the right hemisphere. That the affection must have been due to a definite lesion, either of the cortical centres of sensation, or of the conducting paths in the capsule, could surely not be gainsaid from the preceding description. It would

be an absurdity to assume the existence of hysterical hemianæsthesia in a newly-born infant; nor could the idea of a tumour be entertained, as the affection had never varied throughout life, but had always been present in exactly the same degree.

Electricity appeared to me under these circumstances the most appropriate remedy for the condition with which I had to deal; and amongst the various methods of application which presented themselves to my mind, I selected that which is known as faradisation of the skin. I used Stöhrer's double-celled induction coil, and a large soft gilt-wire brush as the active electrode, while the circuit was closed by a moistened conductor applied to the nape of the neck. The brush was slowly passed over the back of the left forearm, and a current strength which generally produces a smart sensation in a healthy person, was not perceived. The first faint sensation of tingling was felt in the forearm with a current strength of 10°; on gradually adding five more degrees, a more decided feeling of pricking and heat was caused; and this sensation gradually became much stronger during the further course of the application. This latter lasted altogether five minutes, and no part of the body except the left forearm was touched. The result was most astonishing, as immediately after the application sensation was found to have been completely re-established in the entire left side of the body.

The girl was kept under treatment at the hospital for some time longer for the epileptic seizures from which she suffered, and at her discharge some weeks afterwards sensation appeared to be quite equal in both sides of the body, the condition of hyperæsthesia on the right side having greatly abated. The surprisingly rapid effect of a single application of electricity, in restoring an important function which had been in complete abeyance during the patient's previous lifetime, may be explained by assuming that the clot of blood, which was the primary cause of the affection, had been absorbed soon after birth, but that this lesion had left a condition of functional inertia or paralysis in the part originally affected. Pricking, pinching, biting and scratching, which the patient herself had abundantly applied to the suffering parts, had not been able to rouse the dormant energy; but this was easily effected by the more suitable stimulus of electricity, which overcame without difficulty the impediment which had so long existed in the conduction of sensitive impressions to those cortical centres by which they are appreciated.

To illustrate Mr. Charters J. Symonds's Œsophageal Tube *in situ*.

A. Upper aperture of larynx.
B. Œsophagus laid open.
C. Silk thread by which the tube is retained in position.
D. Wide upper end of the tube above the stricture.
E. Narrower lower part of tube below the stricture.
F. Cardiac end of stomach.

XIV.—*A Case of Malignant Stricture of the Œsophagus illustrating the use of a new form of Œsophageal Catheter.* By Charters J. Symonds, M.S. *Read January* 23, 1885.

JAMES M., æt. 40, warehouseman, was admitted into Guy's Hospital under my care June 23, 1884. Seven weeks ago he began to have difficulty in swallowing; this rapidly increased, so that when first seen on Monday, June 23, he assured me that he had not swallowed anything for two days, and that for some time previously he had been restricted to fluids. The man was much emaciated, having lost 2 st. 10 lbs. in the seven weeks. He was very weak and scarcely able to come up to the hospital. He complained of hunger and sleeplessness, and had all the symptoms of œsophageal obstruction. I immediately attempted to pass a bougie, but failed to introduce even the smallest size. The obstruction was met eleven inches from the teeth, and appeared to be about the bifurcation of the trachea. He was ordered, for that night, nutrient enemata every four hours, and to take by the mouth what beef tea and milk he could. On the 24th he was better, having swallowed a pint of beef tea and some milk. He continued to take fluids easily by the mouth, so the enemata were discontinued. By July 10 he had gained 8½ lbs., and though swallowing well, it was impossible to pass a bougie.

July 15.—I succeeded in introducing a long œsophageal catheter about No. 10 gauge. This was the kind of tube recommended by Mr. Durham at a meeting of the Society, and projected from the mouth. He retained this about thirty-six hours, and then coughed or pulled it out. In this particular case the tube produced great distress, partly owing to laryngeal irritation, and partly to his being obliged to expectorate his saliva constantly during the night. Being anxious to avoid the operation of gastrostomy, it occurred to me that the inconveniences of the tube would be removed, and all its advantages retained, by making it shorter and retaining it by means of a piece of silk. I had accordingly a piece of œsophageal tube about six inches long, connected by German silver to a boxwood funnel, with a strong silk thread passing through the wood.

July 22.—This tube was passed through the stricture by means of an ordinary conical bougie fitted into the funnel. The bougie being withdrawn, the funnel end was left resting on the upper face of the stricture. The silk passing from this point upwards out of the mouth, was looped over the ear and fastened by a piece of strapping behind. This tube was retained eight days, and was easily withdrawn by the silk ligature. He swallowed fluids freely through it, enjoying at the same time the taste of his food, and was relieved of the constant expectoration caused by the other tube. No tube was worn during the next twelve days.

August 12.—A tube with an ivory funnel was introduced, and retained ten days. During a week of this time the man returned home, and experienced no inconvenience whatever. Being alive to the possibility of ulceration of the œsophagus from the use of wood and ivory, Messrs. Down Bros. made me some tubes composed entirely of gum-elastic; the earlier ones were lined with silver to afford a firmer attachment for the silk, but in the later ones this lining has been omitted, and no difficulty has been found in withdrawing the tubes.

August 27.—A tube was passed. I was now leaving for my holiday and placed my patient in charge of my dresser, Mr. Campbell Gowan, to whom I am greatly indebted for successfully carrying out the treatment during my absence, and for suggesting a plan of introducing the tubes which has proved of great service. After ten days the tube was removed, cleaned, and reintroduced. Four days later (September 12) a fresh tube was inserted, and retained twelve days. The patient had, up to this date, gained 1 st. 9 lbs., and could, with the tube in, drink a pint of milk at a draught. He went daily into the grounds.

October 9.—The tube removed after fifteen days, was still in good condition (this tube was reintroduced November 1). A fresh tube without the silver lining was now inserted. It was removed on the 23rd, cleaned, returned, and retained another week. It was thus, after three weeks' residence in the œsophagus, still strong and but little altered. An attempt to reintroduce it failed and the same result followed on the 31st.

November 1.—The difficulty in swallowing is now increasing, and he has lost weight. The tube removed on October 9 was introduced, after some difficulty, by directing it along the posterior wall of the œsophagus. It was tightly grasped by the stricture. The man was at once able to swallow freely.

About October 9, the patient began to have cough with

foul expectoration, and to complain of thoracic pain. These symptoms have not increased, and at present (November 10) he is comparatively comfortable, and is able to go out daily.

Remarks.—The tube which I introduce has proved of the greatest service in the treatment of the patient whose case I have related, and there is no evidence that any injurious effect has been produced upon the œsophagus. The funnel end engages the stricture, and has an outside measurement of one half to three quarters of an inch. The tube is six and a half inches long, has an ordinary catheter end and eye. It is prevented slipping down by the silk cord in addition to the funnel expansion, and by means of this cord it can be easily withdrawn.

This form has many advantages over the long tube which projects from the mouth; it is not unsightly, it does not interfere with deglutition in any way, it produces no irritation of the larynx, and retains to the patient the pleasures of taste.

The largest tube introduced in the present case was a No. 12 catheter gauge, but, if taken earlier, a much larger tube might be used. What is the largest that may be safely employed remains to be seen. So complete did the obstruction appear to be that I expected to be obliged to perform gastrostomy the day after admission. This operation is often attended with so much distress from excoriation of the skin around the opening that I was glad to avoid it, and I cannot but think that so long as these tubes can be passed, the result will prove more satisfactory than the gastric fistula. In order to ensure the easy reintroduction of tubes, I propose to have some made with an open end, and before withdrawing, to run through the tube a guide over which it can be removed and a new one introduced. My main object, at present, is to bring the principle of the treatment before the Society, and to solicit opinions as to its value.

Note.—The patient died on April 17.

From January 23, the day on which he was exhibited at the Society he continued to wear the tubes till the time of his death.

February 16.—It was found that the stricture had dilated so that the funnel easily passed through it. I therefore fitted a silver funnel into a large-sized india-rubber tube and inserted this. He was able to take minced meat through the tube, and the cough which accompanied swallowing with the smaller

tubes disappeared. This was removed in three weeks, cleaned, and returned for thirteen days.

March 10.—He weighed 7 st. 13½ lb. and in the next week lost three pounds; the expectoration abundant and very foul and the cough disturbs his rest.

March 23.—There is dulness at left base with râles and general signs of pulmonary complications.

March 25.—He could not swallow at all, violent fits of coughing accompanying every attempt. This appeared due to the growth having extended beyond the limit of the tube. I therefore passed a long gum elastic tube of No. 14 gauge, and appeared to traverse a second stricture. Through this he at once swallowed freely and the tube was worn till his death, and is now preserved with the specimen. He died with symptoms of gangrene of the lung, and the inspection revealed this change in the left lower lobe. There was pus in the pleura, and pneumonia of the right base. The growth in the œsophagus was four and a half inches in length, and began eleven inches from tip of tongue, and four inches from cricoid and reached to within one inch of the cardiac orifice of the stomach. There was considerable narrowing at the upper end, but in the rest there was great loss of substance from ulceration, the aorta being exposed at one point, the pleura at another, and the mediastinal glands at a third. The trachea was not involved. There was no evidence whatever of ulceration above the stricture from the pressure of the tube, which reached through the growth into the stomach.

In reviewing the case it may be shortly added that the man was kept alive eight months in comparative comfort; that he never experienced any inconvenience from the tubes; that the stricture dilated considerably, and that no injurious effect attributable to the tube was apparent at the post-mortem. During the later stages it was necessary to remove the tube more frequently, as it became blocked by the sputum. As to the durability of the tube and silk it may be stated that one tube with its silk was in the œsophagus altogether for three months, and still remains sound and fit for use.

XV. *A Case of Myxœdema with a Post-mortem Examination.* By W. Hale White, M.D. *Read February* 13, 1885.

IN the first place I feel that I must explain the late appearance of this paper. The post-mortem examination was made last June. In the autumn I prepared the microscopical sections and wrote an account of them. My friend Dr. Mahomed was to have written the clinical part of the paper and to have prefaced my pathological account with it, but his sad illness prevented this ever being accomplished, and hence the imperfection of the following account of the patient's condition whilst alive, for I have had only the clinical clerk's notes for the description of the symptoms. The same reason will also I am sure be accepted as sufficient excuse for the pathological results only appearing now, although I had described them in October; it will be noticed that Mr. Victor Horsley's Brown Lectures delivered last December confirm them in every particular.

At page 98 in volume xv of this Society's *Transactions* will be found recorded by the late Dr. Mahomed a case of myxœdema which seemed at first to improve under treatment by nitro-glycerine, but the patient soon relapsed into her former condition, and was not permanently benefited by the treatment. The case was complicated by albuminuria.

On June 12, 1884, the patient was admitted into Guy's Hospital for ascites. She had not been seen since July, 1884, but in that interval had enjoyed good health, and the myxœdematous condition had diminished. A year before admission she first noticed the commencement of abdominal distension which began two months after her being delivered of twins. The abdomen continued slowly to increase in size till admission, when it was noticed that the myxœdematous condition was less marked, although it could be seen in the face and hands, and the speech was slow. Three days before admission she had an epileptic fit.

On June 17, paracentesis abdominis was performed and 5 gallons 5 pints of fluid were withdrawn. Soon after this she had several epileptiform fits and she passed into a status epilepticus and died.

At the autopsy, the brain was anæmic, the cerebral vessels decidedly thick, and there was an old elongated brownish hæmorrhage into the outer division of the lenticular nucleus, at its posterior part, not involving the external capsule. The thyroid was very small and atrophic, it had lost its ordinary juicy red appearance and was pale and shrunken looking, the cervical glands were healthy. The heart and lungs were normal. There was much chronic peritonitis; soft and deeply pigmented lymph lined the abdominal cavity and bound the intestines together. The walls of the stomach, intestines, and abdominal aorta were thick and juicy, but this was probably due to the ascites. Much perihepatitis, the capsule of the liver being uniformly thickened and white, the hepatic tissue itself was fatty; the capsule of the spleen was also thickened. Pancreas, mesenteric glands, suprarenal bodies and abdominal sympathetic all appeared normal. The kidneys might have been a little fatty, otherwise they were quite normal. The tongue seemed normal; there was a distinct blue line on the gums as if from lead. The genital organs and bladder were all healthy. The chief muscles of the body were normal and there was no gout in the great toes. The right middle cervical ganglion was larger than the left, and had a well-marked blood-vessel ramifying down it. The pituitary body seemed large.

Histological examination of the organs.

1. *Lungs* healthy.

2. *Kidneys.*—The only abnormality to be detected is that there is a slight proliferation of nuclei in the intertubular connective tissue and also perhaps on the Malpighian bodies.

3. *Liver.*—The only changes are a proliferation of nuclei in the intercellular connective tissue, as described by Ord, and a fatty change in the liver-cells. This is not extreme, but is peculiar from the fact that the fat is deposited around the intralobular vein rather than around the periphery of the lobule.

4. *Spleen* healthy.

5. *Submaxillary gland.*—Between the cells there is a slight proliferation of nuclei, but the chief change is seen in the larger masses of connective tissue such as those around the blood-vessels and lobes of the gland. The connective tissue in these situations is, I think, best described as having a degenerate sodden appearance; there seems to be more of it than natural, and yet there is no multiplication of any of its

elements. Such an appearance might be produced by effusion into it. Very few nuclei are visible, and in many parts the fibres themselves can hardly be distinguished; it looks just as though they had degenerated into a clear material which did not stain with logwood. As these changes have taken place in connective tissue around blood-vessels it gives the appearance that the tunica adventitia of the vessels is much thickened because that, in common with other connective tissue, is altered and no distinct line of demarcation is observable between it and the surrounding tissue.

6. *Thyroid gland.*—There is little or no proper thyroid structure left; a few bodies, evidently the remains of vesicles, are to be seen, and in one or two instances just a trace of the epithelial lining still remains. These degenerate vesicles are filled with small epithelial cells which have been apparently produced by the multiplication of the proper epithelial lining. The connective tissue between the vesicles would for the most part be suited by the description of the connective tissue in the submaxillary gland, having a sodden, degenerate appearance, but, whilst nowhere is there any evidence of the formation of new connective tissue, yet here and there in patches there is considerable small cell proliferation, rendering the section quite dark. There are to be seen in places some white blood-cells which have evidently wandered out of the blood-vessels. The whole organ has undergone extreme simple degeneration.

7. *Heart.*—The muscle-cells are perfectly normal; in parts the connective tissue appears to have the same condition as has already been described, but the change is not so universal and might perhaps have been passed over had not it been already detected in other organs.

8. *Right middle cervical ganglion.*—The cells are abundant, very few pigmented; in many a distinct nucleus and nucleolus are visible. Considering how cells may vary in ganglia which we have no reason to think abnormal, these cells appear very healthy; it is true that some are blurred and have no distinct nucleus, but this is not more so than is often the case. The connective tissue has the degenerate, ill-defined, sodden appearance already described; the result of this is that it has pressed on the nerve-cells so as to reduce the size of the capsule, and in several instances the connective tissue is brought in such close contact with the cell that the line of demarcation is not very evident. Here and there this condition of the connective tissue obscures the nerve-fibres. There is no small cell proliferation.

9. *Right superior cervical ganglion.*—The description of the middle ganglion applies here, except that the changes described in the connective tissue are not so marked; hence as there is less degeneration a few more nuclei are to be seen; the alteration is most evident in some connective tissue around a vessel, giving the appearance of great thickening of its adventitia. The nerve-cells are healthy.

10. *Right cervical sympathetic nerve.*—In longitudinal section it is to be noted that the nerve-fibres and also a few nerve-cells which happen to be present are quite healthy; the connective tissue around the nerve appears very abundant, and has somewhat of the character already described.

11. *Left superior cervical ganglion.*—The remarks made about the right cervical ganglion apply here also. The condition of the connective tissue is to be noted, and also the large quantity of it around the ganglion.

12. *Semilunar ganglion.*—The cells are well formed and there are plenty of healthy nerve-fibres; the connective tissue has not the same appearance as in the other ganglia, but here and there are plenty of small cells, if anything, more than the normal number, which can be seen in places distinctly developing into fibrous interstitial tissue.

13. *Anterior crural nerve.*—Normal. The connective tissue is abundant, and has much the same appearance as in other parts.

14. *Suprarenal body* perfectly healthy.

15. *Lymphatic gland* quite healthy.

16. *Carotid artery* perfectly healthy.

17. *Pituitary body* normal.

The histological examination of the organs fully bears out the position taken up by Dr. Semon that the atrophy of the thyroid is the cause of the whole disease, because in this case it is the only discoverable pathological lesion, excepting the myxœdematous condition affecting various parts of the organism. The changes in the sympathetic ganglion and submaxillary gland are simply the results, and not particularly important results, of the atrophy of the thyroid. This view is fully borne out by the fact that in Kocher's patient, in whom the thyroid was totally extirpated and no myxœdematous symptoms followed, there was found an accessory thyroid body which underwent compensatory hypertrophy. Our case also shows that the view put forward by Dr. Hadden that the disease is due to a lesion of the cervical sympathetic is untenable, for the specimens show that the essential parts, such as nerve-cells and fibres, are

quite healthy, and also against the sympathetic theory I would urge that if the disease is due to a lesion of the cervical sympathetic, evidences of this, such as vasomotor and pupillary disturbances, should be constant symptoms in myxœdema as they are in all cases of tumours, aneurysms, and injuries of the neck in which the cervical sympathetic is injured. This is not so, and, further, in none of the numerous physiological experiments on the cervical sympathetic have the symptoms of myxœdema ever been observed. Lastly, on the hypothesis of disease of the sympathetic it is quite impossible to explain the case in which the whole thyroid was removed and no myxœdema supervened, but the accessory thyroid hypertrophied. In this case no inference can be drawn from the difference in size of the two middle cervical sympathetic ganglia, because normally the variations in size are so great.

Since the above was written Mr. Victor Horsley has drawn attention to the tremors observable in monkeys after excision of the thyroid. This symptom has not been frequently observed in myxœdema; hence it is interesting to note that the patient is described as having had epileptic fits. What they were like I cannot say; as I never saw them I can only quote the report of the case.

XVI.—*Two Cases of Phlegmonous Pharyngitis.* By R. E. Carrington, M.D. *Notes of Post-mortem* by W. Hale White, M.D. *Read February* 13, 1885.

THE following cases are brought before the Society because they seem to be examples of a disease which hitherto has not been recognised clinically to any great extent. Patients labouring under the affection are admitted into hospital with symptoms of laryngeal dyspnœa, and tracheotomy may or may not be performed. It appears, however, always to be useless, and the patient dies of the general and not of the local condition.

The disease is doubtless of considerable rarity as is evidenced by the fact that Birch-Hirschfeld (*Lehrbuch de Pathologischen Anatomie*) makes no mention of it, although he describes the state of the larynx in over six thousand autopsies at the Berliner Charité.

I may add that I know of at least two other undoubted cases, which, however, I am not able to publish.

Case 1.—T. M., æt. 46, was admitted on the early morning of May 28, 1884. He had been ill the previous seven days somewhat remittently, some days being passed in bed; on others he did his work. On his admission there was laryngeal stertor, but no marked dyspnœa. He was placed in a tent with a steam apparatus and watched. In the course of a few hours, however, he died quite suddenly before the house surgeon could be called in again.

The autopsy was made nine hours after death by Dr. Goodhart, by whose kindness I am able to publish the report. He was a fine, healthy-looking man, well nourished. There were no sores, scars, nor signs of injury. There was much subpleural ecchymosis on the right side. The lungs were somewhat airless and congested. All the soft tissues of the pharynx were œdematous, and on the right side from the tonsil downwards for two inches the mucous membrane was minutely injected and swollen with purulent œdema. Yellow spots of pus pointed here and there, and on section there was a diffused purulent infiltration of the mucous membrane and submucous tissue. The same condition had

spread into the tissue of the epiglottis so that the cartilage was buried in a similar diffuse infiltration. On the left side the œdema was serous only. There was very little narrowing of the rima glottidis.

With the exception that the spleen was rather large, though not soft, and that there were a few cysts in the kidneys, all the other viscera were healthy.

Dr. Goodhart suggested in his report that the man died of asthenia rather than asphyxia.

Case 2.—The patient, a medical man, æt. 49, was admitted into Guy's Hospital on November 10, 1884, under my care, when I was doing duty for Dr. Wilks. We could get little or no history from him, but he told us that three days before he began to suffer from a severe "cold," and this had gradually become worse, so that he himself came up to the hospital urgently requesting tracheotomy to be performed. An old friend, an esteemed member of the profession, knew him well, and felt sure that he was by no means an intemperate man, but it was ascertained that probably for some little time before his illness he had been indulging a good deal in beer. He came up to the hospital at 2 P.M., and was at once placed in a tent before the fire, the air of which was moistened by a steam apparatus. He walked up to the ward, and though there was a good deal of laryngeal stridor, there was no "sucking in" of any part of the chest, so that it was not thought advisable to perform tracheotomy before trying whether any relief would be obtained from palliative treatment. He was in a great state of apprehension and very restless, tossing his arms about, and still urgently desired the operation. At 4.30 he was seen by Mr. Clement Lucas, and operation was decided upon. The patient vehemently shook his head when chloroform was suggested.

At 4.45 when desired to leave his bed to walk to the table, a distance of two or three yards, he was suddenly seized apparently with a spasm of the glottis, fell back and ceased to breathe. Tracheotomy was immediately performed and the tube inserted without delay. Artificial respiration by Silvester's method was at once resorted to, and was so far successful that in a few seconds he began to breathe spontaneously although very feebly. Almost immediately he was seized with an epileptiform attack, his eyes were fixed, his face, at first pale, became blue, and the muscles twitched convulsively. Artificial respiration was still maintained, subcuta-

neous injections of brandy were administered, and hot water applied to the præcordial region. The result was that after some little time the patient began to breathe slowly and very feebly. A couple of capsules of nitrate of amyl were now administered. The heart's action had never ceased, but the pulse had become very feeble, and during the fit almost imperceptible. The feeble, slow respiration continued, and he appeared to regain a certain amount of consciousness, looking now and then at those about him. He was also able to swallow a small quantity of brandy and milk. In fact he appeared to be progressing favorably. The bed was then moved to the tent, his head slightly raised on a pillow, the steam apparatus turned on, and arrangements were made to leave him under proper supervision. But he suddenly again became pale, his eyes turned upwards, and he ceased breathing. He then became cyanosed, and double internal strabismus ensued. The pulse again became almost imperceptible. Artificial respiration was at once resorted to, and I may here remark that it was respiration of a very effectual kind, viz. a combination of Silvester's and Howard's. The arms were raised above the head by one operator, whilst another knelt across the patient and compressed the chest in alternation. The amount of air passing in and out of the cannula by these means was very considerable. This was kept up for a full hour, until 6.20 P.M. Subcutaneous injections of brandy and ether were administered. The faradic battery was freely used, but all was of no avail, he never breathed again, and the heart-sounds became inaudible at least half an hour before efforts at resuscitation were discontinued.

Dr. Hale White furnishes the following account of the autopsy.

At the post-mortem examination the following condition was discovered:

The larynx was congested and dark blue in colour, especially at its posterior part; the epiglottis was reddened, thickened, but not ulcerated; the rima glottidis appeared perhaps slightly smaller than natural; there was some œdema of the glosso-epiglottoidean folds. The right ary-epiglottoidean fold was reddened, and had in it an open abscess-cavity about three quarters of an inch long and half an inch wide, the long axis corresponding with the long axis of the fold; this cavity was shallow, and its floor was coated with pyogenic membrane. The left ary-epiglottoidean fold was thickened and congested, and on making an anterior posterior vertical section through the

soft parts outside it, the fat and cellular tissue were found to be infiltrated with a greenish-yellow purulent material which had not anywhere formed a cavity. There was no ulceration within the larynx, and except for perhaps slight thickening the vocal cords seemed healthy. No disease of the cartilages could be detected after prolonged and careful dissection. The two alæ of the thyroid were much ossified. No membrane was present. The left crico-thyroid and thyro-arytenoid muscles were obviously inflamed but not suppurating. The active disease was, it will be seen, chiefly extra-laryngeal, although there was some swelling of the folds and epiglottis. The tracheotomy wound had gone through the lower quarter of an inch of the crico-thyroid membrane, through the cricoid cartilage, and just down to the isthmus of the thyroid. There was no impediment to the free entrance of air below it. The contiguous lymphatic glands were reddened, the thyroid gland was healthy. There was no evidence that the vagus nerves were implicated.

The lungs were intensely congested and somewhat softened at the posterior parts; no pus was found in the bronchial tubes, but, together with the trachea, they were much congested.

The heart was considerably fatty, and weighed eight ounces. There was no noteworthy change in any other organ.

XVII.—*A Case of Locomotor Ataxy, without Disease of the Posterior Columns of the Spinal Cord.* By A. Hughes Bennett, M.D. *Read February* 27, 1885.

THE case to which I beg to direct the attention of the Society is specially interesting from the circumstance that, although the patient during life presented all the prominent symptoms of so-called tabes dorsalis, the posterior columns and cornua of the spinal cord were found after death to be without a trace of disease. This fact suggests certain important considerations concerning the physiology and pathology of locomotor ataxy.

The patient was a gamekeeper, aged 48, who stated that he had always been a healthy man, never having suffered from syphilis or other disorder. About fifteen years ago he complained of severe pains in his legs, which continued for three months, from which symptoms he completely recovered. He afterwards remained well till nine months before he came under observation. He then for the first time experienced gradual weakness of the legs, so that he was easily fatigued and was unable to do a full day's work. This slowly increased, without, however, actually incapacitating him from his duties. Some months afterwards he was attacked with severe shooting pains in his lower extremities, occurring in paroxysms, and shifting from place to place. With the advent of these the weakness of the legs augmented, and was accompanied with a sensation of numbness in the feet. He was therefore compelled to give up his situation as a nobleman's gamekeeper.

On examination the patient was found in good general health. There was no evidence that his intelligence was abnormally affected, but he was an uneducated and stupid man from whom it was difficult to obtain a satisfactory account of his illness. There were no headaches or cerebral symptoms except occasional giddiness. Vision in both eyes was good, there was no paralysis of the ocular muscles, and the fundi of both eyes were normal. Voluntary movements of the eyeballs were accompanied with slight nystagmus. The pupils

were of moderate size, the left somewhat smaller than the right, and both contracted well to light and accommodation. The muscles of the face and tongue were as in health, and articulation, mastication, deglutition, and all the special senses were normal. The movements of the upper extremities could be all performed, but the grasp of the hands was weak, and delicate actions of the fingers were unsteady and slightly inco-ordinated. The movements of the trunk seemed normal, and the functions of the bladder, rectum, and sexual organs appeared to be intact. The patient felt his lower extremities to be weak. He walked with a slow, unsteady, hesitating, and markedly ataxic gait. Without assistance he staggered, had difficulty in turning or walking in a straight line. He could not stand on one leg, and all his motor difficulties were increased in the dark. When the eyes were closed the patient swayed and would have fallen if not supported. In bed the movements of the lower limbs were vigorous and displayed considerable force, but they were accompanied by the typical phenomena of inco-ordination. The knee jerk on both sides was totally absent and the plantar reflexes were diminished. There was very slight rigidity of the large joints, which was easily overcome by passive movement. The sensibility of the skin to touch and pain appeared to be everywhere normal, except in the feet and legs, where it was both diminished and retarded. The patient complained of a constant dull aching in his back and lower limbs, and this was almost daily accompanied by attacks of lancinating pains which shifted about from place to place, and which were sometimes very severe. The muscles throughout the body were thin and spare but nowhere specially atrophied; their mechanical irritability and electrical reactions were unimpaired. The other organs and functions of the body were normal.

The patient died after a residence of two and a half months in the hospital. From the beginning the weakness of the legs and insecurity of gait increased, so that in about six weeks he could not stand. The lancinating pains were very constant and severe. For many weeks there were attacks of uncontrollable vomiting. Emaciation ensued and the general health broke up. During the last week the patient was feverish, restless, and prostrate; he became semi-comatose, and finally, after a severe convulsive attack, died. Otherwise no new or special symptoms developed.

Post-mortem examination (30 hours after death).—Permission was given to examine the brain and spinal cord only.

The entire body was thin and emaciated, but nowhere was there any special atrophy. On the right gluteal region there was a superficial bedsore about three inches in diameter, and there was commencing change in the skin on the corresponding left side and over the sacrum. On removing the calvarium the membranes of the brain were found deeply congested; there were considerable œdema and sub-arachnoid effusion, but no adhesions. The convolutions were somewhat flattened, especially on the left side, in the frontal and parietal regions, otherwise they were normal. On removing the brain several ounces of cerebro-spinal fluid escaped. The vascular congestion was found to be universal; in other respects the blood-vessels were healthy. On slicing through the hemispheres the cerebral substance was seen to be deeply injected, the lateral ventricles each contained about half an ounce of clear yellow fluid, and the choroid plexuses were œdematous. In the substance of each frontal lobe, involving the anterior cornua of the lateral ventricle, was a circumscribed patch of softening, on the left side about the size of a hen's egg, on the right somewhat smaller. These extended inwards to the third ventricle and longitudinal fissure, downwards to the base of the brain, and upwards to within an inch of the cortical substance. The under and fore part of the corpus callosum, the lamina cinerea, and the tuber cinereum were involved in the softening; but the fornix, the corpora albicantia, the corpora striata, and optic thalami were apparently normal. There was a similar patch of softening in the cerebellum about the size of a walnut, situated at the left side of the anterior free margin of those lobes forming the upper boundary of the great longitudinal fissure. The middle and inferior peduncles on the left side were softened, but the superior remained of normal consistency. To the naked eye the pons, medulla, corpora quadrigemina, cerebral peduncles, and other parts of the encephalon appeared healthy.

Microscopical examination of the softened cerebral matter showed much granular detritus, broken-down cells, Gluge's corpuscles, numerous oil-globules, and shrivelled blood-corpuscles.

On opening the spinal canal the vessels of the membranes were seen to be deeply congested, especially in the lower dorsal region, where there were found small patches of superficial hæmorrhage. There were no adhesions either to the bone or to the cord, and the latter was enucleated without difficulty. The pia mater was thickened, especially at its posterior aspect, most marked inferiorly, and gradually diminishing from below upwards. Behind, the membrane was studded throughout

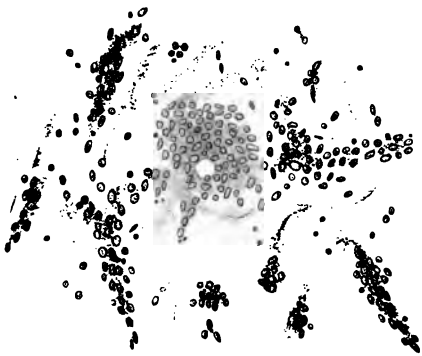


Fig. 1.

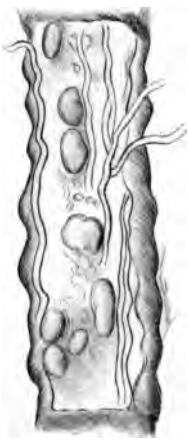


Fig. 2.

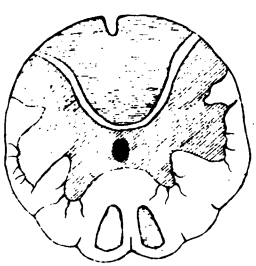


Fig. 3.

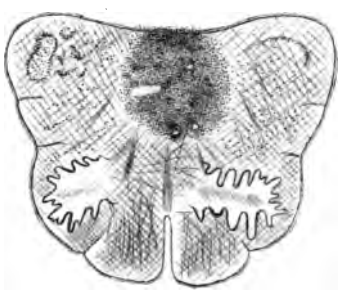


Fig. 4.

Dr Hughes Bennett's case of Locomotor Ataxy.

E. Thurston, delt

Dantelsson & Co lith.

DESCRIPTION OF PLATE VII, ILLUSTRATING DR. BENNETT'S CASE OF LOCOMOTOR ATAXY.

FIG. 1.—Section of morbid growth, 200 diam., showing cellular structure of sarcoma.

FIG. 2.—Naked-eye appearance of the middle dorsal region of the spinal cord, showing multiple sarcomatous tumours scattered over the posterior aspect of the pia mater.

FIG. 3.—Transverse section at the lower part of the medulla oblongata, showing the central canal surrounded by sarcomatous growth.

FIG. 4.—Transverse section at the upper part of the medulla oblongata, showing the floor of the fourth ventricle involved by sarcomatous growth.

with what to the naked eye appeared to be a number of isolated tumours. These were of a round or oval shape, of firm hard consistence, of a white colour, and they varied in size from a mustard seed to a pea. At the medullary end of the cord these were few in number, but on proceeding downwards they gradually augmented, so that in the middle dorsal region they were perhaps from six to eight to the square inch (Pl. 8, fig. 2). Continuing to increase in number towards the lower portion of the cord, they became in the lumbar region quite confluent so as to make the cord in that situation of abnormal thickness and rigidity. These growths were seen to be attached to and to surround the posterior roots. To the naked eye none of these tumours were seen on the anterior or lateral aspects of the cord.

The medulla and cord were first hardened in Muller's fluid and strong methylated spirit. They were then immersed for several days in absolute alcohol, and afterwards embedded in celloidin. Sections were made by Dr. Hebb, Pathologist to the Westminster Hospital, to whom I am indebted for the following description of the microscopical appearances. "There was general thickening of the pia mater most marked at the anterior and posterior aspects. This diminished gradually from below upwards, and at any given level was much more marked behind than in front. In the lumbar region the neoplasm which constituted the thickening of the membranes closely embraced the posterior, and to a slight extent the anterior roots; in the dorsal region the posterior only, and in the cervical region the cell growth had greatly diminished in extent and did not involve any of the nerve-roots. In a limited portion of the lumbar region, one anterior cornu was involved by the disease, the cells of the neoplasm having advanced along the lines of the cornual rootlets and blood-vessels. This seemed to have invaded the normal structures without causing their destruction to any appreciable extent. With this exception the nervous tissues of the cord were normal (Pl. 7, figs. 1 and 2). Some of the anterior spinal roots were involved in the growth, but only to a limited extent in the lower lumbar region. Their structure on section seemed normal. Although the tumour closely surrounded the posterior roots on both sides throughout the dorsal and lumbar regions their histological condition appeared to have suffered little demonstrable change. Examined with a high power (one twelfth of an inch) the only peculiarity that could be detected was unusual swelling of the white substance of Schwann. The

state of the nerve-trunks of the body with their peripheral terminations in the muscles and skin, were not examined.

"The medulla oblongata, which to the naked eye appeared normal, after staining, disclosed in its upper part a morbid mass measuring 0·5 × 0·7 cm., which extended from the floor of the fourth ventricle, into which it projected, to the centre of the bulb, towards the lower portion of which it diminished in size and was limited to its centre, surrounding the central canal, thus being of conical shape, the base upwards (Pl. 8, figs. 3 and 4). The morbid material here also seemed only to infiltrate the otherwise healthy tissues rather than destroy them. In addition to this fairly circumscribed mass there were a number of minute patches of similar nature scattered throughout the substance of the medulla.

"The neoplasm was everywhere composed of round cells, and was sarcomatous in character (Pl. 8, fig. 1)."

Commentary.—In commenting on the facts of this case, special attention is directed to the pathological and clinical phenomena.

1. *Pathological.*—The patches of softening in the frontal lobes, and in the cerebellum and its peduncles, were probably recent, and due to the general cerebral inflammation, which supervened shortly before death.

The sarcomatous mass as seen by the microscope in the centre of the medulla, was in size and shape fairly defined. The morbid process was not strictly limited to this area, but was dotted here and there all over the bulb. Such a pathological condition is extremely rare. The fact that the growth seemed to infiltrate and displace the normal tissues rather than destroy them, may explain the almost total absence of serious bulbar symptoms during life.

Disease of the same nature involving the spinal membranes, appearing to the naked eye as a series of small multiple tumours confined to their posterior aspect, and surrounding the posterior roots, is also an interesting and almost unique pathological condition. Histologically the lesion was found to leave the cord itself absolutely intact, except at one anterior cornu, in a limited portion of the lumbar region, where the tissues, as in the case of the medulla, were displaced but not extensively destroyed. This also accounts for the absence of symptoms due to serious implication of that locality. The anterior roots were seen to be only slightly implicated, which harmonises with the fact that there was no paralysis, wasting of the muscles, or abnormal electrical reactions during life.

DESCRIPTION OF PLATE VIII, I
BENNETT'S CASE OF LOCO. .

FIG. 1.—Transverse section of the spi.. region, showing the posterior roots involv..

FIG. 2.—Transverse section of the spir region, showing the posterior roots, and, n. roots and the anterior cornu involved in s..

ally, the posterior roots were extensively involved, and though the anatomical changes in their structure were slight indeed doubtful, they were sufficient to induce marked ormalities in the functions of the sensory tracts.

2. *Clinical.*—The points of clinical interest may be considered under two heads, namely the phenomena connected a disease of the medulla, and of the spinal cord. From position and extent of the growth in the former it might e been supposed that important bulbar centres would have n involved and corresponding symptoms produced. Yet se during life were conspicuous by their absence. There s no visual disturbance and no facial paralysis or spasm; special senses were intact; articulation, vocalisation, glutition, respiration, and circulation were normal, and there as no optic neuritis or glycosuria. The only phenomena ttributable to derangement of the medulla were giddiness, light nystagmus, and during the last few weeks of life persistent vomiting. The explanation of this absence of symptoms s already been suggested.

The clinical point illustrated by this case, to which it is the ject of this paper specially to direct attention, is the relation ich exists between the spinal symptoms and the lesion of posterior roots of the cord discovered after death. The ient had been examined by many physicians, all of whom d pronounced him to be suffering from tabes dorsalis, and is condition in most respects justified that diagnosis. There as inco-ordination in the movements of the limbs and chiefly the lower extremities, with a typical ataxic gait, increased the dark. There was at the same time no appreciable ilure in the gross power of the muscles, nor were these trophied. The Romberg symptom was present, the patient vaying and losing his balance when his eyes were closed. here were paroxysms of characteristic lancinating pains, and efficient and retarded sensibility in the skin of the lower limbs. he plantar reflexes were diminished, and the knee-jerk phemena totally abolished. This sequence of events, although t constituting a complete clinical picture of tabes, warnted, it must be admitted, the conclusion during life that e patient was suffering from that disease. It is true that re was no affection of vision, or trace of optic atrophy. he pupils, though unequal in size, were not paralysed to he stimulus of light or accommodation. There was no bnormality of the vesical or sexual organs, and the disease d only been in existence for nine months. But clinical

experience has shown that none of these circumstances preclude the presence of those central changes which are usually associated with tabes dorsalis, or that the absence of these symptoms is incompatible with a pronounced specimen of that affection as we now recognise it at the bedside. Now, it is universally assumed that the lesion which causes this train of phenomena is situated in the posterior columns of the spinal cord, and our standard text-book on nervous diseases thus expresses the general belief:—"Sclerosis of the posterior root zones for a considerable portion of their longitudinal extent, is the essential morbid alteration in locomotor ataxia."*

In the case before us there were all the prominent symptoms of that affection, without a trace of degeneration of any portion of the posterior columns, the posterior cornua, or the posterior roots within the circle of the spinal membranes. Evidence is thus adduced to show that, in the production of that group of symptoms comprised under the term tabes dorsalis, disease of the posterior columns of the cord is not an essential factor, and that this may be fully developed, that centre being in a perfectly normal condition. This fact had already been observed by M. Dejerine, who has placed at least two cases on record presenting features during life almost exactly the same as the case at present under consideration, in which after death the cord, spinal roots, and nerve-trunks were found healthy.† In these, however, he discovered that there was parenchymatous neuritis of the nerve-endings, in the skin, and, to a lesser degree, in the muscles of the limbs affected. He concluded from this that the ataxic symptoms were due to disease of the sentient nerves in the skin and muscles, producing anæsthesia of the former, and loss of the so-called muscular sense in the latter. Afferent paths were thus interrupted at the periphery which alone was capable of producing the typical condition of ataxy without central change. This form of the disease he proposed to call "*nervo-tabes périphérique*," in contradistinction to the classical tabes dorsalis. In the present case it is a matter of regret that the cutaneous and muscular nerve-endings were not histologically examined; at the same time facts show that the primary disease did not originate there, the post-mortem revelations proving the lesion to have begun in another portion of the nervous system. There can be no doubt that

* *A Treatise on Diseases of the Nervous System*, by James Ross, M.D., second edition, vol. ii, p. 57.

† "Études sur le Nervo-tabes périphérique," par J. Dejerine, *Arch. de Physiologie*, 3me série, vol. iii, p. 231.

locomotor ataxy, as generally met with in practice, is usually associated with degeneration of the posterior root zones. The observations of M. Dejerine demonstrate that symptoms indistinguishable from this may occur without any appreciable lesion of the cord, and may be due to degeneration of the peripheral nerves only. This case makes it evident that precisely similar results may follow interruption of the co-ordinating paths in a third locality, namely, in the posterior nerve-roots. Van Deen and Claud Bernard experimentally demonstrated that the section of these in animals induced total inability to steadily utilise and co-ordinate movement, without causing loss of motor power; in short, a condition of extreme and typical ataxia. We have here before us a repetition of this experiment, produced by disease, and followed by precisely the same physiological phenomena. The posterior roots throughout the dorsal and lumbar regions were embedded in a sarcomatous mass. Pressure on their fibres was the consequence, accompanied by the symptoms of tabes dorsalis. Thus may the anæsthesia, analgesia, and retardation of sensibility be explained. Irritation of the sensory filaments caused the neuralgia and attacks of lancinating pains. Interruption at this portion of the reflex arc accounts for the alteration of the tendon and other phenomena, as well as the production of ataxy and the Romberg symptom. So far then this case supports the view that those elements which convey the impulses regulating co-ordinate movement, are situated in the afferent paths of the nervous system. The disease in this case being limited to the posterior roots, constitutes a more conclusive fact in favour of that theory even than those in which degeneration of the peripheral nerve-endings alone was demonstrated.

It is admitted that in this observation there are two possible flaws against absolute proof of the preceding proposition, namely the existence of implication by disease of the medulla, and anterior roots. Reasons, however, have already been given for the opinion that these complications, for practical purposes, need not militate against the argument.

Whether Dejerine's lesion was primary or secondary to undemonstrable trophic influences in the nerve-centres, need not be here discussed. But, whatever theory be advanced to explain the physiology of locomotor ataxy, and the prominent symptoms of so-called tabes dorsalis, this case serves to show that the point at which, in the causation of the phenomena, the nervous path is interrupted, need not of necessity, as is

generally asserted, be primarily situated in the posterior root zones of the spinal cord.

Reflection on this fact suggests that we have not yet fathomed the anatomical substratum of that Protean disorder which we at present recognise under the term tabes dorsalis. This assemblage of symptoms probably consists of a combination of different pathological conditions, many of which are represented by phenomena common to all, and each of which in time may be differentiated. It is possible that a case such as the present may be a link in the chain of discovery.

Such considerations are of practical utility, as the tendency of the present day is to assume that certain isolated symptoms of necessity indicate the existence of serious and progressive central disease. In practice we see persons who happen to suffer from pains in their extremities, peculiarities of their pupils, alteration in their reflexes, or from other special nervous symptoms, often pronounced as the subjects of tabes dorsalis. These are either made the victims of prolonged and unnecessary treatment on that hypothesis, or doomed as hopelessly incurable. The case under consideration, although it ultimately ended fatally, proves that our present conceptions of the pathology of locomotor ataxy must be modified, and that the generally believed classical lesion for its production is not without exception. It further suggests that there may be other more innocent or removable circumstances which may give rise to the same series of symptoms.

XVIII.—*A Case of Arrested Rickets.* By Seymour Taylor, M.D. *Read February* 27, 1885.

ALEXANDER S., æt. 6, came under my notice at the out-patient department of the North London Consumption Hospital in June, 1884.

The history of the case is briefly as follows :—His mother has had sixteen children, of which the present child was the twelfth. He was born at full term. His father and mother appear quite healthy people, and the former is a teetotaler. His mother has lost five of her children,—two from whooping-cough, one each from bronchitis, from smallpox, and from inflammation of the lungs. All the other children are perfectly healthy, except that two of his sisters, the sixth and seventh children respectively, are knock-kneed.

When 1½ years old the patient fell out of his cot, and his illness with subsequent bending of bones dates from this accident. He was subsequently treated by different practitioners, first on account of bronchitis and soon afterwards for "enlarged liver." At that time he used to scream terribly, especially if lying flat on his back, and he also suffered from profuse sweatings, especially about the head. I have ascertained that at this period he had a distinct attack of laryngismus stridulus. At the time of his fall he was unable to walk; he could not even push a chair in front of him as his limbs and muscles seemed peculiarly painful. He was not restless at night.

The child will be found on examination to have the typical pigeon-breast, with beading of the sternal ends of the ribs. He suffers from the chronic form of bronchitis usual in rickety children. The heart is depressed and pushed somewhat to the right side; there is no cardiac murmur. He is stunted in growth, being 29½ inches in height only, the limbs are shortened. The clavicles have their curves so pronounced, as to become almost V-shaped. The humeri are curved forwards, and are only five inches in length. The forearms are bent, and held in a position of extreme pronation. Supination is only possible as far as the supinator longus will allow. He is "pot-bellied." The liver projects one inch below the costal arch and is painful to the touch. The spleen cannot be made

out to be enlarged. The spine is curved to the right, and the thorax bulges to the right side posteriorly.

The tibiæ and fibulæ are bowed forwards and he is flat-footed. When he first came under observation the tibiæ and other long bones were very soft and pliant, so much so that I was somewhat afraid to use much force in manipulation. He has not been accustomed to creep on all fours, but has rather a tendency to shuffle along on his buttocks.

The fontanelles are closed, the os frontis is somewhat prow-shaped, and the girth of the skull at the level of the occipital protuberance is 20¾ inches. I have never found any evidence of increased uric acid in the urine.

Remarks.—In connection with the causation and associations of rickets I may again revert to the child's parentage and early history.

The child was born and reared in a house where the ventilation and light were always good. He was well formed and healthy at birth. He was suckled twelve months, and was not given any starchy food till the ninth month. The father and mother are both healthy, and certainly neither show any signs of ever having suffered from rickets. There are no signs of tubercular disease in either parent, nor did the mother

suffer from any deprivation during her pregnancies. She was quite healthy when this child was born. I may note, however, that she had children very fast, sixteen births in twenty-two years. I have carefully cross-examined both parents with a view to ascertain the possibility of a syphilitic taint, but have failed to obtain the slightest evidence thereof. The mother has had no miscarriages, no premature births, no stillborn children, nor have any of the infants suffered from snuffles or rash.

There is no history of what Hilton Fagge called the prodromata of rickets, viz., drowsiness, vomiting, diarrhœa; although drowsiness is a symptom which has supervened upon the bending of the bones. The child has not had alcoholic stimulants given to it; its father wears the blue ribbon. The children born subsequent to this one are all healthy; two of the patient's brothers have enlisted into the army, and have been taken into corps which have picked men only.

It will thus be seen that the evidence which I have brought before the Society as to the cause of rickets is mainly of a negative character. This case only tends to strengthen my belief in Sir William Jenner's opinion that rickets is a diathesis, and quite a distinct condition from syphilis. We have here samples of healthy parents, living with good hygienic surroundings, and producing healthy children with the exception of the one under our notice.

But when we come to remember the rapid pregnancies of the mother I submit that it is not to be wondered at that one at least of her children should show signs of bodily weakness.

In mental qualities my patient does not appear defective. He is intelligent for his age, and in some directions he shows great aptitude for learning. I am informed by his father that he is especially quick at music, and that he sings, with his treble pipe, the bass parts which he has heard his father sing.

Lastly, I may draw the attention of the Society to the opinion offered by the child's mother, viz. that his illness dates from, and is in consequence of, his fall from the bed. This traumatism of rickets as a starting-point is, I believe, one which has been before acknowledged, and is extremely interesting to me, and one on which I hope to make some further observations.

I have ventured to call this a case of arrested rickets, as the child is improving under treatment. The pliancy of bones is less marked, and there can now be felt in the long bones, notably the tibiæ, strengthening buttresses of bone on the concave surfaces of the arches.

XIX.—*A Case of Nephro-Lithotomy.* By Charters J. Symonds, M.S. *Read February* 27, 1885.

J. C., æt. 50, a carpenter and joiner, of a healthy family and abstemious habits, was admitted under the care of Dr. Mahomed, into Guy's Hospital on June 11, 1883. He was a well-nourished, healthy man, and never had any severe injury or general disease.

During the last twenty-four years he has been subject to attacks of pain commencing in the left loin, thence passing down into the groin and inner side of thigh. The frequency of the attacks varied, occurring from once to three or four times in a year. The attack lasted sometimes a day or less, at others it extended over a week with exacerbations and remissions. Sometimes he would be laid up for a month or even two months after an attack, from a feeling of general illness, what he calls "liver," during which time he suffered from loss of appetite and general malaise. There was no diminution of urine during these attacks or during the after-period.

The pain he describes as agonising, and he could obtain no relief until it spontaneously disappeared. This pain has left him for four years, and up to seven months ago he was comparatively well. At this date he had for a time increased frequency of micturition, being obliged to empty the bladder every two hours or less. So sudden was the desire to micturate and so urgent the call that he was often unable to reach a convenient place.

In February last, *i.e.* four months ago, he first observed blood in the urine. Usually clots were present, sometimes being one to one and a half inches in length, and occasionally interrupting the flow of urine by being caught in the urethra.

On one occasion after a sharp walk he passed urine red from blood, but without clots. After a sharp walk or after work which entailed a good deal of stooping, some blood would pass with the first micturition, and afterwards clots. As the man said, "anything which worked the body," meaning movements requiring the use of the erector spinæ muscles, produced hæmorrhage; especially he noticed it when gardening. He was unable to ride in a carriage on account of the pain and hæmorrhage. He found himself easiest while living on one floor,

and walking with a sliding motion of the feet; planting his foot firmly caused an increase of pain.

For the last three or four months he has been resting and is better of the pain, but feels languid and weak. He has a persistent dull aching pain in the left loin. He sleeps on the left side, as resting on the right causes the lumbar pain. When admitted there was no pain on micturition nor increased frequency so long as he kept in bed. There was no fulness or tenderness in the loin.

The *urine* was pale, strongly alkaline, specific gravity 1015. It contained some albumen, and small clots were usually present.

A few days later it is stated to contain pus in considerable amount and phosphatic crystals with mucus.

He was placed upon a milk and farinaceous diet.

I was asked to see the patient by Dr. Mahomed, and sounded the bladder, but found no evidence of stone, a result similar to that obtained on two former occasions by Mr. Ransford, his medical attendant in Bath.

July 6 it is noted that he had improved, had gained flesh, and had a good appetite. He daily went into the grounds. The dull aching pain continued, and the urine had the same characters noticed on admission, the blood always increasing on exertion.

There being little doubt as to the diagnosis in this case it was decided to cut down upon the kidney, to remove the calculus if possible, leaving the kidney, unless from suppuration its condition was sufficiently bad as to render its complete removal advisable. This was accordingly done on July 11.

I made an incision parallel and close below the last rib. The lower end of the kidney was isolated, and the anterior surface of this part and the pelvis examined. At once a hard body was felt at the commencement of the ureter. The kidney was large, bulging in several places, and evidently distended with fluid. By means of a steel director the pelvis was scratched through over the stone, which was then seized with forceps. Being conical in shape, and the small end being seized, it was impossible to extract it through the small opening. On enlarging the hole by means of a raspatory, a large quantity of watery, odourless fluid gushed out, the kidney collapsed and the stone disappeared. The search to recover the calculus lasted a long time; we thought it had fallen back into the dilated pelvis, or had escaped into the wound. Not finding it in the latter place an incision was made

through the cortex, which proved to be but a quarter of an inch in thickness. Through this opening the finger was inserted, but careful exploration failed to discover the stone. As there were many recesses, however, from the dilated calyces this was not surprising. Mr. Howse and Dr. Mahomed both assisted me in this search. Having nearly completely isolated the kidney, the ureter was hooked up into the wound so as to make the upper end of the kidney dependent. On examining now the calculus was found in the pelvis; it was brought up to the cortex, the latter incised, and the stone removed.

Necessarily in such a prolonged manipulation a good deal of injury was inflicted. Three openings were made into the pelvis: one by scratching through it; another by incising the cortex, through which the finger was passed in search of the stone, thus enlarging the aperture by tearing its margins; and a third through which the stone was finally removed. As stated before, the organ was so completely isolated that it would have been easy to remove it. As no pus had been found (and having in view the greatly increased danger of nephrectomy as compared with nephrotomy) we elected to leave the kidney and close the wound. The operation lasted nearly two hours. The wound was closed by silk sutures placed deeply through the muscles, and two large drainage-tubes were inserted, their deeper ends being in contact with, but not within, the kidney.

The carbolic spray was used and the usual antiseptic dressings applied.

In five hours it was necessary to dress the case, as a copious thin fluid had soaked through; this was blood-stained, but without urinous odour.

July 12.—At 9.30 A.M. second dressing, tubes removed, cleaned, and returned.

July 13.—Third dressing 9 P.M. Skin united. One tube shortened. One suture cut, one loosened.

July 15.—9 A.M. fourth dressing. The same thin fluid, no pus. One tube removed and one suture. Two others cut.

July 16.—A simple enema given.

July 18.—Two sutures removed and remaining tube. Eucalyptol dressings substituted on account of persistent carboluria.

July 20.—The wound was practically superficial, and on 24th the spray was discontinued, and boracic lint employed.

In a few days he was up. The pus persisting, together with a little blood, the bladder was examined, but no stone detected.

On August 17 he was discharged. For some time he had been free from pain, but as the urine still contained albumen, which on August 4 had increased from $\frac{1}{10}$ to $\frac{1}{3}$ (after sounding), he was kept in the hospital upon a low diet. On August 14 it was noted that there was slight œdema of the feet. When discharged there was some tenderness, and a feeling to him of fulness in the loin, attributable probably to the incision. A pulse tracing at this time showed a slight increase of arterial tension.

Condition of Urine.

For nineteen days before operation.

Average daily quantity . . .	30 oz.
Smallest	14 oz.
Largest	40 oz.
Average specific gravity . .	1022
Average per cent. of urea . . .	1·5

Reaction alkaline. Albumen about $\frac{1}{8}$th.

Deposit contained mucus, pus and blood arranged in three strata. The two latter always in small amount; the blood forming a stratum about $\frac{1}{16}$th of an inch thick.

Day of operation.

14 oz. passed before operation showed the usual characters.

8 oz. passed afterwards contained more blood than usual.

For nineteen days after operation.

Average daily quantity . . .	43 oz.
Smallest quantity	24 oz.
Largest quantity	60 oz.
Average specific gravity . .	1022
Average per cent. of urea . . .	2·0
And for the first five days . . .	2·5

Reaction generally alkaline, often faintly so, and sometimes acid, especially after the operation.

The *first* day after operation 52 oz. were passed having a smoky appearance, from diffused blood, and about $\frac{1}{8}$th albumen.

On the *third* day carbolic acid appeared in the urine six hours after the fourth dressing, the urine remained dark for five hours.

On the fifth day carbolic acid again appeared about six

hours after the dressing, remaining about the same time as before.

The albumen dropped to $\frac{1}{10}$th, but on August 4 rose to $\frac{1}{3}$rd. This was due probably to his being sounded on the 3rd, for the albumen in four days had again fallen to $\frac{1}{10}$th.

The stone is conical, its greatest length $\frac{3}{4}$ inch, greatest breadth $\frac{7}{16}$, narrows to $\frac{1}{8}$th. It is of a tawny colour, covered with fine rounded nodules, and in particular lights shows bright, crystalline points. (Plate IX, fig. 3.) On section there is no lamination to be seen, but the larger end has in its centre a darker material. The stone is hard and dense, and weighs about 18 grains.

An analysis of the stone carried out under the supervision of Dr. Stevenson, shows it to be composed entirely of phosphates.

Remarks.—The method adopted in searching for the stone was that suggested by Mr. Howse in his paper in vol. xvi of the *Transactions*. The lower end of the kidney was freed, and the pelvis just where it is becoming ureter was examined from the anterior surface. Here a hard substance was at once detected and held by the fingers, while the pelvis was scratched through with a blunt director. The opening was made in the infundibulum on the side next the kidney. The plan adopted for finding the stone seems a valuable one, for, as Mr. Howse has pointed out, exploration is greatly facilitated by the presence of a firm understratum of muscle.

But little alteration took place in the urine after the operation, and as the bladder appeared healthy, it is probable that the blood came from the other kidney. This is the more likely when the distended condition of the left kidney is remembered, for the calculus appeared to have completely obstructed the ureter. Though relieved of the stone, the man is by no means cured, for he writes in January, 1884—six months after the operation—that he still suffered pain in his left side on stooping. The urine still contained blood at times, and the specimen sent up for examination contained some pus, albumen, and blood. The man said in his letter, that though able to work a little he did not consider his health good.

November 10, 1884.—The patient writes that he is much better, is free from pain, except when he over-exerts himself. A specimen of urine which he forwarded showed abundance of pus-cells and some blood-discs.

XX.—*A Case of Nephro-Lithotomy.* By Henry Morris. *Read February* 27, 1885.

THIS case is recorded with the object of adding one more to the series of successful nephro-lithotomies which have been read before this Society since October, 1880. (See vol. xiv, p. 30.)*

Edward G., æt. 24, a jeweller, was admitted, under Dr. Powell, into the Middlesex Hospital on April 10, 1884, suffering from symptoms of calculus in the left kidney. To Dr. Powell I am indebted for the opportunity of performing the operation, as well as for some of the earlier notes of the case.

Since his schoolboy days Edward G. had complained of pain in his loins, but in spite of this he had enjoyed fairly good health till two years ago, when he was attacked by a "kidney complaint." This came on without any assignable cause, and was chiefly marked by pains in the left loin, shooting down, at times, to his left testicle. For three weeks before admission these pains had been too severe to permit of his working, and he attended during this period at the out-patient department. On admission he had pain in the left renal region, and tenderness on deep pressure in the left loin. His urine was neutral, of sp. gr. 1010, and contained blood and triple phosphates. There was frequency of micturition, though the urine was stated to be somewhat deficient in quantity. Five days after admission, however, it was noted that the average daily quantity of urine voided had been 45 ounces. From April 18 to 25 the pain was diminished and less constant, but there was still tenderness on deep pressure in the left lumbar region. When the pain occurred the left testis was retracted; more-

* I desire to take this opportunity of correcting an error which inadvertently crept into my first paper on Nephro-lithotomy in the process of correcting for press; and to which my attention has since been directed.

It is implied on p. 34 of the fourteenth volume of the 'Transactions' of the Society that Charles Bernard was not a medical man. In my MS., however, I wrote that the description of Marchetti's operation which has come down to us "*was written by one to whom it was narrated, but not until ten years after the event, by the patient who was not a medical man.*" I never intended to state—though in my printed paper it would appear that I did—that Charles Bernard, F.R.S., was not a surgeon.

Apart from the fact that he was one of the surgeons of St. Bartholomew's Hospital, his description of what is said to have been Marchetti's operation on the English Consul Hobson is in itself evidence that he was a medical man. (Vide *Medical Times*, February 21st and 28th, 1885.)

over, this testis was discovered to be much atrophied. The urine at this time was acid, sp. gr. 1024, and contained a trace of albumen due to the presence of pus. After consultation with Dr. Powell it was decided that the patient should be transferred to a surgical ward for the purpose of undergoing nephro-lithotomy.

The operation was performed on May 10, 1884.

A calculus was felt, by the finger passed over the front surface of the kidney, near the inner border, and below the hilum. Digital examination of the posterior surface, and acupuncture of the kidney had failed to detect it. The sensation communicated to the finger by the calculus through the renal substance was simply that of increased induration, as compared with the resistance offered by the rest of the kidney. With my finger-nail I scratched through the renal tissue covering the calculus, and thus verified the nature of the induration; then with a bistoury I incised the inner edge of the kidney on its posterior surface; and partly by pushing the calculus with my left index finger (which I kept all the while in front of the kidney), and partly by the aid of a narrow curette, the stone was dislodged and removed. There was no hæmorrhage of any moment from first to last. A large drainage-tube was introduced into the wound, but not into the kidney; the edges of the wound were held together by sutures; and terebene oil on lint and a thick layer of absorbent cotton wool were retained over the wound by a light bandage.

The operation was performed at 2 P.M. At 1 P.M. he had micturated for the last time before the operation, and no urine was passed after the operation by the penis, until 7.30 A.M. on May 11, when 15 ounces of lightly blood-stained urine were voided naturally. At 12 o'clock at noon on the same day (May 11) 9 ounces more were voided; so that 24 ounces were passed naturally in the first twenty-four hours after the operation. The dressings were changed once in the evening of the day of operation, and three times on the following day; but on May 12 there was so little discharge from the wound that this frequency of dressing was considered quite unnecessary, and was therefore discontinued. Urine had in fact ceased to pass by the wound. During the second period of twenty-four hours the urine was passed per urethram as follows:

May 11.—2.50 P.M., 4 ounces; 5 P.M., 4 ounces; 10 P.M., 4 ounces. May 12.—12.15 A.M., 5 ounces; 2 A.M., 4 ounces; 5.15 A.M., 6 ounces; 7.50 A.M., 10 ounces; 10.45 A.M., 6 ounces; 1.21 P.M., 5 ounces. Making a total of 48 ounces.

The urine continued to be blood-stained until May 14. On the 15th it was neither blood-stained, nor did it contain albumen; its sp. gr. was 1023, and there were 40 ounces voided in twenty-four hours. On the 16th the quantity of urea was estimated, and found to be 495 grains in 42 ounces of urine passed in the twenty-four hours. On May 17 the wound was in great part united, and the stitches were removed. The patient had had no pain since the operation, and his only discomfort had been the enforced restriction to the recumbent position. The temperature rose on this day to over 101° (101·8° being the highest). On the following day (the 18th) and on part of the 19th, the temperature remained just below 100°. With these exceptions the temperature throughout never exceeded 99° Fahr.

May 20.—The bowels had not acted since the operation, and so an enema was given. This had the desired effect, and the defæcation was unattended by pain; but on May 21, from the state of the dressings it was inferred that a little urine had again been discharged through the loin wound; 60 ounces of urine were passed per urethram, so that the chief part, if not the whole of the urine from the left, as well as from the right kidney must have come the proper way.

On the 23rd the bowels acted twice, and again a small quantity of urine was thought to have come subsequently through the wound; but the amount so escaping was extremely small. It would seem as if the passage of the fæces along the descending colon caused some disturbance to the wound in the kidney, which, however, must have been nearly healed. This interference was the more probable owing to the situation of the wound in the kidney. Forty-six ounces of urine were passed the natural way; it was of good colour, sp. gr. 1027, acid, and contained no albumen.

On May 28 the patient, who had been on ordinary diet since the 26th, was feeling quite well, and only the track of the drainage-tube remained unhealed.

On June 3 this track had closed so that not even a fine probe could be introduced. On June 10 he got up for the first time, and on the 12th went into the garden, and there took walking exercise. He continued to gain strength daily, and was considered to be well, when on June 26, at 8 P.M., after taking a good deal of exercise out of doors during the day, he passed urine coloured deeply with blood. On June 27, at 4·30 A.M., the urine was still more blood-stained than that passed at 8 o'clock the previous evening. At 9 A.M.

on June 27, the urine was much less mixed with blood; and on June 28, 51 ounces of urine were passed in twenty-four hours; it was acid, and of sp. gr. 1020, and contained neither blood nor albumen. The bowels had been acting twice a day, so that the hæmorrhage could not have been due to pressure on the kidney by an overloaded colon. No pain, sense of discomfort, or feeling of illness of any sort attended this attack of hæmaturia.

For a few days he was kept in bed, but there was no return of the hæmaturia; on July 8 he left the hospital quite well, and with the cicatrix in his loin perfectly firm throughout. On several occasions since this paper was read he has been seen at the hospital. There has been no return of the hæmaturia, he never suffers pain, has been at work regularly since leaving the hospital, and has never felt better in his life than since the operation.

The calculus was divided (Plate IX, fig. 1), and Mr. Thomas Taylor has kindly given me the following account of its composition. "The half of the calculus which I herewith return consists of a small dark-coloured nucleus of oxalate of lime, upon which has been deposited urate of ammonia mixed with a small quantity of oxalate of lime; this is surrounded by a layer of dark oxalate of lime, and the whole is coated by a thin layer of nearly white oxalate of lime, upon which are deposited, in parts, crystals of pure oxalate of lime.

"1. Nucleus, oxalate of lime.
"2. Urate of ammonia.
"3. Oxalate of lime."

DESCRIPTION OF PLATE IX.

Fig. 1.—To illustrate Mr. Morris's case of Nephro-lithotomy. P. 188.

Fig. 2.—To illustrate Mr. Croft's cases of Preputial Calculi. P. 8.

Fig. 3.—To illustrate Mr. Symonds's case of Nephro-lithotomy. P. 184.

Fig. 4.—To illustrate Mr. Symonds's case of Removal of Calculus from the Vermiform Appendix. P. 288.

Fig. 1.

Fig. 2.

Fig. 3.

Fig. 4.

Danielsson & Co. lith.

XXI.—*Case of Nephro-Lithotomy.* Under the care of W. Howship Dickinson, M.D., and J. Rouse. Communicated by Dr. Dickinson. *Read April* 24, 1885.

Henry S., living at Leighton Buzzard, remembered to have occasionally passed dark-coloured, apparently bloody, urine since he was six or seven years old, and was told by his mother that he had done so earlier, "ever since he was a child." In the year 1877 he, then being twelve years old, came under my care in St. George's Hospital with hæmaturia, which was attributed to stone in the kidney.

On April 2, 1884, then having reached the age of nineteen, he again became my patient in the same place with similar symptoms. I had on his discharge urged his parents to put him in some quiet way of life. They responded by apprenticing him to a blacksmith, at whose trade he worked until his admission. Up to this time he had had attacks of hæmaturia with intervals which varied from a week to six months. These attacks were attended or preceded by sharp pain in the right lumbar region in the position of the kidney, passing thence to near the anterior superior spine of the ilium, not to the testicle or thigh. This pain was often present independently of the hæmaturia, though the two were often associated, the pain usually preceding the hæmorrhage.

Both were brought on by exertion, more particularly by lifting, and by jolting, as by riding in an omnibus, not by cold. Beer had been noticed by the patient to bring on both pain and bleeding, in consequence of which he was for some time a teetotaler. In the hospital two pints of porter were found to be effectual in these respects; pain came on two hours afterwards, hæmaturia the next day.

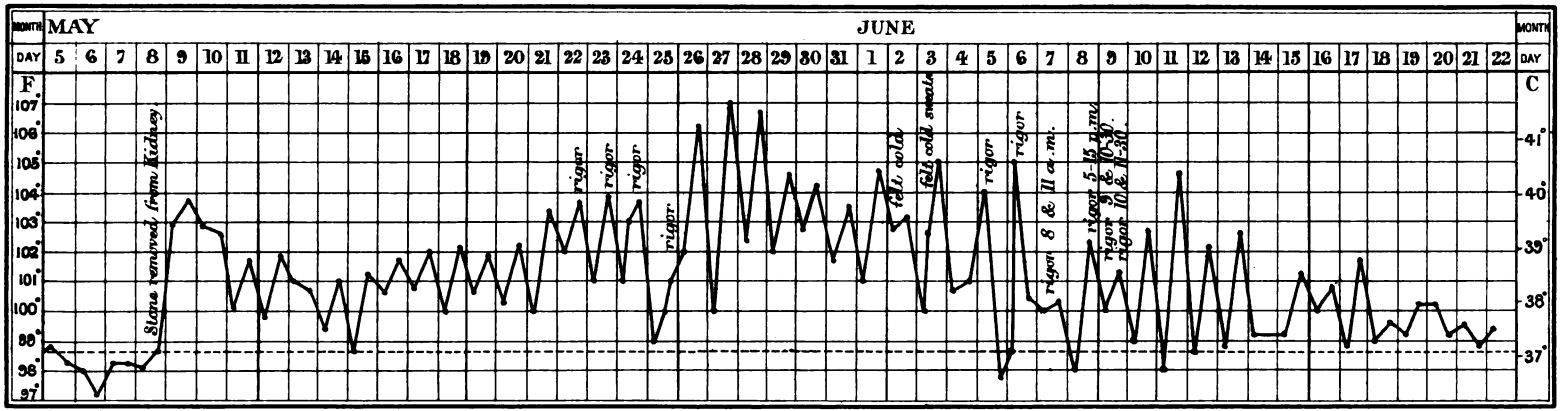
The urine was generally natural in appearance and of good specific gravity, 1016 to 1020. It was sometimes faintly acid, sometimes alkaline, and displayed under the microscope sometimes oxalate, and sometimes phosphate of lime. No casts were at any time found, nor was there any albumen excepting when blood was present. This occurred while he was in the hospital on slight provocation and at frequent intervals, which varied from two to ten days. The urine then assumed a full blood colour; the blood, which was corpuscular, fell on standing as

a bulky red deposit, leaving the supernatant fluid but slightly tinted.

The character of the hæmorrhagic discharge, its association with movement and exertion, the localised pain, and the history of the case were sufficiently indicative of stone in the kidney, and it may be added that the absence of pus, of albumen, save when blood was present, and of casts, were sufficient evidence that but for the stone the kidneys were practically healthy. The patient was in good general health, rather thin than fat, but not extremely so; beyond slight sallowness of complexion he showed little sign of having lost blood, and altogether the freedom from doubt with regard to the diagnosis, and the health of the patient, marked him as a promising subject for renal lithotomy. In these circumstances I suggested to my colleague Mr. Rouse that he should undertake the operation.

After consultation with the other surgeons this was done on May 8, 1884. Under ether, and with antiseptic precautions, a transverse incision was made in the right loin, as in Amussat's operation; the kidney was readily exposed, and a lump which was thought to be a stone was readily felt with the finger through the renal substance. This was punctured with a needle and the grating of stone recognised. The glandular tissue was then incised, with some hæmorrhage, and the stone felt and somewhat displaced with the finger. It appeared to lodge, or to be embedded, in one of the calyces. A second stone, apparently much smaller, was also felt. The larger stone was easily withdrawn by means of a pair of forceps. The second stone could not be readily grasped, and was left in its place in the hope that it would subsequently come out of the wound under the influence of movement and position.

The stone which was extracted was almost exactly of the shape of a heart, the apex and separation of the ventricles being clearly distinguished. The notched base had evidently fitted into the calyces, the apex into the infundibulum. From base to apex the stone measured three quarters of an inch, the same from side to side. When dry it was found to weigh sixty-five grains. It was preserved uncut, but from its dark colour and finely nodulated surface, there could be no doubt that it consisted superficially and probably chiefly of oxalate of lime. The lesser stone came out of the wound on the fourth day after the operation. It was of about the size of a pea; it had a polished and roundly faceted exterior, and weighed



Dr. Dickinson's case of Renal Calculus.

Mintern Bros. lith

two grains. The colour was suggestive of oxalate of lime rather than uric acid.

The patient went on smoothly from May 8 until the 22nd, when he had a rigor and the temperature went up to 103·7°. Next day there was another rigor, with a temperature of 104°, and much rusty sputum such as belongs to pneumonia. The difficulty of moving the patient prevented any adequate examination of the chest. The rigors were repeated on the 24th and 25th, and the temperature continued to ascend with evening exacerbations until on the 27th it reached 107°, which was the maximum attained during the illness. There was now much pain in the left elbow and wrist and the latter joint was red and swollen. There was some effusion in the left knee. The wound itself, in spite of the alarming constitutional disturbances, was going on well; it looked healthy, was granulating steadily from the bottom, and the amount of urine discharged through it, which at first had been most profuse, was diminishing. Alcohol and quinine were given freely.

On the 29th an attack of profuse diarrhœa occurred, the bowels acting twenty times in about the same number of hours. This was controlled by enemata of opium, and from this time there was some improvement in the aspect and temperature of the patient, which from this date never went above 105°. Between May 29 and June 11, the daily range was still very great, often over six degrees, the lowest temperature during this period being 97·6°, the highest 105°. (Plate X.) Meanwhile the local condition of the joints was improving; they were less swollen, less red, and less exquisitely tender, the diarrhœa was in abeyance, the patient took liquid nourishment and stimulants well and had not quite the look of hopeless prostration which commonly attends such a temperature in such circumstances. From June 12 the temperature steadily declined, the general symptoms mitigated and the healing of the wound progressed. By the 20th very little urine passed through the wound, which was almost skinned over. The new surface was powdered with calomel to keep it dry; the

crust so formed generally broke when he turned over, allowing a few drops of urine to escape, which was all that made its way otherwise than by the natural channels.

On July 10 the wound was perfectly healed and the patient up.

On the 29th he was sent to Wimbledon convalescent, whence he passed to his own home, where he resumed work as a blacksmith.

On October 27 he came to the hospital to show himself; the urine was then natural, he had no symptoms of calculus; nothing remained of the disease but the two stones in my possession, and nothing of the operation but the scar.

The case presented no difficulty in diagnosis. Hæmaturia is a better guide to stone than pyelitis; the latter condition may depend on so many causes that it is of little value as a surgical indication. After the operation the most noteworthy occurrence was the alarming attack of what must be called septicæmia, for the recovery without suppuration was, I suppose, enough to show that the disturbance did not attain to pyæmia, however much it resembled it. It may be observed that the antiseptic method of dressing was rendered impracticable by the profuse discharge of urine from the wound.

XXII.—*A Case of Thrombosis of the Basilar Artery, with profound coma, extreme lowering of rectal temperature, and death in five and a half hours.* By H. Charlton Bastian, M.D., F.R.S. *Read March* 13, 1885.

MICHAEL D., æt. 43, a watchman, was admitted into University College Hospital under my care in a comatose condition, at 6 P.M., on November 16, 1884.

The following history of the patient was subsequently obtained:

His health had been generally good, but three years ago he was in Middlesex Hospital on account of ulcers on his legs, just above the ankle-joint on each side. The wife says these ulcers existed when he married eleven years ago, and they continued up to the time of his death. The ulcer on the left leg was said to have followed a kick. No definite history of syphilis, though the patient's wife had had three miscarriages (she could not say at what term) and no living children. Has lived in a good house, and has mostly had sufficient food; has been rather intemperate, getting intoxicated occasionally. His occupation has been that of a night watchman for the parish of St. Giles; his duty being to look after the tools of workmen employed upon the roads. He has not had constant employment, having been out of work for two months preceding the week before his death. During this time he had very little either to eat or drink. Had suffered from a bad cough lately, and has complained of a severe pain in the left frontal region during the paroxysms of coughing.

Had been at work again one week when the illness commenced. On the afternoon of the day on which it occurred he went to his work as usual, and appeared to be in good health. He was seen by a man at 4.30 P.M., and seemed quite well. One hour later the patient was again seen by this same man, who found him in a sitting position groaning, but unable to speak. He was soon put into a cab and sent to University College Hospital.

The following careful notes of his condition were then taken by my late house physician, Mr. Henry Littlewood.

In Casualty Room, 6 P.M.—Patient profoundly comatose,

not to be roused; no distinct stertor, but breathing irregular and at times slightly stertorous; no flapping of cheeks; face pale, cold, and clammy. All limbs flaccid, with the exception of some slight rigidity of right arm. Pupils equal, somewhat contracted. No conjugate deviation of head and eyes; eyes open. No want of symmetry about the face noted. No conjunctival reflex. No knee jerk; no ankle clonus. No involuntary evacuations. Urine drawn off, acid, and containing a small quantity of albumen. Temperature in rectum at 6.10 P.M. 98°; at 6.20 97°. Heart-sounds healthy; its action tumultuous. A few moments later pupils became more dilated, and it was now noticed that they reacted sluggishly to light on both sides, although any such reaction was doubtful when patient was first seen.

The patient was transferred to Ward VII, a mustard plaster being applied to the nape of the neck, and two drops of croton oil on butter administered. The following notes were subsequently taken.

7 P.M., temperature in rectum 96·2° The thermometer does not rise in either axilla above 95°. Pulse 80, fairly full, regular in time, but irregular in force. Respirations irregular—there are a few rapid inspirations, then a pause, followed by a long and deep expiration.

7.50 P.M., patient remains in much the same condition. Temperature in rectum now below 95°. Pulse 84, compressible, rather irregular, 9, 5, and 7 in successive periods of five seconds. Respirations of character above noted, but hampered now by excessive secretion of mucus. Trachea and throat full of mucus of a glairy character. Pupils of medium size, do not react.

8 P.M., rectal temperature below 95°. Pulse 64.

8.45 P.M., rectal temperature below 95°. Pulse 100; respirations 17 per minute. All limbs now quite flaccid, the slight rigidity of right arm, originally noted, having disappeared.

10.15 P.M., rectal temperature still below 95°. Pulse can only be felt with difficulty; irregular, and at times (varying with respirations) imperceptible. Respirations unaltered. Pupils more contracted than at last note, unaffected by light. All limbs flaccid.

10.30 P.M., three spontaneous and momentary dilatations of pupils seen. Pulse cannot be felt at wrist.

10.45 P.M., death occurred. (Fifteen minutes afterwards, the rectal temperature was found to be still below 95°.)

The *necropsy* was made by myself fourteen hours after death. Calvaria and dura mater presented nothing unnatural. Longitudinal sinus almost empty, containing only a small quantity of semi-fluid blood. On removing dura mater some flattening of the convolutions over the posterior two thirds of both hemispheres was seen, more marked on the right than on the left side. On examination of the large vessels at the base of the brain, the first part of the right middle cerebral was seen to be more opaque, and its walls thicker than natural. Other portions of the middle, anterior, and posterior cerebrals presented a fairly healthy appearance. The posterior half of the *basilar artery* was dilated to twice the diameter of the anterior portion. The length of this fusiform aneurismal swelling was one third of an inch, and the walls of the vessel were here opaque and notably thickened. On opening the basilar artery the dilated portion was found to be perfectly occluded by a soft colourless clot, uniformly adherent to the aneurismal walls. Two or three small specks of dark blood were seen in the midst of this pale fibrinous clot. The middle cerebellar arteries were in connection with the aneurismal swelling and were also occluded. Both vertebral arteries were healthy and patent, the right being much larger than the left. The left posterior communicating artery was unusually small, and the right rather large. All parts of the hemispheres were carefully examined without revealing anything unnatural. The lateral ventricles contained a distinct excess of serum, about one ounce in each; and there was a general dilatation of veins over the walls of these ventricles. Sections through the corpora striata and thalami revealed nothing unnatural. Similar incisions made through cerebellum, pons, and medulla also showed no focal lesion of any kind, and nothing distinctly unnatural. No notable excess or diminution of blood could be said to exist in these parts, nor was there any appreciable diminution in the consistence of the pons.

Heart of medium size, right cavities distended with fluid blood, much blood-staining of lining membrane. Tricuspid and pulmonary valves healthy; mitral and aortic valves thicker and more opaque than natural. Root and other parts of thoracic aorta show many patches of degeneration, yellowish white in colour and slightly raised above the general surface; lining membrane generally smooth, no ulcerations.

Lungs congested and œdematous, especially in lower lobes; in that of the right side several small extravasations of blood were seen, whilst in the upper part of the left lower lobe a

large patch 1½″ in diameter was found, in which blood had been recently extravasated into the lung substance.

Liver, Spleen, and *Kidneys* congested, notably tougher than natural; no other distinct change. *Testicles* healthy.

Remarks.—This case is in many respects a remarkable one. It very rarely happens that a patient dies from occlusion of one of the cerebral vessels in less than twelve hours, and I have been unable to find any case on record in which it occurred so rapidly as in this instance. In none of the cases of thrombosis of the basilar artery recorded by Hayem (*Archiv. de Physiolog.*, 1868, p. 270) was the fatal progress of the case so swift, death having taken place in them in sixteen to twenty-one hours. The clinical record of the present case resembles in the closest manner what might be found in a case of very large cerebral hæmorrhage. This is seen especially by the extreme depression of the rectal temperature, which in about two hours and a half from the onset of the attack had sunk below 95°, and continued at this low point till the patient died, three hours later. Unfortunately, the thermometer not being graduated below 95°, the exact minimum was not ascertained.

Last year I brought before the Society the record of a case of cerebral hæmorrhage in which the rectal temperature sank as low as 94·5°, and where, as in this case, it continued thus depressed till death occurred three hours afterwards.

The fact that the thrombosis in the present case occurred in the lower half of the basilar artery, and thus cut off or greatly diminished the supply of blood to the respiratory centres, doubtless accounted for the suddenness with which the fatal issue was brought about. The centres for the third nerves, which are situated in the floor of the fourth ventricle above the upper part of the pons varolii, seem to have been partially paralysed rather than stimulated; thus at 7.50 the pupils were found to be of "medium size and insensitive to light." It is well known that in many cases of hæmorrhage into the pons varolii the pupils are notably contracted, so as to simulate the condition met with in opium poisoning.

The respirations, as in Hayem's cases, were much disturbed, weak, and irregular, but never very frequent. Within an hour and a half of the patient's death they were only 17 per minute; yet in a remarkable case of thrombosis of the vertebral and basilar arteries recorded by Dr. Duffin (*Med. Times and Gaz.*, 1876, vol. ii, p. 622) the respirations are said to have been 105 per minute for about six hours previous to death;

the pulse being at the same time about 156, and the temperature 105°.

The clinical picture presented by thrombosis of the basilar artery varies greatly in different cases, according as the upper or the lower half of the vessel becomes blocked, and also according to the rapidity or slowness with which complete occlusion occurs. In two or three of the cases recorded by Hayem, in which death was brought about rapidly, the patients were, as in this case, found in an apoplectic condition, so that unfortunately we are still in need of information as to the premonitory or initial symptoms. The clinical details of the actual attack given by my late excellent house physician, Mr. H. Littlewood, are more complete than in any of the cases which I have been able to find on record.

The present case affords a further illustration of the extreme difficulty, or even impossibility in some cases, of making a diagnosis between cerebral hæmorrhage and thrombotic occlusion of some cerebral artery. Still, in no other artery except the basilar would such occlusion be likely to reduce the temperature to such an extent as, in this case. The temperatures recorded by Bourneville (*Études Clin. et Thermom.*, 1873) in cases of occlusion of one or more of the other cerebral vessels by embolism or thrombosis were rarely depressed below 98·6°, and never below 98°. Amongst cases of this type, that which has now been recorded is almost, if not quite, unique, both as regards the extent to which the temperature was depressed and the rapidity with which death was brought about.

Lesions in the pons are altogether exceptional in regard to the remarkable amount of variation which they may cause in the general temperature of the body. Here we have an occlusion of the basilar artery (*i. e.* an incipient softening process) depressing the temperature below 95° for some hours; on the other hand, some years ago, I saw the terminal stages of a less acute attack, in which softening of the pons was found, ending with a temperature of 110°.

In regard to the cause of the thrombosis, in two of Hayem's cases it was due to arteritis. Here we had rather a simple aneurismal dilatation of the basilar with a thickened and degenerated vessel wall. The clot which filled the vessel was colourless, soft, and altogether resembled in appearance the clots which often form in the right side of the heart some hours before death.

Microscopical sections kindly made for me by Mr. F. G.

Penrose quite confirmed this view as to the nature of the change, and the colourless thrombosis (wholly composed of fibrin and white blood-corpuscles) was seen to extend into each middle cerebellar artery. Other sections made through the hardened medulla oblongata revealed most typical specimens of endarteritis in some of the vessels on its anterior surface. As may be gathered from the clinical history, there was only a possibility that the patient had suffered from syphilis.

In the case recorded by Dr. Duffin the lateral ventricles contained several ounces of serum, and the veins of Galen were distended; in the present case there was also a distinct excess of fluid in the ventricles, and all the veins on their walls were notably gorged. This is scarcely to be wondered at, seeing that the posterior cerebral arteries supply the walls of the ventricles with blood, and also in part the choroid plexuses. The cutting off of the propulsive force with which the blood is usually driven through the basilar artery may go far to account for these effects, and also even for some evidence of œdema in the posterior part of the hemisphere, to which the slight flattening of the convolutions was probably due in the case now recorded.

XXIII.—*Unusual Sequela of Ovariotomy.* By Richard Barwell. *Read March* 13, 1885.

At the latter end of last October (1884) I removed the left ovary of an unmarried woman, æt. 29. The local results were as good as could be desired; but certain mental sequelæ followed such as have been hitherto unrecorded.

Nevertheless, by questionings I traced the fact that this was not an isolated event, and especially I found, that to Dr. Keith, Mr. Knowsley Thornton, Mr. Dent, and others, cases very similar, if not quite identical, had occurred.

Hence it appears that although rare as a sequela of ovariotomy such perturbation has arisen with sufficient frequency to render it a fact that should be known to the profession.

Louisa H., æt. 29, fair complexioned, flushing easily, and of somewhat excitable temperament, admitted into Victoria Chandos Ward under the care of Dr. Black, September 5, 1884, suffering from large ovarian cyst.

Save for the ovarian complaint the patient appeared in good health, the lungs, heart, and kidneys sound; somewhat constipated.

Owing to circumstances unconnected with the condition of the patient the operation had to be postponed; a week or two afterwards the approach of a catamenial period interfered, so that it was not until October 28 that I was able to proceed.

October 28.—Ovariotomy performed. The case was perfectly simple, the cyst unilocular without adhesion. There were drawn off by the trocar twenty-three pints of fluid somewhat thinner than usual. The disease was on the left side; the pedicle, tied with silk, first by transfixion, then by inclusion of the whole, was allowed to drop back into the abdomen. The other ovary being healthy, the section wound was closed by deep quill sutures and by superficial ones of wire.

October 29.—A considerable amount of blood in the urine, and probably, but with the deep blood-staining, it is difficult to judge, some carboluria. Highest temperature 99·2°.

October 30.—Hæmaturia continues. Deep sutures removed,

wound in good condition. Temperature (highest) 99·2°. She is menstruating.*

November 1.—The hæmaturia almost ceased. Yesterday the temperature at 2 A.M. was 100°, during the next four hours it rose, and at 6 A.M. stood at 102·4°, it then fell steadily again, and at 10 A.M. was at 99°. It will be unnecessary to refer again to temperature, as during all the rest of the case it was normal.

November 4.—The nurse's notes for the night of the 2nd and 3rd state that she was hysterical, but on seeing her I had strong doubts as to the particular form of mental excitement and irritability. The wound had healed, there was no abdominal or pelvic tenderness. In fact the conditions locally were quite satisfactory.

November 5.—Owing to circumstances connected with the service of the hospital and the exigencies of other patients she was moved from the private to a general ward.

November 7.—On the first night of her removal she was fairly quiet; she slept, however, but very little. On the night of this date she was very violent, mistook identity of persons about her, shrieked, struggled, and at last became so uncontrollable that Mr. Dodson (my house surgeon) had to give a little chloroform in order to secure her, afterwards to inject four minims of the hypodermic solution of morphia. The bowels acted for the first time since the operation (eleventh day).

November 8.—The morphia calmed her for about three hours; she did not sleep, and about 5 A.M. became violent again. The condition was not the least like the delirium of fever, nor like delirium tremens, it was unmistakably acute mania. She was again removed to the private ward.

November 16.—There have been various phases of violence alternating with quieter intervals, during which she talked incessantly. She had but very little sleep—in very short snatches—making up altogether in the twenty-four about one and a half hours. Dr. Blandford was so kind as to see her with me; she happened then to be in one of the quieter phases just described. He gave a favorable prognosis, advised that no opium should be given, but chloral and bromide of ammonium, and these only in small doses when absolutely necessary.

November 20.—During the last five days and nights, the

* She had only ceased normal menstruation on the 20th, one week before the operation; the return of the flow was very slight.

record says, "Very restless, sleeping at short intervals so as to make up about two hours in the twenty-four. I detected to-day for the first time a little heat about the head; ordered therefore an icebag. Out of the next twenty-four hours she slept about five."

November 24.—She has been rather quieter during the last three days, sleeping still in short snatches about five hours. It appeared to me a sign of improvement that she volunteered the statement that she had been, and felt then, "mad."

November 29.—Sleep has been gradually becoming longer and quieter. She is, with the exception of a few intervals, rational.

December 6.—After this date she was quite restored to reason and was allowed to go out with a nurse, then with another patient. In spite of a good deal of jactitation and of some violent stuggling there was no sign of hernial protrusion at the wound. As soon as her belt was finished she left the hospital.

Of a case like this several views may be taken, thus:

1. That there may have been proneness or hereditary tendency to insanity.
2. That insanity may follow any of the major operations, ovariotomy not more than any other.
3. That it was the result of disturbance of the urinary organs (kidney).
4. That it was the result of disturbance of the generative organs.
5. That it was merely coincident.

1. I took considerable pains to ascertain the history, in the matter of mental malady, of this patient's family. Her father died of cerebral apoplexy at the age of seventy-four; beyond this there is absolutely no trace of brain trouble. The patient herself is of the temperament called "mobile," flushing easily when spoken to, and evidently troubled with shyness; but before the operation she was very docile, very amenable to regulations.

2. If insanity be an occasional sequela of surgical operation the matter is not known to me, except in a few cases where strong hereditary tendency existed or where the patient had been previously insane.

3. The amount of blood lost by the kidney was not enough

to produce cerebral disturbance. Indeed, a certain amount of hæmaturia not unfrequently follows intraperitoneal operations performed, as was this one, under the carbolic spray, but mania is not a usual sequela of such event, and I shall have to refer to a case of mania after ovariotomy without any kidney disturbance.

4. In disturbance of the generative organs seems to lie the most probable etiology of this condition, but insanity from such cause usually leads to words and actions which betray its origin. Now, Laura H. never let fall an obscene or a doubtful word; her actions were none of them provocative or amatory. On November 6, when she was at her worst, she evinced some affection for my house surgeon, but she mistook him for her brother.

5. There remains the question of coincidence, and such explanation might be accepted if this case stood alone, but I can point to several cases of operation, not on the ovaries alone, but also on the uterus, which have been followed by such result. Thus Dr. Keith writes of a patient whose uterus he had removed under carbolic spray: "There was blood in her urine the next day, the day after albumen, and then came an attack of acute mania from which she died."* I am not sure that I am right in my interpretation of a private letter he was so kind as to send me that another of his patients became maniacal. Mr. Knowsley Thornton informs me that one of his ovariotomies died insane, and that he has at the present time a case of partial removal of the uterus, which he thinks will recover from an attack of mania.

Dr. Bantock kindly permits me to say that one of his patients was for some days insane; he at that time looked on the case as one merely of hysterical mania.

One case operated on in St. Thomas's Hospital died insane.

Mr. Dent had a fatal case of acute mania after ovariotomy. In this there was no hæmaturia nor any hereditary proclivity.

Thus we must recur to the subject of disturbance of the generative organs, and I submit that I have educed evidence to show that this occurrence, though very rare, is too frequent to be classed as a mere coincidence, but that some disturbance of those organs acting on the cerebro-spinal system is capable of occasionally producing mania, which may, as in

* *British Medical Journal*, January 31, 1885.

most of the cases quoted, be fatal, may be very evanescent, as in Dr. Bantock's case, or may, as in my own case, continue for a full month and then pass entirely away.

Since this subject is not, so far as I know, mentioned in any work on ovariotomy or on other branches of gynæcological surgery, it appeared to me desirable to bring the matter under the notice of this Society.

XXIV.—*On Three Cases of Colotomy with Delayed Opening of the Intestine.* By N. Davies-Colley. *Read March* 13, 1885.

In the operation of colotomy as usually performed I consider that there are two great dangers. The first is that of peritonitis which may follow the wounding of the peritoneum, either during the search for the bowel or when it is being incised and stitched to the edges of the skin wound. The second is the suppuration of the deep planes of connective and adipose tissue in the vicinity of the colon. This is no doubt due to the admission of the external air, and frequently also of the gaseous and fluid contents of the intestine to the tissues which have been lacerated in exposing and drawing to the surface the extraperitoneal portion of the bowel. On several occasions I have seen the operation followed by extensive cellulitis, and in some cases in which pneumonia and other visceral diseases have been the immediate cause of death, it has appeared to me that the fatal result was due primarily to blood-poisoning dependent upon the deep-seated suppuration.

Many years ago, therefore, it occurred to me that it would be wise when possible to delay the opening of the intestine until, under antiseptic precautions, time has been allowed for the wounds of the peritoneum and the lacerations of the soft parts to be sealed up by reparative lymph. It was not, however, until very recently that I put this idea to the test of actual experiment. But meanwhile my colleague, Mr. Howse, who had derived such excellent results from a similar change in the operation of gastrostomy, had already applied the same principle to that of colotomy, and I believe that he has been very well satisfied with the success which has followed the adoption of this mode of procedure. In the last three cases in which I have had to perform colotomy I have used various means for attaching the intestine to the skin wound, and have then left it unopened, in the first case for one day, in the second for four, and in the third for as many as six days. I propose to read to you the reports of these cases, and you will easily gather from them that the operation has been attended with much less suppuration than often follows colotomy, and that

the risks to which the patients have been subjected have been considerably diminished.

Case 1.—Edward K., æt. 49, waiter at a public-house, was admitted into Guy's Hospital under my care on November 13, 1883. Nine months before he had suffered much from pain in the loins, and had had to keep his bed. In July, after some difficulty in micturition, he suddenly passed a large quantity of pus in his urine. Since that time he had suffered from frequent and painful micturition, together with bearing-down pains in the rectum. On admission he was pale, thin, and anxious looking. He was passing his urine every ten minutes day and night. It contained pus, and occasionally flatus and fæces. He was also troubled with a frequent desire to defæcate and his stools were of a "slimy" character. On digital examination a smooth, moderately firm rounded mass could be felt bulging into the anterior aspect of the rectum above the prostate, which was of normal size; and about four inches from the anus some polypoid excrescences could be felt which were thought to be the fringe of an opening into the bladder. There was some swelling and tenderness also in the left iliac fossa. As rest and anodynes gave him but little relief it was decided to perform colotomy.

This was done, under ether, upon December 7. A vertical incision was made in the left lumbar region; the bowel was easily found, and then drawn out of the wound. Two harelip pins were passed through all its coats at right angles to each other, and a piece of drainage-tubing was wound around the small knuckle of the colon, which was thus isolated. The rest of the wound was then closed with sutures. The operation was performed under the spray, and carbolic gauze dressings were then applied. There was no escape of flatus or fæcal matter. Considerable abdominal pain accompanied by vomiting followed. The next day the rubber tubing was removed, but the needles were left in their places. On the second day a quantity of fæcal matter was found in the dressings. He felt much better and the vomiting had not recurred after the removal of the tubing. Eight days after the operation the needles were taken out, and a small slough of intestine, which had been strangulated by the tubing, came away. All the fæces now escaped by the wound. The discharge from the rectum consisted only of mucus and a little urine. But little constitutional disturbance followed the operation. His temperature rose above 101° on the seventh and eighth days, but at other times it was

about 100° or lower. The upper part of the skin wound healed by primary union. In the lower part there was for a time some suppuration until a few small sloughs of connective tissue had been discharged.

In January the patient was able to get about the ward, and with short exceptions he continued to do so until April. His bladder symptoms then became more severe. He had to micturate frequently, and occasionally he passed phosphatic crystals or small portions of fæcal matter.

Some relief was given him by perineal section on April 17, but he gradually sank and died on April 30. A cancerous mass was found between the upper part of the rectum and the bladder, communicating by large openings with each cavity.

Case 2.—Anne G., æt. 47, a married woman, was admitted under my care into Guy's Hospital on February 5, 1884. For many years she had had piles with occasional loss of blood. Of late the piles and bleeding had been more troublesome. For eleven months she had suffered from constipation, and for six weeks she also had from time to time severe lumbar pain during defæcation. She was fairly well nourished, with a rather congested condition of the cheeks. At the margin of the anus there were some inflamed external piles, and four inches up the rectum an annular growth could be felt, like the cervix uteri in shape, with a central passage one quarter to three eighths of an inch in diameter. As the obstruction appeared to be increasing, and as she suffered from considerable pain in defæcation, it was decided to perform colotomy.

On February 8, ether was administered, and the descending colon was drawn out of the wound through a vertical incision. It was then fixed by a sort of clamp which held two points of the bowel wall by the rounded extremities of screws. Antiseptic precautions were used, and the extremities of the wound were brought into apposition by sutures. But little disturbance followed. The pulse was quick. There was vomiting for the first evening, and she complained afterwards of some abdominal pain. There was also some feeling of distension, with a desire to get rid of flatus and fæces. The pressure of the screws was relaxed daily, but on February 12, four days after the operation, one of them was found to have penetrated the intestine. The clamp was therefore removed, and a vertical incision made into the bowel.

On the 17th (nine days after the operation) I noted that

the upper part of the wound was healed by primary union, but that below the artificial anus there was a small opening through which a few drops of healthy pus could be pressed. This soon healed, and in less than three weeks from the operation she was able to get up. (In a week from the operation her temperature had fallen to normal, and it remained low all the rest of her stay in the hospital.)

She went out on April 5, wearing a belt with an india-rubber plug for the artificial anus. At this time she was passing half of her motions by the wound and half by the anus. There was also a considerable tendency to contraction in the artificial anus.

In October she came to see me, and I found that the colotomy wound had entirely closed up, the condition of the stricture having considerably improved. She was passing her motions regularly, and she stated that they were as thick as one or two of her fingers. She had lost a little flesh, but she was otherwise in good health, and she suffered less discomfort than before the operation. There was occasionally some hæmorrhage from the bowel, but this could readily be checked by the use of tannic acid suppositories. The parietes bulged a little at the site of the colotomy wound, but the scar was smooth and inconspicuous. I saw her last in the beginning of February, and she was in much the same condition.

Case 3.—James W., æt. 39, a foreman on the railway, was admitted into Guy's Hospital under my care on March 13, 1884. He had suffered from diarrhœa for twelve months. For four months he had been passing blood, and for three there had been bearing-down pains. He had been obliged to leave off work the previous September. On admission he was a tall, strongly-framed man, but rather wasted, having lost 4 st. in weight during the last year. His appetite was very bad, and he was evidently suffering much pain. The anus was relaxed, and from one inch up, the rectum, as far as the finger could reach, was indurated, fixed, and tender, but without much contraction. Ovoid nodules could be felt in its walls about ¾ inch in their longest diameter. He was passing loose motions eight or nine times a day, and there was some incontinence of the fæces.

On March 14 colotomy was performed under ether. The bowel was found, and after it had been with some difficulty laid hold of and drawn out through the vertical incision, it was held in its place by the clamp which I now exhibit. This

consists of two parallel steel bars, each furnished with two rounded ivory studs rather more than ½ inch apart. A screw head at either end approximates the bars and so brings the ivory studs close enough to hold the intestine firm at two points of its surface. The ends of the clamp were prevented from pressing upon the skin by the interposition of small pads of gauze, and the rest of the wound was brought together by wire sutures. The operation was performed under spray, and carbolic gauze dressings were applied.

The next day he looked well, but he complained of repeated attacks of vomiting during the night, and his pulse was 120. The clamp was therefore removed, and the vomiting did not recur. On the 20th, six days after the operation, an incision ¾ inch long was made into the knuckle of bowel, which had remained *in situ* since the removal of the clamp. A week later the wound was found to have almost completely healed by primary union. No deep suppuration occurred, and after the first week his temperature remained about normal. Although nearly all the fæces from that time escaped by the artificial anus, he still continued to pass mucus and blood with small quantities of fæcal matter per anum, and there was but little relief of the pain which he suffered.

He is now (March, 1885) a good deal weaker, and there has latterly been some suppuration in the left ischio-rectal fossa. Nearly all the fæces still come away by the artificial opening.

I have not brought these cases before the Society to illustrate the advantage of performing colotomy for cancer of the rectum, but in order to point out the safety with which this operation may be performed by the method which, with various modifications, has been adopted in these and other cases by my colleagues and myself at Guy's Hospital.

The chief difficulty which I have had to contend with has been that of securing the intestine without causing symptoms of strangulation. If sutures are used they cannot be passed through the whole thickness of the intestine, for they would be sure to cause the escape of its gaseous or fluid contents into the wound. If, on the other hand, they are passed through the outer coats of the intestine, these are so thin that I should fear that any effort of the patient, such as the straining which accompanies vomiting, might cause them to cut their way out, and allow the bowel to retreat again into the abdominal cavity. Two of my colleagues at Guy's Hospital, viz. our President,

Mr. Bryant, and Mr. Howse, have drawn out the knuckle and left it protruding from the wound without any sutures to secure it, but I have hesitated to do so for fear of the difficulties which might arise if the bowel should slip back into the wound. In my first case, as I have related, I used two needles to transfix the bowel, and a piece of rubber tubing to constrict the portion thus isolated in such a way as to prevent the escape of any of the contents of the intestine. This proved to be an objectionable plan on account of the symptoms of strangulation to which it gave rise. In the other two cases I used clamps by which the intestinal wall was pressed upon at only two points and so held in position. These can be gradually unscrewed so as to lessen the pressure, or they may be entirely removed at the first dressing, when the knuckle will probably have contracted sufficient adhesions to retain it in its new position.

I need hardly add that there are many cases of colotomy in which the opening of the colon cannot be postponed. When the patient is suffering from complete obstruction of the lower bowel it will be necessary to open the colon at once. But in a large majority of the cases for which colotomy is performed for cancerous and ulcerative affections of the rectum, there is no immediate necessity for opening the colon, and in such cases I submit that some such plan as that which I have adopted in these, the only three cases in which I have yet applied the principle, will be found beneficial.

It has also occurred to me that still further extensions of this method may be possible. For example, in the case of a tumour of the colon, it might be better to draw out the loop of intestine containing the growth with the investing peritoneum and wait for a few days before excising the loop by the knife, the cautery, or some caustic agent. Again, in order to secure the absolute freedom of the lower bowel from the passage of fæces, a small loop containing the whole calibre of the colon might be brought out of the external wound and after a few days removed. In this way we should be able to prevent all possibility of fæcal matter passing on into the rectum, for the artificial anus thus formed would be the terminus, and not merely a lateral outlet, of the bowel.

XXV.—*A Series of Cases of Spina Bifida treated by Plastic Operation.* By A. W. Mayo-Robson. *Read March* 27, 1885.

In 1881 the following paragraph attracted my attention in one of the leading text-books on surgery:—"Viewing, then, the great danger of any effectual surgical treatment in cases of spina bifida, it seems better to watch the case carefully, and not to interfere unless the tumour is growing." This led me to carefully think over the subject with a view to carrying out some more definite lines of treatment than those hitherto adopted, which seemed to me very uncertain and very frequently fatal.

It was not, however, till the end of 1882 that I had a chance of putting my ideas into practice, the result quite coming up to my anticipation. I have since that time operated on four cases; have seen the operation done by Professor Jessop and Mr. Atkinson, my colleagues at the Leeds Infirmary; and have also had a letter from Dr. Robert T. Hayes, of Rochester, U.S.A., telling me that he has operated successfully by the same method.

My first case was fully described in a paper in the *Brit. Med. Journ.* for March 24, 1883; hence I need only quote it briefly.

Case 1.—A. S., a fairly well-nourished child, æt. 6 days, the subject of a spina bifida in the lumbar region the size of an orange, was operated on at such an early age, because the sac was excessively thin, had become inflamed at the fundus and threatened to burst.

The operation done on October 26, 1882, was as follows: When the infant was fully under the influence of chloroform I made a vertical incision through the skin on each side of the tumour, about half an inch from its base, and then very carefully dissected the integuments from the meninges, until I reached the laminæ of the vertebræ; this required very careful dissection, as the membranes left were so thin as to be perfectly translucent; the fluid was now let out by puncturing with fine scissors, which were also used to cut away the redundant membranes. The cauda equina was fully exposed,

lying on the floor of the spinal canal. I now had two folds on each side, each fold being of a different width, the two inner meningeal folds three fourths and half an inch respectively, and the two skin-flaps of the same width; but whilst the wider meningeal flap was on the right, the wider skin-flap was on the left. Thus, when sutures were applied, the lines of union were not opposite.

Acting on the same principle as is carried out in uniting the peritoneum, I brought together the serous surfaces of the arachnoid by several sutures, so as to completely shut off the spinal canal.

Mr. Mayo had, in the meantime, been dissecting (under the antiseptic spray) the periosteum from the femur and frontal bone of a rabbit, which he had just killed. This periosteum I now placed, with its osteogenic layer undermost, over the closed meninges, and carefully sutured it to the periosteum of the laminæ on each side, and to the bony margins above and below. After this the skin was sutured, a layer of protective applied, and a pad of salicylic wool placed over the wound.

The whole operation, which occupied more than an hour, was performed under the eucalyptus air. Catgut ligatures were employed, and the instruments and sponges were well carbolised. On the second day, the nurse in applying the napkins displaced the dressing; but although the skin-wound slightly opened there was no formation of pus, and no slough came away; in fact, through the small opening I could see that granulations had covered the superficial surface of the interposed periosteum. The child recovered without a bad symptom, and when shown to the Leeds and West Riding Medico-Chirurgical Society in December, 1882, two months after, the skin of the lumbar region was quite flat, and only presented a cicatrix where the tumour had been.

A thin shield was worn in order to protect the tender part of the spine. Six months after, the child was perfectly well in every respect; but when nearly a year old it died from teething convulsions after a day's illness, and no autopsy could be obtained. The portion of sac removed I show you in the jar handed round.

Case 2.—Mary A., æt. 18 days, a puny, ill-developed child of weakly parents, with a questionable history of syphilis, but without any family history of deformity, was brought to me with a spina bifida in the lumbar region, the size of a large orange, the coverings being so thin and translucent quite up

to the margin of the tumour that it was quite evident that no integumental cover could be obtained from the surface of the swelling, and as the skin all round was inflamed and beginning to ulcerate the case was not of a promising nature, but as it was quite evident the child must die shortly unless relieved I decided to operate.

Operation December 5, 1882.—The translucent membrane was partly cut away, just sufficient being left to form the meningeal cover, which was sutured with catgut over the opening into the spinal canal, that being three quarters of an inch wide and two inches and a quarter long; the skin was then dissected up over the loins so as to allow two flaps to be slid towards the middle line, where they were joined together over the meninges by silver sutures.

The whole operation was done strictly antiseptically. The after-progress locally was all that could be desired, the wound apparently healing by first intention without any signs of inflammation, but when the silver sutures were taken out on the third day the centre of the skin wound reopened a little, showing granulations springing from the meninges; the wound was supported by strapping.

The general condition was not altered, and although there was no elevation of temperature and no purulent discharge from the wound, the condition of marasmus present on admission continued, and the child died, apparently from asthenia, on the evening of December 8, 1882.

Post-mortem report by Dr. Griffith, house physician.—"All the lumbar and the two upper sacral vertebræ were found to be bifid; the meningeal wound had healed and the skin wound almost. There was no evidence of meningitis, and no apparent increase or diminution in the amount of cerebro-spinal fluid." The bifid spine which I pass round shows well the extensive deformity.

Case 3.—Elizabeth J., æt. 16, residing in Armley, was admitted on November 4, 1884, into the infirmary, suffering from spina bifida and talipes equino-varus. She said that she had no relations similarly affected. Up till the age of seven her feet were perfectly normal, after which time she commenced walking on her toes, the pedal deformity having from that time gradually increased.

During the last month the spina bifida had slightly enlarged, but had given no pain except when handled, and she had been entirely free from all head symptoms.

She sought advice on account of the deformity of the feet, and was admitted as an in-patient to Leeds General Infirmary in order to have tenotomy performed; but the day after admission the spina bifida, which was situated in the lumbar region, and was about the size of a large fœtal head with somewhat thin covering, began to weep from several points at its fundus; a clear watery non-albuminous fluid transuding in such quantity that a thimbleful was easily collected for analysis. This continued for three days, during which time the tumour was dressed with salicylic wool changed from time to time. The tumour was now much increased in size, very tense and hot to the touch; and at the same time, as shown by the chart, the temperature in the axilla was raised.

The patient now complained of headache, was restless, cross, and peevish; answered sharply when spoken to, looked pinched and extremely ill; the pupils were dilated, eyes blood-shot, skin was cold and clammy, pulse feeble and flickering, breathing rapid. Potassium bromide was ordered to be taken. Ice was applied to the tumour and hot bottles to the feet; these gave relief to a certain extent, but on November 13 the tumour was so tense and the headache and other nervous symptoms so severe that it was felt that unless relief could be given the patient must soon die. Aspiration was performed with full antiseptic precautions, 16 oz. of a clear transparent fluid (cerebro-spinal) being withdrawn; the pupils immediately gained their normal condition although the pain in the head became very much increased, but after half an hour the patient slept comfortably, and the temperature fell from 102° to normal. When the sac was empty the opening into the spinal canal could be felt to admit three fingers. The relief was of short duration, all the symptoms returning by the 15th, when the patient was very restless and constantly moaning, refusing food and complaining of intense headache, the pupils being dilated and sluggish.

November 16.—Aspiration was again performed, when 6 oz. of clear fluid were withdrawn, giving relief as before; on the 19th aspiration became again necessary, and 14 oz. of fluid were removed. A general consultation was held with a view to further operative treatment, but it was decided that the patient would stand the best chance by continuing the same treatment; hence the aspirations were repeated, and on the 21st 8 oz., 23rd 10 oz., 25th 10 oz., December 5th 12 oz., and 8th 7 oz., of fluid were withdrawn. At the fifth aspiration the fluid was slightly cloudy, at the sixth, seventh and eighth, it

was decidedly purulent but sweet, strict antiseptic precautions having been observed throughout.

On December 10, as the patient was getting decidedly worse and the temperature was keeping high, the headache and other nervous symptoms still continuing, it was felt that unless something more decided was done the patient must shortly sink.

On December 11, the following operation was performed. A large crucial incision was made, so as to raise four triangular flaps of skin, with their bases outwards, from the surface of the tumour, the dissection being performed so as to separate skin from sac without puncturing the latter; after the base of the tumour had been reached the thin fundus was opened, when it was found that there were no nerves in its walls, and as the sac was inflamed, and was lined with a thick layer of lymph, it was completely removed quite down to the base, which was in some parts nearly an inch thick. There being a good deal of hæmorrhage, about twenty catgut ligatures were applied to vessels. The base of the sac (as proved by passing a probe) communicated with the spinal canal, the opening being surrounded by lymph. The skin-flaps were accurately brought together by silver sutures and a large drainage-tube was inserted; the whole operation was done antiseptically and the wound was dressed on the 13th, 16th, 20th, when the drainage-tube was removed, and the 24th, when the wound was perfectly healed. From the time of the operation the patient gradually gained strength, lost her nervous symptoms, and was discharged cured on January 5, twenty-four days after operation. The accompanying temperature chart very clearly shows the progress of the case.

February.—At the present time the patient wears an oval vulcanite pad over the site of the tumour; she is perfectly well, and intends to come into the infirmary after a time to have her feet operated on.

Patient exhibited to the Society.

Temperature Record.

	A.M.	P.M.
Nov. 5 ...	98·4° ...	99·4°
6 ...	98·4 ...	100·0
7 ...	98·2 ...	99·2
8 ...	98·2 ...	99·4
9 ...	97·5 ...	99·8
10 ...	97·9 ...	100·6
11 ...	99·8 ...	101·8

	A.M.	P.M.			
Nov. 12	100·8°	101·2°			
13	102·4	102·0	Aspiration.	16 oz.	Watery fluid.
14	98·0	98·8			
15	98·9	100·9			
16	100·9	98·0	Aspiration.	6 oz.	
17	99·6	100·0			
18	100·0	99·8			
19	100·6	98·4	Aspiration.	14 oz.	
20	100·4	101·2			
21	99·8	102·4			
22	100·4	100·2	Aspiration.	8 oz.	
23	99·6	101·6	Aspiration.	10 oz.	
24	99·0	100·2			
25	102·3	103·2	Aspiration.	12 oz.	
26	103·0	99·6			
27	98·2	101·6			
28	99·8	100·4			
29	99·8	101·2			
30	100·4	100·8			
Dec. 1	100·0	101·0			
2	100·0	101·0			
3	100·4	101·2			
4	100·2	101·2			
5	100·4	102·4	Aspiration.	Pus 12 oz.	
6	98·4	100·2			
7	99·0	99·0			
8	101·4	100·8			
9	99·2	102·2	Aspiration.	Pus 7 oz.	
10	103·2	102·8			
11	101·6	98·4	Operation of excision of sac at 3 p.m.		
12	100·4	100·4			
13	99·4	100·4	Dressed.		
14	99·8	99·4			
15	98·2	100·0			
16	99·2	100·8	Dressed.		
17	98·2	99·2	After which a normal temperature.		
24	—	—	Dressed and found healed.		

Case 4.—Notes of this case were furnished me by my house surgeon, Mr. A. Atkinson, to whose care and attention the successful result of this and the last case are in a great measure due.

Daniel C., æt. 7 weeks, the child of healthy parents, was admitted into the Leeds Infirmary on January 31st, 1885, suffering from a large spina bifida in the lumbar region. The child was puny and badly nourished. It had a large head with open fontanelles and dilated veins, the face was thin and small, but the eyes were not prominent. The tumour measured seven and a quarter inches in circumference and three and a half inches across, the coverings being very thin and ulcerated at the fundus.

On February 5, Mr. Robson, operating under the eucalyptus air, made a vertical incision along the right of the tumour and tried to dissect the skin from the meninges, but, owing to the coverings being thin, the sac was perforated and cerebro-spinal fluid escaped; the dissection being continued, a wider meningeal flap was taken from the left and made to bridge over the opening in the spinal canal, which was one inch long by half an inch wide, being there united by a continuous catgut suture to a narrow meningeal fold on the right; the redundant integument having been excised, the skin-flaps were united along the middle line by silver sutures. The nervous contents of the spinal canal were in no way interfered with. Before the operation was completed, the child became suddenly collapsed, apparently owing to the chloroform; suspension by the feet and artificial respiration restored it, and the operation was completed.

A drainage-tube was inserted between the skin and meninges, and the wound was dressed with a layer of gauze dipped in carbolic solution and covered with salicylic wool, this being well covered with adhesive plaster to prevent soiling of the dressings.

February 7.—Drainage-tube and wire sutures removed, child looking well and wound apparently healed.

February 8.—Temperature rose to 104·5,° but sodium salicylate gr. j, every hour reduced it to 99⅘° in a few hours.

The temperature fluctuated till the 12th, after which it was normal, but during the whole time the wound looked well and showed no sign of inflammation.

On the 11th, *i.e.* six days after operation, the fontanelles were depressed as they appear in a child suffering from choleraic diarrhœa; this lasted two days, and the infant rolled its head from side to side as if uneasy, but it did not cry.

On the 16th the fontanelles were well filled and the head looked large, but the mother said that it was no larger than before operation. Discharged from the infirmary on the 18th, thirteen days after operation, apparently well but weak.

March 20.—The child was brought to the infirmary to have a pad adjusted.

The site of the tumour presented a linear scar showing the old line of incision, and the skin was on a level with that over the loins and sacrum.

Child exhibited to the Society, March 27, 1885.

Temperature Chart.

	A.M.	P.M.	
Feb. 5	—	98·2°	Operation. Excision of sac and suturing of meninges and skin-flaps.
6	100·0°	98·2	
7	101·0	100·8	
8	103·0	104·8	
9	104·8	99·8	Temp. reduced by gr. j Sod. Salicyl. every hour for six doses.
10	102·0	98·6	Sod. Salicyl. repeated.
11	101·8	99·4	
12	101·0	99·4	
13	98·6	99·6	
14	98·6	99·0	
15	99·6	99·6	
16	98·6	98·8	
17	98·6	98·8	Discharged cured.

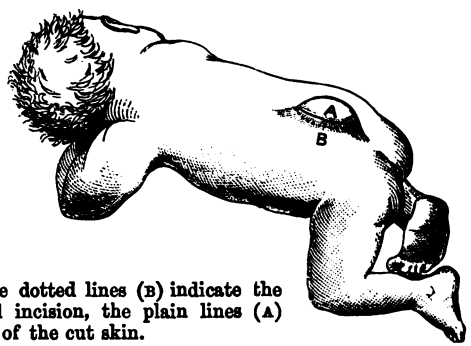


FIG. 1.—The dotted lines (B) indicate the meningeal incision, the plain lines (A) the edges of the cut skin.

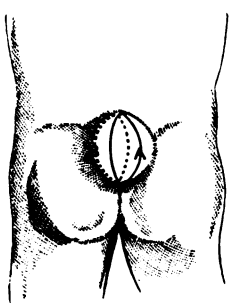


FIG. 2.—The dotted lines (B) indicate the meningeal incision, the plain lines (A) the edges of the cut skin.

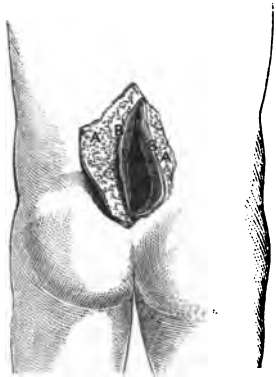


FIG. 3.—Showing the meningeal and skin flaps.

The points to which I would draw attention are:

1. The principle of closing the meninges by bringing together two serous surfaces as in peritoneal surgery, and superimposing separate skin-flaps, the meningeal and skin lines of suture not being opposite.

2. The great importance of observing strict antiseptic precautions, as a septic condition would probably end in the same way as these cases usually do when they spontaneously ulcerate, viz. by meningitis and convulsions.

3. The success attending the plastic operation in cases which are absolutely not amenable to any other form of treatment, *e.g.* when the coverings are thin or the opening into the spinal canal large.

4. The possibility of transplanting periosteum and its capability of surviving; as in the case read first; although in that example bone did not form, one may hope that the use of human periosteum (*e.g.* from a recently amputated limb) will give better results.

5. The successful issue of Case 3, where, although the sac was acutely inflamed, its complete removal with efficient drainage effected a cure. This case presents several points of pathological importance, *e.g.*:

(*a*) The increase of temperature without septicity, apparently due to tension of, or pressure on, the great nerve centres.

(*b*) The great relief given by aspiration.

(*c*) Suppuration in the sac, possibly due to simple tension, probably not due to septicity, as the fluid was quite sweet on every occasion.

(*d*) The entire absence of brain symptoms after operation, although the pressure on the cerebro-spinal centres must have been considerably interfered with during the time the wound was healing.

I would suggest the following as a practical classification of cases of spina bifida for purposes of treatment:

1. Where no operation can or should be done.
2. Where no operation need be done.
3. Where an operation should be done.

Class 1.—Where no operation should be done.

(*a*) Where the deformity is very extensive, as in fissure of the whole or a considerable portion of the vertebral canal.

(*b*) Where there is complete paraplegia, as in a case my friend, Dr. Libbey, of Horsforth, asked me to see with him, where the sac was large and excessively thin quite to its

margin, and where the lower extremities hung absolutely powerless.

(*c*) Where the sac is large, the fissure extensive, and the coverings excessively thin quite to the edge of the tumour, and no skin can be obtained to cover the meninges. But that such extreme examples may stand a chance of cure is proved by Case 2.

Class 2.—Where nothing need be done.

Where the sac is small, and the coverings are so dense and firm as to form a good pad over the opening in the spinal column, as in the case of a girl of fourteen, whose mother brought her to see me a few months ago, where I advised a thin silver shield to be worn over the swelling to protect it from injury and prevent further bulging.

Class 3.—Where some operation should be done.

(*a*) Where the sac only communicates with the spinal canal by means of a small opening; here it is a simple matter to dissect off the skin, expose the neck of the sac, ligature it by means of one circular ligature, and cut off the redundant meninges, bringing the skin over so as to have the line of skin sutures quite at the side away from the pedicle. Such an operation was performed by Mr. Edward Atkinson, one of my colleagues at the Leeds Infirmary.

(*b*) Where the sac has a good skin cover and communicates with the spinal canal by a large opening it is quite easy to perform the operation described in Case 1, carefully closing the meninges, and if possible placing the line of skin sutures away from the meningeal line of union. Such cases have been operated on successfully, not only by myself but by Professor Jessop, of Leeds, and by Dr. R. Hayes, of Rochester, U.S.A. Human periosteum might be placed between the meninges and skin, but I am not at all sure that a thin plate of bone if formed would be very serviceable, although I hoped to obtain it in the first of my cases. If the expanded neural arches be large, I think it might be advisable to bend them towards the central line, and by uniting them with thin silver wire to obtain a truly physiological closure of the spinal canal. I have not had a chance of trying this plan as yet.

(*c*) Where the coverings are excessively thin quite to the margin of the tumour, as in Case 2, the operation is more difficult and uncertain, as the skin can only be obtained by a process of sliding from the contiguous parts, and the tension necessarily present is not conducive to healing.

(*d*) Where the spinal cord or the nerves are blended with

the sac, a condition which often cannot be diagnosed until the dissection of the skin from the meninges is made, I should advise excision of portions of the redundant meninges between the nerves, replacing the nervous structures in the spinal canal and bringing over the skin cover, keeping up free drainage between the membranes and integuments; or, if this could not be done, the membranes might be punctured, the collapsed sac with the nervous structures intact be placed in the canal, and the skin cover made as before.

It is important to remember that a silver or leather shield should be worn over the site of operation in order to protect the parts from injury and to prevent the cicatrix from stretching or giving way.

XXVI.—*A Case of Choreiform Movements Supervening in Infancy, and probably of Congenital Origin.* By W. B. Hadden, M.D. *Read March* 27, 1885.

A. C., female, æt. 22, first came under my notice in December, 1884, when she was an in-patient under the care of Dr. Stone, at St. Thomas's Hospital. On January 12, 1885, she was admitted under me at the Royal Hospital for Children and Women. Her father and mother are alive and healthy. There are four other children in the family, all healthy. The eldest child, a girl, died from convulsive fits at the age of ten months.

The mother has had no miscarriages. The patient is the second child. The mother is said to have suffered from albuminuria for two months before her confinement. Labour occurred at full time and lasted twelve hours, no instruments being used. It was noticed soon after birth that the child's head used to fall back, and that she did not sit up like other children. She cut her teeth regularly and without trouble. She never had any fits, and there is no history of head injury. She never could walk better than she does now. Movements were first observed when the patient was seven months old, but it was not until the age of two that they attracted serious attention. They have persisted ever since without much alteration. She began to talk between the age of two and three. The catamenia appeared at fifteen, have always been scanty and accompanied by pain, but are regular every four weeks.

The patient is a short, thick-set girl with large mammæ. She seems quite intelligent. She reads fairly, but has never learnt to write, on account of the condition of her hands. Her head is rather large and flattened at the top. On measurement it is symmetrical in all diameters. The hard palate seems somewhat arched. There is no deformity of the spinal column.

When lying down in bed the movements are very slight, and during sleep they are quite absent. But if excited by being watched or on attempting movement they are at once noticeable. The condition affects almost entirely the face and upper extremities.

On making an effort to speak the face becomes much con-

torted. The angles of the mouth are retracted, the upper lip and alæ nasi raised, the forehead wrinkled, the eyelids partially closed. These grimaces give rise to various and rapidly succeeding expressions. An appearance of gaiety is followed by a frown, by a sniff of disdain, or by a momentarily lachrymose aspect. Her speech is interrupted by the violence of the spasm; the words are brought out explosively, and she is soon out of breath. With effort, however, she can pronounce pretty clearly. The muscles of the neck, especially the sterno-mastoids, are rigid, but those of the back and abdomen appear unaffected. There are slight movements of the tongue, but it is protruded straight and not atrophied. The fingers are flexed at the metacarpo-phalangeal joints and extended at the phalangeal. But this position is not fixed. The fingers are often separated, and there are simultaneous to and fro movements. The movements often strongly suggest athetosis, but they are more rapid and more various.

The muscles of the forearms and arms are tense, the rigidity being especially marked in the flexors at the elbow- and shoulder-joints. She usually keeps the left hand, which is more affected than the right, behind her back or pressed firmly against the coverlet, possibly with a view to restraining its movements. All the muscles of the upper extremities are well developed and respond to a continuous current of moderate strength. Each hand measures seven inches across the palm. Her grasp is decidedly feeble.

The legs are short, but well developed, and as she lies in bed she has very fair muscular power. She can neither stand nor walk without help. When supported on each side she shuffles along, the legs tending to overlap and the knees to rub together. At the same time the thighs are adducted and the left foot turned outwards nearly at a right angle to the leg. During progression the movements of the face and hands are well seen. After a few steps she gets short-winded and has to rest. On making passive movement of the lower limbs much resistance is felt. Occasionally there are slight, barely appreciable, movements of the toes. The patellar tendon reflexes are very brisk, apparently exaggerated, and more marked on the right side than on the left. Now and then ankle clonus could be elicited. The muscles of the lower limbs respond to a continuous current of about twenty cells. Sensation is perfect all over the body. There is no loss of control over bladder or rectum. Taste, smell, and hearing are good on each side. There is no colour-blindness, no nystagmus or

squint, and the fundus of each eye is quite healthy. Lastly, there is no sign of any visceral disease.

The case just described belongs to a group which, although having many striking points of difference, possesses one characteristic condition. I refer to the spasm of the lower limbs and the peculiar gait.

Spastic paraplegia may exist alone, with or without affection of the upper extremities, constituting the disease known as spasmodic tabes dorsalis (Charcot) or infantile spasmodic paralysis. Very frequently, however, other symptoms are superadded. In *Brain*, October, 1883, I recorded two instances in which there were atrophic changes in the upper limb, but I do not know of any similar published cases.

Including the present, I have seen three instances in which there were choreiform movements. Two of these are quite public characters. One, who sweeps the crossing at the corner of Bond Street and Grafton Street, has the characteristic gait and movements of the left hand. I examined him about a year ago and noted as follows. "As he walks the thighs are adducted, and the knees rub and overlap, the feet scrape along the ground and the great toes are directed inwards. The fingers of the left hand are flexed at the metacarpo-phalangeal joints and extended at the phalangeal, and are moved constantly backwards and forwards." In addition his articulation was very indistinct, his speech being like that of a child learning to talk. He told me that the condition of his limbs dated from birth and that he did not speak until he was thirteen.

The other patient is half beggar, half vendor, who is usually to be seen outside the Mansion House Station. The upper limbs are in constant movement as he walks, and his grimaces are such that they attract the attention of passers-by. His gait also is characteristic. Although I have not had the opportunity of entering into his case I have no doubt that it belongs to the category of the one just mentioned.

In connection with the spasmodic paraplegia of infants other symptoms are often found. Fits in early life, which may or may not persist later, are very common. In many intelligence is not impaired, but mental deficiency and even absolute idiotcy are not infrequent. I have also seen nystagmus, squint and inequality of pupils. In my experience the tendon reflexes are well marked, usually exaggerated, and ankle clonus is frequently present. Among occasional symptoms are, deformity of chest, asymmetry of skull, arching of palate, delayed dentition, and defects of speech.

But to revert to the first case I have described. Where, it may be asked, is the lesion, and what is its nature? I confess I am not prepared to discuss this question, but I think it probable that the affection is dependent on some congenital defect in the brain, or a porencephalous condition. I discovered that the patient had been under Dr. Hughlings Jackson's care at the London Hospital nine years ago. Dr. James Anderson was good enough to search for the case and found it noted as one of "Congenital Double Hemiplegia with Slow Choreiform Movements."

I must mention that Dr. Ross describes a very similar case in *Brain*, Oct., 1882. Here the affection appeared to date from an injury in infancy. The writer thinks it probable that there was a lesion on each side involving the operculum or that part of the cortex which lies between the two limbs of the fissure of Sylvius and which forms the roof of the island of Reil.

It has been suggested that those motor disorders in children known as infantile spasmodic paralysis and congenital athetosis are dependent on injury to the cranium or spinal column during birth. I have made careful inquiries on this point in about a dozen cases, and have found that there is no justification for this supposition. I could not satisfy myself in a single instance that violence during delivery, either by forceps, or by traction on the legs, had any influence.

The case which I have described in many respects resembles athetosis, but I think it preferable to limit that term to the motor disorder occurring after a distinct hemiplegic attack.

As regards treatment, I may say that drugs, galvanism, and ether spray to the back have been, as might be expected, without the least benefit.

XXVII.—*Calculus and Tumour of the Bladder (Carcinoma?); Lithotomy; death on the ninth day.* By John R. Lunn. *Read March* 27, 1885.

J. C., æt. 35, married, by trade a stoker, was admitted September 8, 1884. He had rheumatic fever fifteen years ago, but had not been laid up since with any serious illness. Four weeks before admission he noticed his water began to pass away involuntarily. He attended as an out-patient at the University College Hospital, where he was sounded. Two weeks before admission into the infirmary he gave up work in consequence of the increasing incontinence of urine. The smell of his water was very offensive, but it never contained any blood. He thought he had lost flesh lately.

On admission.—Patient was thin and anæmic, had a careworn expression of face. He complained of shooting pain in his perinæum, some difficulty with his bowels, and constant dribbling of urine. The meatus externus urethræ was swollen and excoriated. A No. 10 catheter was passed without difficulty, but caused a good deal of pain. The prostate was apparently enlarged and tender. The urine was loaded with pus and albumen. The temperature on admission was 99° Fahr. Tongue very dry and furred. All the other organs appeared healthy. No enlarged glands were discovered. The bladder was ordered to be washed out daily. The appetite slightly improved during the next few days. On sounding the bladder on September 17 a stone was struck. The next day lateral lithotomy was performed, and a large stone, weighing one ounce and a half (the size of a pewit's egg) was extracted from the bladder without much difficulty or bleeding.

In addition to the calculus a new growth was felt, which was too extensive to remove. On the evening of the operation the temperature was 100° Fahr., respiration 20, and pulse 92, and the patient expressed himself much relieved and free from pain. The wound and bladder were ordered to be washed out with boro-glyceride (1—40) twice a day. The second day after the operation acute orchitis set in which soon yielded to treatment. Vomiting occurred at intervals. The urine still contained pus and was the colour of claret wine. The pulse ranged between 92 and 124 a minute, and the day

before he died the left kidney was noted to be enlarged (September 26).

Post-mortem notes (forty-eight hours after death).—Body very emaciated; rigor mortis passed off; the wound in the perinæum looked very unhealthy and showed no signs of healing. The bladder weighed 8 oz. and was empty; almost the entire surface was occupied by a villous growth, the wall of the bladder being greatly thickened and apparently consisting of dense fibrous tissue. No enlarged glands were found anywhere. The ureters both were distended to the size of one's little finger and full of pus. Both kidneys were completely disorganised and full of purulent matter. The right kidney weighed 8 oz., the left 14 oz. All the other viscera were healthy.

The calculus has been tested chemically and is formed of phosphate and oxalate of calcium and a little uric acid.

I am indebted to Mr. Eve for the following account of the bladder and description of the growth:

"A urinary bladder opened by a horizontal incision from before backwards. Its walls were infiltrated with a soft cancerous growth, which in the interior wall has attained an inch and a quarter in thickness. The growth projects from the inner surface of the bladder in irregular and papilliform shreds. One of these, an inch in length, formed a tabular prolongation from the origin of the right ureter as if a prolapse of, or a growth from, its mucous membrane had occurred. In microscopic structure the tumour had the character of a soft cancer. The cells were rather small, spheroidal, with large nuclei, and were in no part observed to be arranged in balls or nests. In the softer parts of the tumour they were aggregated in large, diffuse, smaller, and rounder masses, in spaces formed by a scanty stroma, while in the finer parts the stroma was abundant and fibrous and the cells formed elongated rods and masses."

Remarks.—This case appears worthy of note: 1st. On account of the existence in the same individual of the two conditions, calculus and extensive new growth in the bladder.

2nd (if the man's statement be taken as correct), on account of the short period during which symptoms were present.

3rd. The growth in the bladder appears to be of a purely local character, no doubt caused by the irritation set up by the calculus, and this again accounting for its very extensive and general disposition over the entire surface of the organ.

In Sir Henry Thompson's latest work on *Tumours of the Bladder*, the new growths were all limited in size and position. As regards the growth, I have not been able to find any record of a similar case to the present one.

4th. The fact that the patient had been sounded for stone before and on admission, but none detected, his statement that there had been no hæmorrhage, and the short period during which any symptoms had appeared, and the presence on rectal examination of a rounded fulness of an indefinite character at the anterior part of the base of the bladder, led one to suspect an abscess of the prostate spreading backwards.

5th. The slight obstruction of the bowels and the incontinence and character of the urine would appear to agree with this condition. From the history and symptoms of the case there did not seem to be any distinct evidence of a tumour of the bladder.

6th. On discovering the stone at a later examination it was determined to perform lateral lithotomy as giving the patient a chance of recovery, and as presenting also an opportunity of further exploring the bladder. After removal of the stone, however, the growth was found to be so extensive and firmly fixed to the wall of the organ that it was not thought advisable to interfere with it. Although the patient ultimately sank he was greatly relieved by the operation and suffered little pain afterwards. On this account it would appear to have been perfectly justifiable.

The bladder and calculus I have given to the Royal College of Surgeons (No. 3701*a*).

XXVIII.—*Three Cases of Bullet Wound.* By Richard Barwell. *Read March* 27, 1885.

Case I. At about 11 o'clock of the night, November 14th, 1875, a young gentleman, after taking a copious dinner in apparently the highest spirits, shot himself with a Derringer.* Presumably he aimed at the heart, but went a little too low. He vomited immediately, and was soon after taken to the hospital. I found him greatly collapsed; over the seventh costal cartilage was a small black wound from which oozed a drop or two of blood. No probe could be passed—nor could one have been unless force had been used—through the opening in the cartilage, which evidently had split to let the bullet pass, and had closed again. The patient survived twenty-two hours, dying of prolonged shock and of almost constant vomiting, which no remedy would control.

After death there was found in the cartilage an opening formed by three lines diverging at equal angles from a centre; behind this was a scarcely perceptible rift in the diaphragm, without any ecchymosis, through which the bullet, leaving the chest, entered the abdomen. It then passed through the anterior and posterior walls of the stomach, and notching the upper inner edge of the kidney went between the first and second lumbar transverse process, and was found behind the former projection. The case interested me much as showing the power of such small weapons, and of projectiles driven only by the fulminate in the copper cap.

Since the above date various cases of pistol-shot wound of limbs, or superficial ones of the head and trunk, have occurred in my practice. Most of these have not been very severe nor important; but two of a serious nature will, it is hoped, prove of interest to the Society.

Case II. Thomas H. B., æt. 35, a gentleman against whom his partners in business had brought some charges (unfounded as was afterwards proved), shot himself in the Charing Cross Station with a Derringer, October 22, 1880. He was brought

* The weapon originally bearing this name, the bore of which is $\frac{4}{10}$ inch in diameter.

at once to the hospital. I saw him very shortly after. He was much collapsed, pulse 74, very weak, surface cold. On the right side of left nipple and within the circle of the areola was a bullet wound from which arterial blood flowed pretty freely. He coughed occasionally, not very violently, and this caused the blood to spurt forth, so that when he was sitting nearly upright at the head of the bed (for he could not lie down) the blood was driven out and fell to the floor two feet nine inches beyond the foot of the bed. There was also considerable hæmoptysis. The wound was dressed with a single layer of lint dipped in carbolised oil very lightly applied, and surrounded by a ring pad. Strict silence and immobility enjoined, ice frequently given by the mouth.

October 23.—Bleeding from wound gradually diminished and ceased a little after two this morning. Collapse at 2 P.M. nearly passed off. Much surgical emphysema round wound, also extensive pneumothorax, but prolonged examination unadvisable; hæmoptysis continues. Temp. 98·3° (evening); pulse 112; resp. 40.

October 27.—Surgical emphysema occupies all the left side down to the eighth rib and half the front aspect of the chest. There is pneumo- and hæmo-thorax. A line (horizontal in his semi-recumbent position) almost coincident with the fourth rib separates the hyper-resonant from the dull portion of the chest; in this latter part absence of respiratory murmur, but marked œgophony is present. I detected by palpation the bullet a little outside the angle of the scapula, just superficial to the ribs. I shall not at present interfere with it. Hæmoptysis continues, but the blood is no longer fresh, save an occasional small quantity.

November 4.—Has been going on well; the pneumo- and hæmo-thorax diminishing, the temperature steady and normal. But at this date he was evidently weaker and last night had severe sweating.

November 16.—The sweating has been diminished by sulphuric acid, and the strength has improved. During the last ten days the hæmoptysis has changed its character to dark and then to discoloured blood. Expectoration now slight and consists almost entirely of unstained mucus. The surgical emphysema has disappeared, also, except quite at the upper part of the chest, the pneumothorax; round the lower portions there is still dulness on gentle percussion, but more powerful taps elicit a clearer note. The respiratory murmur is a little distant.

November 26.—Removed the bullet; it was flattened on one side, and had here embedded in it a fragment of bone which probably it rent from the back part of the rib as it passed out of the chest.

December 3.—Nothing but gradual progress to record. The patient left to-day, save for some slight weakness, perfectly well. The treatment had consisted in the administration of acetate of lead, small repeated doses of opium, sulphuric acid; also as debility and pneumonia came on, of small doses of ipecacuanha and of brandy; later still of quinine and iron.

Case III. Carl Anel F., æt. 31, shot himself with a small pistol; he had for some years been suffering from the head, hearing voices, &c. He hardly appeared to know how or why he unlocked the pistol and made such use of it; after doing so at 2 A.M. on October 24 he lay on his bed for four hours, then woke his wife and told her. At 9 A.M. husband and wife walked together to the hospital.

There were found two small wounds, one traversing the left side of the soft palate, the other three quarters of an inch down the right auditory meatus; from this latter a little blood flowed. It may as well be said at once that the first-named wound gave no trouble. I once, by lifting the palate, caught sight of a corresponding wound at the back and side of the pharynx; this, as well as the one in the velum palati, healed quickly. The situation of the bullet is unknown; it is surmised from the direction to be among the deep muscles at the back and left side of the spine.

I examined the wound at the ear; the bullet had passed three quarters of an inch down the meatus without damaging the skin on either side; then it left that channel very obliquely, continuing its directly transverse course. On passing a probe I came upon a hard substance which gave to my hand the impression of being metallic. This substance was touched at a depth of just over two inches, not from the tragus or antitragus, but from the margin of the meatus.

Now, the direction and depth of this tract being considered, it seemed to me that the bullet must lie in very dangerous proximity to the lateral sinus as it passes inside the mastoid bone; it might even have wounded that vessel, and be acting like a cork to prevent hæmorrhage. It seemed to me the safe course to leave it alone.

October 26.—The flow of blood almost ceased on the second day (25th). On the 26th this became mixed with a consider-

able quantity of clear, watery fluid, either cerebro-spinal or liquor Cotunnii; some was collected for testing, but was too much mixed with blood. On the 28th it was mingled with pus.

November 1.—A spiculum of bone came away. The parotid and the parts behind the jaw swelled.

November 14.—During the past fortnight the patient has been suffering from inflammation of parts overlying the ascending maxillary ramus, and I this day detected a softer spot with deep fluctuation near the angle of jaw. The temperature has been high at night, 102°, on two occasions but down to 99° in the morning. He is singularly apathetic, and hardly takes notice of anything.

November 23.—Some troublesome cough caused me to ask Dr. Murray to examine the chest. He had bronchitis and some emphysema; phthisis doubtful. With a Nélaton's probe I obtained a clear mark of lead.

December 4.—There is now a somewhat large abscess over the ramus of the jaw, and though temperature is not far from normal the man is getting weaker and thinner; it seemed to me time to endeavour to extract the bullet. To do this from the bottom of the narrow channel was evidently impossible; no sufficiently powerful forceps could be introduced and opened. When, therefore, after a few failures a sufficiently characteristic lead mark on a Nélaton's probe had been obtained, I made a curved incision immediately behind the pinna, turned the flaps backward and forward, and trephined the mastoid process to the depth of $\frac{1}{8}$ of an inch. The cavity left by removal of the bone-plug was examined by touch and with Nélaton's probe, without result., but I found with my finger a narrow opening. The probe, passed along this, was again blackened, but at a great depth—over an inch from the floor of the trephine hole. This track was now very cautiously enlarged with a gouge, a pair of fine strong sinus forceps passed down till it seemed to me I got a touch as of metal; they were opened, and I felt that I had hold of the bullet, which, loosening with a rocking movement, for it was tightly fixed, I now extracted. On the night of this operation the temperature rose to 100·8°.

December 5.—Temperature fell last night after 12, and at 10 A.M. to-day was normal; it rose afterwards, and was at 100·4° from 2 to 6 P.M., and then began to fall again; patient has some pain.

December 9.—At my request, Mr. Dodson, my house

surgeon, to whose unremitting care I am much indebted, opened the abscess over the jaw.

After this I have nothing to report, the trephine opening filled quickly, the sinus contracted. He had no abnormal temperature, and was discharged on January 5 perfectly well. Indeed, he might have gone a week earlier, but for some police arrangements.

On the two last cases much might be said did time permit. A few words are inevitable. Both men shot themselves; the pistols, therefore, were very close, and in a medico-legal point of view, especially bearing on a recent case, it is important to observe that although the muzzle was in this last case on the skin, no clothes intervening, there was neither scorching nor powder-tattoo, and this is, as far as my observation goes, the usual condition with projectiles driven only by the power of the fulminate.

In the former case (the thoracic wound) the bullet passed through about seven inches of lung tissue, as near as I could measure, inflicting, therefore, very grave injury to a vital organ. In the latter case, although there is no positive proof, yet there is high probability that the projectile entered, though it did not pass entirely into, the cranial cavity. Yet both these men recovered.

These results must be, I think, attributed to the shape of the projectiles. Conical bullets, though they split bones more widely than round ones, must bruise soft parts through which they pass less.* Still more may the result be due to the small size of the bullets—that extracted from the thorax of Thomas B. measures a little less than a quarter of an inch in diameter. That taken from the head of C. F., is No. 230 Eley's gauge, which measures exactly a quarter of an inch.

It must not be supposed that the projectile possesses but little force; the bullet that went through T. B.'s lung is flattened on the side which struck the back part of the rib, and as it left the chest it ripped off a piece of bone, burying it in the lead.

The bullet which I took out of Carl F.'s head is, as you see, flattened and twisted; it is indeed very much misshapen by its passage for two inches through bone; the drainage-tube passed and kept in after the operation measured from the skin inward

* As evidenced by the singular absence of ecchymosis in the first case of this series. Only the edge of the kidney wound, and that to a very small degree, showed signs of bruising.

two and an eighth inches. The weapon has, indeed, considerable power. Carl F.'s pistol—here it is— is a beautifully made rifle barrelled, seven chambered revolver (American). It looks like a toy, and can be hidden completely in the hand. At ten paces I fired at a beech-wood board exactly one inch thick; the bullet went clean through the wood and flattened itself against the wall behind, out of which it raised a little cloud of brickdust.

It is evident, however, that unless some immediately vital part be hit, such as the heart or certain parts of the brain and spinal cord, such small bullets have but little immediate effect. A man might have several such bullets in him, yet still be good for some little fighting, and may after all recover even though long and deep wounds like those described have been made.

XXIX.—*Amputations at the Hip-joint by Furneaux Jordan's Method.* By Lewis W. Marshall, M.D. *Read April* 10, 1885.

ON October 27, 1882, a paper upon Amputation at the Hip-Joint by Furneaux Jordan's Method was published by me in the *British Medical Journal,* having been previously read at a local meeting of the Midland Branch, on June 24, 1880. Four cases formed the basis of this paper. Since that time this operation has become much more widely known and generally adopted. A list of cases has been recorded by Dr. Maclaren, of Carlisle, in a paper read by him in Edinburgh, and afterwards published in the *British Medical Journal* of June 7, 1884.

I am permitted by your Society to lay before you those cases already published, and to add to them the amputations done by me since October, 1882. Before doing so, I will briefly describe this method of amputation.

If excision of the hip has been previously practised, the existing incision is prolonged to about the middle of the thigh; the femur enucleated, the soft parts cut through with a circular sweep of the knife, and the femoral tied. This is the first method suggested by Mr. Jordan, but he goes on to say, in his report of a case in the *Lancet,* March 23, 1879, that "the surgeon may if he choose, make the circular sweep before the shaft of the bone is turned out, if precautions against hæmorrhage have been complete." Of the two methods I have more often adopted the latter than the former for reasons to be given hereafter.

Summarised my cases stand thus:—ten in number. Three after excision, and the remainder when this operation could not be practised on account of extensive implication of the femur or pelvis, or from the general condition of the patient being such that more conservative measures were likely to prove futile. In all, it may be briefly expressed, that the main object of the operation was to save a life which would otherwise have been sacrificed. Their ages were as follows:—two cases at eleven years, one case at ten years, two cases at eight years, two cases at seven years, one case at six years, one case at five years, one case at three years.

In all but one the fingers were used to compress the femoral; in two Esmarch's bandage was first applied, the fingers being finally substituted, because the bandage was insecure. Davy's lever has been used by me twice, once in this operation, and once in a double flap amputation. Of the bleeding I may say that although it is not excessive, there is extreme variation in the quantity lost in each case, because where large abscess cavities are present, much oozing is apt to occur from them; the actual number of vessels requiring catgut ligature being usually not more than two, the femoral included.

In this list, one death—the direct result of the operation, an amputation on the right side in which Davy's lever was used—has to be placed. Death occurred in a few hours, the patient never rallying, the blood loss being excessive. With the exception of two other deaths, one of which took place three months after operation from visceral lesions, and the second, nine days after from the effects of the rupture of an abdominal abscess, all are, I believe, living and well.

Drainage was secured by horsehair, and in addition, when an opening into the pelvic cavity existed, an india-rubber tube was used at the upper end of the incision. It is my custom to secure the flaps loosely together by wire sutures at three or four points only; and as a dressing, a loose covering of carbolic oil and lint, with an outer layer of oakum has been most commonly chosen. The lint is kept soaked by repeated applications of carbolic oil. The earlier cases were done under the spray. In the later ones it was omitted. This gives a general outline of my success in this operation, and also, very briefly, of the details of after-treatment. I would supplement the remarks made in reference to the dressing, by a full statement of the means adopted by me for limiting the shock of operation and securing a comparative speedy reaction. To these details and to the care with which they have been carried out, I attach much importance, and consider that, to some extent, my success is due. Half an hour before operation I order to be given a dose of opium in proportion to the age of the patient, the customary abstention from food usual before the giving of an anæsthetic being observed. The bed to be occupied by the patient after his removal from the table is kept thoroughly warm by hot-water bottles, and an enema of beef-tea and milk, containing aromatic spirits of ammonia, is at hand to be administered immediately. If the collapse is great, both brachials and the femoral are ordered to be held. I give a suppository of morphia within half an hour of the opera-

tion. In my earlier cases—indeed in all—vomiting has come on at an early period and has given much trouble and distress. To meet this difficulty I am now, and have been in the last four or five cases, led to withhold all feeding by mouth, if small quantities of iced champagne be excepted. Nutrient enemata either with or without ammonia as the case appears to demand, are given every half hour until such time as it is found by a careful use of Brand's beef jelly, whey and cream, or a little milk, that the stomach is capable of doing its work. At the risk of being thought prolix, I have felt it necessary to insist on these details because, as I have before expressed, I feel so much is due to them.

Mr. Jordan remarks upon his single case in support of his method that, "as compared with the ordinary operation of two flaps, the wound was less severe, the cut surfaces were less extensive, and in a manner further removed from the trunk; it was followed by less shock, less hæmorrhage, less opportunity of septic infection. The vessels cut were more easily dealt with. The thigh might be simply cut through with a circular sweep of a few sawing movements. The boneless thigh should be firmly held and somewhat flattened if cut across. The muscles may be cut on the same level as the skin; the bone being absent, they retract so strongly that the skin readily covers them; its vitality is less endangered; and a large cellular plane is not opened. The bulk of the soft parts of the thigh, especially near the pelvis, lies at the inner side of the femur. Why put a knife through these parts? It is better to enucleate the femur when it is covered, and cut across the limb where it is smaller and further removed from the trunk. In removing the thigh very low down, the area of the wound is no doubt increased; but even then it would be a much less dangerous wound in character and locality." He goes on to say that the operation is more suitable for those cases in which the soft parts can be freely left. He also calls attention to the value of the operation as giving safe access and free drainage for any length of time.

I have given these quotations from Mr. Jordan's paper because I feel that in his own words the advantages of the operation introduced by him are more likely to be conveyed accurately than by any words of my own. There are some points in connection with the carrying out of this method which I wish to mention.

I stated in the earlier part of this paper that the second plan recommended by the writer has been most commonly

employed by me. I mean the performance of the circular sweep of the soft parts before the enucleation of the femur is attempted. My reason for doing this is that the increased length of leverage given by freeing the limb from the main attachment of the muscles enables the fingers and the knife to pare the muscles off from the bone with much less risk of wounding vessels unnecessarily. Also, when the hip-joint is arrived at, separation is rendered much easier and more rapid of the tough bands which in old-standing cases of hip disease held the trochanter and what exists of the neck of the femur so closely to the pelvis. The chief advantage gained by Furneaux Jordan's operation is, that the gluteal vessels remain untouched, the wounding of these in the double flap operation and the consequent bleeding being the cause of much risk to the patient. In all discussions upon the subject of amputation at the hip-joint, that I have read or heard of, great stress has been laid on the anxiety to which the bleeding from these vessels has given rise. The value of abdominal torniquets and Davy's lever for the compression of the aorta and common iliac artery has been freely discussed, and the conclusion arrived at was that Davy's lever was most to be preferred. I regret to say that my only death occurred after the use of this instrument in a boy æt. 11, and in better health than any other patient operated on by me, but with the disease requiring removal of the right leg. This last fact may probably account for its incomplete action, and my mind therefore may have been somewhat unjustly prejudiced against the lever. Whilst admitting this, I however see good cause for omitting its use. Handling per rectum does not appear to me to be desirable if not absolutely needed. Where—and we never know when it may not exist—intra-pelvic inflammation or suppuration is present, harm may result by the pressure and manipulation of the lever. The efficacy of nutrient enemata directly after operation might also be interfered with.

In all my cases I have had the valuable help of Mr. Wright, senior surgeon to the General Hospital, Nottingham, by whom the common femoral has been controlled most satisfactorily. The presence of the hand in the groin has in no way been a hindrance to the operator. It must of course be remembered that my cases have been collected from children's work, and these remarks may not therefore apply with equal force to adult patients. The femoral artery has always been tied by me immediately on completion of the circular incision before enucleation of the femur is commenced. Although of secondary

importance, amputation at the hip-joint being undertaken principally for the saving of life, I may state that the stump left by Jordan's method is in every way all we could wish for. I am aware of the late Mr. Shuter's case and his suggestion that the periosteum should be left. Whilst such a measure would probably add to the usefulness of the stump, the time occupied by the operation must be lengthened, and this is undesirable. One important matter I must refer to before concluding. It is that one of my cases, a girl, who had been in hospital nine months, and whose hip-joint had been previously excised, was the subject of extensive lardaceous disease. Her liver reached well towards the umbilicus, and a heavy deposit of albumen was found in her urine, of which traces could be detected up to ten months after amputation. She was seen by Dr. Goodhart amongst some other cases shown by me in June, 1880 (eleven months after removal of her limb), by whom her liver was then found to be enlarged, although at that time much reduced from its original size. This girl is now hale and hearty, and seen constantly by me, walking on her crutch and enjoying life thoroughly. Her liver is now to be felt just below her ribs. The albumen in her urine has not returned. In two other cases large deposits of albumen were present when amputation was done.

In conclusion, I may say that by giving us this plan of operation Mr. Jordan has conferred a boon on the patients and a welcome addition to surgery. My object in placing my cases before the Society is to add to the previous record of cases as yet confined, I believe, to the publication of papers written by Mr. Jordan, Dr. Maclaren, of Carlisle, and myself. I have to thank my friend Dr. Goodhart for his suggestion that I should read this paper here, and the president and members of this Society for permission to do so.

Dr. Marshall's Amputations at Hip-joint by Furneaux Jordan's Method.

No.	Name.	Age.	Previous operation.	Amputation.	Artery controlled by.	Hæmorrhage.	Shock.	Result.	General complications.
1	Sam. Perry	8 yrs.	Excision, Oct. 9, 1878	April 20, 1879	Tourniquet and finger	Small	Not great	July 9, 1879*	Pelvis involved. Sequestra. Sinuses 8 months. Exhaustive diarrhœa 7 months before. Amputation.
2	Ch. Saxton	7 yrs.	Excision, Dec. 11, 1878	Aug. 13, 1879	Finger	„	Great	O.P. Oct. 8, 1879	Lardaceous liver. Albumen 4 days before operation, lasted 10 months. Sinuses on admission.
3	W. Wilkinson	8 yrs.	None	Oct. 24, 1879	„	„	Not great	O.P. Jan. 21, 1880	Sinuses 2 months.
4	Geo. Martin	6 yrs.	Excision, Feb. 26, 1880	April 8, 1880	„	„	„	O.P. June 5, 1880	Sinuses 1 month. Hæmorrhage after excision.
5	M. Cotton	8 yrs.	None	Dec. 29, 1881	„	„	„	O.P. May 5, 1882	Albumen 3 months before, lasting a short time. Sinuses 5 months. Chest involved.
6	Jas. Owen	11 yrs.	„	March 31, 1881	Davy's lever	Very free	Great	*Death*, March 31, 1881	Numerous sinuses.
7	H. Fox	11 yrs.	„	Feb. 4, 1883	Finger	Much	Much collapse	Feb. 13, 1883†	Sinuses 16 months. Pelvis involved and perforated.
8	Sarah Hardy	10 yrs.	„	Sept. 25, 1884	„	Small	Slight	O.P. Dec. 6, 1884	Sinuses 3 months.
9	Lily Foster	5 yrs.	„	Oct. 12, 1884	„	„	Not great	O.P. Dec. 15, 1884	Sinuses 4 months.
10	John Francis	7 yrs.	„	Jan. 25, 1885	Esmarch at first, slipped, finger afterwards	In excess	„	Feb. 7, 1885‡	Large discharging wound 6 months. Pelvis involved. Intra-pelvic abscess opened through acetabulum at operation and sequestra removed.

* Died 3 months after from visceral disease.
† Died 9 days after from bursting of an abdominal abscess which discharged through parietes exhaustion.
‡ Still in hospital, but doing well up to February 7, 1885.

XXX.—*On Amputation at the Hip in certain desperate cases of Disease of the Joint or Bone.* By Jonathan Hutchinson, F.R.S. *Read April* 10, 1885.

MY object in the present paper is to elicit the opinion of the surgical members of the Society as to whether the amputation of the entire limb in certain desperate cases of disease of the hip and femurs might not be advantageously performed more frequently than it is. Especially I think is this the case when things are going badly after excision of the hip-joint. I feel sure that in my own practice amputation has in several instances snatched the patient from an otherwise certain death, and I can look back on several others in which I much regret that it was not performed. It is astonishing how well, in cases of even extreme exhaustion, this formidable operation is borne, and how rapid and satisfactory is the convalescence. I do not indeed recollect that I have ever lost a patient after amputation at the hip-joint for suppurative disease. The difference is very marked between this class of cases and those of amputation for new growths.

I shall not attempt to produce statistics either from my own practice or from the narratives published by other surgeons, since I have not the facts before me, but shall content myself with the brief record of three or four illustrative cases.

The first of these is one which, having been already published, I shall allude to very briefly.

It occurred many years ago, at a time when I had the good fortune to have the services of Mr. R. W. Parker as my house surgeon at the hospital.

Our patient, a young woman of about twenty, was enfeebled and emaciated to the last degree by combined disease of the hip, knee, and femur. We had thought her too ill to bear any operation, and had been for some time expecting her death. It was finally determined to give her the chance. She was too weak, I well recollect, to be taken to the theatre, and I amputated at the hip-joint on her bed. Thanks to Mr. Parker's assiduous care, she recovered, and is at the present time stout and healthy.

My next case is one in proof that even advanced amyloid

disease of the viscera is no bar to the operation. In this instance the hip-joint itself was not involved, but there was necrosis of the femur up to the great trochanter. The patient was a boy æt. 10. I had removed the sequestrum and then sent him to Margate. He returned after a stay of some months with profuse suppuration still going on, with his liver enormously enlarged, and so bloodless that his face and lips were of waxy pallor. His feet were somewhat swollen, but there was a good point,—he had no albumen in the urine.

I amputated at the hip-joint by Furneaux Jordan's method, detaching the periosteum from the shell of new bone over considerable parts of its surface. He recovered easily from the operation, but the point of most interest was that his liver afterwards diminished to half its bulk, and that he regained to a very remarkable degree his strength and colour. There was some reproduction of bone at the stump.

I had him under observation for about a year after the operation, but have not seen him lately. Several of my friends have mentioned to me other cases in which, as in this, there was definite proof of diminution in size of an amyloid liver after removal of the diseased bone or joint which had caused it. I am bound, however, to admit that this is not invariably the case, for in another instance in which, after amputation at the hip-joint, the parts healed perfectly and the patient went to the seaside, I found some months later that the amyloid cacchexia was decidedly advancing and the liver getting larger.

My third case is that of a young gentleman whom I attended with Mr. Benjamin Duke at Clapham Common.

He was æt. 15. I had excised the head of the femur one year before the amputation at the hip-joint on account of disease attended by a very large abscess. Most of the interval had been spent at the seaside, and at one time there had seemed good hopes that recovery would ensue. These, however, faded away. He lost strength, became extremely thin, and had still profuse suppuration. He was as pale as he could possibly be, but there was no evidence of visceral enlargement and no albuminuria. On the day that Mr. Dukes and myself decided to recommend the amputation we did not think it at all likely that he would live another week. On the following day when we met for the performance of the operation, a most untoward complication presented itself, for we found the leg below the knee attacked by erysipelas. Feeling sure that this was our only chance, we decided to go on with the intended operation.

I again adopted F. Jordan's method, and although for a

few days after the operation our patient remained in a very critical condition, he ultimately made a fair recovery and has since undertaken a voyage to Australia for his health. Although much improved, I believe that sinuses still exist.

In this case, as in the first, the acetabulum was carious, but in neither of them were any conditions found at the amputation which could have been treated with benefit by any other measures.

By the side of these very encouraging cases, permit me to place two others in which the operation was not done, and which are, I fear, fairly illustrative of a considerable group.

About four years ago I excised the head of the femur in the London Hospital for a young woman aged about twenty three, who had long suffered from suppurative disorganisation of the joint. After the excision she remained in the hospital about six months, with profuse suppuration all the time and steady deterioration of health. At length, amputation being declined, she was taken home by her friends for the benefit of change of air, and some time afterwards I heard that she had died.

In another case I attended ten years ago with Mr. Benjamin Clark, of Clapton, a young gentleman æt. 19, who was in a very advanced condition of exhaustion from suppurative disease of the hip-joint when I first saw him. He was too ill for us to think of excision, and I urged as strongly as I could that we ought at once to relieve him by the removal of the entire limb. His parents naturally thought this very serious advice, and it was decided that another opinion should be asked. I met in consultation a very distinguished surgeon, since deceased, who entered into the consideration of the case in a most careful and thorough manner. The result was, however, that although he agreed with me that the patient was too weak to bear excision he could not bring himself to recommend an amputation. He reminded us that although patients often became very ill from hip-joint disease, that yet they but seldom died, and recommended that we should wait. Within a fortnight of our consultation, nothing having been done, the patient died.

The conclusion which I would wish to submit for the consideration of the Society is this :—That amputation at the hip-joint, when done in the state of hectic produced by suppurative disease, is attended usually by but little shock to the system, and is often followed immediately by definite indications of relief, and in the sequel by perfect restoration to health.

That it would be well if this operation were resorted to

more frequently than it is, and that it ought to be considered as distinctly indicated whenever in a patient otherwise free from disease death seems likely to occur from the joint mischief.

Thirdly, that the existence of even advanced amyloid disease of the viscera does not preclude the operation, since it does not prevent the recovery of the patient nor prevent the restoration of the general health.

In conclusion, let me say that I am well aware that there is nothing novel in the recommendation of this operation in the class of cases referred to. Indeed, I quite hope that in the discussion which will follow, other surgeons will relate yet more satisfactory results than those which I have brought forward. My sole desire in introducing the subject to your notice is to recommend a practice which I feel sure is good, and to elicit the experience of others respecting it.

XXXI.—*On Pityriasis circiné (Horand) and Pityriasis circiné et marginé (Vidal).* By T. Colcott Fox, M.B. *Read April* 10, 1885.

In recent years attention has been directed to an affection of the skin, interesting from a diagnostic point of view, though otherwise of a comparatively trifling nature, which approaches somewhat in aspect the well-known roseola annulata of Willan, but, unlike it, pursues a chronic course. It is not my intention on the present occasion to occupy the time of the Society with a recital of the history of the disease, variously described as pityriasis rosea, pityriasis maculata and circinata, &c., but I will refer those interested in the matter to the theses of Mettou (1877) and Nicolas (1880), and to the papers by Vidal ("Du Pityriasis," *Progrès Méd.*, 1877), by Duhring (*Amer. Journ. Med. Sci.*, Oct., 1880), by Behrend (*Berl. klin. Woch.*, 1881–2), and by myself in the *Lancet* for 1844.

As I shall deal with the affection only as seen in children, I will take as my text the paper entitled "Notes pour servir à l'histoire du Pityriasis circiné," by Horand, of Lyons, in vol. vii of the first series of the *Ann. de Derm. et de Syph.*

Horand there gives a detailed description of seven cases in children, whose ages ranged between eight and thirteen years, and accurately portrays an affection which seems to me a distinct morbid entity, and which I have several times recognised in this country. Horand says that the affection can have for its site the neck, the trunk, and the limbs, but it shows itself especially about the scapular, deltoid, and subclavicular regions. It is constituted by discrete or confluent patches, of which the dimensions vary ordinarily from a lentil to a two-franc piece. They exceed these dimensions only in rare cases. The patches are slightly raised, rounded, the smaller uniform in aspect, the others depressed in the centre with only the borders raised, which gives them a distinctly circinate form. Their colour varies from a pale rose to a yellowish white, approaching to the tint of the surrounding skin, so that one can sometimes with difficulty distinguish them from the rest of the integument if the patient be not placed obliquely. Often the border alone is coloured. These macules are constantly dry, their surface is more or less furfuraceous, and the tissues upon which they are set are supple, and not thickened or inflamed. The skin which separates them is

healthy. They are accompanied by a little pruritus sometimes, and then under irritation they redden. They give rise to no other inflammatory phenomena during their evolution of which the duration cannot be determined precisely. The eruption can persist many months, if not treated, but in spite of chronicity the characters do not change, the patches seem to preserve throughout the dimensions they first presented, and on their disappearance the affection leaves no trace of their presence. The nature of the eruption is quite obscure, but it seems to occur only in those who do not practise scrupulous cleanliness. The subjects of it are often apparently quite well, though sometimes below the standard of health. It is not contagious and no fungus can be discovered.

To this accurate descriptive summary I will add that it seems to commence nearly always on the trunk and thence to extend to the limbs, as it pales away in the regions originally invaded. In its extension down the limbs the eruption first occupies the inner aspect of the upper arms and thighs. I have never traced it on the palms or soles, nor in the buccal cavity. I must point out again the delicacy of the eruption, for it is much less marked than even slight cases of psoriasis in children and even commencing macules of tinea circinata. The majority of the lesions indeed, as Horand says, can often be only detected in certain incidences of light, and the skin simply looks dirty from a faint roughening and yellowish pigmentation. After a bath, however, and when the circulation through the skin is stimulated, the original pale rosy aspect of the older macules is brought out and the eruption shows very plainly. As a rule the macules, which are more or less rounded or oval when freshly evolved, remain isolated and are very closely set, being separated by at least an equal area of healthy skin. As to its frequency Horand met with seven cases in seven years, and I have recognised as many in two or three years, so that whilst not very rare it cannot be very common. The youngest child affected in my experience was four years old.

Without going into details I will ask the Society to accept from me the statement that it is not a roseolar or erythematous syphilide, though closely resembling it in aspect except for the rings, and it is not caused by any of the well-known fungi which attack the skin. Nor do I think it can be classed with the now clearly-defined group of the erythemata. It corresponds closely to what one would imagine a *chronic* roseola annulata (Willan) would be like, and as such I think it would be best to group it.

One of my objects in bringing to notice this affection is to contrast it with another. In July, 1879, Vidal of the Hôpital St. Louis, demonstrated to the Société de Biologie a form of P. circiné et marginé which he stated to be a perfectly defined and easily diagnosed affection. In the *Annales de Derm. et de Syph.*, vol. iii, 2nd Ser., 1882, he further figured in connection with such cases a minute fungus called by him the Microsporon anomæon ou dispar, consisting of round spores averaging one thousandth of a millimetre in size, but particularly irregular in volume, disposed in circles round the epithelial cells, and arranged also in groups or masses of spores irregularly distributed on or between the cells of the superficial layer, and especially the middle layer of the epidermis and about the orifice of the hair-follicles. Vidal could never detect with any certainty any mycelium, in this particular differing widely from the fungus associated with Erythrasma. This fungus was found in patches on the face, beard, and neck, and I do not gather that any cases in children were noticed. The hairs were unaltered. Vidal distinguishes the eruption from the maculate and circinate pityriasis of adults (Bazin) by the facts that the parasitic form is never symmetrical, its distribution has the capricious irregularity of parasitic affections, and it commences on the limbs or trunk, its course is irregular and its duration is many months. He does not, however, contrast it with Horand's pityriasis circiné.

I have met with two cases lately in children of four years and seven years respectively, closely corresponding to Horand's affection, in which I have found a micro-organism apparently identical with that described by Vidal. The only difference from Horand's disease is that the rings seemed to attain a somewhat larger size, some being nearly an inch in diameter. I show a water-colour drawing of one case and will briefly describe it.

Agnes S., æt. 7, a child with reddish hair, very fair complexion and delicate skin, developed a pink-ringed macule the size of a shilling on the inner side of the left knee in September, 1884. She was at the time apparently in her usual good health. The macule was thought to be a ringworm and was treated as such by two medical men. The eruption, however, gradually spread down and up the leg and appeared on the other limb, and then extended upwards over the trunk. The child was observed to scratch occasionally in the daytime and at night considerably.

In October, 1884, I found the whole surface of the body and limbs studded closely with faded rosy macules from a split pea to a shilling in size, rounded or oval in outline, roughened

or faintly scaly, and hardly at all raised. Here and there they were ringed, and coalesced to form gyrate patterns. A few existed on the face and one in the scalp, but the hair remained unaffected. There was an appearance of considerable symmetry. It was difficult to estimate the duration of all the macules, which were first noticeable when the size of split peas, as they appeared to slowly spread and intersect one another and remain in a semifaded condition for a long time. The body seemed almost clear at times except for some patchy roughness and tawny staining, but after heating of the skin or a bath they showed up again quite brightly. I watched the child carefully until the middle of January, 1885, in the hospital, and by that time most of the eruption had disappeared by constant bathing and under arsenic, but since her discharge many new macules have evolved irregularly, especially about the loins. The child suffered from dyspepsia and constipation. None of the girl's brothers and sisters were affected.

In both the cases I found a great quantity of a minute organism in every patch I examined, and not in the intervening healthy skin. It is a very minute organism, as I show under the microscope, and occurred mostly in the dense aggregations of which here is a drawing. I have never been able to detect any mycelium. My friend and colleague Mr. Watson Cheyne was kind enough to undertake a cultivation, a specimen of which, permanently stained, I also show. It is presumably the same organism, but repeated attempts at inoculation have so far failed.

I have no intention of jumping to the conclusion that this fungus is the cause of the eruption; indeed, I am disposed to think otherwise, as I have already found a similar organism in two cases of psoriasis in children, and it is possible that it is commonly to be found amongst desquamating epithelial scales on children's skins. Very similar organisms have been met with by other observers in several affections of the skin, but I will not now detain the Society with a review of this subject, but content myself with the foregoing remarks in the hope that other members may investigate the matter as occasion arises.

August, 1885.—Since the foregoing remarks were contributed I have systematically sought this organism in the desquamating cuticle of psoriasis, ringworm, &c., in children, and can affirm that it is almost invariably present. It is probably, as Dr. Stephen Mackenzie has suggested, only a micrococcus flourishing in a shedding epithelium.

XXXII.—*A Case of Sporadic Cretinism.* By Sidney Phillips, M.D. *Read April* 24, 1885.

N. P., a female child, æt. 10¼, first came under my observation in the out-patient department of St. Mary's Hospital in February of this year. She was born at Holloway, of healthy parents, each æt. 25 at the time of her birth, and between whom no blood-relationship existed. For the first four years of her life she lived at Holloway, since then at Ealing.

There have been six other children of the marriage, two older and four younger than the present patient; all these children, except the youngest, who died of bronchitis, are living and healthy.

The father was, previous to the birth of the child, occasionally intemperate in the use of alcohol.

There is no evidence of rickets in any of the children and no indication of phthisis or syphilis can be traced.

When seven months pregnant the mother was much frightened by a child falling into a well, but pregnancy continued the full term, and the child—the present patient—was born after a natural labour and without instrumental assistance.

From the mother's account it appears that she was a fine child at birth, and progressed very well till about the age of nine months; she was then observed to become quieter than heretofore and less active than other children of the same age.

She also grew more slowly than natural and ceased to grow altogether at about 2½ to 3 years of age.

Dentition was not difficult nor delayed, but she did not attempt to speak or to walk till six years old.

Her habits which had previously been very dirty, improved about this time. Since then she has not altered in any way, either mentally or physically. She has never been able to learn even her letters, and attempts to teach her anything have been discontinued.

She was admitted early in February into the hospital, and I am indebted to the kindness of my colleague, Dr. Cheadle, for allowing her to remain under my care.

She is now æt. 10¼ years, her height is 2 feet 7¾ inches (the average height of a child of 2½ to 3 years of age), her weight is 2 st. 7 lb.; her head is large, measuring 20½ inches round the forehead and occiput; the vertex is very flattened, and the anterior fontanelle is still unclosed over the upper part of the head, the hair is very scanty, and the scalp is covered with thin dirty-looking scales. The eyebrows and eyelashes are well developed.

The face is broad, with an absence of expression; the cheeks large and flabby; the bridge of the nose is broad and depressed and the nostrils expanded.

No thyroid gland can be felt, and though it is often difficult of detection in children of this age, the circumference of the trachea can here be so nearly surrounded by the fingers that I think there can be no doubt of its absence in this case.

The skin is everywhere very harsh and dry, and in some places scaly; it feels, too, decidedly thickened. The mother says she never perspires, and a hot bath failed entirely to make her do so.

Above the clavicle on each side of the neck is an elastic rounded mass fully the size of a large orange and on both sides to some extent moveable.

The whole body presents a condition of firm œdema; both upper and lower limbs, but especially the calves, are much enlarged, and, though fairly hard, will pit on using considerable pressure, but the pitting disappears again on removal of the pressure much more rapidly than in cases of ordinary dropsy. The hands are much thickened and may be fairly described as "spade-like." The feet also are very short, thick, square, and œdematous, and the skin of the sole is somewhat wrinkled. There is also œdema of the face and forehead, and a thick elastic roll in the submental region gives her the appearance known as "double chin."

The tongue is large and cannot be fully protruded; its substance is very hard and almost leathery. The voice is hoarse and croaking and her speech almost impossible to understand. There is very marked lordosis, and this with the enlargement of the limbs gives her somewhat the appearance of a child with pseudo-hypertrophic paralysis; and after being laid on her back she protrudes the buttocks in the act of rising very much as do patients suffering from this disease, but she does not elevate the trunk by climbing up her own lower limbs in the characteristic way.

She can walk fairly well, but is weak on the legs and easily pushed over. There is some curving of the tibiæ which has only recently come on. She rarely moves about the ward, but sits in bed or by the fire all day in a state of perfect contentment, playing with toys, of which, unlike other children, she never tires. She rarely speaks, and never unless addressed, and only answers by a word or two such as "yes" or "no."

She shows very little emotion of any sort. She appears to recognise her mother when she visits her in the ward, but stares stolidly at her and gives no evidence of satisfaction or the reverse.

She seems somewhat insensitive to pain, presenting her finger for blood examination with apparent pleasure.

She sometimes, but not always, passes the urine under her. The urine is clear, sp. gr. 1018, acid, free from albumen.

When first admitted she was very anæmic, but during five weeks' stay in the hospital, under treatment with small doses of tincture of iron, the corpuscles increased from 61 to 78 per cent. of the normal. There is no increase in the white corpuscles, and the red discs readily run into rouleaux.

The circulation is feeble; the nose and extremities become blue in cold weather; and on one occasion a soft systolic apex bruit was audible. The appetite and general health are fairly good; the fundus oculi normal. Temperature 98°.

This case is an example of the condition of sporadic cretinism of which Mr. Curling in 1850, and the late Dr. Hilton Fagge in 1871 recorded cases before the Royal Medical and Chirurgical Society. Similar cases have also been recorded by Dr. Langdon Down,* Dr. Fletcher Beach,† and Dr. Routh.‡ In all ten cases, inclusive of the present one, have been communicated to one or other of the Medical Societies of London since Mr. Curling first commented on the disease.

The symptoms have varied somewhat widely in degree and in mutual association.

In one of Dr. Fagge's cases the disease is said to have been congenital, in others to have first shown itself during early childhood, but in none later than the age of seven or eight years. Eight of the ten cases have occurred in females, only two in males. In all there was defective intelligence, but in some the mental condition was that of complete idiocy with or without deaf mutism; in others there was a fair degree of intellectual power. Thus in one case Dr. Fagge says, "The

* *Trans. of Path Soc.*, vol. xx. † Ibid., vol. xxv.
‡ *Proceedings of Medical Society*, vol. vii.

mental faculties are very good and the patient is fond of reading all sorts of books and converses freely."

In *all* the cases the thyroid appeared to be absent, though it should be mentioned that in one case a post-mortem examination subsequently revealed its presence. The supra-clavicular swellings were present in all the cases, but in Dr Fletcher Beach's case they gradually disappeared while the patient was under observation.

The enlargement of the tongue, the croaking voice and the scanty growth of hair were noticed in some cases but not in others.

The exact symptoms then which are essential to the cretinoid state are rather difficult of definition. It differs from mere idiocy in not being always congenital, and in other symptoms it differs from mere dwarfishness, for there is arrest of mental development in cretinism as well as stunting of bodily growth. The condition seems to consist in arrested bodily growth with degeneration of intellect, associated in most cases with absence of thyroid gland, croaking voice, scanty hair and supra-clavicular swellings.

The present patient is, if I may so call it, a very complete example of the disease; in her all the symptoms which have been distributed among the other recorded cases are strikingly marked. But she presents in addition a condition closely allied to, if not identical with, the disease known as myxœdema.

The hebetude of mind, the general firm œdema of limbs and face, the spade-like hands, the enlarged tongue and scanty hair, with the occasional flushings of the face, combine to form a strong resemblance to this condition.

It is true that the mental state here is somewhat different from that usually observable in myxœdematous adults, but it must be remembered, as Dr. Ord has pointed out, that if the œdema should begin with early life, the nervous symptoms would naturally differ from cases where it was developed at a later period. In the case of the adult there is, as he says, "a sleep or torpor of a central nervous system already built up," but in the child this central nervous system is undeveloped from the beginning and remains, as he describes it, "shapeless." The condition of myxœdema had not at the time when Dr Fagge's paper was read (1871) received the attention which has since been devoted to it, but there can, I think, be no doubt that it was present in three of Dr. Fagge's cases of cretinism.

The engravings which accompany his paper in the 'Transactions,' of the Royal Medical and Chirurgical Society, as

well as his description of the cases are sufficient evidence of this. Thus in one case he speaks of the "hands being short and broad;" in another, "the limbs are short and thick as also are the feet and toes;" in a third case, "the skin of the hands and legs looks as if too large for him." In one of Mr. Curling's cases "the body was thick and the limbs disproportionately large and the tongue swollen;" in another case, "the tongue was protruding." In Dr Routh's case exhibited before the Medical Society last year the œdematous condition was well marked.

It appears then that of the ten recorded cases of cretinism, œdema was present to a greater or less extent in at least six of them; and this result is quite in accordance with the statement of Foderé, quoted by the Sardinian Commission, that "cretinous infants mostly become œdematous."

And in connection with this subject, perhaps I may be allowed to point out the remarkable prescience with which Dr. Fagge almost foretold the disease now known as myxœdema. Reasoning from what he had observed in cases of sporadic cretinism he writes in the paper to which I have already alluded as follows. He says: "It may be interesting to speculate as to what characters would be present should the disease sporadic cretinism (if this be possible) arise still later in the course of adult life. The peculiarities in the form of the cranial and facial bones and in the bony framework generally would then probably be absent, the development of the skeleton being unalterable when once completed. And I think we must conclude the most marked features in such a case would be a coarseness and thickness of the soft parts of the face, especially the lips, and perhaps of the subcutaneous tissues of the hands and feet, besides the presence of supraclavicular tumours and possibly a wasting of the thyroid body." Here the disease myxœdema is anticipated with a remarkable approach to accuracy.

There is then good evidence both here and on the Continent, that myxœdema is not an exceptional association with cretinism, but rather one of the usual symptoms of that condition, present at least as often as some of the other symptoms.

And this clinical association of myxœdema and cretinism is quite in accordance with what we might be led to expect from the result of experimental investigation. It has been demonstrated by Kocher that ablation of the thyroid gland will produce a cretin condition in human beings. Mr. Victor

Horsley's experiments on monkeys have shown that in them at any rate myxœdema, or something much allied to it, follows the same operation.

That the condition of myxœdema then should be associated with other cretin manifestations in the subjects of deficient thyroid is only what might be expected.

Clinical evidence then accords with experimental observation that myxœdema is one of the usual evidences of the cretin state, and I have brought forward this case to-night, not as an exceptional example of sporadic cretinism, but rather as a typical case exhibiting prominently and strikingly the characteristic symptoms of the disease.

As to the causes to which the absence of the thyroid gland may be referable I can offer no suggestion; the cause is as obscure here as in all the other recorded cases. I would only point out that this is the third among ten cases in which there has been a clear history of fright to the mother during pregnancy, and that the conditions which in *this* case at any rate are accountable for the deficiency of the thyroid must be in their nature rather transient and accidental than connected with the constitution of the parents or the locality inhabited, since there are six other children of the marriage all healthy and living under the same conditions as the present patient.

XXXIII.—*A Case of Inguinal Aneurism. Ligature of the External Iliac Artery with two Kangaroo-tail Tendon Ligatures and Division of the Artery between them. Suppuration of the sac. Ultimate recovery.* By W. J. Walsham. *Read April* 24, 1885.

W. F., æt. 33, a strong and muscular man, came under my care on February 4, 1884, for an aneurism in the right groin. For twelve years he had been in the army, but for the last six had worked as a dock labourer. He admitted having been a hard drinker, and having some years previously contracted syphilis. About two months ago he fell whilst carrying a sack of coals and hurt his right groin, and immediately afterwards perceived a small lump in that situation the size of a walnut. This lump gradually grew larger, but he followed his employment till a week before his admission, when he had to give it up on account of the pain and swelling of his limb. On examination a prominent globular pulsating swelling the size of a small cocoa-nut was found in the right groin in the course of the main vessel. It measured five and a half inches in its longitudinal diameter, seven inches in its transverse, and projected two inches above the level of the thigh, while its upper margin extended about an inch above Poupart's ligament. Pulsation was visible over the whole swelling, and was of a forcible and expansile character, but could be stopped, although with some difficulty, by pressure upon the external iliac artery. A loud bruit could also be heard in all parts of the tumour. The right leg and the penis were very œdematous, the right calf measuring two inches more in circumference than the left, and pulsation in the tibials could not be felt. The heart, lungs, and kidneys, it may be added, were examined and found normal. The man was put to bed, the limb raised and bandaged, his diet restricted and potassium iodide in ten-grain doses given three times a day.

On February 7 (three days later) the swelling and œdema of the limb had markedly diminished, but the aneurism itself had clearly increased in size while the pulsation was more forcible and could be felt as far outwards as the great trochanter. The following day (the 8th) I tied the external iliac artery, making my incision a little higher than usual, so as to avoid

the sac of the aneurism which encroached upon the lower part of the vessel. Having exposed the artery and found it not perceptibly dilated, I applied, about one inch from the sac, two strong kangaroo-tail tendon ligatures three eighths of an inch apart, and divided the artery between them with blunt-pointed scissors. Pulsation entirely ceased on tightening the first ligature. The ligatures were then cut off short, a drainage-tube was inserted, and the wound closed by four stout catgut sutures and dressed with antiseptic gauze and absorbent cotton. Owing to an accident with the steam spray apparatus the carbolic spray failed in the middle of the operation.

The limb, which had been previously swathed in cotton wool and flannel bandages, was raised on a pillow. Thirty minims of tinctura opii were given immediately and five minims ordered to be taken every two hours while the patient was awake.

The wound was dressed for the first time on the second day after the operation, and was found to have united by the first intention except at the situation of the drainage-tube. The temperature was 102°, but the patient's condition in all other respects was excellent. From this time he progressed favorably. The temperature rapidly fell to normal, the swelling of the limb disappeared, he slept and ate well, and appeared in every way perfectly comfortable. But the aneurism still continued soft and fluctuating, though slightly smaller in size, and the wound in the situation of the drainage-tube would not close, but remained as a sinus down which a probe could be passed for about two inches. The patient was therefore not allowed to get up, and in this state he continued till March 27th (seven weeks), when his temperature rose from normal to 101°, and the next day, the 28th, to 103°, whilst his pulse was found to have increased from 72 to 130. The wound was then dressed. It appeared healthy, and nothing fresh was noticed in the aneurism. On the evening of the 29th the temperature had fallen to 99·6° and the pulse to 120; but the patient complained that the bandage felt tight, and it was loosened by the house surgeon Mr. Power, who was sure that he had not applied it more tightly than usual at the last dressing.

On the 30th the temperature was 99·4°, the pulse 120.

On my visit on the morning of the 31st I found him with a temperature of 100·4°; a pulse of 120, and a thickly furred tongue but otherwise comfortable. On removing the dressings the aneurism was evidently larger and the thigh swollen, discoloured, and scored by the bandage.

Over the most prominent part of the aneurism the cuticle was raised into a blister the size of a penny-piece by fluid black blood which oozed up through a very minute pin-prick opening in the skin below. The sinus left in the situation of the operation wound looked healthy and was nearly healed. A pad of lint well soaked in collodion was applied over the small hole in the aneurism, the limb bandaged, the patient placed on low diet and ordered to be carefully watched. On April 1 the aneurism was smaller, but the aperture in the sac had increased to the size of half a crown and through it a black clot the size of a walnut was projecting. A probe dipped in a strong solution of perchloride of iron was thrust into the clot in six or eight different places, and the aperture covered with a flat pellet of cotton wool soaked in collodion, the aneurism being left exposed so that should any bleeding occur it might be at once detected by the nurse on guard.

On April 2 there was a slight oozing of brickdust red fluid from under the pellet of collodionised cotton wool, and a probe dipped in perchloride of iron was again thrust into the clot in different directions, and the pad reapplied. On April 3 the sac was evidently sloughing, and a linseed poultice was ordered.

On the 10th the aneurism was level with the thigh and freely suppurating.

On the 17th it was reduced to a healthy granulating sore.

On May 5th it had contracted to a small sinus, and on May 17th the patient was discharged with both wounds soundly healed. The man's allowance of stimulants during his stay in the hospital had been one pint of ale daily, but on the night of his discharge his friends got up a benefit for him and he was drunk for three days or more. The debauch did not appear to do him any physical harm, and when his benefit money was all spent and he had become sober he at once returned to the laborious occupation of a dock labourer, and when last heard of was still continuing that employment in good health.

Remarks.—The aneurism had formed so rapidly, had attained so large a size, its increase was so marked during the few days I had the patient under observation, and its sac was so thin, that I did not hesitate to tie the artery at once without making any attempts to cure it by pressure. There are some surgeons who hold that it is hardly justifiable, as a general rule, to tie the external iliac without previously trying e effects of pressure. Had the aneurism not presented the aracters given above I should probably have followed their

teaching, and subjected the patient to a course of pressure before ligaturing the vessel. But since this case was under my care I have collected all the cases of aneurism of the groin that I could find published since 1870, in the *Lancet, British Medical Journal, Medical Times, Dublin Medical Journal,* and *American Journal of Medical Sciences;* and from a review of them I am inclined to think that pressure should be the exception and ligature the rule.

Of the fifty-eight cases thus collected thirty-seven were treated by pressure, or by pressure and subsequent ligature of the external iliac. But in only ten of these was the pressure successful. In two of the remainder pulsation ceased in the sac; but one of these died nine days after leaving the hospital from the rupture of an aortic aneurism, which Mr. Clutton, under whose care the patient was, seems inclined to attribute to the increased blood-pressure thrown upon the aorta from the use of the Esmarch's bandage. In the other, just as consolidation was thought to be taking place, death occurred from the rupture of an aneurism which had formed just above the spot where the abdominal aorta had been compressed. An Esmarch's bandage had here, also, been applied to the limb. In two others the pressure not only failed to stop the pulsation in the aneurism, but led to the death of the patient, one man dying nine days after the last attempt of pneumonia and exhaustion with the upper lobe of the lung infiltrated with pus, and lymph on the sigmoid flexure of the colon; and the other, seven days after the last compression, of exhaustion with suppuration in the sac and thrombosis of the femoral vein. Both were healthy and well nourished previous to the commencement of the treatment. In the rest (twenty-three) a ligature was applied, but not only in these twenty-three cases did the previous treatment fail to stop the pulsation in the aneurism, but it seems in many to have placed the patient in a worse condition for subsequent ligature, and in some to have been the direct cause of death after the artery was tied. Thus in three the parts were found so matted together that much difficulty was experienced in separating the fascia transversalis from the peritoneum, and the latter structure in two out of the three was in consequence wounded. In one the artery gave way at the seat of ligature and former pressure (Bellingham's), and the patient died of hæmorrhage. In one the parts were found greatly congested at the operation in consequence of the pressure, and the patient died of diffuse cellular inflammation extending upwards as high as the left kidney. In one the sac of the aneurism became

very thin, and in another actually burst whilst under the pressure treatment, (but in this latter case fibrin ferment had also been injected into the sac). In three extensive sloughing of the wound took place. In one sloughing of the skin occurred followed by cicatricial contraction of the parts, rendering the subsequent operation of tying very tedious and difficult; whilst in another the parts were found much swollen and congested at the operation, though the patient ultimately did well. In the remainder, beyond the delay and irritation to the patient from failure, no apparent harm from the pressure treatment ensued.

To sum up: Of the whole thirty-seven cases subjected to pressure ten only were cured; four died apparently as the direct result of the treatment, and twenty-three had to have the artery tied. And of these twenty-three, twelve were placed by the pressure treatment in a worse condition for the ligature, and two actually died, the fatal result being attributable entirely or in great part to the pressure. In face of these facts it seems to me a question whether, as generally taught, pressure ought to be attempted before resorting to the ligature unless, other things being equal, pulsation through the aneurism is very easily controlled, and by very moderate pressure—cases, which would appear to be the exception in aneurism in the groin rather than the rule.

With regard to the operation, as far as I know this is the first instance in which the method of securing an artery with two ligatures and dividing it between them has been applied to the external iliac since, at any rate, its revival in our own times. I have now tied five large arteries in this way without any mishap, and I have seen sixteen others similarly tied by my colleagues at St. Bartholomew's Hospital, all with like success. The advantages claimed for the method are, first, that it diminishes the risk of secondary hæmorrhage by removing the longitudinal tension of the vessel, and ensuring that no part of the artery above the upper, and below the lower ligature is deprived of the nourishment it receives from its sheath; and secondly, that the artery, being divided completely across, there can be no chance of its calibre being restored through the slipping of the knot, the too rapid absorption or giving way of the ligature, or the failure of division of the internal and middle coats. Such a restoration of the artery, as is well known, has now in several instances led to a return of pulsation in the aneurism and as a consequence in some cases to the giving way of the sac and fatal hæmorrhage.

I find that of the thirty-three large arteries tied with one ligature at St. Bartholomew's Hospital since 1870, secondary hæmorrhage has occurred in no less than four cases, whilst of the twenty-one tied with two, with division of the artery, there has been no case of secondary hæmorrhage.

The last point in the case to which I would refer was the suppuration and sloughing of the sac. This did not appear to depend in any way on the method of tying the artery. It occurred seven weeks after the operation, the operation wound being then practically healed. This complication appears to be not very uncommon after ligature of the external iliac for inguinal aneurism. Thus of 153 cases in Norris and Cutter's tables as quoted in Holmes' 'System of Surgery,' three died of this accident which occurred in no less than thirteen instances. In the cases I have collected it also occurred three times. I did not adopt the method I believe generally recommended, of free incision and turning out the clots, as it seemed to me that in so doing there must be considerable risk of disturbing the clots sealing the vessels entering the sac. I find on looking up the published cases where this has been done that hæmorrhage has in several instances ensued. I would therefore submit that it is better to be in no hurry to open the inflamed sac, and turn out the clots, but to promote suppuration and the breaking down of the clots and the formation of firm coagula in the vessels communicating with the sac by some such method as was adopted here, of course taking care that there should be a free exit for the pus when fully formed.

APPENDIX.—37 *Cases of Aneurism in the Groin, in which pressure was employed either alone or was followed by ligature of the External Iliac Artery.*

PRESSURE ONLY.

No.	Reference.	Sex.	Age.	Aneurism.	Artery compressed or tied.	Date of ligature or pressure.	Complications.	Kind of pressure.	Date of discharge.	Result and remarks.
1	Lancet, 1876, i, 596 (Annandale)	M.	37	Femoral, left	External iliac	May 21 and 29	—	Digital, 5 hours, May 21st; 7½ hours, May 29th	—	Complete cure.
2	Lancet, 1876, i, 596 (Annandale)	M.	38	Femoral, right	External iliac	Oct. 3	—	Digital and instrumental, 8½ hours; last three under chloroform	—	Complete cure.
3	Lancet, 1879, ii, 791 (Staples)	M.	39	Ilio-femoral	Common iliac	June 5	—	Esmarch's bandage; Liston's torniquet on common iliac (rapid)	—	Complete cure.
4	Lancet, 1873, ii, 380 (Holt)	—	—	Femoral, 26 inches in circumference	External iliac and common iliac	—	—	Digital and instrumental on common and external iliacs for 52 hours, with intervals of rest; tourniquet	—	Aneurism became diffuse, but was ultimately completely cured.
5	Lancet, 1873, ii, 8 (Elliott)	M.	25	Femoral	External iliac	—	—	Carte's above and below; flexion and pad; then continuous under chloroform	—	Tumefaction and ecchymoses from pad; ultimately complete cure
6	Brit. Med. Jour., 1870 (Russell)	M.	38	Groin, size of clenched fist	External iliac and common iliac	—	—	Digital 24 hours, tourniquet 9 hours; relaxed at intervals under anæsthetic; then tourniquet 5 hours	—	Pulsation ceased, but returned in 16 days; cured after last five hours of tourniquet.

7	Amer. Jour. Med. Sci.; Med.-Chir. Trans., lii, 309 (Hilton)	M.	45	Iliac and popliteal	Common iliac and femoral, below aneurism	—	—	Pressure, common iliac, 6 hours, failed; tourniquet on common iliac and femoral at same time for 10 hours	—	Intense pain and hyperæsthesia of limb; 11 weeks before he could walk down stairs; when last heard of still occasional painful sensations.
8	Med. Times and Gaz., 1879, i, 645 (Couper, reported by Treves)	M.	25	Femoral, left, immediately below Poupart's ligament; large, rapid growth; injury; no syphilis; œdema of limb	External iliac	May 2nd, 5th, and 7th	—	Digital, 40 hours, May 2nd, under morphia; ditto, 25 hours, May 5th; direct (10 lb.), 2 hours, May 7th	—	Pulsation entirely ceased a minute after direct pressure removed
9	Amer. Jour. Med. Sci., 1870, lix, 69 (Gordon Buck)	M.	30	Groin, right; size of hen's egg	External iliac	June 24, 1867	Sloughing of the skin	Instrumental	Sept. 12	Pulsation ceased; cured. Readmitted January 14th, 1869, with return of aneurism; iliac artery could not be felt; cured by flexion.
10	Lancet, 1885, i, 472 (Berry)	M.	40	Ilio-femoral above Poupart's ligament and 2 inches below it; 1 inch in width; injury; no syphilis	—	—	—	Oct. 9th.—Lister's tourniquet for 4½ hours continuously; latter part of time under chloroform. 12th.—Flexion. 13th.—Flexion. 14th.—Lister's tourniquet 3½ hours, with aid of tobacco and chloroform; pulsation entirely ceased	Nov. 3	Circulation returned in limb on 16th; tumour quite firm; no pulsation.—Jan. 27th. Still no pulsation.

No.	Reference.	Sex.	Age.	Aneurism.	Artery compressed or tied.	Date of ligature or pressure.	Complications.	Kind of pressure.	Date of discharge.	Result and remarks.
11	Brit. Med. Jour., 1880, i, 441 (Clutton)	M.	34	Femoral, left; very large; injury; syphilis	External iliac, on brim of pelvis	July 11	—	Esmarch 1 hour; tourniquet, Petit's, 9 hours; digital; 36 hours altogether	Sept. 4, *death*	Pulsation ceased; aneurism shrank, but fluctuated. Left hospital Aug. 26. Died Sept. 4, second aneurism having burst in pericardium. Mr. Clutton asks, "Did Esmarch's bandage do this?"
12	Brit. Med. Jour., 1877, i, 496 (Heath)	—	—	Common femoral	Abdominal aorta	—	—	Tourniquet; Esmarch on limb	*Death*	Consolidation occurring when second aneurism (abdominal aorta) gave way, just above the spot where the pad of the aortic tourniquet had compressed the vessel.
13	Roy. Med. and Chir. Soc. Trans., lii, 317 (Birkett)	M.	41	Femoral, groin; intemperate	Common iliac and femoral below	—	Spot where pressure had been applied infiltrated with inflammatory products and ecchymoses	Tourniquet; pressure 8½ to 10 hours, on three occasions, at intervals of 8 or 9 days	*Death*	Patient died, 9 days subsequently to the last attempt, of pneumonia and exhaustion. Upper lobe of lung infiltrated with pus; lymph on sigmoid flexure of colon; aneurism not consolidated; pulsated as strongly as ever.
14	St. Bartholomew's Statistical Tables	M.	72	Femoral, groin, left; healthy and well nourished	External iliac	June 26	Partial suppuration of sac; tissues much indurated; suppuration	Tourniquet; digital	*Death*	Pressure stopped June 27th. Death July 5th. Thrombosis of left femoral vein.

PRESSURE FOLLOWED BY LIGATURE.

No.	Reference.	Sex.	Age.	Aneurism.	Artery compressed or tied.	Date of ligature or pressure.	Kind of ligature.	Complications.	Previous pressure.	Date of healing of wound.	Date of discharge.	Result and remarks.
15	Brit. Med. Jour., 1880, i, 518; Clin. Trans., 1880, 193 (Clutton)	M.	46	Femoral, right, immediately below Poupart's ligament; size walnut; slight arterial degeneration; no syphilis	External iliac	Jan. 11	Carbolized silk, ends cut short, catgut drain	—	Every variety	Jan. 18	Mar. 15	Aneurism consolidated 5 wks. after operation; wound opened 6 weeks after operation; very slight discharges continued till 4½ months after operation, when ligature separated; wound again healed.
16	Lancet, 1876, ii, 213 (Watson)	M.	30	Femoral, right; size walnut	External iliac	Jan. 19	Carbolized silk, ends cut short; antiseptic; oil-silk drain	Parts matted together; peritoneum wounded	Abdominal compression, Signorimi's, Carte's; aneurism increased under it.	Feb. 10	—	No peritonitis; good recovery.
17	Brit. Med. Jour., 1883, ii, 321 (Southam)	M.	38	Femoral; healthy; drinker	External iliac	Nov. 26	Chromicised catgut; two ligatures close together; artery not divided; antiseptic; drain tube	Aneurism burst after injection of fibrin	Tourniquet, weights; fibrin ferment injected	—	Feb. 26	No tendency to gangrene; wound readily healed; complete cure.
18	Brit. Med. Jour., 1877, ii, 474 (Fleming)	M.	28	Femoral, left, size 3¼ × 3¾ in.; injury; healthy	External iliac	July 9	Silk, ends left out; antiseptic	Parts matted together and ecchymosed from pressure; great difficulty in separating peritoneum	Read's; Esmarch's bandage; aneurism increased under it	—	—	Ligature came away 58th day after operation; complete cure.

No.	Reference.	Sex.	Age.	Aneurism.	Artery compressed or tied.	Date of ligature or pressure.	Kind of ligature.	Complications.	Previous pressure.	Date of healing of wound.	Date of discharge.	Result and remarks.
19	Lancet, 1880, ii, 300 (Heath)	M.	45	Superficial, femoral, left, 2½ in. below Poupart's; very large, 15½ in.; 4 months' duration; injury	External iliac	Jan. 16	Silk, ends cut short; antiseptic; horsehair drain	Carboluria from 17th to 22nd; gangrene two inner toes on Feb. 29th; suppuration of sac?	Shot bag	Feb. 8	—	On Feb. 16 throbbing pain fluctuation, and some discoloration of aneurism; temp. 101°; sac laid open; no pus; blood-clot and fluid blood; tube and antiseptics. Feb. 25. — Great gush of blood; tourniquet over femoral for 2 hrs.; hæmorrhage not repeated. Wound healed May 1st.
20	Lancet, 1870, i, 729 (McFarlane)	M.	30	Groin; healthy	External iliac	Jan. 26	Hemp; carbolic dressings	—	Pressure of all kinds	—	—	Ligature came away Feb. 20th; upper 3rd, wound healed first intention; cure.
21	Lancet, 1872, i, 148 (Terry)	M.	36	Femoral, right, reached above Poupart's ligament, size hen's egg	External iliac	Aug. 25	Silk	—	Various forms	Oct. 12	—	Ligature removed 32nd day after operation.
22	Brit. Med. Jour., 1879, ii, 693 (Curgenven)	M.	24	Superficial femoral, right; injury; had lasted 4 months	External iliac	Aug. 1	Silk	—	Lister's	—	—	Ligature came away 20th day; wound healed first intention; no recurrence of pulsation.

23	Lancet, 1874, i, 873 (Johnson Smith)	M.	27	Femoral, right, 4 × 3¾ in.; syphilis, bubo, otherwise healthy	External iliac, middle third	Mar. 18	Smooth twine	Epigastric artery wounded in ligature; much sloughing	Flexion; Carte's, but caused great pain and restlessness; flexion from March 3rd to March 10th; Carte's 1 hour; Read's 15 hours; Carte's 20 min.	May 1	June 1	Ligature came away April 2nd; wound nearly healed. April 6th, wound opened and sloughed.
24	Lancet, 1874, ii, 118 (Sanders)	M.	55	Femoral, size of two fists; injury	External iliac, lower third	Feb. 1	Silk	—	Flexion for hour or two every day for 5 or 6 days	—	Apr. 24	Considerable suppuration of wound and burrowing of pus. Ligature separated Feb. 25th.
25	Lancet, 1874, i, 509 (Diver)	M.	35	Femoral, size of large walnut, had lasted 5 mos.; also aneurism of popliteal, size of child's head; healthy	External iliac	Jan. 27	Silk	In 48 hours gangrene came on in lower third of leg; amputated 21st day after operation	Tourniquet for 48 hours	—	—	Ligature came away 28th day after operation.
26	Lancet, 1875, i, 269 (Bell)	M.	34	Femoral; healthy	External iliac	—	Hemp	—	Canister and shot; aneurism increased in size, and walls became thin.	—	—	
27	Lancet, 1871, ii, 191 (Lund)	M.	42	Femoral, left, high up	External iliac	—	Catgut, ends cut short; antiseptic; lac plaster dressings	Operation difficult on account of adhesion of tissue to sheath	14 mos. before pressure for 30 hours, followed by slough, which left a white cicatrix	—	—	Healed well, except where wound dipped into cicatricial tissue.

No.	Reference.	Sex.	Age.	Aneurism.	Artery compressed or tied.	Date of ligature or pressure.	Kind of ligature.	Complications.	Previous pressure.	Date of healing of wound.	Date of discharge.	Result and remarks.
28	Lancet, 1873, ii, 7 (Elliot)	M.	32	Femoral; spare; syphilis	External iliac	Aug. 20	Carbolized catgut, trebly knotted, ends cut short, antiseptic	Pulsation returned in 5 days. On Nov. 26 artery again tied with double-twisted hempen ligature antiseptically; no trace of catgut seen	Tourniquet, digital; acute flexion before first operation; after failure of first operation pressure on sac by air-pad (12 lb.) and on femoral below	On separation of ligature	Apr. 2	Ligature separated after second operation on Jan. 24; artery appeared perfectly sound. Second operation very difficult; peritoneum opened; intestine escaped.
29	Brit. Med. Jour., 1883, ii, 1131 (Rose)	M.	—	Femoral; injury; no syphilis	External iliac	Feb. 18	Antiseptic	—	Digital 24 hours	Mar. 10	May 15	Aneurism much smaller, but slight pulsation; subsequently recurred and dissected out.
30	Med. Times and Gaz., 1873, i, 140 (McCormac)	M.	28	Groin, size large orange	External iliac	Oct. 16	Carbolized catgut, spray	Suppuration of wound copious; piece of aponeurosis of external oblique came away; suppuration extensive throughout	Digital 12 hours, Oct. 10th; flexion, Oct. 11th; aneurism larger	—	Jan. 6	Complete cure; up on Dec. 16th.
31	Lancet, 1878, ii, 546 (Pinkerton)	M.	40	Inguinal, left; lasted 6 months; injury; no syphilis	External iliac	Oct. 2	Catgut, ends left out; carbolic dressings	Parts matted together from pressure; peritoneum wounded	Carte's and digital from July 22nd to Sept. 9th	—	—	Upon Oct. 22nd pressure had caused a depression in walls of abdomen.

32	Brit. Med. Jour., 1879, ii, 713 (Barron)	—	—	Femoral	External iliac	—	Carbolized catgut; Lister's precautions	—	Tourniquet, digital	—	—	Very rapid recovery; temp. never rose more than a degree above normal.
33	Lancet, 1875, i, 12 (Bryant)	M.	45	Femoral; healthy; no injury	External iliac	Jan. 30	Carbolized catgut; no drain	—	Weight	—	May 14	Cure.
34	Lancet, 1876, i, 775 (Sydney Jones)	M.	30	Ilio-femoral, right; had lasted a month; healthy	External iliac	Oct. 6	Carbolized catgut; ends left out; almond oil and cotton wool	2 oz. clotted blood, Oct. 15th; oozing continued throughout day, 16th; discharge tinged with blood, 22nd and Nov. 1st	Flexion	Nov. 6	Dec. 20	Ends of ligature came away Oct. 14th.
35	Lancet, 1875, i, 47 (Bryant)	M.	28	Femoral, size duck's egg; injury; healthy	External iliac	Apr. 28	Carbolized catgut; no drain	—	Flexion, pressure, ice; parts swollen and sore	—	—	Ultimately did well.
36	Brit. Med. Jour., 1873, i, 199 (White)	M.	34	Inguinal; injury	External iliac	May 28	Carbolized catgut; carbolic oil dressing	Hæmorrhage, June 4; death in two hours	Bellingham's method (14 lb.) for a month	—	*Death*, June 4	Discharged June 2. Secondary hæmorrhage June 4; wound plugged, but died in two hours; artery had given way at seat of ligature.
37	Brit. Med. Jour., 1875, ii, 413 (Elliott)	M.	37	Femoral, left, 6×4 in., pushing up Poupart's ligament; cicatrix of bubo over it; syphilis	External iliac	June 12	Hemp; carbolic acid dressings	Tissues found at operation greatly congested by pressure	Digital, flexion; tourniquet, Carte's; pressure begun on June 7th, and continued on and off for five days	—	*Death*, June 15	Diffuse cellular inflammation extending upwards as far as left kidney.

XXXIV.—*A Case of Obstruction of Arteries and Veins extending over many years.* By W. B. Hadden, M.D. *Read May* 8, 1885.

THE patient is a healthy-looking man, æt. 51, formerly a commercial traveller. He has been married many years and has had two children, who are alive and healthy. His wife has had some premature confinements. His paternal grandfather and grandmother had gout. His father died at forty-two of heart disease; and his mother at seventy, of bronchitis; a brother died of heart disease, and had varicose veins for many years. The patient denies syphilis and alcoholic excess.

In 1862 he had his first illness since childhood. From his description it appears to have been renal colic, although he had no hæmaturia.

The disease from which he now suffers dates from 1868, when he was suddenly seized with a sharp pain in the left foot and heel. He was said at the time to be suffering from rheumatic gout. Phlebitis of the same leg followed and mortification was feared. The left leg was swollen and tender up to the groin. He was laid up for about five months.

In 1874 he injured his leg, and to this cause he ascribes the pigmented scars to be mentioned presently.

In 1880 he came under the care of Dr. Mitchell Bruce at Charing Cross Hospital, and remained under his observation for three or four years. I have to thank Dr. Bruce for most of the details in the history of the patient before he came under my notice.

When first seen by Dr. Bruce he had obstruction of the right brachial artery. The attack began suddenly with cramp in the fingers, weakness of the hand, and pain in the upper brachial region. To the patient the fingers appeared dead, numb, and useless, and the limb swollen, although it was visibly flabby and shrunken. The pulse at the radial artery was small. The heart was irregular. The man states that the nails became black at this time, but did not come away.

During improvement the right radial and ulnar veins

became painful, converted into solid cords, and evidently thrombosed. In August, 1881, that is, about eighteen months after he was first seen by Dr. Bruce, he was returning home one day when he suddenly dropped a bag he was carrying in his left hand. He was confused, but was not convulsed and did not fall.

The left leg was not affected, but the doctor who then saw him thought that the face was drawn. The patient says he had no pain in the arm at this time. He was put to bed at once and was unconscious for some days, not knowing anybody. A complete recovery seems to have followed.

Between 1881 and 1884 he had attacks of arteritis or phlebitis, according to his medical attendant, but of the details I am ignorant. Between June and November, 1884, he is stated to have had phlebitis of both femoral veins, and also of the veins of the right buttock and pubes.

It was at this time the man first came under my notice, as an applicant for an out-patient letter at St. Thomas's Hospital. I then thought that his case resembled very closely one that I described before this Society last year as obliterative arteritis. I only made a casual examination as the man refused to come into the hospital and went away without treatment. I did not lose sight of him, however, and on February 2nd of this year he was admitted into St. Thomas's Hospital under Dr. Stone, who kindly allows me to use the case.

He complained of pain in the right groin. On examination there was tenderness in this position and feeble pulsation of the right femoral artery. No beat could be felt in the right popliteal and posterior tibial arteries, but the dorsal artery of the foot was pulsating strongly. The right internal saphenous vein was thickened, cord-like, and evidently thrombosed. There was very slight œdema over the dorsum of the foot and some enlargement of the superficial veins.

There was no pulsation whatever in the right brachial artery, which was thickened, but it was good in the third part of the subclavian. At the back of the arm, just above the external condyle, there was a strongly pulsating collateral branch. The pulse at the right radial was very feeble, and that at the ulnar could not be detected at all. There was no marked enlargement or thickening of the superficial veins. The right hand was cold and blue, and the grasp was not so good as that of the left hand. On the left side the femoral artery and vein were unaffected, but there was some dilatation of the superficial veins.

There was nothing abnormal in the vessels of the left upper extremity.

Just above the pubes the superficial veins were slightly prominent. There was no enlargement of veins over the buttocks. On the front of both legs, and on the inner and outer sides of both ankles, there were pigmented scars, some of which were large, others small and outlying. Those on the right side he ascribed to injury when a boy, but he confessed to ulcers of the left leg ten years ago. He indignantly denied the imputation of acquired syphilis, stating that if he had had syphilis it must be hereditary.

There was no hypertrophy of heart, and no bruit, but the rhythm was irregular. There was no sign of aneurism. The urine was free from albumen whenever examined.

For about six weeks after admission the pain in the right groin continued, but no fresh obstruction in the course of either artery or vein was noticed.

Three weeks after admission he was suddenly seized with a rigor, and his temperature, which had been previously normal, rose to 103·8°. At this time he had headache and tenderness limited to the right frontal and parietal regions. He complained too of sorethroat and the tonsils were red and inflamed. On one occasion he was slightly delirious at night. The attack passed off without further mischief. About a fortnight later he had a recurrence of the headache, which was localised, as before, to the right side. Since his admission he has had occasional attacks of diarrhœa, the explanation of which was not altogether clear.

The treatment adopted consisted mainly in the administration of mercury and iodide of potassium, and of hot soda baths on alternate days. At the same time he was confined to bed.

At the present moment he is free from pain and tenderness and says that he is better than he has been for two years. I am not prepared to say what share the anti-syphilitic treatment has had in bringing about the improvement.

As to the cause and nature of this affection I am much perplexed.

Is it gouty? His paternal grandparents were said to have suffered from this disease, but I am far from satisfied that the sharp pain in the left foot which occurred in 1868, and which was clearly the beginning of his present affection, was really gout. It is quite possible, probable I think, that it was phlebitis.

The question arises whether the disease is syphilitic. In spite of the patient's emphatic denial the scars on his legs are highly suggestive. There is, however, no evidence of old disease in the throat, eyes, or testicles. Whatever may be the constitutional taint or state underlying this affection its pathological nature is by no means clear.

The sudden obstruction in the right brachial artery suggests embolism, and this assumption is, to some extent, supported by the condition of his heart, which was irregular. But although irregular there was no hypertrophy and no bruit. In addition there was no history of arterial obstruction in the viscera, with the exception of a suspicion of a cerebral block giving rise to loss of power of the left arm.

Two explanations may be put forward.

First, there may be a twofold agency, embolism affecting the arteries, and phlebitis. Secondly, we may assume a common cause, an inflammatory condition involving the coats of both arteries and veins. This hypothesis has an *a priori* probability. In its favour may be urged the absence of a cardiac bruit, the relation in time, on one occasion, between the arterial and venous obstruction, and the prolonged tenderness in the course of the right femoral vessels without corresponding obstruction.

XXXV.—*Four Cases of Osteitis Deformans.* By JOHN R. LUNN. *Read May* 8, 1885.

IN the *St. Thomas's Hospital Reports* for 1883, vol. xiii, I have given a full account of a case of osteitis deformans which came under my care in the new St. Marylebone Infirmary. In that article I gave a description of the clinical features and physical changes during life and a report of the condition found after death, including the minute changes in the bone as seen under the microscope.

Having four other cases under my care at the present time I thought it would be interesting to bring them to the notice of the Clinical Society. The four cases to be shown this evening, I think, illustrate very well the changes which take place during life in the different stages of the disease. The admirable description of the disease by Sir James Paget in the sixty-fifth volume of the *Medico-Chirurgical Transactions*, so fully agrees with my own cases, that I need not weary the Society with the details of the clinical histories of the cases shown to-night. Since 1876, when Sir James Paget drew attention to the disease, numerous other cases have been recorded, and evidence goes to prove that the disease is more ancient and of more common occurrence than was at first supposed. The reason why it was so long unnoticed is the fact that the disease occurs late in life and rarely gives rise to symptoms requiring active medical treatment, death usually being due to some intercurrent disease. The majority of cases recorded have either occurred in public institutions or been discovered accidentally.

The chief symptoms noticed by the patient are pains of a rheumatic character in the affected limbs, loss of height, and in male patients increased size of the head, rendering the hat too small.

The facts which strike the eye of the observer are the large size of the head, prominent eyebrows and chin, drooping position of the head, the chin resting on the sternum or approaching it, the curvature of the long bones of the lower limbs, and the curvature of the spinal column. I believe these conditions are due to the fact that at some previous time the bones of support have lost their normal strength or solidity.

Fig. 1. Case II. H.S. æt. 68

Fig. 2. Case IV. M.W. æt. 70.

Fig. 3. Case I. C.S. æt. 75

I have drawn my conclusions from an extended observation of the disease during life, the conditions found after death, and the changes which are shown to have taken place on examination of the bones by the microscope. If we look at the bones themselves to see how to account for the weakness and curvature, we find a definite change in their structure sufficient, I think, to explain this yielding, in fact a great part of the natural firm bone has disappeared, and in its place is a large quantity of porous and spongy bone. In my fatal cases the morbid process was nearly universal, but its chief stress seems to have fallen upon the bones about the main axis of the body, the cranial vault, spine, pelvis and long bones of the lower limbs being most damaged, the face, fingers, and feet least. The absorption and disappearance of the original bone seems to have been the primary event in the course of the disease, and this must have taken place not by any coarse process, but by very delicate interstitial changes. The latter had affected not only the compact tissue of the shafts, but also the cancellous tissue of the extremities of the bones.

The formation of the porous bone must be regarded as secondary, but its presence is difficult to explain. As to the nature of the morbid process which produces the anatomical alteration, the view advanced by Sir James Paget is that it is a kind of chronic osteitis, which I believe has been generally accepted. But while fully admitting that chronic inflammation may have some share in the process, I scarcely can think that it altogether accounts for the changes found after death. The conclusions I have arrived at are that osteitis deformans consists of:

1. A constitutional disease producing atrophy and absorption of a large part of the osseous systems.
2. Consequent weakening of the bones, so that they yield when exposed to strain.
3. Compensatory strengthening by the growth of what may be looked upon as a variety of callus.
4. The occasional formation of definite tumours.
5. A fatal cachexia.

Case 1 (male). Pl. XI, fig. 3.—C. S., æt. 75, a coachman by trade, married twice, but has had no children. He was admitted May 10, 1884. Father dead, cause unknown; mother was a lunatic in Glasgow Asylum for many years before she died; one brother, who was quite well when he was last seen. The patient had been in the workhouse for fourteen years. He

was in Hanwell Lunatic Asylum seven years ago, for four years. Never had any serious illness, but states he suffered a good deal with his brain.

It is impossible to get a trustworthy history of him. He has had one or two attacks of gout whilst a patient here. The patient is very quiet and reserved in his manner and slow in his movements. He has the typical aspect of a case of "osteitis deformans;" he walks with his chin almost touching his sternum and has some difficulty in turning his head to either shoulder. His shoulders are raised and thrown forward, he stands with legs apart, his fingers are nearly on the level with his knees, his head looks too heavy for his body; his chest small and square-shaped and does not move well. On breathing the costo-sternal cartilages appear ossified and his posture is similar to the photographs in the sixtieth volume of the Royal Medical and Chirurgical Society's *Transactions*.

The head measures twenty-three inches in circumference at the widest portion, appears enlarged and bossy, the supra-orbital arches look enlarged and stand out boldly, the facial bones appear free from disease at present. Both femora enormously enlarged and massive at their lower half, curved outwards and forwards. Both tibiæ appear in the same condition, but the right is more advanced than the left; the right leg measures thirteen and a half inches in circumference, the left thirteen inches. No ankylosis of knee- and ankle-joints; the bones of the feet appear free from disease at present. Both humeri more thickened than natural, but not much curved. The left radius and ulna thickened and curved with the convexity on the dorsal aspect; hands free. Some ankylosis of both shoulder-joints, and inability to raise the arm above the head.

Both clavicles appear enlarged and thickened, and some irregularity at the acromial end of the left one, viz. old fracture. The pelvis feels broader than natural and both crests massive and thickened.

All the ribs feel thickened and larger than natural; intercostal spaces small. Viscera appear normal, urine contains no albumen; a small fatty tumour on the back of the neck.

Case 2 (male). Pl. XI, fig. 1.—H. S., æt. 68, married, a labourer, was admitted into the Marylebone Infirmary, July, 1884, with an ulcer of leg. Father died at the age of seventy; mother died at ninety, causes unknown; one brother subject to gout, another said to have died of cancer in Paddington Infir-

mary, fourteen years ago. (I am not able to find any notes of this case there.)

The patient has always enjoyed good health, with the exception of a winter cough, and a bubo which he had thirty years ago (scars still in the left groin). He thinks his hat has got too small of late, suffers from pains of a rheumatic character in both legs, generally worse at nights. The last eighteen months he noticed his legs curving and thinks he is getting shorter; his height at present is five feet five and a half inches. Enlarged glands in both groins. Superficial and deep reflexes normal. No anæsthesia, walks with his head inclined forwards and downwards, his chest seems small and square shaped, his arms hang low. His shoulders are raised and the upper dorsal vertebræ appear curved and rigid, giving him the appearance of having an angular curvature of his spine. Both femora curved forwards and outwards and thickened at the lower half. When he stands with his feet touching, the distance between the two internal condyles is three and a quarter inches. Both patellæ normal. Both tibiæ seem enlarged and their anterior borders rounded. The right measures fourteen and a quarter inches, the left fourteen inches. Feet quite free at present. No ankylosis of hip-, knee-, or ankle-joints. Neither arms nor forearms seem affected. The clavicles seem thicker and more massive than natural. The head appears enlarged at every part and measures at the widest circumference twenty-two inches. The orbital arches look larger than natural, bones of the face appear free from disease. No disease of viscera with the exception of bronchitis; urine normal. Ectropion of both lower lids. Since examining the above case I have seen his brother, James S—, aged 68, who tells me that he has noticed his own head getting larger and his hat seems too tight for his head. I find both femora enlarged in their lower halves, and the right clavicle is decidedly larger than the left. Now and then suffers from attacks of gout.

Case 3 (female).—S. S., æt. 53, a woman, married, admitted into St. Marylebone Infirmary for rheumatism; father and mother both dead; causes unknown; has had four children, one daughter still living and quite well. No miscarriages or difficult confinements. No history of gout, consumption, syphilis, or cancer in the family. Always had excellent health until ten years ago, when she says she was frightened by the Regent's Park explosion, and was struck in the back by a Venetian blind; she states that whilst in bed she has

grown shorter, also her legs have gradually become bent and thighs thicker. They have been crossed for four years, and she has been confined to bed for the last twelve months, and is rapidly becoming more helpless, bent, and in pain.

The patient is just able to stand with her legs crossed, the left over the right; her chin inclines towards her suprasternal notch, she is not able to rotate her head from side to side. Her trunk seems very short, there seems a good deal of tenderness over the lower dorsal vertebræ, and over the right arm, where pressure is made. She thinks her head is larger than it ought to be. Both femora are curved and thickened, ankylosis of both hip- and knee-joints (the latter only I think is due to being confined in bed); generally lies with her left foot out of bed. Both clavicles enlarged and thickened, the right seems larger than the left. The right humerus feels thickened and solid, curved on its posterior surface (the patient generally rests on her right arm, the circumference of which is nine inches). The left humerus not so much affected. The right elbow-joint is stiff, and gives the patient a good deal of pain when moved; both radii and ulnæ curved in their posterior surfaces. Right hand and wrist stiff, and skin over hand and fingers very glossy and tense; left hand seems free from disease. The head measures twenty-three inches in circumference at the widest part and looks too heavy for her body, and feels bossy over the parietal regions. The chest is very square-shaped, and the last ribs seem nearly to touch the crest of the ilium. The ribs appear broadened and nearly touching each other, costal cartilages ossified; the pelvis much wider than normal and both crests of the ilia appear thickened and massive. The patient weighs 7 st. 4 lb. Urine normal. Slight bronchitis, liver slightly displaced and other viscera normal.

Case 4 (female). Pl. XI, fig. 2.—M. W., æt. 70, married, was admitted September 20th, 1884, into St. Marylebone Infirmary with bronchitis. Granular lids, and the right eye destroyed from old inflammation, commencing cataract in the left eye with some old choroiditis, *i.e.* senile. My attention was drawn to the condition of her legs by the nurse of the ward, as she seemed to resemble the patient (Case No. 3) in the next bed, who was suffering from osteitis deformans. The patient has had four children, two sons and two daughters (one of whom has been a patient here with diphtheria, and who seemed rather weak-minded); no history of insanity, syphilis, or cancer.

The last two years she has noticed her legs becoming gradually bent and has suffered from shooting pains, which she attributed to rheumatism. Her attitude resembles the two male patients, Cases 1 and 2. Her chin inclines towards the sternum, and her head looks too large for her body; shoulders are raised and thrown forwards; lies in bed with her legs crossed, the right resting on the left. Her chest is square-shaped and moves a little on respiration; the ribs seem larger and broader than natural. Both clavicles feel massive and enlarged; the bones of the upper extremity are not much affected yet. No ankylosis of shoulder- or elbow-joints. Both femora are bent, but not so much thickened as Cases 1, 2, and 3. Head measures twenty-two inches in circumference; both tibiæ are bent and not much thickened, their anterior borders feel rounded, feet free from disease. Both patellæ normal and movable; the pelvis seems thickened and rounded. Bones of the face appear normal at present; urine contains no albumen or sugar; bronchitis signs in chest.

Case 5.—Since preparing my paper the brother of Case No. 2 has come under my care for gout, from which he has suffered a good deal at times.

J. S., æt. 65, married, no children, no history of syphilis. Has been always a hard worker, but lately he has noticed his hat getting too tight, but has never noticed his legs curving. The right femur (lower half) seems larger and more massive than natural. Both clavicles feel enormous: chest square-shaped and ribs appear broader than natural. His head looks large and his supra-orbital arches stand out prominently. He cannot move his head well from side to side. Feet and upper extremities appear free at present.

XXXVI.—*A Case of Hæmoptysis treated by the Induction of Pneumothorax so as to Collapse the Lung.*

By W. Cayley, M.D. *Read May* 8, 1885.

FREDERICK W., æt. 21, a porter, was admitted into the Middlesex Hospital on February 9, 1885.

Family history.—Father and other relatives suffer from rheumatism. A cousin on the father's side died of consumption. Mother dead, but he does not know from what disease. Has six sisters who are all alive and well.

Previous history.—Had rheumatic fever at the age of 11, and has been liable to rheumatic pains ever since, but has otherwise been strong and healthy. Had another attack of rheumatic fever last winter, and was an in-patient in this hospital from December 18 to December 29. Nothing wrong was noticed with his lungs while in the hospital, and there was no cardiac complication. He has since suffered from a slight cough, but his general health has been good.

Present attack.—On February 7 he was at work in his usual health. At nine in the evening he had an attack of hæmoptysis, which recurred the following norning, and again in the night of the 8th. He estimated the whole quantity which he brought up as about a pint.

State on admission.—Patient was a pale-faced young man, well nourished and of good muscular development, pulse 96, resp. 18, temp. 100°. He was quite free from malaise, tongue clean, appetite good, breathing tranquil. The expansion of the left side of the chest was very deficient, and the breath-sounds in front were extremely feeble; percussion was not attempted. Heart-sounds were normal.

Shortly after his admission he coughed up about 2 oz. of florid blood.

He was ordered to keep the recumbent posture, ice was applied to the left front, and he was ordered gr. x of gallic acid every three hours.

At 11.30 P.M. he coughed up about 2 oz. of florid blood and some clots. He was given a grain of ergotine subcutaneously.

February 10.—Slept well, is free from malaise, has a slight cough and spits transparent bronchial mucus containing pellets of blood. Expansion of left side of chest extremely deficient, and breath-sounds very weak. Over the scapular region there is dulness on percussion and feeble bronchial breathing. These signs were attributed to the lung being clogged with blood.

At 2.30 P.M. he coughed up 4 oz. of bright red blood, and at 9 P.M. 6 oz. more, and he continued to spit blood in small quantities during the night. Two hypodermic injections of ergotine were administered, and a sixth of a grain of morphia.

February 11.—Pulse 84, resp. 18, tranquil, slept fairly, crepitant râles, attributed to blood in the bronchial tubes, are audible all over the left back. At 1 P.M. he coughed up 8 oz. of florid blood. Gr. j of ergotine was administered subcutaneously. Ordered ℥. x of Ol. Terebinth. every four hours.

February 12.—Has brought up no large quantity of blood since yesterday afternoon. Expectoration is viscid and blood-stained. Last night, and again this morning, had retention of urine necessitating the use of the catheter. Urine turbid with lithates, not albuminous, slept well, feels comfortable. Pulse 80.

At 8 P.M. he brought up 8 oz. of blood, and at midnight 6 oz. After each was given a hypodermic injection of ergotine.

February 13.—Pulse 72, resp. 20, tranquil, slept fairly well, but required the catheter. Has brought up during the night about an ounce of viscid blood-stained mucus, has no malaise, but has become very anæmic. During the day he coughed up 16 oz. of florid blood.

February 14.—At 5 A.M. he brought up 6 oz. of blood. Sputa still viscid and blood-stained. Pulse 84, dicrotic.

Expansion of left side continues very deficient, breath-sounds very feeble, and accompanied, both back and front, with moist râles, dulness, and feeble bronchial breathing over scapular region.

Ordered gr. x of pot. iodide with ℥. x of Ol. Terebinth., three times daily.

At 3 P.M. brought up 8 oz. of blood. Ergotine gr. j was administered subcutaneously.

February 15.—Pulse 112, dicrotic; resp. 20, tranquil; passes urine naturally.

At 10.30 P.M. brought up 9 oz. of blood.

February 16.—Pulse 100, weak and dicrotic; resp. 28,

tranquil. Is much blanched, complains of feeling of weakness, is thirsty and has a sensation of tightness across the sternum. Cough less troublesome, sputa continue viscid and blood-stained. Urine turbid with lithates, sp. gr. 1030.

Ordered infusion of digitalis, 2 drachms, three times daily; ergotine to be discontinued.

February 17.—Brought up 6 oz. of blood in the night. Pulse 104, resp. 32. Condition much the same.

February 18.—Has brought up 4 oz. of blood.

February 19.—Had an attack of profuse hæmoptysis in the night, bringing up 24 oz. of florid blood. Is much blanched and very weak. Pulse 108, dicrotic; resp. 18, urine, sp. gr. 1035, loaded with lithates.

February 20.—Continues much in the same state, has had no hæmoptysis, but the sputa continue blood-stained, of a somewhat brownish colour. Ordered ℥. xx of Liq. Ferri. Pernitrat. three times daily.

Patient now had no return of the hæmoptysis till the 26th, and during this time his general condition decidedly improved, though, as will be seen by the chart, he had slight evening febrile exacerbations. The anæmia became less marked, the pulse of better quality, he took his food well, and expressed himself as feeling much better, the expectoration continued blood-stained and the physical signs remained much the same.

At 8 A.M., February 26, he had another attack of hæmoptysis, bringing up 12 oz. of florid blood. There was no return till March 2, when, at 2.30 P.M., he again had a profuse attack, bringing up 32 oz. The blood came up in gushes, and he seemed in great danger of being suffocated. The blood was bright red and frothy, and rapidly coagulated. After this he was much exhausted and was again extremely blanched.

March 3.—Pulse 112, dicrotic; resp. 24, tranquil. Is in much the same condition.

March 4.—Pulse 96, dicrotic; resp. 20. Expectoration more profuse; it presents the same blood-stained character. Complains of pain in the second left intercostal space above the nipple. At 11 P.M. he brought up 12 oz. of blood.

March 5.—Pulse 92. Pain in chest subsided after the hæmoptysis.

March 6.—Brought up 3 oz. of blood in the night.

March 7.—No return of the hæmoptysis.

March 8.—No return of the hæmoptysis, feels better. At 4.30 P.M. brought up 10 oz. of blood.

March 9.—At 9.30 A.M. brought up 8 oz. of blood, is very

prostrate and anæmic. Pulse 104, small and dicrotic; resp. 36, laboured. Scarcely any expansion of left side of chest, coarse râles heard all over left front and axillary region, and now numerous moist râles, finer than on the left side, are audible over the right front and axilla. It was not considered safe to raise him in order to examine the back.

March 10.—Patient had another attack of hæmoptysis this morning, bringing up 4 oz. His condition was now one of great prostration. He was much blanched, pulse 96, very small; resp. 28, temp. 98·6°, and it was evident that he would soon succumb to repetition of the hæmoptysis, the source of which was considered to be in all probability either a pulmonary aneurysm or the ulceration of an artery of some size in the left lung. He had brought up, since his admission, a gallon of blood besides a large quantity of blood-stained sputum.

The moist râles which had become audible first over the left, then over the right lung were attributed to the presence of blood in the bronchial tubes. Acute tuberculosis was considered as excluded by the absence of emaciation, the slight amount of general constitutional disturbance, the tranquil breathing, and the very moderate degree of fever.

It appeared to me that if the left lung could be collapsed, the consequent great diminution of the circulation through it would afford a fair prospect of arresting the hæmorrhage. It would, moreover, probably check, at any rate for a time, the development of tubercle in this lung, supposing such to be in progress. The extreme anæmia of the patient would, I thought, render this proceeding less dangerous, as less oxygen would be required for the aëration of the blood, and he would therefore be better able to support the collapse of the lung.

Accordingly, at 6 P.M. on the 10th, chloroform was administered, and Mr. Hulke made an incision over the sixth intercostal space, an inch behind the anterior axillary fold, and opened the pleural cavity, and then introduced a double tube made by uniting two pieces of elastic catheter about 3 inches long by means of a shield; the opening was protected by a small cage lined with carbolic gauze, and the whole covered by a carbolized gauze bandage. The operation was performed under the carbolic spray with all the usual antiseptic precautions. The air passed freely in and out of the tube and the apex of the heart became displaced to the mid-sternal region. The respirations at the same time became much accelerated.

In the night following the operation he twice brought up blood, 4 oz. and 2 oz.

March 11.—Has slept well after a hypodermic injection of morphia. Pulse 112, resp. 48, jerky in character. There is tympanitic resonance over the left front.

March 12.—Pulse 128, resp. 44. Tympanitic note over left front less marked, and breath-sounds more audible. The tube was found to be completely blocked with fibrinous material; it was accordingly removed and a silver tube, in form like a flattened tracheotomy tube, was introduced in its place. The air now passed in and out freely, the resonance became markedly tympanitic, and a bell-sound could be elicited. The breathing over the left front became inaudible.

After the introduction of the silver tube he complained of much pain, which was relieved by a hypodermic injection of morphia. His temperature rose to 104·6°, and much serous fluid began to flow through the tube.

March 13.—At 10 P.M. last night the silver tube was removed. Much serous fluid had flowed through the wound. He passed a good night, but is very prostrate this morning. Pulse 128, resp. 52. Sputa less abundant, continue to be glairy and blood-stained. Physical signs unaltered.

March 14.—Pulse 120, resp. 40. Slept pretty well after morphia, but sweated a good deal. Serous fluid continues to ooze from the wound, but in smaller quantities. Physical signs unchanged.

March 15.—Pulse 124, very feeble; resp. 44, irregular and jerky. This morning had a severe attack of dyspnœa which was relieved by a hypodermic injection of morphia. There is still tympanitic resonance and absence of breath-sounds over the left front. The apex-beat can be felt at the fourth interspace, immediately to the right of the sternum. Abundant crepitation is present over the right front. Serous fluid continues to ooze from the wound. At 3.45 P.M. he died quite suddenly, having previously seemed rather better.

Post-mortem examination.—There were firm fibrous adhesions at the left apex which extended down the posterior border of the upper lobe to below the interlobular fissure. The anterior extremity of the lower lobe was also adherent to the parietal pleura. This had evidently prevented the complete collapse of the lower lobe towards the spine. The collapse extended over the greater part of the lower lobe and the lower part of the upper one. The pleural surfaces were in a state of inflammation and covered with recently effused lymph, but there were only two drachms of non-purulent fluid in the cavity. There was a free opening into the pleural

sac, which would admit the finger, through the left sixth interspace.

The left upper lobe was partially divided by a fissure into two lobes. In the extreme apex was a small cavity with smooth walls round which the lung was puckered. The whole lung, except quite at the base, was thickly studded with grey miliary granules.

In the lower part of the upper lobe, two inches from its anterior extremity and abutting on the interlobular septum, which was bulged downwards by it, was a cavity the size of a walnut, filled, except at its centre, by concentric layers of tawny fibrin, so as closely to resemble an aneurysm. In the centre of this laminated clot was a small round cavity containing some loose black coagula, and this communicated with a considerable branch of the pulmonary artery. A large bronchus opened into the cavity in the lung which contained the laminated clot, but there was no communication between the cavity in the centre of the clot and the bronchus, though at the upper part the clot was soft and had no doubt recently allowed a communication to take place. No trace of any aneurysmal sac could be detected, the cavity in the lung had a smooth wall and showed but little trace of any chronic disease round it.

The right lung showed no signs of old tubercular disease, but was thickly studded with recent grey miliary granules. The other organs were normal.

As the case turned out to be one of acute miliary tuberculosis, it is evident that no treatment could have prevented a fatal termination, and the patient did not live long enough to enable any positive conclusions to be drawn as to the effects of the operation on the hæmoptysis. He had two attacks in the night following the operation, but no subsequent recurrence, and the post-mortem examination showed that the communication between the false aneurysm and the bronchus was closed, and not improbably, if the patient had lived, complete solidification of the aneurysm would have taken place. The lung round the aneurysm was quite collapsed.

When we consider how largely the pulmonary circulation is influenced by the respiratory process, and the small amount of blood which circulates through a collapsed lung, I think such a mode of treatment affords a fair prospect of arresting otherwise uncontrollable hæmorrhage. Extensive consolidation of the lung would no doubt, by preventing the collapse

of the lung, be a contra-indication, and thus would restrict the applicability of the operation to a few cases. The probability of exciting pleurisy is of course another objection. In this case, notwithstanding antiseptic precautions, pleurisy was set up, but it was of a non-suppurative form, and but for the tuberculosis would not, I think, have added much to the patient's danger.

Temperature, pulse, and respiration.

	Temp.	Pulse.	Resp.
Feb.			
9.—M.	100·0°	96	18
E.	100·6		
10.—M.	99·2		
E.	101·0		
11.—M.	98·4	84	18
E.	100·0		
12.—M.	97·8	80	
E.	98·8		
13.—M.	98·2	72	20
E.	101·8		
14.—M.	98·6	84	20
E.	100·6		
15.—M.	102·8	112	20
E.	100·2		
16.—M.	100·0	100	28
E.	100·0		
17.—M.	100·2	104	32
E.	102·0		
18.—M.	99·0		
E.	101·2		
19.—M.	101·0	108	18
E.	103·2		
20.—M.	98·6	96	20
E.	99·6		
21.—M.	98·6	92	24
E.	100·0		
22.—M.	97·8	104	20
E.	99·8		
23.—M.	97·8	76	20
E.	101·0		
24.—M.	98·0		
E.	101·0		
25.—M.	98·0	84	18
E.	101·8		
26.—M.	99·4		
Feb.			
26.—E.	100·6°		
27.—M.	99·0	96	20
E.	102·2		
28.—M.	99·0		
E.	100·6		
March			
1.—M.	99·6	80	
E.	100·4		
2.—M.	99·2	96	18
E.	100·4		
3.—M.	99·6	112	24
E.	101·6		
4.—M.	98·2	96	20
E.	101·6		
5.—M.	98·6	92	
E.	101·6		
6.—M.	98·2	96	
E.	101·6		
7.—M.	99·2		36
E.	101·2		
8.—M.	99·0		
E.	101·6		
9.—M.	98·6	104	38
E.	102·0		
10.—M.	98·6	96	28
E.	102·6	128	44
11.—M.	100·4	112	48
E.	102·4		
12.—M.	101·2	128	44
E.	103·8		
13.—M.	101·4	128	52
E.	102·8		
14.—M.	99·6	120	40
E.	99·0		
15.—M.	100·0	124	44

XXXVII.—*A Case in which (at the suggestion of the late Dr. Mahomed) a Calculus was removed from the Vermiform Appendix for the relief of Recurrent Typhlitis.* By CHARTERS J. SYMONDS, M.S. *Read May* 8, 1885.

CHARLES S., æt. 23, basket-maker, was admitted into Philip Ward, Guy's Hospital, under the care of Dr. Mahomed, July 16, 1883. The following history which he gave of himself, and all the early clinical notes, are compiled from the careful report made by Mr. Vernon.

Both parents were dead, the one from heart disease and the other from cancer of the uterus. Two sisters were living. Two years ago he had typhoid fever. Six months ago (January, 1883) he was seized during the night with pain in the right iliac region; this increased in severity and he became ill generally, and at the end of a week was unconscious, remaining in this state four days. The illness lasted seven weeks. During the first week he vomited everything, and his bowels were not opened for ten days, and the whole time there was great tenderness in the right iliac fossa. During the latter part of this illness he was in the Camberberwell Infirmary, where he was told he had typhlitis. When he got up he observed a hard lump in the right groin, about the size of a walnut; sometimes this was tender, but for the most part painless. Since this illness he has had repeated attacks of pain, which come on suddenly and last one or two days. At first these attacks recurred about once a month, but during the last five weeks he has had six, and they have been increasing in severity. Three days before admission (July 13) he was seized while at work with severe pain in the right iliac fossa, which he said "doubled him up." He felt sick but did not vomit, and was obliged to leave his work. He applied poultices with turpentine and obtained some relief, remaining in bed till the day of his admission (July 16).

On admission there was to be felt in the right groin on deep pressure a small, hard (slightly tender) lump, two inches

long, and parallel with Poupart's ligament, and three fourths of an inch wide. His bowels acted regularly, and all his organs appeared healthy. The man was placed on a fluid diet, was soon relieved of all pain, and by July 20 he was up. About this time I was asked by Dr. Carrington, who was temporarily in charge for Dr. Mahomed, to see the case with a view to some operative interference. We decided, however, that, as the case was recent, there was a fair probability of the complete disappearance of the swelling and so of the recurrent attacks of pain.

On July 26 pain returned in the groin and continued up to the 30th, occasionally shooting over the abdomen. It was increased by resting on his left side. The abdominal walls became rigid and the swelling enlarged. He was again restricted to a milk diet, had opium internally and hot fomentations over the abdomen. On the 30th he was much better, and the lump could again be felt, and by August 4 it had decreased to the size observed on admission.

Dr. Mahomed, having returned, considered fully the question of operation, to which the man had no objection whatever so long as there was a prospect of being relieved from the recurrence of pain, which prevented his continuing long at his work. Dr. Mahomed proposed that the appendix should be approached by an incision in the right iliac region, as he was strongly of opinion that there existed an abscess-cavity containing a concretion, and that the periodical occlusion of a communication with the cæcum determined the recurrence of pain and the symptoms of peritoneal irritation. This view was strengthened by the apparent enlargement of the swelling during the attack observed in the hospital, and by the fact that the swelling remained dull on percussion. I was of opinion myself that while the small lump to be felt was probably a concretion, that the recurrent attacks were due to some peritoneal adhesion or band constricting the last portion of the ileum. On account of urgent home affairs the man left the hospital on August 10, to return as soon as possible.

On August 11, the day after his return home, he resumed work, and after two hours was seized with the old pain and obliged to go to bed. The next day (August 12) he was readmitted, with extreme tenderness in the right iliac region, shooting pains across the abdomen, and a temperature of 102·1°. He was placed on a milk diet with opium. On the 18th the pain was less, but the tenderness remained, and there

was a visible swelling above Poupart's ligament too tender for manipulation.

August 14.—The swelling was hard, with a distinct upper edge, about two inches and a quarter above Poupart's ligament, with which structure it ran parallel for about three inches.

August 16.—He was easy again, and on the 18th the lump decreased to its smallest size.

As everything now seemed quiescent we decided to explore the swelling through the iliac fossa. Dr. Mahomed planned the operation, and it was on his recommendation that the incision adopted was selected. The lump was oval in shape, could be distinctly felt, but could not be taken hold of; it was well defined and only slightly tender. We concluded that we had to deal probably with a concretion, but whether it lay inside the appendix or rested in a cavity bounded by peritoneal adhesions and bowel it was impossible to say. We also thought that it might be a small abscess, with or without a concretion. Our plan was to approach the mass from behind, to remove a calculus if present, or to drain the cavity should we find pus.

On August 24 chloroform was administered, and under the carbolic spray an incision was made, commencing two inches above and one internal to the iliac spine, curving downwards and forwards for about four inches, being much like that used in ligaturing the external iliac artery. It was so arranged that its centre corresponded with the position of the swelling. The various structures were divided and all recognised distinctly, except the transversalis fascia. Being particularly anxious to avoid the peritoneum, the structures were at once raised out of the iliac fossa, when the lump was plainly felt as a hard rounded body. A hand pressed deeply from the front steadied the swelling and brought it further into the wound. A vertical incision was now made down on to the mass and a hard and calcareous body exposed. Before removing the calculus a fine silk suture was passed through the tissues just above the opening, lest when the body was extracted we should lose the entrance into the cavity. The opening was now enlarged and a calculus removed. No pus at all was seen, and the cavity from which the calculus was removed seemed smooth and free from deleterious material. The soft and purplish lining was evidently mucous membrane, and as moreover we could trace the tortuous and cord-like appendix upwards towards the cæcum, there seemed no room to doubt

that the appendix had been opened. Exploration of the cavity, which seemed only large enough to contain the calculus, failed to detect a channel leading towards the cæcum or in any other direction. There was no fæcal or other unpleasant odour. We therefore decided to close the aperture in the appendix, which was done by silk sutures introduced after Lembert's manner. Our object in doing this was to diminish the chance of a fæcal fistula. At no time did we recognise the peritoneum, so that we presume the cavity lined by this membrane was not opened. It appeared that the appendix had become adherent to the parietal peritoneum, and that through the adhesions we had entered its canal. It was the probable existence of such an arrangement that led us to approach the concretion from behind. The wound was closed by deep silk sutures, a large drainage-tube inserted, and the usual gauze dressings applied. The calculus is oval and much like a small bird's egg. It measures three quarters of an inch by half an inch. It had a brownish-yellow colour well shown in the drawing (Plate IX, fig. 4). On section it shows a laminated capsule, enclosing an irregular mass of putty-like material which has partly shrunk away from the wall. This gave a slightly fæcal odour when sawn through. The calcareous material is composed of phosphate of lime.

August 26.—First dressing. Tube removed, freed from coagulum and reinserted. Some of the sutures were loosened.

August 28.—Second dressing. Tube removed, as there was every sign of primary union; no fæcal odour from lower end of tube.

August 29.—He had for two days been complaining of aching pain in the wound, which became severe on this day. The respirations were hurried and the abdomen hard, but there was no sickness and no fever. The opium he had been taking was renewed.

September 1.—Some pus escaped from the wound, and he was relieved. A fresh tube was introduced and two sutures removed. The pus had no fæcal odour.

September 5.—He was nearly free from pain, the tube was shortened to an inch and a half, and the wound was healing.

September 11.—He had pain again, probably due to the retention of a little pus.

September 14.—He was free from pain and was on full diet.

September 20.—He got up, a small scab only remaining over the centre of the incision.

September 25.—After walking about the ward he felt something escaping from the wound and had pain all that night; the next morning it was found that about two drachms of pus had escaped from the wound. The antiseptic dressings, which had for some time been discontinued, were reapplied, and as the pain was still severe the wound was reopened by a probe and a piece of small drainage-tube inserted.

October 7.—He was discharged at his own request. There remained some induration to be felt on deep pressure, but the wound had apparently healed.

During the whole of his stay the temperature never rose above 99·4° F. Fearing some further trouble might arise so long as any perceptible induration remained, and being alive to the possibility that the real cause of all his pain might still exist, he was directed to return at the first sign of a recurrence of his former symptoms.

The man resumed work in a fortnight, and suffered no inconvenience till November 3, when he had a return of the old pain in the iliac region; gradually it spread as before over the abdomen and became so severe that he was obliged to take to his bed. Two days later he observed a yellowish fluid escaping from the sinus.

He was readmitted into Job Ward under my care on November 5th. There was a sinus (three inches deep) discharging a thin yellowish fluid, and the hard swelling still remained. By November 11 he had improved, and now we could feel the appendix passing upwards from the induration beneath the centre of the scar. The rounded swelling that had been observed before November 3, and which had varied in size, had now completely disappeared. On November 22 he was discharged, the sinus having closed and the man being in good health. He soon after returned to his work, and when last seen some weeks later he had been able to follow his occupation without any pain. There was at this time scarcely any induration to be felt, no more than could be explained by a deep cicatrix. When last heard of, April 1885, though an inmate in the Barming Heath Lunatic Asylum, he was well as regards his old disease, and had never had any trouble since November, 1883.

Remarks.—I believe I am correct in saying that this is the first case in which a concretion or calculus has been removed from the appendix vermiformis without, at the same time, the

opening of an abscess, and the credit of whatever value rests in the procedure must be given to my late and lamented colleague, Dr. Mahomed, at whose suggestion the operation was undertaken, and who further advised the inguinal incision, in opposition to that in the linea semilunaris proposed by myself. It is a matter to me of great regret that I am deprived of the association of Dr. Mahomed on this occasion, and his untimely death has lost to us much information upon disease of the appendix, more especially with a view to operation, which I know he was collecting. That the operation in this case was justified I think few will deny, when it is remembered how frequently a concretion is found to be the cause of fatal typhlitis. This fæcal concretion was present in eleven out of seventeen fatal cases, in all of which the disease originated in the appendix, either as a sloughing ulcer with a concretion, or a sloughing ulcer without a concretion. I mention this to exclude tubercular ulcers. I think this case so individual that it is not expedient to attempt to formulate conditions under which we should undertake similar operations. The guide throughout was the hard mass, and without this I scarcely think we would have undertaken the proceeding. In none of the eleven cases mentioned above was the concretion calcareous, so that I imagine it is rare to find a calculus, and so it will not be often that the foreign body in the appendix will serve as a guide. We directed our dissection to the posterior surface of the cæcum and appendix, not because we imagined that under normal circumstances we might avoid the peritoneum, but because here, if anywhere, we would probably meet with adhesions, and so possibly enter a cavity containing the appendix and concretion shut off from the general peritoneal cavity. Again, without such a guide as we had in this case there would be great uncertainty in reaching the appendix, when we remember how variable is its position. Though it is not my intention to enter to-night into the general subject of surgical interference in typhlitis, I would, after the study of twenty-three fatal cases, and with a knowledge of how frequently large iliac swellings due to typhlitis disappear, suggest that some cases at least might be saved by earlier incision, before, I mean, fluctuation is felt. Such cases are recorded, and success has followed the treatment. This treatment has been more especially advocated by American physicians, and applies chiefly to those cases where there is iliac fulness, and the low general condition resulting from the accumulation of pus, the case at the same time lasting longer

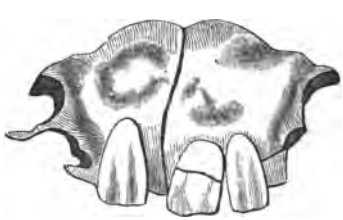
than usual. The difficulty very often in diagnosing the cause of sudden abdominal pain, and the number of cases of typhlitis that recover will, I imagine, long deter us from opening the abdomen and removing the appendix, and yet post-mortem inspection shows that if such a procedure could be undertaken a recovery might have been possible in some otherwise hopeless cases.

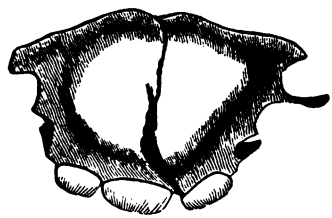
XXXVIII.—*Œsophagotomy for the Removal of a Plate with three Artificial Teeth which had been accidentally swallowed, and was impacted in the Œsophagus.* By George Lawson. *Read May* 22, 1885.

Mary N., æt. 55, a milkwoman, was admitted under my care into Queen Ward, Middlesex Hospital, on January 14, 1885, having about half an hour previously swallowed a plate with three false teeth.

On examination externally something hard could be felt in the œsophagus, about the level of the cricoid cartilage, by deep pressure with the fingers on the left side of the neck. The patient was brought into the theatre, and I tried to catch the foreign body with a pair of long curved œsophagus forceps, but although I could just feel the plate, yet I could not grasp it, so I decided at once to open the œsophagus. The patient having been placed under chloroform, a long No. 10 elastic catheter was passed into the œsophagus, so as to be used as a guide if necessary. The patient's head being turned to the opposite side I made an incision about three inches in length along the lower prominent border of the sterno-mastoid and exposed the edge of that muscle and a portion of the omo-hyoid. The sterno-mastoid and the omo-hyoid with the carotid sheath were then drawn outwards, whilst my colleague Mr. Gould drew the trachea in the opposite direction, and with his fingers on the right side of the neck pressed the œsophagus towards the incision. The recurrent laryngeal nerve was then seen, and this was also pushed outwards. The œsophagus was now visible, and the plate could be easily felt with the fingers. I then made a vertical incision through the œsophagus on to the plate, and seized it with a pair of forceps, but it was so firmly fixed into the walls of the œsophagus, by the sharp clips which had held it to the neighbouring teeth, that I could not readily remove it through the incision. I therefore enlarged the opening upwards, and in doing so, divided a thyroid artery, probably the superior thyroid as the current of blood was from above, which bled sharply. This was at once arrested with pressure-forceps. As the plate was still firmly held, I divided it with a pair of bone-forceps and

removed it in two portions, taking care to maintain a hold of each portion with a pair of pressure-forceps.





The dark lines across the woodcuts indicate the division of the plate with the bone forceps.

A ligature was then placed on the thyroid artery and the lips of the wound brought together with four sutures, and into the lower end of the wound a drainage-tube was inserted. No sutures were put into the œsophagus, as owing to the stretching caused in drawing out such an irregular body which was impacted across the œsophagus, I felt that there must necessarily have been some laceration of muscular tissue, and I thought that the parts would fall together better than I could adjust them with sutures.

As soon as the plate was felt with the fingers in the wound, the catheter was withdrawn from the œsophagus. The wound was covered with boracic lint charpie, and over this was placed carbolic gauze with oilsilk. The patient was then sent to bed and ordered to be fed with nutrient enemas, and with Slinger's nutrient meat suppositories. No food was to be taken by the mouth, but from time to time the lips and tongue were to be sponged with iced water to allay thirst.

The nutrient enemas were composed of peptonised beef tea and gruel; a three-ounce enema to be given every four or six hours, and three of the meat suppositories in the twenty-four hours.

January 15.—The patient was very restless during the night and complained of great thirst. The dressing was changed; there was a very free discharge of saliva and mucus from the wound. The drainage-tube was removed and the wound covered with iodiform wool, into which the secretion from the wound could drain. To relieve the thirst the patient was allowed to suck a little ice.

3 P.M.—Temperature 101·6°, pulse 110. She has retained the enemas, and the beef suppositories.

January 16.—Patient passed a very restless night, and has a distressing and frequent cough. Before her admission into the hospital she was suffering from a chronic bronchitis, which had now become aggravated.

Has had the enemas and suppositories regularly, and has retained them.

A quantity of saliva and mucus continue to flow from the wound. Three sutures removed. Ordered to go on with the enemas and suppositories and to each alternate enema to add ten minims of Tinct. Opii.

Temperature 100·4°, pulse 120.

January 17.—Has had a bad night, scarcely sleeping at all. A large quantity of saliva, mucus, and some pus flows from the wound. Has had a mustard poultice over the chest, which seemed to afford some relief.

Temperature 99°, pulse 108.

To continue enemas and suppositories, but as the pulse was a little flagging to add three drachms of brandy to each enema.

January 18.—About the same; still much secretion from the wound. As the patient was feeling very weak, Brand's essence of beef to be given frequently by the mouth in teaspoonfuls at a time. To continue the enemas, but omit the suppositories.

January 19.—Passed a much better night. Not quite so much discharge, but it has a very offensive, gangrenous smell. Around the wound there is a blush of redness. Has taken two tins of Brand's essence in the twenty-four hours. To continue the enemas and Brand's essence. Temp. 101°, pulse 108.

January 20.—The redness around the wound has increased. The discharge has diminished in quantity, but is very offensive. Patient refused to take any more of the Brand's essence. Ordered milk to drink in small quantities at a time, and to continue the enemas with the brandy, but without the Tinct. Opii except when restless. The cough still continues troublesome; a mustard poultice is applied daily and seems to give great relief. Temp. 100·2°, pulse 100.

January 21.—The redness around the wound is less, but the discharge continues from the wound and is very offensive. The patient had a good night. As a portion of all fluids taken by the mouth escaped out of the wound, I passed an œsophagus-tube, such as is used in cases of stricture of the œsophagus, with a funnel-shaped extremity which projected about six inches from the mouth, and allowed it just to enter

the stomach, but not to press against its walls. This was to be kept in, and through it all food was to be introduced into the stomach. For about half an hour after its introduction the patient had cough and some irritation, but the discomfort soon passed off and she was able to retain it without any apparent inconvenience. Three ounces of peptonised beef and an egg were then given by the tube, and fluid nourishment was regularly administered through it every four or six hours. Temp. 99·2°, pulse 100.

January 22.—The cellulitis around the wound about the same as yesterday. The discharge still very offensive. The patient suffers no inconvenience from the œsophagus-tube which has been kept in, and through which liquid food is passed into the stomach. During the twenty-four hours she had administered one and a half pints of peptonised beef tea and gruel, two eggs, four ounces of milk, and two ounces of brandy. Temp. 99·2°, pulse 88.

January 23.—Passed a good night. There is still redness and some swelling around the wound. The nutrient enemas discontinued. Three pints of beef tea and gruel and two eggs were administered by the tube. Temp. 98·2°, pulse 96.

January 24.—This morning an abscess was opened at the upper part of the cicatrix of the wound, and about half an ounce of pus escaped. Took two and a half pints of strong beef tea and gruel, two eggs, and two ounces of brandy. Temp. 98·4°, pulse 88.

From this date the patient made a steady progress towards recovery. From the wound several small sloughs of cellular tissue escaped. The discharge ceased to be offensive and was healthy, and diminished daily in quantity. The wound gradually closed and by February 22 was completely healed. The œsophagus-tube was worn continuously until February 8. About every four or five days the tube was removed and a fresh one introduced. The wearing of the tube did not produce any real discomfort. The patient slept well and did not complain of annoyance from its presence. As the patient improved in health her appetite increased, and the quantity of food given by the tube was, on February 1, three pints of beef tea, one pint of milk, two pints of gruel, two eggs, and two ounces of brandy. This was administered in quantities of one pint every four hours. On some days she would take only five pints through the tube in the twenty-four hours.

On February 8 the opening in the œsophagus had appa-

rently quite closed, as only a small quantity of pus escaped through a small sinus at the lower part of the wound. The tube was now removed, but it was ordered to be reintroduced for the administration of food.

The patient from this date was fed by the tube five times during each twenty-four hours, and this was continued until February 22, when the wound was quite cicatrised.

The patient has since been able to take her food without any discomfort. There is a deep puckered cicatrix in the site of the incision, and the parts are firm and healthy.

Remarks.—From the experience of this case the questions of interest which arise are: Is it better to introduce sutures to close the wound in the œsophagus, or to leave the cut edges to fall together of themselves? I certainly think that, when the body which has to be removed is an irregular and jagged one, so as to necessitate some bruising or tearing of the tissues in its extraction, it is better not to use sutures, but if the foreign body could be extracted through a clean cut wound, then sutures might be applied with advantage.

One point which struck me in this case was the amount of saliva which was constantly flowing through the wound, and which was no doubt the cause of the little cellulitis which followed. My impression is that this cellulitis might have been avoided if, immediately after the operation, I had introduced an œsophagus-tube and fed the patient through it. In this patient, the way in which an œsophagus-tube can be tolerated was well shown. For three weeks she wore a tube continuously and without any real discomfort. The patient began to improve from the time that the tube was introduced, and this no doubt was due to two causes. 1st, the patient was better nourished, and 2nd, only saliva and mucus escaped through the opening in the œsophagus, as no food was given by the mouth.

The successful termination was no doubt greatly due to the œsophagotomy having been performed soon after the false plate was swallowed, and before any ulceration or inflammation had taken place at the locality in which it was impacted.

XXXIX.—*A Case of Œsophagotomy.* By H. A. Lediard. *Read May* 22, 1885.

WILLIAM W., a postman, æt. 49, living at Ambleside, was asleep on a sofa on the evening of March 22, 1885, when he was awakened by finding that the plate, bearing one artificial tooth, had slipped into his throat. He attempted to reach it with his finger, but failed, and shortly after he was seen by Dr. Redmayne, by whom he was put under chloroform whilst extraction was attempted, without success. He was sent to the Cumberland Infirmary, and was admitted on the evening following the accident.

Patient was a healthy-looking man, of probably abstemious habits. It was found that he had great pain on any attempt to swallow, chiefly in the region of the thyroid cartilage, and emphysema of the neck was present, but there was no projection felt in the neck to indicate the presence of a foreign body.

Upon my seeing him I ascertained that before coming to the Infirmary emetics had been given, and that our house surgeon had made several efforts to extract the plate, which he had felt and grasped about the thyroid region; a horsehair probang had been used unsuccessfully, and the patient was altogether in a very uneasy state, being anxious that no further attempts should be made through the mouth. On his removal to the theatre I endeavoured to find a guide to the point of impaction, but could feel nothing externally; I passed an œsophageal bougie into the stomach (about the size of the little finger), and thought I felt the instrument scrape slightly as it was withdrawn. Finding that the patient was unable to swallow a mouthful of water, and that there was evidence of some injury to the pharynx from the emphysema, and blood on the forceps used, the patient was put under ether and œsophagotomy performed on the left side of the neck.

After dividing the platysma muscle the knife was put aside and the cellular tissue separated with the fingers only until the spine was reached, the omo-hyoid muscle was pushed downwards, and one small vessel only was tied. The gullet was next searched with a finger, behind and below the larynx, but nothing felt, and after passing a bougie through the mouth

into the œsophagus, I cut into it behind the cricoid cartilage; the finger was now used inside the gullet with the same result as in the examination from outside, the bougie was next passed through the wound downwards into the stomach, and at or about the cardiac end of the stomach I believed that the plate was felt, but without giving to the hand the impression that the plate had been pushed into the stomach.

The cut in the gullet was closed with two catgut sutures, but the skin incision was left gaping slightly at the centre, and a drainage-tube passed down to the bottom of the wound.

Very little need be said as to the subsequent progress of the case as the recovery was steady and uninterrupted. Rectal alimentation only was allowed for the first three days and then fluids were given by mouth, a little milk or beef tea sometimes leaking through the wound, and one day a little orange pulp he had been eating; a wood-wool pad was kept on the neck and this served to absorb the discharge from the wound or leakage from the gullet. Enemata were occasionally given and a little purgation employed, and after running a painless course the plate appeared at the anus on April 11th, *i.e.* nineteen days from the date of impaction.

There was very little febrile reaction following the operation; the chief trouble seemed to be some coughing with discharge of mucus from the pharynx, but swallowing seemed fairly easy from the moment that liquids were first allowed.

Patient left the hospital on April 20 feeling and looking well in all respects.

The plate will be seen to measure 1½ inches long by ¾ inches broad, and was made to hold two incisors, one of which is wanting. The margins of the plate present numerous sharp points in addition to a formidable-looking hook at one end.

The whole subject has been ably dealt with by Dr. Church in the nineteenth volume of the *St. Bartholomew's Hospital Reports;* and two cases have been recently brought before the Society by Dr. McKeown in vol. xi and Mr. Butlin in vol. xvii. The present case bears out Dr. Church's conclusion from statistics, viz. "that there is very little risk in the operation itself, and that a good result may be fairly expected if the operation is done shortly after the foreign body becomes impacted,"* whilst the circumstances in which the plate became dislodged are similar to Dr. McKeown's and Mr. Butlin's cases.

* *St. Bartholomew's Hospital Reports*, vol. xix, p. 67.

There is nothing to be said as to the operation, which seemed to be surprisingly simple and free from hæmorrhage, whilst there was little or no difficulty in separating with the fingers the layers of cellular tissue lying between the carotid sheath and the windpipe.

It may seem to have been an error to have operated in this instance, because the plate was not found in the pharynx or œsophagus, and I am unable satisfactorily to explain by what means the plate became dislodged whilst the patient was on the table, or if the finger unconsciously disturbed the plate from its resting place.

There is another point. Was it justifiable to open the gullet after the finger in the wound detected nothing? I think it was, for the plate might have been lodging in the thoracic portion of the œsophagus and been extracted with ease.

I have omitted to mention that there were no laryngeal symptoms from the first, yet there can be little doubt that the plate was lying behind the lower end of the larynx when grasped in the forceps, and from the fact that the mucous surface of the gullet about the cricoid cartilage presented several ecchymosed patches.

XL.—*On two Cases of Raynaud's Disease.* By T. Colcott Fox, M.B. *Read May* 22, 1885.

In 1862, Raynaud wrote as follows: "I propose to demonstrate that there exists a variety of dry gangrene, affecting the extremities, which it is impossible to explain by a vascular obliteration; a variety characterized especially by a remarkable tendency to symmetry, as well as that it always affects similar parts, the two superior and inferior limbs, or the four together; moreover, in certain cases, the nose and ears; and I shall seek to prove that this species of gangrene has its origin in a vice of innervation of the capillary vessels, which it will remain with me to specify." He believed it to be essentially a neurosis characterized by an exaggeration of the excito-motor power of the central parts of the cord presiding over the vascular innervation. The existence of such morbid conditions was established by Maurice Raynaud in his masterly Thèse de Doctorat in 1862, under the title *De l'Asphyxie Locale et de la Gangrène Symmetrique des Extrémités;* in the article "Gangrène" in the *Nouveau Dictionnaire de Médecine,* 1874, and in *Archives Générales de Médecine,* 1874. But notwithstanding the papers by Dr. Southey in the *St. Bartholomew's Hospital Reports,* vol. xvi, and in the Clinical Society's *Transactions* for 1883, and the record of three cases by Dr. Barlow in the same volume, the disease has as yet attracted so little attention in this country that I hope the exhibition of two cases, rather different from those already shown here, will be of interest to this Society.

Case 1.—Mrs. S., æt. 41, was sent to me by my friend Mr. P. F. Gilbert. She states that as a girl she always had cold hands and perspirations, but not blue hands and chilblains. Friends would frequently remark, "How cold your hands are!" or, when kissed, "How cold your nose is!" She was, however, little conscious, from her own sensations, of cold extremities. There is nothing otherwise of note in her early life. Her family history is good. Her father died of apoplexy aged 77, and her mother, though never robust, is still alive at the age of 78. All have good blood circulation and I have

failed to bring to light any neuroses. She never had rheumatic fever or any special illness.

At the age of 18 she came to London as a kitchenmaid, and had frequently to wash the doorsteps. She was married at the age of 27, and has had six children, including twins at the last birth. Her functions have been generally in good order, and she has not had any miscarriages.

Mrs. S. dates the commencement of her disease from June ten years ago, on moving into a damp house. Her fingers then began to go at frequent intervals white and dead, all of them simultaneously on both hands, "like wax" as she expresses it, and they pained her excessively. I think there is some doubt whether she did not suffer from "dead fingers" at a much earlier date, but her memory fails her a good deal. Such, however, is her own account, and, at any rate, the pain seems at that time to have specially attracted her attention to her hands. Gradually her feet also became involved, and up to the present time the recurrences have been getting more frequent, the local syncope has given place to local asphyxia, and the condition has become more pronounced as the ravages of the attacks accumulate.

She is now a woman of spare habit and with an anxious face and intensely nervous temperament. She says she is a "dreadful sleeper," especially after the slightest worry or excitement, and she has had several severe hysterical attacks on similar provocation, but I cannot connect these with any local attacks of asphyxia. Her heart and other organs are healthy. There is no diabetes.

When I saw the patient in February, the condition of the fingers might easily have been mistaken, at first sight, for scleroderma. All the fingers were icy cold, of a slightly blue tinge, fusiform in shape; the skin over them was shining and shrunken; the nails were variously affected and some of the bones atrophied. The history, however, at once made the nature of the malady clear. On subsequent occasions I have seen all the fingers slightly swollen and of various shades of lividity up to the metacarpo-phalangeal joints, with the course of some of the veins traced out by blue lines. The colour is always deepest at the palmar surface of the ends of the fingers. The attacks of asphyxia are now so frequent as to be almost continuous. The fingers are always the seat of pains, which are intensified with each fresh spasm of the vessels and are often excruciating as the relaxation sets in. Then the patient is much distressed and will frequently burst into tears.

Occasionally the asphyxiated condition of one of the fingers will lead to the formation of a blood blister, the contents subsequently become puriform, and on the rupture of the bullæ an ulcerated surface is left which is very slow to heal. At the time of writing, the left ring finger is ulcerated at the end and under the nail.

These attacks, so constantly repeated over ten years, have led to striking deformity of the fingers. I have already alluded to their fusiform shape and the atrophied condition of the skin generally. On the right hand, the distal phalanx of the thumb is shortened, and the nail incurvated over the scarred and puckered end. The end phalanx of the ring finger is nearly atrophied away and the nail shrivelled up. The little finger is in a similar condition and has been much attacked. The ring and middle fingers are conical with the nails curved over their ends. On the left hand the state of things is closely similar, but perhaps the changes are hardly so pronounced. As I have already remarked, the end of the ring finger is ulcerated. All the fingers are now in a chronic state of asphyxia and lividity, which is intensified with each attack. The joint movements are also very limited.

In the toes the disease is less severely felt, but their ends are somewhat blue and cold. The tip of the nose is rather blue, with dilated venules. There are scars over the right external malleolus.

The influence of cold is very marked. Her ears and nose go blue and cold directly she goes out of doors; her hands and feet get intensely livid with an "agony of pain." She dare not take her very warm socks off even in the house, and she keeps her hands muffled in cotton wool and warm gloves.

She also states that her extremities go blue "in a second" if she is startled by a sudden knock at her door or any unusual occurrence, and her heart "feels as if in a vice." Her pulse is thready and compressible. There has been no hæmoglobinuria, and her blood appears to be normal.

The patient has now been for some weeks under observation in the Westminster Hospital, and her excessively "nervous," hysterical, and emotional condition fully noticed. She has had many attacks of asphyxia of the fingers and toes, accompanied by excruciating pain, and for the most part ending in the formation of a crust at the end of the fingers beneath the nails. Some purpuric stains about the insteps at the present moment mark the site of symmetrical patches of congestion. The extremity of the nose is often involved, and

in one attack became covered with a thick crust. These attacks seem to occur without any regularity, but are mostly traceable to a colder change in the weather.

I would call attention to the condition of the face where the skin seems more pigmented than natural, covered with dilated venous radicles, and thinned, giving a rather expressionless aspect, which reminds one of scleroderma.

She has been treated according to Raynaud's method by descending constant currents to the spine, and with manifest improvement.

Case 2.—Joseph A., æt. 51, a man of most regular life, who had been for twenty-five years in the police force, consulted me in the Department for Diseases of the Skin at the Westminster Hospital in February, 1883. He is a stout, florid-complexioned man with a remarkably good physique, but yellowish conjunctivæ, and he then and still complains of feeling weak and often very unwell. There is nothing of interest in his early history. He applied to me on account of two oval, symmetrical, gangrenous sores, superficial, about the size of a half crown, one at the junction of each middle and lower third of the shins. These sores were covered with an adherent black eschar, and they had originated suddenly in "blood blisters." There were also a few pustules scattered around. His feet and hands were cold, and his pulse soft and weak, but with one exception I could find no other diseased condition. He was suffering from marked diabetes, but was not aware of it. I may here remark that a brother, æt. 41, also has diabetes, and it is perhaps significant that his father died comparatively young of phthisis, although the family are not phthisically inclined.

To continue the narrative, the sores were very obstinate to heal, and on March 28 another congestive patch appeared on the right shin which seemed to threaten gangrene, but eventually subsided. At the end of May the skin lesions were quite well.

On January 2, 1884, he applied to me again for a renewed outbreak of blood blisters about the feet and shins, but at that time, not appreciating their true significance, I did not make exact notes of them. His circulation at this time was very weak. The fact that he was suffering from Raynaud's disease was disclosed to me by the following occurrence.

On the 5th January his left great toe became suddenly black and swollen and intensely painful. Redness, which

gradually changed to lividity, extended up the side of the foot and leg to the junction of the middle and lower thirds of the tibia.

On the 9th a blister formed on the toe, and the pain and swelling subsided. The right foot was unaffected. On making further inquiries I now found that in the winter of 1883, before I saw him, he had suffered from a severe attack in his left foot which laid him up for five weeks. His doctor told him he had a severe frostbite, and that he was a lucky man not to lose his great toe. A month before Christmas of the same year four "black blisters" appeared beneath his toes. I gathered also that he has suffered for years from sudden blanching and numbness of the digits, in fact from "dead fingers," and occasionally from asphyxia of the fingers. The patient was a doorporter at some well-known stores and therefore much exposed. He has given up his occupation, and during the past winter has clothed himself with great care and carefully protected his hands and feet. Moreover, he has not ventured out unless the weather was genial. Consequently he has been almost free from these attacks of cramp of the vessels; indeed he has suffered from little else than the usual attacks of local syncope.

Remarks.—I will detain the Society with but few comments on these cases, as I have little to add to the masterly elucidation of the disease by Raynaud. The case of the man illustrates the advisability of denominating the affection Raynaud's disease rather than symmetrical gangrene of the extremities, a name which Raynaud fully recognised as not completely satisfactory. The conjunction of the disease with diabetes is of much interest. Raynaud only met with one such case in a woman, aged 31, who passed seventy-six grammes of sugar per litre of urine. The diabetes was completely cured and the gangrene arrested by an exclusively nitrogenous diet, the exhibition of alkalies internally, and a sojourn at Vichy three years running. The local asphyxia in her case preceded by eight years the first definite signs of diabetes, but the influence of the latter on the nutrition of the tissue was seen in the increased severity of the gangrenous process. I would call attention to the asymmetrical character of some of the man's attacks. Raynaud rightly insisted on the remarkable tendency to symmetry usually present, but that asymmetrical attacks also occur is certain and in such cases the nature of the malady may be far less obvious. In neither of my cases

could I get any clue to the occurrence of hæmoglobinuria, although I have seen a remarkable case in a girl of thirteen years under the care of my friend Dr. Leslie Ogilvie in which such a coincidence existed. With her the patches occurred on the body and did not implicate the digits.

The case of the woman is very well marked and the condition typical of incessant, long-continued, and moderately severe attacks. I show for comparison a woman of the same age with extreme atrophy of the fingers and other parts of the body left by a long-standing generalised scleroderma. The histories of the two affections are quite dissimilar and the appearances, as seen to-night, quite distinct to the careful observer. However, the diagnosis does not appear to be always so clear, as one may see from the interesting discussion on Prof. Ball's case at the Soc. Méd. des Hôpitaux. I find under the head Scleroderma several cases which read like Raynaud's disease. I may refer to the cases described under the name Sclérodermie in 1871 by Ball and Dufour and mentioned in the article "Main" in the *Nouveau Dictionnaire de Médecine*, and those by Verneuil, Ball and Liuville and Hallopeau collected in Beaurégard's thesis *Des Déformités des Doigts*, 1875, also by Hardy and by Vidal (*Gaz. des Hôp.*, 1878), a very severe and interesting case by Grasset and Apolinario of Montpellier reported by Brochin (*Gaz. des Hôp.*, 1878), in which brown taches and cicatricial patches simulate morphæa, and by L. Rapin under the term Sclérodactylie (*Revue des. Sci. Méd.*, 1878). The polished, stretched, atrophied condition of the face is referred to in the latter case of undoubted Raynaud's disease. Vidal says that his case proves the intimate relation between scleroderma and Raynaud's disease, and Brochin points out that the symptoms of the two diseases can be united in the same patient. With reference to this point I may mention that in two unquestionable cases of generalised scleroderma under my observation in which the hands were involved both women had been long subject to "dead fingers," and one of them continued to have mild attacks of asphyxia of the fingers after the onset of the scleroderma.

In conclusion, I may add to the references given by Raynaud and Southey the following:—

Marroin (of Constantinople), "Observation d'asphyxie locale des extrémités" following intermittent fever, in the *Archives de Méd. Nav.*, 1870, xxiii p. 341.

Behier's Thèse de Paris on *Asphyxie locale*, 1875.

L. Vaillard ("Contribution à l'étiologie de l'asphyxie locale

des extrémités," *Recueil de Mém. de Méd. et de Chir. Mil.* 1877, p. 585, from the *Provence Méd.*, 1877).

Camilo Nielson (*Ugeskr. f. Läger* 1877 copied to *Schmidt's Jahrb.*, 1878.

I would also call attention to the following references :—

"Ein Fall von Lepra maculosa mutilans," by Kochler of Kosten (*Berl. Klin. Wochen.*, 1877, p. 676.)

Deux Cas de lèpre anæsthétique mutilante, by Thaon, of Nice, *Progrès Méd.* 10 Nov., 1877.

XLI.—*Sequel to paper on Three Cases of Raynaud's Disease,* '*Clin. Trans.*,' vol. xvi, p. 179. By Thomas Barlow, M.D. *Read May* 22, 1885.

I PROPOSE in this communication to give some brief notes of the further progress of the three cases of Raynaud's disease which I described in the 16th volume of the *Transactions*, and to deal especially with the results of treatment.

The first case, Elizabeth N., originally under the care of my colleague, Dr. Poore, came again under my observation August 29, 1884. She was admitted into University College Hospital with the fingers of the right hand almost black, the dorsum of the hand of a dark purple, and the palm of a light purple colour. The forearm for the lower two thirds was blue, the colour gradually fading away. There was slight distension of superficial veins.

The left upper limb was similar to the right, but the colouration was less intense. The limbs felt very cold. The radial pulse was almost imperceptible at the wrists. The feet were also of a purplish colour, which was most marked over the toes, but not so intense as the colour of the fingers. The colouration extended upwards for at least an inch above the ankles. There was also a little distension of superficial veins, and the child complained of aching in both feet and arms.

The face was sallow and pale. Temp. in axilla at 12 noon, 98·8°. The heart-sounds were natural, the second being, perhaps, a little accentuated, 84 per minute. The urine passed at 2 P.M. was pale, acid, free from deposit of any kind, and contained no albumen.

I cannot give the exact duration of this attack, but I believe it was not more than three hours. It is to be noted that August 29 was very chilly, succeeding a very hot day.

Next day, August 30, between 6 and 7 A.M., body temp. was 100°, and at 9 A.M. 99·8° in the rectum. The urine passed in the night was of a dark amber colour, but there was no deposit of pigment. It was of slightly alkaline reaction, sp. gr. 1018, and free from albumen. There was no play of colours with nitric acid.

The limbs felt warm and the skin showed no pigmentary changes. The child was kept in bed until September 2, when

she was allowed to get up at 8.30 A.M. At 9.55 A.M. the hands and feet began to get blue and gradually became quite purple, the colouration extending to 1 in. above the wrists and just above the ankles. The child cried out with pain and nothing could quiet her.

At 10.5 A.M. I placed the child's right hand in a large basin of hot salt and water, the positive pole of a Leclanché constant current battery being applied to the inner side of the child's arm and the negative pole in the water. The current was rapidly made, broken, and reversed by a commutator and the number of cells increased until a moderately vigorous contraction of muscles occurred. At the end of thirty minutes the right hand was perfectly normal in colour, or rather, of a pale pink tint. The thumb and two radial fingers lost their lividity first, then the little finger, and finally the ring finger. The left hand was still as purple as before. The same treatment was then applied to it, and in about the same time recovery took place and in the same order. Nothing was done to the feet except that they were surrounded with cotton wool. The colouration had not been so intense as that of the hands, but it is noteworthy that it did not clear up entirely until 6 P.M. The urine passed at 12 noon was pale in colour, contained slight deposit of mucus, but no pigment and no blood corpuscles; it was of sp. gr. 1016, and was free from albumen. The temp. was, at 7 A.M. 99·4°, 1 P.M. 99·2°, 3 P.M. 100·4°, 8 P.M. 101·2°.

Patient was kept in bed on September 3, but on September 4 was allowed to get up. She was quite comfortable till 2 P.M., when she complained of slight pain in the right foot and said she felt an attack coming on. A slight colouration of the right sole was found. She was allowed to sit by the fire and in half an hour this attack passed off. It is noted that the temperature of the ward was higher that day than on the 2nd.

September 5.—Patient was allowed to get up at 3 P.M. She was playing about in the ward until 5.15 P.M., when she complained of her feet. The soles were found bluish and the colouration rapidly extended over the dorsum up to the middle of the leg. The colouration was more intense on the left leg than on the right. The child complained of much pain. There was nothing abnormal about the hands. I was anxious to ascertain the effect of nitrite of amyl, and Mr. Fleming, the house physician, allowed the child shortly after the beginning of this attack to inhale three minims. For about ten minutes the radial

pulse became perceptibly fuller and the face became markedly flushed, but no change whatever took place in the appearance of the lower limbs. The constant current was then applied to one leg, but the child struggled so much that the application could not be persevered with. The feet were wrapped in cotton wool and the child was put in front of the fire. By 8 P.M. the feet were normal.

September 7.—The child got up to-day at 2 P.M. She was comfortable until 4.30 P.M. and then complained of her feet. The distribution of the lividity was about the same as that of the last attack and the hands were quite normal.

The feet were wrapped in wool and she was allowed to sit by the fire. The feet were normal by 6 P.M. Urine normal. The mother volunteers the statement that the feet are attacked about three times as often as the hands.

From September 8 to September 15 she was allowed to be up and was free from attacks. She was then discharged from the hospital. I learnt that on October 9 and 10 she had attacks, but she was not brought to the hospital.

On November 3, she was brought to me in the out-patient room, five minutes after the commencement of an attack. The left foot was cold and blue up to the ankle and the child was crying with pain. I placed the child's foot in a galvanic bath as before described, and in ten minutes the foot was quite right again. The second and third toes recovered first and the big toe last.

I satisfied myself, whilst the child was in hospital, that in her case there was no initial pallor observed, and that whenever she complained of pain at the onset of an attack there was already some blueness which generally increased up to lividity. The freedom from paroxysms when in bed, as stated in the previous paper, was confirmed.

The second case, Lillie C., recommenced her attacks on October 1, 1883. During the time she was under observation her attacks occurred as before, generally in the middle of the day. They were chiefly confined to the lower extremities. They lasted from half an hour to an hour and were repeatedly followed within an hour or two by the passage of a small quantity of dark urine. I had several opportunities of examining such dark urine, and found as before no red blood-discs, but pigment and oxalates, marked reaction to the guaiacum test, and the characteristic spectrum of methæmoglobin.

The child living some distance from the hospital and the attacks on the whole being of shorter duration than the former

series, there was no opportunity of trying the effect of galvanism on the shortening of an individual attack. She attended daily for galvanism for a fortnight or more, but I cannot say that the result was conclusive, because it was obvious that independently of any treatment the asphyxia of the limbs was less severe than formerly, and the child was able to go to school regularly, so that it seemed unnecessary to insist on her continuing the treatment.

The third case, John P., came under my observation again in August, 1883, and I then determined to give galvanism a thorough trial. I first applied the poles to the spine as Raynaud directs, but not obtaining any definite result, after a few days I applied them locally to the blue extremities themselves, holding the two sponges a few inches apart and painting the limbs vigorously for about a quarter of an hour daily. Fifteen to twenty cells of a Leclanché constant current battery were employed and the plan was sometimes varied by the bath method before described.

The patient very soon testified to the benefit of the treatment. He began to walk better; he was comparatively free from the burning pains, which had rendered him miserable both by day and night, and he was soon able to go to his work, for which he had been quite incapacitated. The tonic effects on the peripheral circulation were obvious directly; the colour of the skin became much less blue, though months elapsed before it became a healthy colour. There was at first a degree of anæsthesia to the galvanic as well as to the faradic current, but this gradually passed away and he became normally sensitive. There were no degenerative reactions, although there was a certain amount of wasting of the leg muscles as well as of the feet. Under the galvanic treatment and increased exercise the feet gradually became much better nourished.

The man attended regularly six days a week from August, 1883, to the end of January, 1884, and subsequently three or four times weekly until the end of May, and I have to thank my assistant, Mr. Roberts, for his systematic and regular application of the galvanism. There were three days of very cold weather in January, when it was certain that, according to his statement, in previous years he would have been exceedingly bad. He had a slight attack of blueness, tingling, and pain in his right foot, but it did not interfere with his work and it soon passed off.

When he was allowed to attend less frequently for galvanism, viz. at the end of January, great pains were taken

to show his wife how to shampoo the feet, and this was done daily by either the patient or his wife till the end of May. He was then dismissed and told to show himself again in October, 1884. At the latter date he was free from pain and his feet, though not plump, were moderately nourished, and were only a little colder and bluer than natural. He could walk well and do his work.

To these cases I may add two others, perhaps allied, but I think not strictly coming under the category of Raynaud's disease. The first was that of a little girl who had a deep cyanosis of both lower limbs extending nearly up to the knee. The affection was not paroxysmal and was not attended with pain. The child had always had a feeble circulation, but the condition had become greatly aggravated shortly before she came under my care. There was no morbus cordis to be made out and the only other noteworthy clinical feature was the occurrence of some ill-defined epileptoid attacks followed by some paresis of the lower limbs. It was for one of these attacks that the child was brought to the hospital.

The daily use of the constant current, applied alternately by "painting" and by means of the bath, was followed by very marked benefit to the cyanotic condition of the limbs. The child's legs were also shampooed, but there could be no doubt about the benefit of the galvanism to the local condition and she expressed herself as more comfortable after every application and walked much better.

The other case was that of a middle-aged woman who had severe tinglings and pains in both hands, and a condition something like "dead fingers." The pains often kept her awake at night, and this and the numbness from which she suffered had seriously interfered with her work as a needlewoman. There was no actual lividity, but the fingers were of a pale leaden colour, also there were no definite paroxysmal attacks.

My assistant, Mr. Roberts, galvanised her several times weekly for more than two months with very great benefit. She was taught to rub her fingers systematically at home and to use alternate hot and cold water douches, but she herself was emphatic as to the comfort that galvanism gave her.

In her case as in some others the curious fact was noted that certain fingers (symmetrical on the two sides) were more affected than others and that in the healthy glow which followed any given application of galvanism certain fingers became pink sooner than the others.

To sum up, although the observations which I have nar-

rated are very imperfect, and the experiments themselves were not all of them sufficiently absolute for scientific purposes, I think they support the recommendation of systematic daily employment of galvanism for the condition of local asphyxia.

I venture to think, with all deference to Raynaud's authority, that the local application is better than the method recommended by him of passing the current over the spine, or, as he would state it, "through the spinal cord."

It seems hardly necessary to add that galvanism ought to be supplemented by the employment of every other method which can be devised of improving the peripheral circulation, whether locally, by shampooing, hot and cold douches, &c., or centrally, by the timely administration of food before any exposure to cold.

XLII.—*A Case of Papilloma of the Bladder successfully removed by operation.* By WILLIAM ANDERSON. *Read May* 22, 1885.

JAMES S., æt. 53, caretaker of a lecture-hall, was admitted into St. Thomas's Hospital on August 9, 1884, with symptoms of vesical tumour.

The first indication of the presence of vesical disease was a spontaneous attack of hæmaturia in the summer of 1872, during convalescence from a railway accident. The hæmorrhage was unaccompanied by pain or marked constitutional disturbance, and subsided at the end of a few days, leaving the patient perfectly free from symptoms for a year, when a second and precisely similar paroxysm made its appearance. This also passed away speedily, but the succeeding intermission was abbreviated to six months, and subsequently recurrence took place at fairly regular intervals about four times yearly.

In July, 1882, the man attended as an out-patient at St. Thomas's Hospital. He was then in the midst of an attack of hæmaturia, losing a considerable quantity of blood, usually fluid, and voided principally with the final contractions of the bladder, but sometimes also in the form of clots, which escaped at the commencement of the stream. There was neither pain nor increased frequency of micturition, the condition was not affected by ordinary exercise, and there were no indications of urethral obstruction. He was somewhat anæmic and debilitated, but was able to discharge the duties of his office.

The introduction of a sound into the bladder revealed on the first and subsequent occasions a peculiarly acute sensibility localised to the region of the trigone (an area afterwards found to coincide with that of the tumour), but was otherwise negative in its results. Rectal and abdominal examination gave no indications of disease, and the urine was found to contain no abnormal elements except blood-discs. Neither in the first nor in later investigations were any fragments of villi detected. The symptoms yielded at the end of ten days under the adminstration of perchloride of iron, and for a time the patient was restored to a fairly satisfactory state of health, but attacks of a similar character recurred again and again at

intervals of about three months, always subsiding readily under treatment and not inducing any serious loss of strength. In June, 1884, however, the disease assumed a new phase; during a paroxysm of more than usual severity symptoms of catarrh became superadded, and from this time the intermissions ceased. The hæmorrhage became less profuse at the end of a few days, but never entirely disappeared, and the urine continued to present a copious muco-purulent deposit. The patient was now tormented by hypogastric pain and irritability of the bladder, and his strength began to fail rapidly. He was advised to enter the hospital with a view to operation, but did not determine to do so until August.

On admission the local examination and the investigation of the urine were carefully repeated without obtaining any new evidence, but the symptoms were regarded as sufficiently indicative of vesical tumour to call for an exploratory operation. This was accordingly undertaken on August 14.

An incision being made through the perinæum into the membranous portion of the urethra, the finger was introduced into the bladder and at once impinged upon a soft floculent growth involving a considerable portion of the rectal wall of the viscus. The mass extended from near the internal urinary meatus upwards for a distance of about an inch and a half, and laterally for about an inch on either side of the median line. It appeared to consist of long villous processes springing from a broad and slightly elevated base, and during

Diagrammatic view of the posterior wall of the bladder showing region involved by the tumour.

the manipulation all doubt as to the nature of the disease was set at rest by the detachment and escape of two or three slender semitransparent fragments having the usual character of papillomatous fimbriæ.

The prostatic urethra dilated under steady pressure sufficiently to allow the introduction of a pair of forceps (with long narrow straight blades and a slight bulbous extremity) into the bladder by the side of the finger, and by careful manipulation of the instrument the root of the tumour was crushed, segment by segment, and the villi twisted away until the whole of the growths appeared to have been removed. The bleeding was severe but not dangerous, and ceased spontaneously after the expulsion of a mass of clot by a powerful reflex contraction of the bladder. A drainage-tube was passed through the wound and the patient was removed to bed.

In the evening the temperature rose to 100·4° and a slight rigor occurred. The rigor returned twice on the following day, the temperature rising on the second occasion to 104°, but there was nothing alarming in the condition of the patient. Some bloody urine was passed by the urethra and a little oozing of blood took place from the wound.

For a few days after the operation the urine contained an admixture of pus and blood, and the passage of the secretion by the normal channel induced an acute pain referred to the glans penis, but these symptoms gradually disappeared and the further progress of the case was complicated only by the development of a small perineal abscess at the end of the third week, associated with febrile disturbance and rather severe rigors.

On September 10, twenty-seven days after the removal of the tumour, the patient was able to leave his bed, and on the 21st was discharged from the hospital free from all trouble save a minute fistulous opening in the perinæum and a trace of blood in the urine.

Histologically the growth was found to consist of a mass of closely aggregated processes of various lengths springing directly from the mucous membrane. Some of the fringes were long and slender, the longest reaching three quarters of an inch, others shorter and more solid, often branched, and containing a more or less considerable basis of loose connective tissue. The vessels were for the most part of large size and with thin walls. The tumour may be considered to represent a combination of the fimbriated and fibrous papillomata figured in Sir Henry Thompson's work. (See Plate VI, fig. 3.)

instances it has permitted the satisfactory removal of all the tumours it has brought within the surgeon's reach, and has given marked relief to pre-existing symptoms in a certain number of cases where it has failed to reveal their cause.

It is maintained by the advocates of the high operation that the suprapubic incision confers much greater advantages for the ablation of tumours, but this has yet to be demonstrated by results, and is by no means proved even in theory. If it be borne in mind that nearly all vesical growths spring from the lower half of the viscus, and the great majority from the rectal wall, which is almost in a direct line with the axis of the prostatic urethra (Woodcut, p. 314), it will be seen that there are few cases that are not sufficiently accessible by the perineal route. There is, however, no doubt that it is less eligible for the removal of growths implicating a very extensive area, and in those which spring from the anterior or upper walls, but the latter regions are not often primarily involved, and the timely employment of perineal exploration would reduce the number of widely diffused vesical neoplasms by facilitating their discovery in the earlier stage of their existence.

The statistics of mortality are of little value, as the result of operation is so frequently complicated by extreme anæmia, renal disease, and other grave constitutional conditions sufficient in themselves to account for a fatal termination to the case. Taking the figures for what they are worth, we find in twenty-seven cases six deaths which may have been caused or accelerated by the surgical intervention. On the other hand, of eight suprapubic operations four ended fatally, one from extravasation of urine attributed to too rapid absorption of catgut ligatures of the vesical wall, the others from exhaustion within a few days of the removal of the tumour, and probably due in great part, if not wholly, to the loss of blood preceding it.

We are still to a great extent in the dark as to the ultimate results of the ablation of vesical tumours, as nearly all the more systematic operations are of very recent date, but it may be serviceable to analyse the published examples of non-malignant growth removed by surgical means, with a view to trace, firstly, the frequency of recurrence, and in cases of relapse, the duration of the interval between the operation and the reappearance of symptoms; and secondly, the length of the period of immunity in cases in which no return has been noted.

Theoretically it seems probable that a relapse would take place sooner or later in a rather large proportion of cases,

considering the exceptional difficulty that must frequently arise in ensuring the removal of every portion of the tumour by any mode of operation yet devised. Bearing in mind also the very slow development of the primary growth in many instances—extending over a long term of years—it might be expected that the signs of recurrence would occasionally be deferred until a late period, long after the apprehensions of the patient had ceased, and after the surgeon had lost sight of his case.

An examination of thirty-eight cases (male and female), in which the nature of the tumour was sufficiently indicated in the reports, shows four instances of undoubted and four of probable recurrence, all within a year of the operation; complete relief in one case for five years up to the date of record; in one for four years, in five for two years, and in four for periods ranging between fifteen and twelve months. In nineteen other cases no signs of recurrence had appeared at the time of the report, but the observations had been limited to a term of less than twelve months. There are hence as yet only eleven cases out of thirty-eight in which we possess evidence of an immunity of one year and over, against eight cases in which relapse is noted as certain or probable. We may hope, however, that the operators will at some future time favour the profession with supplementary information upon those cases which can be followed after the publication of the earlier details.

XLIII.—*A Case of Tumour of the Bladder; Removal; Cure.* By Bernard Pitts. *Read May* 22, 1885.

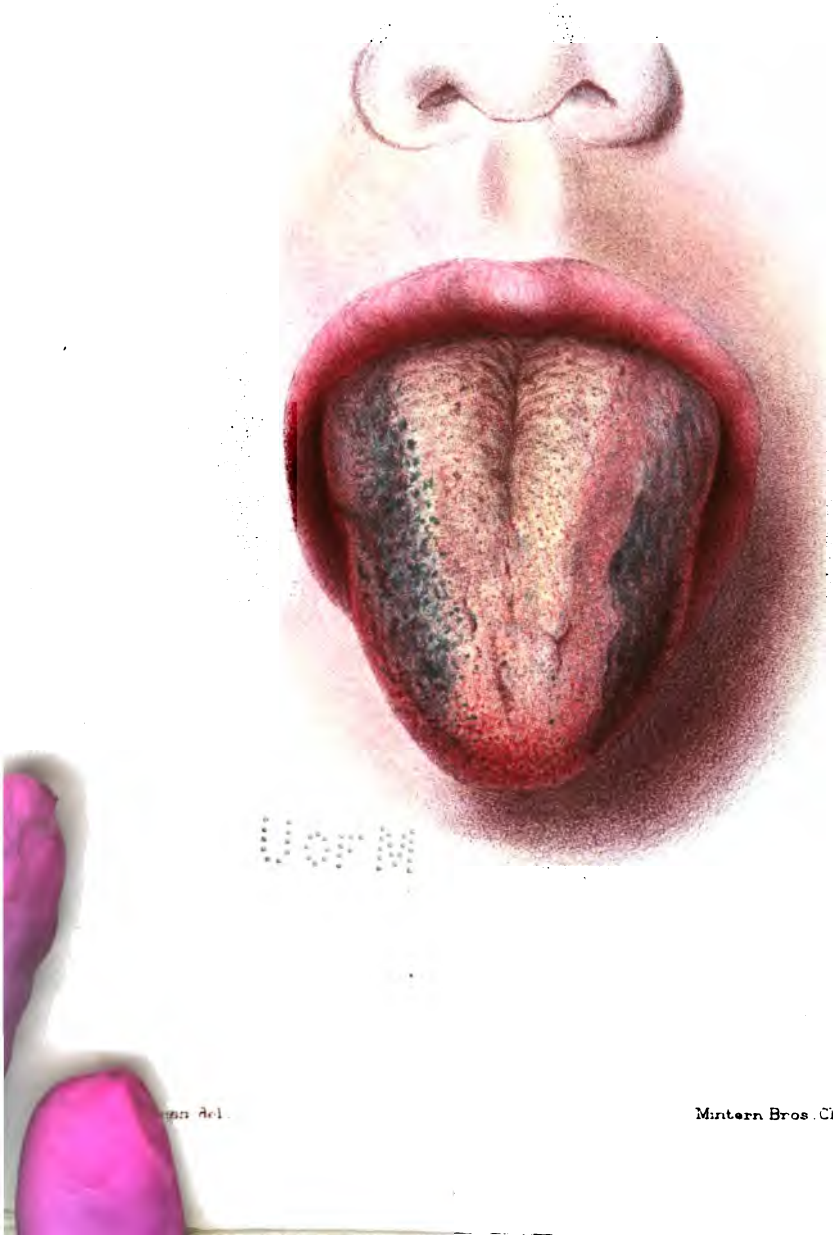
W. B., æt. 43, watchmaker, was admitted on September 10, 1884, to St. Thomas's Hospital. Patient had a very blanched appearance, and was evidently in a debilitated condition, having been losing flesh and strength for many months. He stated that for more than two years he had been passing blood with his urine, but that of late the quantity had much increased, and become alarming in amount. He had never had any pain beyond a tickling sensation in the penis at the end of micturition when passing clots. Of late his water had been very offensive, and there had been a constant desire to empty the bladder. For some months he had been unable to follow his occupation. The urine was of sp. gr. 1020, alkaline, and very offensive, of a deep red colour, and always containing a quantity of clot. A microscopical examination showed possible traces of villous growth. Examination by sound and by rectal exploration gave no indications.

On September 15 I made a perineal exploratory incision and on introducing the finger into the bladder, at once felt a tumour situated on the right side and just below the orifice of the ureter. The tumour seemed circumscribed and soft, and of about the size of an orange, but in reality was no doubt smaller, adherent clot giving one a false impression of size. With serrated forceps I was able to munch off portions of the growth, but no satisfactory progress was made, and a further examination revealed the presence of a very firm pedicle of the thickness of a finger. The pedicle was divided by means of a strong wire écraseur. The bladder was then examined for any further evidence of growth, and washed out, and a soft tube tied in. There was a good deal of bleeding during the early part of the manipulation, but none after the wire of the écraseur was attached. The patient was fairly comfortable for the next two or three days, the urine contained no clot, but was very red, and became more offensive, and with considerable increase of the mucus. The tube was left out on the 19th, and on the 20th he had a rigor lasting ten

minutes, with temp. 102·6°. The next day the temperature became normal, and continued so during the remainder of his convalescence.

On October 7 he was passing his water entirely the right way and without pain. The urine was clear and free from any trace of blood. He rapidly put on flesh and regained a healthy appearance, and has remained perfectly well since. On no occasion has he had the slightest trace of any blood in his water. An examination of the tumour showed it to be what Sir H. Thompson calls a fimbriated papilloma. On floating the portions detached by the forceps in water, the delicate long fimbriated processes were very evident. Microscopical examination showed each of these processes to consist of a fine membrane covered by layers of columnar epithelium, with blood-vessels running up into the process and branching directly under the membrane. The diagnosis in this case was quite clear. One was confident from the amount of bleeding, and from the prominence of this symptom, that there must be a vascular tumour. The microscopical examination gave indications of imperfectly formed villi, but previous to the operation I did not place great reliance on this evidence, for I was quite inexperienced as to the value of such evidence, and had made up my mind to explore on the consideration of the hæmorrhage alone. Following the directions given by Sir H. Thompson in his valuable book on *Tumours of the Bladder*, I was provided at the operation with a gorget director and a pair of serrated forceps. The gorget was of no use to me. I tried it, but found I could manage far better with an ordinary straight director as a guide for the finger. After finding the tumour, I spent a long time, and occasioned a good deal of unnecessary bleeding by tearing off portions of the growth with the forceps. Finding that the stalk was a substantial one, I pulled it as far as possible into the neck of the bladder, and then made use of an écraseur, applying the wire quite close to the expanded portion of the growth, and as the portion of pedicle left was about an inch long, I again used the écraseur and detached the remaining part as close as possible to the wall of the bladder. The lesson I learnt was that, however desirable crushing and biting with forceps may be, when the attachment of the growth is broad, that in a case like the one I met with, where there is a firm, strong pedicle, it would have been far better to use the wire in the first instance. The prognosis in this case is most favorable. If one had been contented with breaking off the softer parts

from the stalk there would have been risk both of after-hæmorrhage and of possible recurrence of the growth. By a division of the pedicle close to its attachment there can hardly be any fear of a recurrence at that particular spot. The rest of the bladder surface was smooth, and there was no difficulty in examining every part of it with the finger.



Mintern Bros. Chromo lith.

LIVING SPECIMENS

DESCRIBED BY CARD.*

I.—*Pigmentation of the Tongue (? Addison's Disease).* By J. K. Fowler. *Exhibited October* 24, 1884.

WILLIAM C., æt. 42, a butcher, attended as an out-patient at the Middlesex Hospital, complaining of cough and hæmoptysis.

The patient is a fairly-nourished man, of a sallow complexion.

The tongue presents the following appearance (Plate XII): The central part is covered with a moderately thick yellow fur extending from the circumvallate papillæ to within half an inch of the tip. The mucous membrane of the sides of the tongue is free from fur and the surface quite clean, but deeply pigmented of a bluish-black inky colour, along a strip from half to three quarters of an inch in breadth, narrowing toward the front of the tongue and not quite reaching to its tip. The discolouration is not uniform, pinkish spots being present here and there.

There are also a few small patches of pigment in the central furred part. At the junction of the hard and soft palates there is a small brown spot in the middle line, and the buccal mucous membrane and that of the lips is slightly mottled with pigment. The only other part of the body presenting any unusual traces of pigment is the forehead, where there is a band of brown discolouration which the patient states has been present for many years.

The areolæ of the nipples are not pigmented. The dis-

* Published in accordance with the Regulation relating to the exhibition of living specimens at the meetings of the Society, viz. that "each case shall be accompanied by a card containing a brief description of the points it illustrates, such card to be retained by the Secretary for publication or not in the *Transactions* at the discretion of the Council."

colouration of the tongue was first noticed six months ago; it is thought to be of a rather lighter tint now than then. He has never had syphilis. He has not suffered from any of the constitutional symptoms of Addison's disease.

Remarks.—It is unusual in Addison's disease to find such an amount of pigmentation of the mucous membrane of the tongue as is present in this case without pigmentation of the areolæ and of other parts usually affected. The absence of any constitutional symptoms of the disease is to be noted, but a confident opinion cannot be formed on that point alone, as the order of appearance of the local and general signs of the disease is very variable.

I am disposed to regard the case rather as one of unusual pigmentation associated with tuberculosis than as one of Addison's disease.

Postscript.—This patient remained under observation for six weeks after being shown to the Society, during which time the pulmonary disease progressed rather rapidly, but no change occurred in the degree or site of the pigmentation. He was reported to have died suddenly from syncope a fortnight later. No *post-mortem* examination was made. The mode of death makes it, on the whole, probable that the case was really one of Addison's disease.

II.—*Aberrant Form of Psoriasis.* By W. Hale White, M.D. *Exhibited November* 29, 1884.

C. A., æt. 32, presented himself to me at the out-patient department of Guy's Hospital, with an eruption which he had had five years, and which had first appeared as a small pimple on the leg. Now it is situated chiefly on the extremities, but the knees and elbows are remarkably free. It is more on the extensor than on the flexor aspect and is remarkably symmetrical on the two extremities. This is best seen in the fold between the buttock and the back of the thigh. It is present on the back also. There the symmetry is so marked that it looks as though one side were an impression of the other, radiating as it does like the letter X from the centre of the back. There is a well-marked patch on the front of the body at the level of the xiphoid cartilage, extending round like a belt, most marked on the left side; it is slightly to be seen at the back of the head at the margin of the hair, slightly on the

face at the junction of the nose and orbit on the right side. It is absent on the scrotum, the palms of the hands, and soles of the feet. In many places it consists of circular rings with a margin one third to half an inch wide, with dark skin in the centre; the margin consists of a reddened base with several fine scales on it. Even where the circular arrangement is not evident the patches have the same characteristics. The dark skin in the middle shows that the disease spread from a centre in a circular manner; this is seen on the back, where the **X** like arrangement is probably due to the coalescence of a circle on the upper part of the back with one on the lower part, for the skin is dark in both the upper and lower angles of the **X**. It is only irritable in cold weather; it has got slowly worse during the last five years. There is absolutely no history of syphilis. The eruption was never moist. The treatment proposed to be adopted is tar ointment and large doses of arsenic.

III.—*A Case of Hypertrophy of the Subcutaneous Tissues of the Face, Hands, and Feet.* By Charles A. Ballance and W. B. Hadden, M.D. *Exhibited January* 23, 1885.

THE patient was a married woman, æt. 35. She had had three children in ten years, but no miscarriages. There was nothing noteworthy in the family history. Up to two years and a half ago she had had no illness and was a strong hale woman. At that period scarlet fever broke out in the house and one of her children died. The patient herself had sorethroat and œdema of the feet, but no rash. She states that there was also dropsy of the hands at this time, but not of the face; but of this Mr. Dismorr, the practitioner in attendance, makes no mention in a letter which he was good enough to write us on the subject.

The patient ascribes her present condition to the scarlet fever, although she admits she had tingling in the hands previously. It must be mentioned also that the catamenia ceased a few months before the attack of scarlet fever, and have never reappeared.

On examination we found the face large and puffy, and strikingly different from a photograph taken a few

months before the scarlatinal attack. The nose was broad and the alæ nasi thickened. The lower lip was thick, rose coloured, and everted. The submaxillary glands were easily felt but not certainly enlarged. The neck was full, short, and the subcutaneous tissues seemed to be too abundant. The thyroid gland was normal in size. There was slight prominence above the clavicles. The clavicles themselves were much curved and distinctly larger and thicker than normal. The hands were remarkably large, the enlargement being due to a thickening of the subcutaneous tissues, which was especially evident at the inner border of the hand. The skin was moist, and wrinkles were present on the backs of the fingers. The nails were large but otherwise appeared healthy. The phalanges and metacarpal bones were not enlarged. Each hand measured across the palm in circumference nine inches. Formerly she used to wear No. 7 gloves.

The feet were more affected than the hands. The measurement around the foot about the centre was twelve inches. Before her present illness she used to wear large 4 boots, afterwards large 6, and now large 8. There was much subcutaneous thickening at the outer border of the foot, on the plantar surface, and below the internal malleolus, but very little over the dorsum. The thickened parts formed pads, which could readily be taken up by the finger and thumb. The hair on the scalp was soft and natural, and had no tendency to fall. The heart and lungs were healthy, and the urine was not albuminous.

The speech was not slow, but was distinctly guttural. This was due to a very unusual enlargement of the tonsils, which interfered with speech, deglutition, and respiration. Indeed, it was for this affection that she came under the care of Mr. Ballance, who removed the tonsils. At the same time his attention was called to the appearances above mentioned. The woman was active both in body and mind. The skin of the body was natural and the perspiration free. There was no anæsthesia.

The case in some respects resembled myxœdema, but many of the most striking characteristics of this disease were absent. The enlargement of the hands and feet was not like that we have seen in myxœdema. The skin was soft, moist, and supple, quite different from the dry scaly surface in myxœdema.

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The growth extends down below the epiglottis, and involves the right side and the posterior wall of the pharynx, and also the right side of the posterior half of the tongue. The teeth have been removed on account of pain.

February 5.—The growth is extending rapidly and the patient does not take any solid food, and only a very small amount of liquid, on account of the pain.

February 17.—Gastrostomy performed. The stomach presented at once, and the lower part of the great curvature was attached to the peritoneum and skin by fourteen silk sutures.

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R. [illegible]

There was a [illegible] months. [illegible] fourteen days [illegible] ised the [illegible] years ago, [illegible]

The [illegible] the right side and the [illegible] the right side of the [illegible] have been removed in [illegible]

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The gastric juice never escapes, nor are the edges of the wound digested.

Weight, March 4, 8st. 7lb.; 11, 8st. 5lb.

He feels satisfied with the amount of food given.

The pain in the throat is less since the operation, and the patient looks much better.

P.S. Patient lived five months after the operation, by which he was much benefited.

V. *Two Cases of Wiring Ununited Fractures.* By C. Macnamara. *Exhibited March* 13, 1885.

I AM anxious to bring these patients before the members of the Clinical Society, to demonstrate the advisability of employing early passive motion of the knee- and elbow-joints, after wiring an ununited patella or olecranon process.

Case 1 is that of F. G., æt. 25, admitted into the Westminster Hospital, on October 22, 1884. This patient was a seaman in the Royal Navy; he fell and fractured the patella of his left leg on the 9th of the previous March. After the accident F. G. was under treatment for nine weeks; he then left the hospital still very lame, and he asserts he could pass his two fingers between the extremities of the ununited bone. On October 3 the patient slipped and inflicted further injury to the left leg. In these circumstances he was taken into the Westminster Hospital for operation.

Case 2 is that of W. H., æt. 32, a labourer, admitted into the Westminster Hospital on January 23, 1885. In the previous October he fell and fractured the olecranon process of his right ulna; he was under treatment for some weeks, but when the splints had been removed it was found that union had not taken place between the ends of the broken bone. The patient attempted to resume his work, but the strength and use of his arm was so much impaired that at his own request an operation was undertaken on January 28, in order to retain the fractured olecranon in contact with the ulna. I employed the Listerian system of dressing in both cases; and, with the valuable aid of Mr. A. B. Barrow, removed a thin slice from the surface of the fractured bones. Holes were then bored through the ends of the ununited bone,

and thick silver wire having been passed through the holes into the extremities of the ununited fracture, were securely brought together. I bored directly through the bones so that in both cases a loop of silver wire pressed into the joints. This enabled me to use considerable force in bringing the ends of the bones together, without the fear of the wire cutting through the bone from traction made upon it by the muscles. I found in the case of the patella, however, that the rectus exerted so much force in preventing the upper fragment from coming into apposition with the lower one, that I divided a considerable portion of the tendon at its insertion into the patella. The knee- and elbow-joints were laid open and exposed to the air and a certain unavoidable amount of rough treatment during the operation, but in neither case were there any subsequent local or constitutional symptoms. I may mention that in both cases at the time of the operation interrupted splints were secured to the limbs by means of gum and chalk bandages. These splints were not removed until three weeks after the operation, when passive motion of the knee and of the elbow-joint was commenced. I wish to lay stress on this method of treating these cases (not that the practice is anything new), but from my own experience their success depends largely on keeping the limb after the operation absolutely at rest; and this can only be done by means of some such bandage as that to which I have referred. Passive motion also must as a rule be commenced from three weeks to a month after the operation. The silver wire can be felt beneath the skin on the surface of the patella and of the olecranon in both these patients, but the fractured ends of the bones have united firmly and so completely that it is impossible to feel where the disunion had occurred. The patient's limbs are as strong and as useful as they were before the bones were fractured.

VI.—*A Case of Ununited Fracture of Right Olecranon Wired Antiseptically. Close Union.* By Rushton Parker, B.S. *Exhibited April* 24, 1885.

MR. S., æt. 21, fell off a bicycle in Wales about the middle of June, 1884. Was skilfully treated by a medical man, but disturbed the fragments himself after a fortnight by

improper use of the limb. By the end of September, hopeless non-union was established. Antiseptic suture under Listerian carbolic acid arrangements, with copper bell-wire embedded and left in October 7, 1884. Uncomplicated recovery after a two or three days' simple traumatic fever; union chiefly primary, the rest by granulation, but with no suppuration, tension, or avoidable inflammation. Drainage-tube removed at the third dressing on the third day. The elbow treated at an angle throughout; rather more than a right angle the first day, flexed up to rather less than a right angle on third day and kept at that. Wire remains in.

VII.—*Paralysis of the Left Fifth Nerve.* By F. W. Strugnell. *Exhibited April* 10, 1885.

ON February 18, the patient, a man æt. 49, had violent pain of neuralgic character over left side of head with slight numbness of the part affected.

February 19.—Complete loss of sensation of the left side of the face and parts supplied by the fifth nerve. Partial dilatation of the left pupil, which does not act. Earache on the left side; severe catarrhal ophthalmia. Does not feel continuous current from thirty-cell battery.

February 24.—Cornea of left eye has become hazy.

March 17.—Pain of severe character in left arm and forearm, cornea sloughing. Thickening of zygoma on left side.

March 27.—Small tumours noticed in different parts of the body.

The patient has had syphilis.

Treatment.—Large doses of potass. iodidi.

VIII.—*Successful Thyrotomy for Papilloma with preservation of voice.* By R. W. Parker. *Exhibited April* 24, 1885.

MARTHA G., æt. 5, was operated on seven months ago. At the time she was suffering from urgent dyspnœa due to papilloma of the vocal cords. The child had been "gruff"

since she began to talk, and there had been increasing dyspnœa with exacerbations from time to time. The child has recovered with *preservation of the voice.*

IX.—*A Case of Osteitis Deformans.* By Stephen Mackenzie, M.D. *Exhibited April* 24, 1885.

M. C., a woman æt. 48. About five and a half years ago the leg bones were noticed to gradually become bent and bowed, and at the same time she experienced in them aching pains. Four and a half years ago she broke both bones of the left leg by slightly knocking against a stool; she did not fall. Seven and a half years ago she had some internal illness, which left her weak for a long time.

There is no history of similar affection nor of carcinoma in family. Her father died of phthisis.

Both femurs, tibiæ and fibulæ, bend outwards and forwards so as to form a continuous curve, causing extreme bowing of the lower extremities. The heels and toes can be apposed, but the knees remain widely apart. The bones appear thickened. There is a forward stoop, and some stiffness of neck. No enlargement of the head. No affection of the upper extremities, with the exception of some thickening of the left clavicle.

Aching pain is more or less constantly experienced in the affected bones.

X.—*Hereditary Multiple Tumours.* By Stephen Mackenzie, M.D. *Exhibited April* 24, 1885.

E. G. B., æt. 35. At the age of nineteen the patient accidentally discovered a lump in the buttock, and subsequently found numerous others in various parts. He has counted from fifty to a hundred. They have increased in number and size since he first observed them but not for some years. They appear to follow no regular order in distribution and have caused him no pain or inconvenience except when pressed upon. He has

had rheumatoid arthritis. A brother, aged thirty-seven, also has a number scattered about body; they were first noticed when he was fourteen years of age. He thinks they have remained stationary in size and that no new ones have been developed for many years. He has no pain or inconvenience from them unless they are pressed upon. He has never had rheumatism in any form. An elder brother, now about fifty or fifty-two, has one tumour for certain and possibly more.

Their mother, aged seventy-two, seen in 1881, when she had one tumour, about the size of a pea, on the inner side of the arm, and another on the extensor surface of one forearm. She stated she had one on the thigh, but it could not be discovered. They were all painless unless pinched. She has had more but they have disappeared. She had rheumatic fever when thirty, and attacks of subacute rheumatism subsequently.

The tumours in all cases have felt firm, somewhat lobulated, and subcutaneous. They appear to be lipomata or fibrolipomata. The chief interest in the cases is their hereditary, and probably congenital origin.

XI.—*Two Cases of Myxœdema.* By J. Hopkins. *Exhibited May* 8, 1885.

CASE 1 kindly supplied by Dr. Suckling, of Birmingham (copied from Myxœdema Schedule issued by Clinical Committee).—Mrs. H., æt. 57. Working class. Mother died of phthisis. Father of plumbism. Sister died of myxœdema. No syphilis or alcoholism. No mental disturbance. Considerable trouble and worry. Catamenia, excessive at the last, ceased at forty-seven. Pregnant three times. Two children survive. Had one miscarriage. The children were born before the establishment of myxœdema. She lived in Birmingham fourteen years; previous to that sixteen years in Hammersmith. The symptoms were first noted when she was fifty-four years old; first in eyelids. The face is large and œdematous and the skin clear, translucent, with œdema of both eyelids, especially the lower.

She is a big woman. Skin of body dry and scurfy. Supraclavicular regions fatty, and great prominence of these regions.

Skin of extremities dry and does not perspire; hands not spade-like, nails normal. Hair scanty, has come off much lately. Thyroid body she says has been enlarged. At present it cannot be felt. Temperature in the mouth 99° F. No dropsy. Sensation normal; feels cold; no occipital headache; no paralysis; retardation of motion. She had a bad fall at Christmas, 1883. No convulsions. Superficial reflexes diminished; deep reflexes normal. Slowness of intellect. Bad memory for recent events. Placid disposition. (Here several negatives omitted.) Speech slow and when she talks she feels a difficulty, as if her tongue would not move. Eyes, &c., normal.

Heart normal; pulse 60. Respiratory and digestive system normal. No teeth at all for the last three years; gums atrophied; tongue large and pale; uvula and soft palate normal. Defæcation normal. Lymphatics normal.

Urine clear, copious, about three pints daily. Sp. gr. 1024. (This was exceptional urine; this day being scanty.) Acid and no albumen. Colour pale yellow. Micturition two or three times during the night and frequently during the day.

The lower lip is very thick, pendulous, and cyanotic.

Notes by Exhibitor.—Mrs. H. has two daughters. The eldest, twenty-seven, has bad sick headaches; the other, twenty-one, has bad headaches, not accompanied by sickness.

Mrs. H. already had a broad parting of the hair when a young woman. Urine at present time contains a little albumen.

(For family history see accompanying case, Mrs. B.)

Case 2.—Mrs. B., æt. 47, myxœdema in two sisters.

Present state.—Cheeks and lower part of face thick and pendulous. Eyelids œdematous; nose broad; speech thick; teeth loose; gums retracted and atrophied; body fat, and abdomen large.

Absence of lumps above clavicles, of waxy blue lips, of loss of hair, of patches of dilated capillaries in cheeks and of slowness of movements, &c.

Symptoms.—Flushing and sweating of face, which sometimes becomes the blue-red of "pickled cabbage." Slavers upon her pillow at night. Difficulty in swallowing small morsels, and food often sticks in the throat, producing choking. Sleep is light and troubled by worrying dreams. Awakes with distressing headache and heaviness of eyelids. Morning vomiting. "Pins and needles" in hands and feet, and bouts of sudden involuntary movements of legs. Tinnitus aurium and muscæ volitantes. Painful indigestion.

History.—Was always very thin till of late years. Attacks

of headache whole life, often with sickness. Thyroid enlarged when a young woman. Menstruation irregular before marriage. One pregnancy. The patient has had a good deal of pain about sacrum since eighteen years old and tender spines of vertebræ. Has noticed fulness of lids and of lower part of face for some years. Has lost many teeth during the past two or three years; they have "turned to chalk" and come away whole. Her hair came out in great abundance during 1883. No history of syphilis or alcohol.

Family history.—Father had lead-poisoning and is said to have died at sixty-three of abscess of liver. Mother died at thirty-five of phthisis. One brother phthisical and another had had rheumatic fever and neurosis of jaw. Two sisters older than patient. One, Mrs. H. (exhibited), has myxœdema; the other, Mrs. K., died of it in 1884. (Case reported in *Lancet*, vol. ii, 1881). Mrs. K.'s eldest daughter has bad headaches, flushes and perspires much about the face, has an enormously large abdomen, severe pain about the sacrum, and tumbles about very much. The patient's son suffers from headaches.

Remarks.—Many of the symptoms of myxœdema point to the sympathetic system. The history of this family does so more definitely. All except Mrs. H. suffer from headache or sick headache. Two flush and perspire abnormally. The two exhibited have had enlarged thyroids.

Perhaps flushing, inordinate sweating, sacral pain and enlarged abdomen will be found to be, in some cases at least, the first symptoms of the disease to appear.

XII.—*Cervical Rib.* By W. Arbuthnot Lane, M.S., and W. Hale White, M.D. *Exhibited May* 22, 1885.

THE man has a movable cervical rib on the left side. Its full length is about $1\frac{3}{4}$ inches. It appears to terminate in a pointed extremity from which a short tendinous band extends to the upper margin of the first thoracic rib. The scalenus medius is inserted into its whole length, and the scalenus anticus has a less defined lower origin than that on the right side. The scalene tubercle is very indistinct. There is no marked displacement of the subclavian artery. It seems to

cross the first rib at the inner limit of the attachment of the fibrous prolongation from the cervical rib. The lower cords of the brachial plexus lie on the cervical rib and are in consequence much more easily felt than on the opposite side. The span of the first left thoracic rib is not markedly greater than that of the right, although its cartilage is distinctly thicker and larger; this causes the inner end of the clavicle to be more prominent than its fellow. The clavicles are of equal length. There is no indication of a cervical rib on the right side, nor any particular prominence of the transverse process of the seventh cervical vertebra. The right first thoracic rib is apparently normal. There is no disproportion in the size of the manubrium and gladiolus. Seven cartilages articulate symmetrically with the sternum. Twelve ribs are present on either side, the last is about 4 inches long. The cervical rib produces a distinct and characteristic prominence in the left side of the neck, which presents a striking contrast to the normal curve on the right side. This appearance is exaggerated by the absence of subcutaneous fat. This case presents none of the more obvious modifications in the form of the ribs and sternum which have been shown to be characteristic of the presence of cervical ribs ("Cervical and Bicipital Ribs in Man," *Guy's Hosp. Rep.*, vol. xlii); at the same time it must be remembered that the left rib is a small one and that there is only one present in this specimen.

XIII.—*Fracture of Larynx.* By W. Arbuthnot Lane, M.S. *Exhibited May* 22, 1885.

F. A.—Eight months ago this man received a heavy blow from a fist on the right side of his neck. This caused him very great pain, and a choking sensation in his throat with inability to speak, This lasted for a few minutes. For twenty-four hours he was only able to speak in a whisper, and it was not for three or four days that he was able to speak with his natural voice. For three days he suffered from pain in swallowing, which he referred to the region of the larynx. After these few days all difficulty in swallowing and all feeling of discomfort passed away, and his throat felt as natural as it did before the injury.

The angle between the alæ of the thyroid cartilage is somewhat increased. Both superior cornua have been broken obliquely from the angles of the alæ, which are seen to be rounded, as in those specimens I showed at the Pathological Society, and which are described in my paper, "Fractures of the Larynx and Hyoid Bone" (*Trans. Path. Soc.*, 1884).

The right great cornu of the hyoid presents a distinct irregularity at its centre, the posterior half passing obliquely inwards from the direction of the anterior. It is impossible to to say whether this irregularity is traumatic or not, owing to the thickness of the structures over it.

XIV.—*A Case of Raynaud's Disease.* By A. T. Myers, M.D. *Exhibited May* 22, 1885.

I HAVE been most kindly allowed by Dr. Cavafy to bring forward a case which is under his care, and before I attempt to describe it I ought to acknowledge my obligations to him and to Dr. H. T. Griffiths, the Medical Registrar of St. George's Hospital, to whom I owe many of the particulars.

The patient, Tracy T., is a boy æt. 12, rather pale, but otherwise healthy looking, who bears the scars of symmetrical gangrene of the ears, and is suffering at present from paroxysmal hæmoglobinuria with some tendency to recurrence of the gangrene. The parents and the five brothers and sisters, whom I have seen, exhibit no similar symptoms. There is no syphilis in the family and no ague. They have lived since before this boy's birth in a suburb of Cheltenham in which no malaria is to be found after careful inquiry.

About five years ago, when the boy was seven years old, and apparently soon after measles, the first attack of paroxysmal hæmoglobinuria took place. About the same time, or soon after it, the ears were noticed to be very much cyanosed when the boy was chilly, and ached much as he got warm. A little later the physician in charge of him at the Cheltenham General Hospital, Dr. E. T. Wilson, describes gangrene of both ears, with a mottled and pasty look, probably the *tachetée* of Raynaud. This gangrene recurred or recrudesced some eight or ten times between 1880 and 1883, always in the winter, and destroyed a small portion of the helix and concha

of each ear, very nearly but not absolutely symmetrically. For the last two years there has been no more actual gangrene, but extreme cyanosis on exposure. Meanwhile the attacks of paroxysmal hæmoglobinuria have never been absent for very long during these last five years, though much more frequent in winter than in summer. There is an imperfect history of occasional red itching patches scattered over the skin which may possibly represent one of the skin manifestations of the *asphyxie locale.*

Since January 14, 1885, he has been under Dr. Cavafy's care in St. George's Hospital. The scars of the first symmetrical gangrene of the ears are plain, and almost any exposure to cold is followed by cyanosis, tenderness, and aching in them. During these four months in the hospital he has had twenty-two attacks of paroxysmal hæmoglobinuria which have been similar in all their main points. The first symptoms have been what have been so often described as premonitory in this disease, viz. yawning and lazy stretching of the limbs with little rigor or subsequent flush. With these the patient is very familiar as leading almost invariably to the passage, within an hour or so, of water of the colour of porter or old ale and as a rule tolerably clearly transparent.

The abnormal colour is rarely observable for more than three or four hours; the water is passed often, scalds him slightly, and changes back from the abnormal to the normal very rapidly, the albumen, which sometimes amounts on boiling to as much as one half in the paroxysmal attack, disappearing as completely and nearly as rapidly as the discolouration. The interparoxysmal urine has been perfectly normal. Repeated examination with the microscope has failed to show any complete blood-cells in the water; once or twice thin, almost colourless stromata of blood-cells have been noticed, from which probably the hæmoglobin has been separated. There is also a considerable amount of granular detritus, varying in colour from tawny yellow to dusky red, and, with this, occasional renal casts containing pigmented granules.

The blood also was examined several times during the attacks, taken from the ears when cyanosed and from the hand. The coloured corpuscles were fairly normal in outline, but sometimes had crenate edges; there was always an abnormal disinclination to form rouleaux, as Boas and others have noticed; and sometimes, but not always, blood-flakes, as they have been called, were found, varying in colour from a deep, reddish black to a thin transparent red, and in size from about

four to ten times as large as a normal coloured corpuscle. On one occasion they seemed to be contained in a transparent envelope, and to be themselves somewhat granular, shading off into the colourless envelope. These were the chief symptoms of the blood dissolution.

On May 12, after the boy had been free from any attack for six weeks, his left hand was put into a bowl of ice and water for ten minutes, and, though there were no immediate effects, yet next morning and on the mornings of the two following days he had a recurrence of the paroxysmal attacks with a feeling of numbness in the left hand, and the same symptoms of dissolution in the blood drawn from his left hand. In other cases where similar experiments have been made the results of artificial chill have been more rapid and the causal connection more indisputable. Still in this case it is worth notice that the only attacks during the last nine weeks were on those three days following the chilling of the left hand.

It is also worth record that for five days (April 23—28) he had a sharp attack of pleurisy and pneumonia, following apparently on slight exposure; the lower half of the left lung was consolidated, the temperature reached 104·5°, and it ended favorably by crisis; but during this sharp fever there was no hæmaturia of any kind nor even albuminuria. The paroxysmal attacks in some cases, but by no means in all, were accompanied by a slight rise of temperature to about 100° F., for a few hours; after the attacks there was slight but distinct icterus lasting as a rule about twenty-four hours. The spleen is very slightly enlarged, but no definite temporary enlargement during the paroxysms has been established.

REPORT

OF A

COMMITTEE OF THE SOCIETY NOMINATED NOVEMBER 10, 1882, TO INVESTIGATE

SPINA BIFIDA

AND ITS

TREATMENT BY THE INJECTION OF DR. MORTON'S IODO-GLYCERINE SOLUTION.

Read May 22, 1885.

Before attempting to discuss the results of the treatment of spina bifida by Dr. Morton's or other methods, it has been thought of essential importance to determine more clearly than has hitherto been done the pathological conditions included under this term.

With this object we have undertaken an examination of all the specimens contained in the London museums as well as those in Cambridge and Glasgow, and sundry others which have been placed at our disposal by different contributors to this report. The importance of this inquiry may be judged from the fact that in England alone, as may be seen from the Registrar-General's report, no less than 647 deaths from spina bifida occurred in 1882, of which 615 were in children under one year of age.

Although the literature of spina bifida is large it is so unsatisfactory and inexact that the Committee have decided to found their report on the examination of the specimens already alluded to, and on cases which have either been under their own immediate treatment or especially reported to them for the purpose of this investigation.

The subject will therefore be considered in two parts: 1. Pathological. 2. Clinical.

1. Pathological Anatomy.

The term spina bifida, which was first used by Nicolai Tulpius* about the early part of the seventeenth century, is employed to define certain congenital malformations of the vertebral canal with protrusion of some of its contents in the form of a fluid tumour. With very rare exceptions the malformation affects the neural arches of the vertebræ, and the tumour projects posteriorly; in rare cases, however, the malformation involves the bodies of the vertebræ, the tumour in such cases protruding anteriorly into the thorax, abdomen, or pelvis between the lateral halves of the bodies affected.

The main bulk of specimens may be classed under three chief divisions, of which the second is by far the most numerous:—

1. Protrusion of the membranes only, *spinal meningocele.*
2. Protrusion of the membranes together with the spinal cord and its appertaining nerves, *meningo-myelocele.*
3. Protrusion of the membranes together with the spinal cord, the central canal of which is dilated so as to form the sac cavity, the inner lining being constituted by the expanded and atrophied substance of the cord, *syringo-myelocele.*

The pathological anatomy of these lesions is strictly parallel with those occurring in the head, and known under the terms "meningocele" and "encephalocele." The affection, indeed, may implicate both head and spine, as in the occipito-cervical cases, of which specimens exist in the museum of the Royal College of Surgeons. The present report, however, has reference to the spine only. The specimens are discussed under some of the following headings:

1. Position of the tumour.
2. Size and form of the tumour, whether pedunculated, sessile, or intermediate. Presence of an umbilicus, or of a longitudinal furrow or other subdivision.
3. Coverings of the sac; the extent of the cutaneous investment, of the "membranous" area, of "ulceration" (if any).
4. Disposition of the meninges within the tumour. Seat of the fluid. Nature of the fluid.
5. Size and configuration of the deficiency in the neural arches.

* *Observationes Medicæ*, Liber 2, caput xxx, p. 231; "Spina dorsi bifida," Amstel., 1685.

6. Disposition of the cord and nerves.
7. Unusual variations. (*a*) Subdivision of sac, complete or partial, by partitions. (*b*) The presence of bony outgrowths across the spinal canal in the neighbourhood of the tumour.
8. The process of cure.
9. Theoretical considerations, drawn from the anatomical facts.

1. **Spinal meningocele.**—Of the 125 specimens of spina bifida which we have examined in the various museums before referred to, 10 were examples of meningocele. The deformity may occur in any region. So far as our examination of specimens allows us to generalise, the deficiency in the neural arches in these cases is usually limited to a small area; sometimes the protrusion occurs between the arches of two adjacent vertebræ without their being in any way deficient.

On the other hand, the neural arches may be deficient for a considerable extent, the neck of the sac being comparatively small, as in Specimen No. 1 of our List (R. C. S. Teratological Series, No. 271), p. 364, where the arches of the last lumbar and all the sacral vertebræ consist chiefly of membrane, and the neck of the sac does not exceed the size of a goose-quill. We have seen no specimen illustrating what we assume to be the typical form of spinal meningocele, that is to say, a sac composed of dura mater and arachnoid (the so-called visceral layer) communicating with the general cavities of the spinal membranes, invested with normal skin, and tending to be pedunculated. In some probably rare cases the sac is double within, composed of distinct portions beneath a single cutaneous covering, the pedunculated communication with the general spinal cavities being also double, as in Specimen No. 2 (St. George's Hosp. Museum, No. 15h). In other cases the spinal cord opposite the opening becomes slightly prolapsed or displaced back so as to lie in the neck of the sac and to more or less occlude it. This condition is represented in Plate XIII, from Specimen No. 3 (Glasgow Royal Infirmary Museum, No. 145); the line of origin of the nerves from the spinal cord deviates but little, the nerves not being in any way involved in the protrusion. A similar but less marked condition is shown in Plate XIV, fig. 1, Specimen No. 4, a cured meningocele (Glasgow Western Infirmary Museum, Series I, No. 2). Specimen No. 5 (St. Bartholomew's Hospital Museum, No. 3486) is the greater portion of the sac of a meningocele, suc-

cessfully excised (and reported upon in *Pathological Society's Transactions*, vol. xiv, p. 214, 1863). In this specimen the sac is uniformly thick except at its summit, where there is a longitudinal depression, unpigmented and smooth. There is no corresponding eminence on the opposite aspect of the sac, that is to say, the depression affects the proper substance of sac-wall. Microscopic examination shows this depressed portion to be devoid of hairs and glands, in marked contrast to the adjoining healthy skin. This portion of the sac is composed of fibrous tissue continuous with that of the corium and having a similar arrangement except that there is no papillary structure. The deeper layer of the epidermis is continued over this surface, the outer layers being lost through partial maceration, and the same is true of the adjoining normal skin. Vessels in this depressed area are visible and appear proportional to the amount of fibrous tissue of which it is composed.

Specimen No. 6 (St. Bartholomew's Hospital Museum, 3483) represents the parts concerned in a sacro-coccygeal meningocele, after destruction of the sac; the terminal portion of the cord, cauda equina and filum terminale can be seen lying at the bottom of the vertebral furrow in the normal position. We have under observation a child presenting a small tumour in this region, which it may be assumed is a simple meningocele; the skin over it is perfectly normal. The tumour does not increase; it measures at its base 1·5 cm. in diameter and it is elevated about ·5 cm. at its centre. The child is healthy and free from all symptoms.

From the scarcity of specimens of meningocele in museums it may be inferred that this form of lesion (which sometimes remains in *statu quo*, and sometimes undergoes spontaneous cure) is much less fatal than the more ordinary form of spina bifida.

2. **Meningo-myelocele.**—The pathological anatomy of this division offers more complex phenomena, but, though subject to minor variations, it is remarkably constant. Of 125 specimens examined the arrangement about to be described was found in seventy-six cases (63·2 per cent.). This arrangement is illustrated by Plate XV from Specimen No. 7 (R. C. S., No. 273 B), taken from the body of a female child aged eighteen days; the lower limbs were well formed but paralysed, as were also the bladder and rectum. Death took place from general marasmus, with retraction of the head due to meningitis. No treatment was adopted. The neural arches are unclosed from the fifth lumbar vertebra to the end of the

spinal column inclusive. The bodies of the vertebræ are all well formed, and the spinal nerves normal. The wall of the protrusion is formed, except at its summit, of skin and dura mater, the latter being continued uninterruptedly from the theca surrounding the spinal cord within the intact part of the neural canal. The arachnoid membrane is continued over the interior of the sac, the cavity of which corresponds to the subarachnoid space. The spinal cord is traceable from the entire portion of the vertebral canal across the upper part of the sac to its posterior wall, with which it becomes inseparably incorporated at a point a short distance above the middle. The last of the nerves (third lumbar) arising from the cord within the intact canal passes horizontally forwards to emerge by its proper intervertebral foramen. The fourth lumbar nerve arises from the spinal cord within the sac, and is applied to the side of the cord in its forward course to the succeeding intervertebral foramen. The nerves succeeding this arise in a double longitudinal series along the back of the sac. Of each nerve both roots are present, and at their origin are separated by a falciform fold of pia mater, an exaggerated continuation of the ligamentum denticulatum. The nerves pass forwards with slight convergence to their respective intervertebral foramina; their course through the bone has been displayed by the removal of the left lateral mass of the sacrum; as they lie in the foramina the posterior roots present the usual ganglia, and the posterior primary division arises as usual. On the roots of some of the lowest nerves within the sac are some small additional ganglia (*G. aberrantia*) similar to those at times met with as a variation in normal anatomy. There is no coalescence of the sacral ganglia.

It will be seen that the anterior and posterior nerve-roots of each nerve, arising along the back of the sac, are separated horizontally by a considerable interval (in which lies the falciform process of pia mater described), and the same is true of the anterior roots of each pair of nerves. It is to be concluded, therefore, that the nervous tissue of the spinal cord, much thinned out, extends commensurately with the area so included, being limited on either side by the line of attachment of the posterior roots. The sac contained a considerable quantity of lymph which lined its interior, and ensheathed the nerves passing through it. During life the swelling presented externally a shallow median furrow, due probably to the resistance here offered by the nerve-roots passing through the middle of the sac to the intervertebral foramina.

Around such a specimen almost all examples of meningo-myelocele may be grouped, the main exception being that in which the central canal is greatly distended and the expanded cord lines the interior of the sac (syringo-myelocele), a condition which will next be noticed.

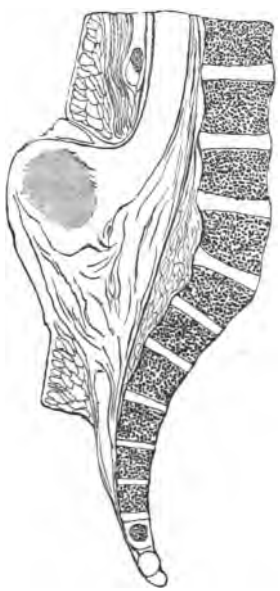
3. **Syringo-myelocele.**—The cases in which the cavity of the sac is a dilatation of the central canal of the cord are very rare. Among the 125 specimens examined, we have only found two unequivocal examples. One is in University College Museum (Specimen No. 8), and was situated in the sacral region. The sac, collapsed and wrinkled, measures 1·5 cm. in diameter; its interior is lined with soft pulpy substance, and neither cord nor spinal nerves pass through its cavity; the innermost lining of the sac is well defined and fibrous. On dissecting off this latter a series of nerves is displayed coursing round its outer surface and connected with it; at the mouth of the sac the dura mater is distinct and lies superficial to the innermost membrane referred to. The other is in St. Bartholomew's Hospital Museum, No. 3481 (Specimen No. 9). It will be observed from the description that the spinal cord and nerves do not lie within the sac, from which it might be concluded that the specimen is one of meningocele simply. Dissection, however, shows that there exists an innermost well-defined membrane, on removing which the nerve-roots are exposed lying in loose connective (? subarachnoid) tissue between the first-mentioned membrane and the other component layers of the sac-wall. In this manner the nerve-roots pass round the sac towards the intervertebral foramina. A minute foramen leads from the sac into the substance of the cord, and presumably represents the central canal of the cord. Such a specimen illustrates the difficulty of diagnosing, without dissection, between a simple meningocele and a dilatation of the central canal. The anatomy of the three other specimens referred to in the table is too doubtful to permit a fuller description.

It is well known that the central canal of the cord above the protrusion is not infrequently dilated, but we would point out that this condition is not confined to spina bifida, and may sometimes exist to a very considerable degree, and even extend throughout the cord without being accompanied by deficiency in the neural arches (syringo-myelus). In Specimen No. 3 (Glasgow Royal Infirmary Museum, No. 145) this condition is observed above the protrusion, at the seat of which, however, it

abruptly terminates (Plate XIII). Hence it must be concluded that such a condition above the protrusion affords no criterion of the exact anatomy of the sac.

From Specimen No. 10 (St. Thomas's Hospital Museum, No. LL 13[1]), a partially cured case (Fig. 1), it would appear as if a dilatation of the central canal might affect the portion of cord lying within the sac, in cases where otherwise the anatomy is that described under the preceding heading of meningo-myelocele, and shown in Plate XV.

FIG. 1.



A partially cured spina bifida. The shaded space represents an unobliterated portion corresponding to a dilatation of the central canal.

We may next proceed to notice the anatomy in more detail under the headings already alluded to (p. 340).

(1) *Position of the tumour.*

An analysis of the 125 specimens examined shows the various regions of the spine to have been affected as follows:

The whole spine in 1 case, ·8 per cent.

The dorsal region in 9 cases, 7·2 per cent.

The lumbar region in 9 cases, 7·2 per cent.

The dorsi-lumbar region in 3 cases, 2·4 per cent.
The lumbo-sacral region in 68 cases, 54·4 per cent.
The sacral region in 21 cases, 16·8 per cent.
The dorsi-lumbo-sacral region in 7 cases, 5·6 per cent.
The sacro-coccygeal region in 1 case, ·8 per cent.
Not determinable in 6 specimens.

(2) *Form and size of the sac.*

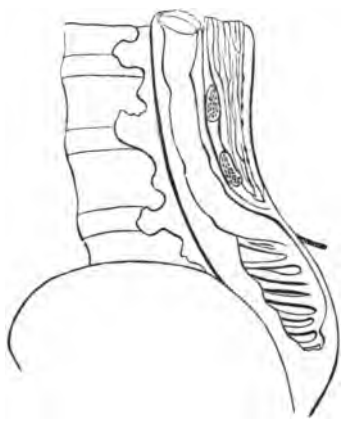
In the great majority of cases, at the time of birth, the size of the sac rarely exceeds that of a Tangerine orange, *i.e.* from 3—5 cm. in diameter. As a tumour of exceptional size at birth may be noted Specimen No. 10, in which the sac measured 8 cm. across, 8·5 cm. longitudinally, and 7 cm. from before backwards; the child was born dead. In some cases the size does not exceed that of half a walnut. In a few cases, where the patients reach adult life, the tumour may assume very large proportions, as in a case shown to the Society on November 22, 1884, sent from Portland by Dr. McLean. The tumour, which was situated in the lumbo-sacral region, measured round its base, where it was somewhat constricted, 22½ inches (57 cm.) In King's College Museum there is a cast (No. 1744) of a spina bifida, which measures 27 inches (69 cm.) around its base.

In form sessile, somewhat constricted at the base, circular, ovoid or cordiform in outline, these tumours occasionally present a slight median longitudinal furrow, or sometimes a more localised depression at or near the summit—the so-called umbilicus. The latter indicates the spot at which the spinal cord meets and becomes incorporated with the posterior wall of the sac; but whilst its presence indicates that the tumour is a meningo-myelocele, its absence cannot be held to show the contrary. The same is true of a median furrow; this corresponds to the attachment of the cord and series of nerve-roots (Plate XV). By no means constant, these depressions depend much on the distension of the sac, the free parts yielding to the pressure within more readily than those which are tied down to the vertebral column by the cord and nerves. The presence of furrows, however, in the sac is of importance not only as evidence that the spinal cord and nerves are incorporated with its walls, but as showing the possible existence of internal partitions. A median furrow may indicate a median partition of the sac, while a bilateral longitudinal furrow may indicate the presence of a double partition or the subdivision of the sac into a central and two

or more lateral portions; to these points fuller reference will be made further on.

Still more rarely the surface of the protrusion presents not an "umbilicus," but a more clearly defined and deeper depression, an excellent example of which is represented in Plate XVI, from Specimen No. 13 (Middlesex Hospital Museum, No. 725). A dissection of this specimen has shown that the depression corresponds precisely to the point at which the cord meets the sac, and a probe passed through it leads into the mid-substance of the cord, from which it may be inferred that this foramen represents the opening of the central canal of the cord upon the surface of the tumour, as shown in outline in Fig. 2.

FIG. 2.



A profile view of the specimen figured in Plate XVI, showing origin of nerve-roots from the posterior wall of the sac.

The importance of this fact in relation to the developmental morphology of the malformation will be noticed elsewhere.

(3) *Coverings of the sac.*

With regard to the character of the external investment, it is very seldom that a normal cutaneous covering exists over the whole protrusion. As a rule, the base alone is covered with normal skin, while in the rest of its extent the covering consists of a thin white glistening membrane-like tissue. In some specimens the skin ends abruptly, in others it merges gradually into the membrane noticed. More rarely, however, the cutaneous covering is continuous over the whole tumour, even

when large, as in Specimen No. 14 (St. Mary's Hospital Museum, $G\frac{4}{8}$). We have seen this condition in a living adult, with a tumour measuring $22\frac{1}{2}$ inches (57 cm.) round its base, and in which case subsequent post-mortem examination showed the spinal cord and nerves to be involved in the sac-wall.

Very little can be judged regarding the presence or absence of ulceration, and the vascularity of the sac-wall from museum specimens. At birth in a considerable proportion of the cases the summit of the sac is more or less "raw;"* occasionally it is even gangrenous. Specimen No. 19 (St. Bartholomew's Hospital Museum, No. 3480) shows the separation of such a slough and the consequent exposure of the cord and nerve-roots which lay within the sac.

In Specimen No. 15 (St. George's Hospital Museum, 15 *a*) the summit of the sac over an area of about 2·5 cm. presents a minutely shreddy surface bounded by a sharply-defined, slightly overhanging edge formed by the tissue composing the surrounding part of the sac, as though another superimposed membrane had been torn off. In support of this view it may be noticed that specimens of anencephalus are by no means uncommon in museums, in which the head of the fœtus is adherent to the membranes. Considered in its immediate connection with the spinal cord and nerves of the sac, great importance attaches to the exact nature of this membrane-like area, and this is further increased when the opening of the central canal at the higest part of the area, as seen in Specimen No. 13 (Plate XVI), previously referred to, is recalled to mind. It will be remembered also that in the typical specimen first described (Plate XV) the nerves were attached in regular series in two longitudinal lines to this membrane, and that between the anterior and posterior roots lay a continuation of the ligamentum denticulatum, unusually exaggerated in development. There is strong presumptive evidence, therefore, that the membrane in question contains the attenuated neural tissue of the spinal cord from which the nerves lying within the sac really take origin. This probability is strengthened when it is remembered that the cord, if the protrusion occurs in the dorsal region, may be found directly continuous with the membrane and normally constituted in

* In the majority of cases, this raw surface, confined to the most prominent part of the sac, is sharply limited, and the presence of blood upon or within it suggests the possibility of sudden rupture of an adhesion between it and the fœtal membranes.

the lowest part of the sac, from which it is continued below into the intact portion of the vertebral canal. We have observed this condition in two specimens. In one, which was not preserved, the tumour was situated in the mid-dorsal region and presented the typical anatomy already described. In the other Specimen, No. 16 (St. Thomas's Hospital Museum, LL 12[1]), the protrusion involved the spine between the tenth dorsal and fourth lumbar vertebræ, and is represented in Fig. 3.

FIG. 3.

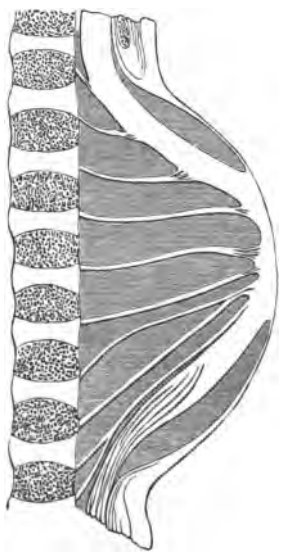


Fig. 3 shows a normal constitution of the cord below the area of its incorporation in the sac-wall.

As strictly analogous examples of this condition of the median part of the sac-wall may be mentioned anencephalus and ectopia of the abdominal viscera. In both these conditions the skin is continued into a similar membraniform tissue, independent of the amnion, the proper appendages of the skin —hairs, glands, &c.—ceasing abruptly at the junction of the two. From this consideration it may be assumed that the same explanation—whatever that may be—is common to all the cases.

(4) *Disposition of the meninges.*

In all the specimens minutely examined it has been possible to trace the arachnoid through the neck of the sac on to its interior, in some cases almost to the summit; in others it has ceased to be separable shortly after entering the sac. This last fact should be remembered in determining the nature of the sac-wall at some distance from its mouth. The fluid, therefore, in these cases is contained in the subarachnoid space.

The fluid withdrawn from cases of spina bifida hardly needs any description in this report. We nevertheless append three analyses, which were kindly undertaken for us by Dr. Halliburton, of fluid from cases referred to in the Report.

Report on the chemical investigation of Cerebro-spinal Fluid from cases of Spina Bifida. By W. D. HALLIBURTON, M.D., B.Sc., Sharpey Physiological Scholar, University College, London.

(From the Physiological Laboratory, University College, London.)

CASE.—Female, æt. 19 years.

The liquid was very faintly opalescent; this opalescence was not at all lessened by filtration.

Its specific gravity was 1007.

It was very faintly alkaline.

Total solids.—The percentage of solids was found to be 1·025. This includes organic and inorganic matter.

The salts consisted of sodium chloride, the most abundant, phosphates, and carbonates. No sulphates were present.

Proteids.—The percentage of proteids was found to be ·0842: that is only a small fraction of the total solids consists of proteids.

Boiling the liquid increases the amount of opalescence originally present.

Boiling after rendering the liquid faintly acid with weak acetic acid produces a small amount of flocculent precipitate.

On saturating the liquid with powdered magnesium sulphate, a precipitate of proteid was obtained; it is the characteristic of proteids of the globulin class to be precipitated in this way; the precipitate was washed, collected, re-dissolved; the solution was opalescent, like solutions of globulins generally: on heating it coagulation was found to occur at 75° C. Thus this globulin resembles, and probably is identical with the globulin of serum, or paraglobulin. The fact that a globulin is present in such relatively large quantities to other proteids explains the fact that the original liquid was opalescent.

Nearly all the proteid present consists of globulin; on filtering off the precipitate produced by the addition of magnesium sulphate, a clear filtrate was obtained; on boiling this even after rendering it faintly acid, no precipitate took place; but on the addition of nitric acid, a very faint precipitate occurred, which turned yellow on boiling, and orange on the addition of ammonia. This proteid, which is present in exceedingly minute quantities, is peculiar in not being coagulable by heat. It is, however, not a peptone, as it does not give the characteristic colour with copper sulphate and potash, and moreover peptones are not precipitable by nitric acid.

Carbo-hydrates.—A small quantity of a substance which reduces copper salts, like dextron, is present. It, however, is present in very small quantities; three hundred cubic centimetres were taken, the proteids coagulated filtered off, and the liquid concentrated; this was added to two cubic centimetres of Fehling's solution, but were insufficient to produce the reduction of all of it. The quantity of cerebrospinal fluid that I had was insufficient to enable me to add more. Two cubic centimetres of Fehling's solution require for their complete reduction ·01 gramme of sugar; three hundred cubic centimetres contain therefore less than ·01 gramme of sugar; that is the percentage is less than ·003. It may be put approximately as ·002, as the amount of copper not reduced was very small.

Fats.—A small amount of fat is present, ether extracting a small amount: the quanity was not estimated.

The composition of the fluid may therefore be thus expressed in a tabular way: the numbers are parts per 1000:

Water	989·75
Solid matters	10·25
Proteids (consisting almost exclusively of globulin) .	·842
Sugar (approximately) . . .	·002
Extractions and soluble salts . . .	9·406
Insoluble salts	·218

Cerebro-spinal Fluid.

Case.—Emily F—, æt. 11 days. Lumbo-sacral spina bifida. *First tapping.*

The fluid was perfectly clear, with a faint yellow tinge: its reaction was slightly alkaline.

Boiling rendered the liquid slightly cloudy.

Trommer's test showed that a distinct trace of sugar was present.

The following is the composition of the fluid (in parts per 1000):

Water . . .	919·877
Total solids . .	10·123
Proteids* . .	1·602
Soluble salts . .	7·544
Insoluble salts . .	·346
Extractions . .	·631

In the above calculation the specific gravity of the fluid was taken at 1007, the average specific gravity of cerebro-spinal fluid. The quantity of fluid obtained in this case was too small to admit of the specific gravity being taken in the usual way.

Case.—John S—, æt. 13 weeks. *Fourth tapping.*

Iodine had been injected on three previous occasions.

Fluid.—Perfectly clear, colourless, faintly alkaline.

The composition of the fluid, in parts per 1000, was as follows:

Water . . .	991·658
Total solids . .	8·342
Proteids . .	0·199
Sugar . . .	0·165
Extractions (minus sugar) .	2·863
Soluble salts . .	4·776
Insoluble salts . .	0·339

* This proteid contains globulin as is seen by the fact that saturating with magnesium sulphate produces a precipitate.

The points of interest in the above are as follows:

1. Proteids, diminished in quantity as compared with other specimens. They consisted ENTIRELY OF GLOBULIN, which coagulated at the same temperature as that at which serum globulin or paraglobulin coagulates (75° C.).
2. Sugar, *very greatly increased* in quantity.
3. Soluble salts, diminished in quantity, as are also the total solids.

These analyses are very similar to those previously made by Hoppe Seyler. Hoppe Seyler's analyses are appended.

Analysis of the Cerebro-spinal Liquid, obtained by puncture in two cases of Spina Bifida (Hoppe Seyler).*

	I.			II.	
	1st puncture.	2nd puncture.	3rd puncture.	1st puncture.	2nd puncture.
Water . . .	987·49	986·88	986·72	989·33	989·89
Solid matters .	12·51	13·12	13·28	10·67	10·20
Proteids . .	1·62	2·64	2·46	·25	·55
Extractions and salts	10·52	11·30	11·14	10·42	9·65

(5) *Size and configuration of the deficiency in the neural arches, and of the aperture of communication between the sac and the vertebral canal.*

As a general rule, it may be stated that the size and shape of the tumour depend on the extent to which the neural arches are deficient. In no specimen have we observed the deficiency limited to a single vertebra or even to two vertebræ. In the greater number of the dried specimens examined, the cleft is found to involve several vertebræ; most usually the last lumbar, and all the sacral. Perhaps the most important fact to be noticed is that the actual extent of the protrusion forms no *certain* criterion of the extent of deficiency in the neural arches; for in Specimen No. 17 (R. C. S. 271 *e*) the deficiency involves the lowest four dorsal, and the whole of the column below, while during life the protrusion, measuring 5 cm. by 6 cm. in its largest dimensions, was limited to the lumbo-sacral region.

As a rule, in the highest parts of the cleft, the laminæ are fairly well developed, and may be united by a dense fibrous membrane; lower down they become stunted and ill-formed. In extreme cases they are so everted as to lie in a transverse vertical plane, and the normal convexity of their inner surface is slightly exaggerated. This alteration in direction is associated

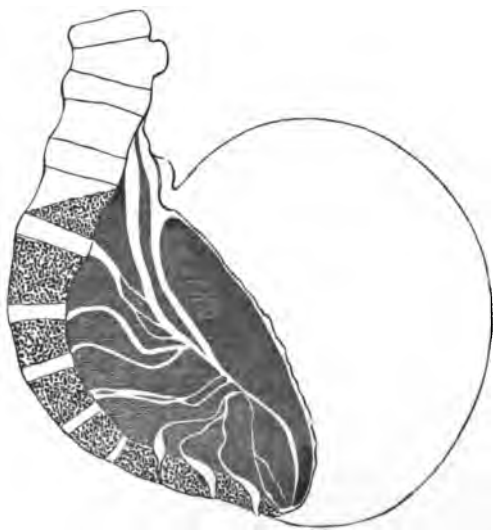
* Hoppe Seyler, *Physiologische Chemie*, p. 601.

with a diminution of the angle of junction between the pedicle and the lamina. It may be noted that the actual size of the aperture by which the sac communicates with the sound portion of the vertebral canal, and thence with the central nervous system, however large the cleft in the spinal column may be, can never exceed the size of the neural ring of the last entire vertebra, and that it is generally very considerably lessened by the deposition of lymph.

(6) *Disposition of the cord and nerves within the sac.*

In the total of 125 specimens examined, leaving out of consideration the cases of syringo-myelocele, it has been found that the cord enters the sac in 79 cases, *i. e.* in 63·2 per cent. The level at which the cord crosses the sac is variable; as a rule it is somewhat above its centre. Occasionally, however, the cord, as it emerges from the vertebral canal, becomes immediately connected with the roof of the sac without the intervention of any space.

FIG. 4.



An unusually large portion of the sac above the cord.

In other cases, the portion of sac above the cord forms the chief part of the whole as in Specimen No. 14 (St. Mary's Hospital Museum, No. G[4]), represented in Fig. 4.

In many specimens, the cord is attached to this upper portion of the sac by a falciform fold of membrane, which forms a median septum, imperfect anteriorly. The contained cord varies in size, sometimes it seems to have its normal size, in other cases it appears attenuated from the traction made on it by the distending sac. Beyond its point of attachment, though incorporated with the sac-wall, it cannot be further traced by dissection.

In a typical specimen taken from a child twelve days old, sections of the spinal cord, including pia mater and nerve-roots crossing the sac, are 4 mm. in their longer and 2 mm. in their shorter diameter at 1 cm. from its attachment to the sac-wall. Under the microscope they display well-marked grey matter of normal structure containing groups of normal ganglion cells, the chief of which lie in the anterior horns; other healthy cells occur scantily scattered through the rest of the grey matter. The central canal is transversely elongated; in places it tends to be T-shaped. The white matter contains groups of well-developed medullated fibres, but these in the greater part are very small and in many parts axis cylinders alone are detectable; the neuroglia corpuscles are normally distributed through the grey and white matter. The sections of the nerves lying upon the cord are quite normal.

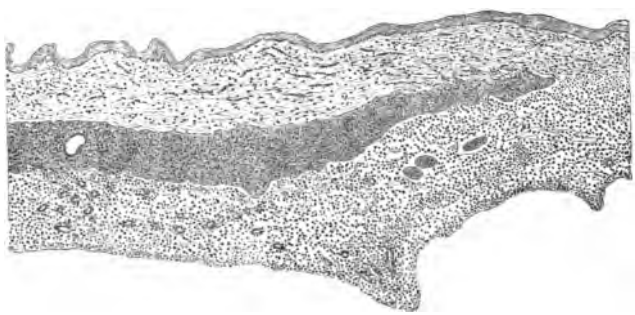
In Specimen No. 18 (Charing Cross Hospital Museum, No. 250 B) the sacral part of the cord impinges on the centre of the sac-wall, while the nerve-roots run forward applied to the cord; very few only arise from the sac-wall and these are in the immediate neighbourhood of the attached cord.

The disposition of the nerves varies with that of the cord; sometimes those arising from the cord, as it crosses the sac, are directed forwards upon it to reach their proper intervertebral foramina, as in Plate XV, whilst those arising from the sac-wall are attached in double series, the anterior and posterior roots being often quite distinct and partially separated by a well-defined falciform fold, corresponding in position to, and continuous with the ligamentum denticulatum. After the nerves perforate the dura mater to reach the intervertebral foramina, they present the normal anatomy; the ganglia on the posterior roots, and the posterior primary divisions of the nerves have the usual disposition; in some cases, however, the ganglia are found displaced within the vertebral canal. In the Cambridge Museum there are specimens showing approximation and fusion of two or more sacral ganglia, a result appa-

rently due to the backward traction made upon the nerves by the distending force within the sac.* In one instance, Specimen No. 19 (St. Bartholomew's Hospital Museum, No. 3483), the lowest nerve-roots are furnished with "*ganglia aberrantia*" within the sac; the conus medullaris terminates in a filament 2 mm. in diameter, upon the lower end of which is a well-marked gangliform enlargement.

Considering the close connection between the spinal cord (and nerve-roots) and the median portion of the sac-wall, the importance of a histological examination of the latter will be evident. Transverse sections made through the sac-wall 1 cm. below the point at which the spinal cord becomes incorporated show an expanded extension of the latter in the fibrous tissue forming the rest of the sac-wall (Fig. 5). The

FIG. 5.



Microscopic section of sac-wall in the middle of which lies a portion of the neural tissue of the flattened cord. A complete central canal will be seen near the left hand extremity.

central canal is seen to be continued through the centre of the extension; its epithelium presents the normal characters. In other sections the canal is much flattened or otherwise

* Professor Humphry has kindly favoured us with the following particulars of these cases:

1034A. Acephalus and Spina bifida in the whole length of the column. Cervical ganglia in one mass on each side. Ganglia in other parts separate.

1035. Spina bifida in sacrum. Three upper sacral ganglia on either side united so as to form one trilobed ganglion enclosed in one sheath of dura mater.

1036. Like preceding.

1037. Spina bifida in the sacrum. The two upper sacral ganglia on the right side and the three upper on the left are united.

1038. Spina bifida in the upper part of the sacrum. The lowest lumbar and the first sacral ganglia on the left side are united.

altered in form. The flattened tissue of the cord gives origin to anterior and posterior nerve-roots, which at their origins are contained within the fibrous tissue of the sac-wall, and most conspicuous on the posterior aspect of the cord. The drawing shows two nerve-bundles in section lying in front of the cord. The origin of these from the cord is distinctly traceable in other sections. The epidermis is continuous over the middle line, but the skin in this situation presents no hairs or other appendages. The nervous tissue is of uniform structure, presenting no division into white and grey parts; it consists throughout of corpuscles uniformly scattered through a finely fibrillar basis, the whole having the general appearance of neuroglia. Groups of normal ganglion cells, however, occur here and there in the sections; the nerves lying in the sac-wall in the same manner present no medullated fibres, but resemble closely in structure the tissue forming the expanded cord. It may be inferred from these appearances that the development of medullated fibres has not been perfected.

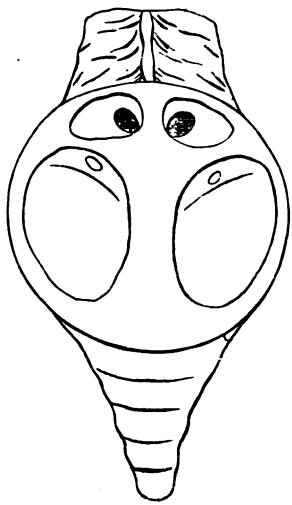
(7) *Unusual variations.*

(*a*) Subdivision of sac, complete or partial, by partitions. Of great importance in relation to treatment by injection is the occurrence of partitions within the sac. These partitions have various arrangements, and exist in different degrees; at times some portions of the sac are completely closed off from the rest, but in most cases small fenestræ or apertures of communication exist in the septa. The presence of these subdivisions will have the effect of confining fluid to the space into which it has been injected. That they do not result from operative interference is proved by the fact that in the cases in which they have been found most marked, no treatment had been at any time adopted.

The chief partition may be median and include the cord lying within the protrusion, together with the nerves passing forwards from the median portion of the posterior wall of the sac to the intervertebral foramina. In one specimen of this kind, which we dissected (Fig. 6) the lateral portions of the sac were symmetrically subdivided by a horizontal partition which crossed them about the junction of their upper and middle thirds; in the centre of each partition there was a small well-defined oval aperture. In other cases the partition is multiple and consists of two main lateral parts; the partition of each side corresponds in position to the series of nerve-roots;

the lateral cavities, thus more or less closed off from the central portion, may be subdivided by cross partitions into

FIG. 6.



A multilocular spina bifida.

many secondary spaces. Plate XVII, Specimen No. 20 (University College Museum, No. 5195). As a rule, all the subdivisions of the sac communicate by small well-defined circular openings, but they may however be completely closed.

(*b*) Presence of bony outgrowths across the spinal canal in the neighbourhood of the tumour.

The most noteworthy variation in the pathological anatomy of spina bifida is one of which we have seen four examples. A fifth specimen exists in the Musée Dupuytren in Paris.

This variation consists in the presence of a distinct osseous or osseo-cartilaginous process, crossing the vertebral canal in an antero-posterior direction. The specimen in St. Thomas's Hospital Museum, No. 21 (Plate XVIII), represents very clearly the osteological aspect of the variation, and that No. 22 in the R. C. S. Museum the associated condition of the spinal cord (Plate XIX) which presumably existed also in the former case. In Specimens Nos. 20 and 23 in University College Museum (Plate XVII) and in St. Bartholomew's Hospital Museum a similar bony process is present, but the spinal cord

is bifid, the bony process lying in its cleft and the halves of the cord diverging as they enter the highest part of the sac.

It is interesting to observe, in the St. Thomas's Hospital Specimen (Plate VI) that there is a duplication of the centra of the bodies of the last two dorsal vertebræ with which the intercalated element is connected. This appears to be related to the condition in which a portion of the vertebral column is completely cleft, of which Specimen No. 24 (R. C. S. Museum, No. 277) offers a good example. This is the skeleton of a human hydrocephalic fœtus with spina bifida involving the dorsal, lumbar, and sacral regions. In this specimen the bodies of the vertebræ are deficient, so that the spine is completely cleft as high as the cervical region. The bodies of the lower cervical vertebræ have two centres, these serving apparently as points of departure for the bifid column below; there is no exostosis crossing the canal at the point of division. Below the point of division the vertebral canal on the inner side of either half is unclosed by the formation of laminæ in this situation. It is to be regretted that the soft parts in this specimen are wanting.

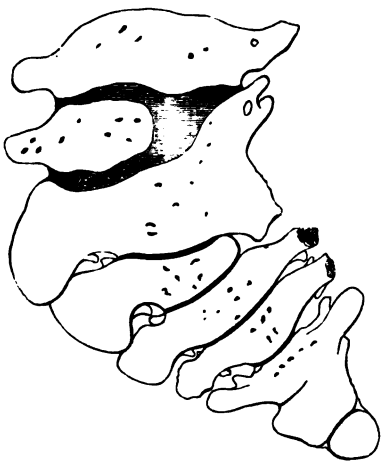
There are no specimens in the museums referred to of anterior spina bifida, that is, of protrusion through a cleft in the bodies of the vertebræ, though such specimens are described by a few authors, and it is possible that in the case last referred to some allied condition of the soft parts existed.

As another somewhat uncommon condition of the osseous system may be noticed that in which there is defective development of one or more of the vertebral bodies at the seat of the cleft. Specimen No. 25 (Plate XX) represents this condition. The sacrum is twisted to the left owing to a malformation of its upper three segments (Fig. 7), consisting in a partial absence of the left halves of the first and third segments, while the left half of the second segment is produced upwards and downwards, as if in some measure to compensate for the deficiency. Specimen No. 26 (R. C. S. Museum, No. 278) shows a double lateral curvature in the cervical region of a fœtus, due to a similar cause; the vertebral canal is throughout unclosed, and the cleft extends also between the supra-occipitals.

In some cases unnatural curvatures in an antero-posterior plane exist. Specimen No. 27 (R. C. S. Museum, No. 274) shows the six lower dorsal, the lumbar and sacral vertebræ of a newborn child. In the lumbar region the spinal column is

strongly concave anteriorly; that this is a permanent condition of some standing is shown by the fact that the body of the

FIG. 7.



Anterior view of the parts represented in Plate XX (reduced).

vertebra at the summit of the curve is so reduced in thickness anteriorly that the contiguous intervertebral discs lie almost in contact.

(8) *The process of cure.*

a. Meningocele.—There are no specimens of meningocele cured by injection in the London museums. Plate XIV, fig. 1, from Specimen No. 4 (Western Infirmary Museum, Glasgow, Series 1, No. 2), represents the parts concerned after cure by Dr. Morton's method. Specimen No. 22 (R. C. S. Museum, Plate XIX) is a meningocele, into the mouth of which the posterior part of the cord has prolapsed. The sac, which was ligatured during life, is completely filled with a homogeneous fibroid tissue, a microscopic examination of which shows it to be composed of delicate connective tissue containing abundant corpuscles.

b. Meningo-myelocele. The following is a description of the appearances seen in the case of an ordinary lumbar spina bifida after cure by the injection of Dr. Morton's fluid. Specimen No. 28 (R. C. S. Museum, No. 276*a*). Externally there is a deep puckered depression at the centre of the sac which is every-

where covered with cuticle; this cuticle is smoother and more shining over the centre of the sac than elsewhere. On section (Plate XIV, fig. 2) the sac cavity is seen to be obliterated by a mass of fibroid tissue, bounded in front by dura mater: the spinal cord crosses the highest part of the sac in the midst of this fibroid tissue. In the lower part of the sac anteriorly are sections of the nerve-roots passing towards the intervertebral sacral foramina. The cord above the sac appears healthy.

From this specimen it may be concluded that the obliteration of the sac is effected by an inflammatory effusion into the sac, and its subsequent organisation. The microscopic examination of the obliterating material (the child died 11 weeks after the first injection) shows it to be composed of young fibrous tissue, elongated corpuscles lying amongst wavy fibres of connective tissue; in places the corpuscles are more numerous and spheroidal, the development of the tissue being less advanced.

c. Syringo-myelocele. In another specimen, No. 10, a similar process of obliteration had taken place in the greater part of the sac after treatment by the same method. In the upper and median portions (Fig. 1, page 345) there persisted a small unobliterated space, which on section proved to be due to a dilatation of the central canal of the cord within the sac (St. Thomas's Hospital Museum, LL 13[1]).

(9) *Theoretical considerations drawn from the anatomical facts.*

The points of prime importance disclosed by the microscopic examination of the sac-wall in a typical case of meningo-myelocele are the continuation of the central nervous system within the median portion of the sac-wall, the integrity of the central canal of the cord within this part, as also the absence of true skin over it. Moreover, it displays the absence of any meningeal cavities behind the incorporated portion of the spinal cord. It is clear, therefore, in the first place, that the nerve-roots which traverse the sac arise from this intramural portion of the central nervous system, and that all expressions of descriptive pathological anatomy which imply a distribution of the nerves *to* the sac-wall are a reversal of the facts, since the sac-wall is really their source, and the nerves within the sac are the proper anterior and posterior roots. But more important than this, the histology of the sac-wall in a typical case (Fig. 5), by demonstrating the integrity of the

central canal of the included portion of the cord, settles beyond doubt what must otherwise be matters of conjecture only, that neither does the neural furrow* remain unclosed in spina bifida, nor, after having been closed, is it subsequently distended by dropsy and ruptured, this rupture being accompanied with the disappearance of that portion of cord (with or without the superjacent integuments) which lies behind the line of origin of the nerve-roots from it. Lastly, this examination serves to complete the refutation of the view held by Förster and many subsequent German writers, viz. that spina bifida in the great majority of cases is due to a dropsy of the central canal of the cord.

The absence of true skin from the central portion of the sac-wall which occurs in almost all cases of meningo-myelocele, as it does also in anencephalus, is of much interest. The surface in question we do not regard as cicatricial, that is, as following an ulceration of normally-formed skin, since there is no histological evidence of any inflammatory process. It is not denied that ulceration may not in some cases be superadded. The junction of the skin proper with the membranous area may be most aptly likened amongst natural structures to that of the skin with the amnion round the attachment of the umbilical cord. The membrane implies, in fact, that the mesoblastic basis of the true skin and the structures lying in subjacent connection with it has not been developed. Among pathological conditions, besides anencephalus, related to this condition may be cited ectopia of the viscera, the membrane which directly encloses them being continuous with the true skin in the same manner, and representing the ill-developed substitute of the cutaneous and muscular systems of the anterior body wall.

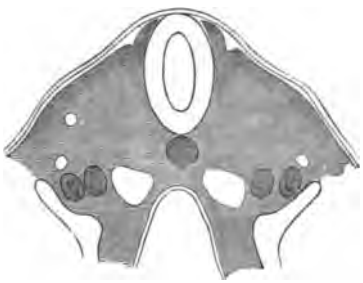
The presence of the spinal cord within the sac-wall in the case of spina bifida offers no difficulty of explanation when the epiblastic origin of the central nervous system is remembered. The theory therefore which best explains the pathological anatomy of spina bifida is that which assumes a primary defect of development of the mesoblast from which the structures closing in the vertebral furrow are developed. After the closure of the neural furrow it would appear that the processes of mesoblast which subsequently insinuate them-

* By NEURAL FURROW is meant the groove of involuted epiblast from which the spinal cord is developed. By VERTEBRAL FURROW is meant the groove resulting from the extension backwards, from the protovertebral mass of the processes of blastema from which are developed the neural arches and membranes of the spinal cord.

selves between the primitive spinal cord and its overlying epiblast are formed in an insufficient degree to meet and combine, or that these processes, should they meet, are not formed in

Diagrams representing the formation of spina bifida.

FIG. 8.

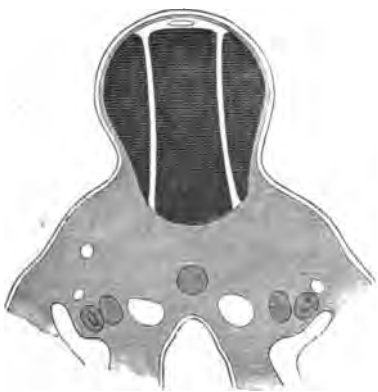


Normal disposition of the parts at 3rd day before closure of the vertebral furrow (after Kölliker). $\frac{40}{1}$.

FIG. 9.

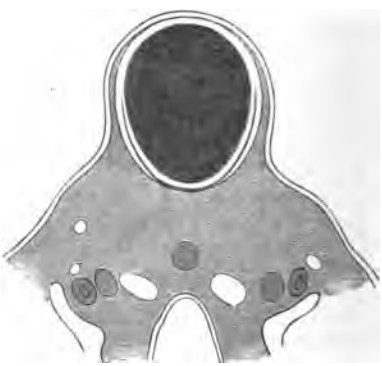
Shows accumulation of fluid in front of spinal cord, no membranes having been differentiated behind.

FIG. 10.



Shows displacement backwards, and flattening of the cord with stretching of the nerve-roots from further accumulation of fluid.

FIG. 11.



Shows dilatation of the central canal of the cord, the nerve-roots passing round its outer surface.

sufficient proportion to serve as a basis from which the various structures subsequently to be produced over the spinal cord can be developed. Hence different degrees of deficiency are

met with, from those in which the spinal cord lies within the posterior wall of the sac, scantily embedded in fibrous tissue (covered with epidermis), to those in which a perfect cutaneous covering exists over the whole sac with, it may be, an abundance of fat in the subcutaneous tissue. Muscular tissue, however, does not appear to be produced in the median portion of the sac-wall, *i.e.* there is no extension of the muscle plates to the mid-line; whilst the absence of a mesoblastic basis for chondrification and osseous formation constitutes, according to the definition, a *sine quâ non* of the malformation. Among other evidences of arrested development of the neural arches may be noticed the association of partial deficiency of the vertebral bodies, corresponding with the situation of the protrusion, Plate XX (R. C. S. Museum, No. 271A), and also Specimen 26 (R. C. S. Museum, No. 278).

The absence of meningeal spaces behind the cord, to which in meningo-myelocele we know of no exception, may be explained on the ground that no sufficient mesoblastic basis has been produced for the formation of the spinal membranes, as there appears also to be no proper basis for the development of the corium, since although the epidermis is continued across the sac, it is not involuted to produce the appendages proper to normal skin. It is of importance to distinguish between the summit of the sac and the other parts. The developmental defect is limited to the former, the rest of the sac being produced by the displacement of the healthy parts during the process of protrusion of the spinal cord, with which the superjacent tissue is incorporated. In the rare cases in which the central canal of the cord opens on the summit of the sac, Specimen No. 13 (Middlesex Hospital Museum, No. 725), Plate XVI, it may be assumed that the neural furrow has never been closed in, and that the median portion of the sac below this is represented by the attenuated tissue of the spinal cord furnished only perhaps with an epithelial investment. In this process of displacement it is clear that the nerve-roots connected with the cord will be drawn backwards with it, their ultimate position within the middle of the protrusion being thus satisfactorily accounted for (*vide* diagrams). There will be no difficulty in explaining the presence of cord elements, even in the lowest part of the sac, when the very early age from which the malformation must date is considered; for at this time the spinal cord is commensurate in length with the vertebral column. The great proportional frequency of the protrusion in the lumbo-

sacral region may be associated with the fact that it is in this region the normal closure of the neural furrow last occurs.

Those cases in which the membranes only protrude indicate a far less severe defect in the developmental process. The cord and its membranes, together with the overlying soft parts, are normally formed, and the protrusion appears to be like a hernia in other parts, occurring through an unsupported spot of the confining cavity; the cord is retained in position by the nerves passing from it, and being quite free of its membranes no traction is made upon it by the protrusion, and it retains its normal position or is but slightly displaced into the neck of the sac.

In syringo-myelocele, the original deficiency would appear to be the same as that described under meningo-myelocele, but the accumulation of fluid here occurs within the central canal to the exclusion of that within the subarachnoid space.

Descriptive List of the Specimens Referred to in the Foregoing Part of the Report.

The specimens are placed in the order, in which they are referred to in the body of the Report.

No. 1.—*Royal College of Surgeons,* No. 271.—The lower portion of the vertebral column of a child; dry specimen. A thin sac, issuing by a narrow neck not wider than a goose-quill in the lower lumbar region. The laminæ of the last lumbar and of the sacral vertebræ are deficient, the arches being completed by membrane.

No. 2.—*St. George's Hospital,* No 15 H.—The sac of a meningocele removed by excision; it consists internally of two divisions, the capacity of one being about two drachms, that of the other about four drachms. Each division has a separate orifice of communication with the spinal canal, of the size of a large goose-quill; the two apertures in the divided surface of the pedicle are about a sixth of an inch apart. From a patient, aged 5 months, under the care of Mr. Holmes; the tumour, which was increasing rapidly, was removed under the carbolic spray.

No. 3.—*Glasgow Royal Infirmary,* No. 145.—Dr. Newman thus describes it: "Spina bifida and hydrorachis interna situated about the middle of the dorsal region." Plate XIII shows the preparation in section. The dilated central canal (*a*) is seen to occupy nearly the whole thickness of the cord, and is traversed by transverse bands of nerve tissue (*b*), which form incomplete septa. The hydrorachis interna ceased abruptly about a quarter of an inch above the sac, with which it does not in any way communicate. A small communication exists between the sac and cavity of the spinal membranes. A careful examination showed the complete absence of nerve tissue from the wall of the sac.

No. 4.—*Glasgow Western Infirmary,* Series 1, No. 2.—The following account of this specimen was kindly furnished by Dr. Coats. A solidified spina bifida in the dorsal region. The tumour forms a flat mushroom-like mass with a narrow pedicle, which passes between two of the spinous processes (Plate XIV, fig. 1), and is continuous with the dura mater spinalis. On palpation it does not appear that any of the proper substance of the cord passes out, its contour being preserved. The case was successfully treated by the late Dr. J. G. Lyon.

No. 5.—*St. Bartholomew's Hospital,* No. 3486.—The sac of a meningocele successfully removed by operation (*vide* Dr. Wilson's Report, *Path. Soc. Transactions,* vol. xiv). There is a longitudinal depression, smooth and unpigmented on the summit, with no corresponding eminence on the opposite surface. Microscopic sections show this depressed part to be composed of fibrous tissue, continuous with the corium, and furnished with an epidermal investment.

No. 6.—*St. Bartholomew's Hospital,* No. 3483.—The parts concerned in a sacro-coccygeal meningocele, after loss of the sac-wall. The extremity of the cord and the cauda equina, occupying the lower portion of the neural canal, are completely exposed; the cornus medullaris terminates in a filament, which presents a gangliform enlargement two mm. in its shorter diameter near its lower end. The lowest nerve-roots are furnished with "ganglia aberrantia." The aperture is bounded by an irregular ulcerated margin of skin.

No. 7.—*Royal College of Surgeons,* No. 273 B.—A specimen (Plate XV) illustrating the typical anatomy of meningo-myelo-

cele, affecting the lumbo-sacral region. For full description see text, pp. 342-3.

No. 8.—*University College.*—A specimen of syringo-myelocele in the sacral region. The sac measures 1·5 cm. in diameter. Its interior is lined with soft pulpy substance; no spinal nerves pass through the sac. The innermost lining of the sac is well defined and fibrous, and on dissecting it off a series of nerves is displayed coursing round its outer surface and connected with it. A probe passed through the neck of the sac emerges from the centre of the spinal cord. The dura mater is distinct at the mouth of the sac and lies superficial to the innermost membrane referred to. (Mr. Godlee's case.)

No. 9.—*St. Bartholomew's Hospital,* No. 3481.—Syringo-myelocele. The sac is about the size of an orange, and situated over the sacrum. At the anterior part are two minute apertures, through which bristles have been passed into the substance of the cord. No nerves traverse the sac cavity. On removing the innermost lining of the sac a series of nerves is exposed lying in loose, probably the subarachnoid, areolar tissue.

No. 10.—*St. Thomas's Hospital,* LL 13[1].—A longitudinal section of the parts concerned in lumbo-sacral spina bifida after incomplete obliteration by the injection of Dr. Morton's fluid (Fig. 1, p. 345). The lower part of the sac is occluded by young fibrous tissue. In the upper part there is a small cavity due apparently to a dilatation of the central canal of the portion of cord crossing the upper part of the sac. From a child eight and a half weeks old. (Mr. Ballance's case.)

No. 11.—*St. George's Hospital,* No. 15 G.—Spina bifida involving the dorsal, lumbar, and sacral regions. With the exception of the three upper, the laminæ of all these vertebræ are deficient. The tumour consists of two distinct cysts, separated by a horizontal septum, the upper about twice as large as the lower. The cyst wall is extremely thin. In the upper sac, the nerve-roots lie beneath a thin membrane, and course round the sides of the sac closely applied to it. This inner membrane possibly represents the pia mater; the sac cavity thus corresponding to a dilatation of the central canal of the cord. In the lower sac, the nerve-roots are applied to the wall, but some are free between their origins on the wall of

the sac and their foramina of exit. The specimen was taken from a child aged 6 months; during life very little, if any, movement was observed in the legs; there was hydrocephalus, and the child was very marasmic.

No. 12.—*St. Thomas's Hospital,* LL 5^1.—Spina bifida of unusually large size; the sac measures transversely 8 cm.; in the vertical direction 8·4 cm., and from before backwards 7 cm.; situated in the lumbo-sacral region, membraniform over its centre; and presenting a longitudinal median furrow; there is a considerable extent of skin round the base; its edges are undulating and sharply defined. The cord crosses the upper part of the sac, to the upper wall of which it is attached by a falsiform membranous fold; the cord subsequently becomes incorporated with the sac-wall and gives origin to the nerve-roots passing through the cavity. On the exterior, besides the median furrow already noticed, there is a median umbilicus about 1 cm.; below the upper edge of the skin which corresponds to the point at which the cord is attached to the sac. Removed from the body of a male fœtus at term, with double talipes varus and "genu recurvatum" on left side. (Presented by Dr. Allan McLean, of Portland.)

No. 13.—*Middlesex Hospital,* No. 725.—Parts concerned in a lumbo-sacral spina bifida. The swelling is cordiform (Plate XVI), and presents a well-marked depression in the middle line at the seat of attachment of the cord, and corresponding with its central canal. There is a bilateral furrow, in the situation of partitions, which more or less separate the median part of the sac from the lateral. The nerve-roots arise from the sac-wall below the seat of depression noticed, and pass forwards through the sac to the intervertebral foramina. Fig. 2, p. 347, is a reduced profile representation of the condition.

No. 14.—*St. Mary's Hospital,* No. G $\frac{4}{5}$.—Spina bifida of the size of a fœtal head, involving lumbo-sacral region. The anatomical arrangement is sufficiently like that described at page 342 (Plate XV) to need no further description. The condition chiefly requiring notice is, that by far the greater part of the sac lies above the spinal cord and nerve-roots which occupy a comparatively small lower portion (Fig. 4, p. 353).

No. 15.—*St. George's Hospital,* No. 15 *a*.—Spina bifida in

lumbo-sacral region, presenting the typical anatomy. The sac is deeply grooved in the median line; over a sharply-defined area, the size of a shilling, its surface is minutely shreddy, as though a superimposed membrane had been torn off.

No. 16.—*St. Thomas's Hospital,* No. LL 12[1].—Spina bifida, involving the spine between the tenth dorsal and fourth lumbar vertebræ inclusive, the sac measures 6 cm. in diameter, and is membranous over its centre. The spinal cord crosses the upper part of the sac, is incorporated in the sac-wall for a distance of about 2 cm., after which it again lies free in the lower part of the sac, terminating as a cone in the usual manner (Fig. 3). The nerve-roots arise from the cord in a continuous series; those arising from the adherent portion pass horizontally forwards, those from the lower portion pass obliquely downwards to the sacral foramina. Passing from before backwards through the sac are broad membranous partitions by which it is partially divided into lesser cavities. From a male child, aged 10 weeks. (Presented by Dr. Archer.)

No. 17.—*Royal College of Surgeons,* No. 271 *c.*—A portion of the vertebral column from a case of spina bifida. There is a deficiency in the neural arches, extending from the eighth dorsal to the end of the column; the laminæ are widely everted, and form an angle of 45° with the antero-posterior plane. There was nothing in the size of the tumour to lead, during life, to the suspicion of so large a cleft in the vertebral arches.

No. 18.—*Charing Cross Hospital,* No. 250 *b.*—Spina bifida in lumbo-sacral region; sac the size of an orange. The cord is attached to its centre; the nerve-roots run forward applied to the cord; none appear to arise from the sac: a few fine nerves arise from the sac in the immediate neighbourhood of the attachment of the cord.

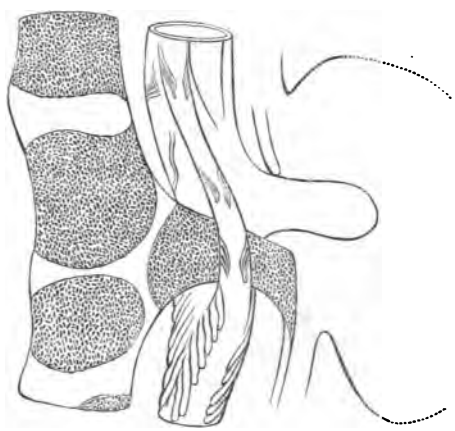
No. 19.—*St. Bartholomew's Hospital,* No. 3480.—Spina bifida in lumbo-sacral region. The centre of the membranous portion of the sac-wall has been destroyed by sloughing. The lower portion of the spinal cord projects from the vertebral canal backwards and presents with some of the nerve-roots within the opened sac.

No. 20.—*University College,* No. 5195.—Dorsi-lumbo-sacral spina bifida. The nerve-roots arise in double series from the posterior and lateral wall, whence they pass straight forwards to the intervertebral foramina. The sac is partitioned into a central and two lateral portions, these latter being again divided by transverse septa. On the right side the lateral and central portions communicate by an opening, about 6 mm. in diameter, above the spinal cord as it emerges from the vertebral canal, and by two or three small openings in the lower third of the partition. The right lateral space is completely subdivided into an upper and a lower portion, the lower portion being again subdivided into non-communicating cavities. In the highest lateral division there are two or three nerve-roots forming pairs with which are roots lying on the inner aspect of the septum, which therefore corresponds in position with the ligamentum denticulatum. On the left side the disposition of parts is almost symmetrical. The spinal cord is traceable into the highest part of the sac, and for about 6 mm. is free; beyond this it becomes incorporated with the sac-wall. At its entrance into the sac the cord is bifid, a process of bone 16 mm. in antero-posterior direction, crossing the vertebral canal about 1 cm. above the sac and lying between the halves of the cord in this situation. The bifurcation of the cord occurs below the level of the seventh dorsal vertebra (Plate XVII).

No. 21.—*St. Thomas's Hospital,* No. LL 12^3.—The skeleton of a fœtus in which there is a spina bifida affecting the last two or three dorsal and all the lumbar and sacral vertebræ. In connection with the arch of the tenth dorsal vertebra there passes directly forwards to the posterior surfaces of the bodies a narrow cylindrical process of bone by which the vertebral canal is nearly symmetrically bisected. The posterior part of the process is expanded laterally and fills the interval between the extremities of the laminæ of the tenth dorsal vertebra; its anterior extremity is expanded in the vertical direction. The element is structurally discontinuous with the parts to which it is apposed (Plate XVIII). The laminæ of the vertebræ concerned are in the upper part of the cleft slightly deficient in length; those in the lower part are about normal. Viewed from the front the bodies of the eleventh and twelfth dorsal vertebræ have a marked median depression, as though possibly possessed of two ossific centres. The lumbar curve is concave forwards; the sacral curve is normal.

No. 22.—*Royal College of Surgeons.*—The last two dorsal and the two upper lumbar vertebræ. The neural arches of the vertebræ are deficient, those of the eleventh dorsal and second lumbar are closed by fibrous tissue. Through the deficiency in the arch of the twelfth dorsal vertebra there protrudes the sac of a spina bifida. The deficiency in the neural arch of the first lumbar vertebra is closed by the expanded posterior end of an osseo-cartilaginous element, which crosses the vertebral canal from before backwards and perforates the spinal cord. This element abuts anteriorly against the posterior surfaces of the bodies of the twelfth dorsal and first lumbar vertebræ with the intervening fibro-cartilage. The division of the cord occurs unsymmetrically; on the left side the anterior column is alone represented in the upper part of the division; the left lateral column is traceable for a short way on the right division. In its lower part the left division of the cord becomes nearly equal in size to the right, the lateral and posterior columns reappearing. The central canal is largely dilated above the point of division; the dilated canal

FIG. 12.



A profile view of the specimen figured in Plate XIX. The osseo-cartilaginous process perforating the spinal cord is seen crossing the vertebral canal.

traverses the upper part of the right division, the dilatation ceasing too in its lower half. Into the mouth of the sac (Plate XIX and Fig. 12) there projects a diverticulum of the dilated right division of the cord. The roots of the last dorsal

nerve arise, the anterior from the left division, the posterior from the right division of the cord; of the nerves below, both the roots arise from the reconstituted division of the cord of the left side. The cavity of the sac is obliterated by young connective tissue. Dr. Batterham kindly furnishes the following clinical details:—A well-nourished female child, three years old; she was regarded as "delicate;" there was no paralysis of motor, nor of sensory nerves, nor of sphincters. The tumour had not grown since birth. The tumour was sessile, and the skin around it presented a few long locks of coarse hair; it was bluish and glossy on the surface, and somewhat doughy to the touch. Treatment consisted in the application of an india-rubber cord. Death took place on the eighth day from convulsions. (Presented by Mr. Vincent Jackson, Wolverhampton.)

No. 23.—*St. Bartholomew's Hospital,* No. 3485.—A specimen of spina bifida in the lumbo-sacral region. A process of bone extends from before backwards completely across the vertebral canal immediately above the deficiency. This process of bone perforates the spinal cord, which appears to reunite below; the cord terminates in a flattened expansion which rests upon a mass of fat on the inner aspect of the dura mater; the nerves come off irregularly from the front of this expansion. Above its perforation the cord presents a greatly dilated central canal. A thin, smooth membrane lines the dura mater forming the sac.

No. 24.—*Royal College of Surgeons,* No. 277.—The skeleton of a hydrocephalic fœtus with spina bifida involving dorsal, lumbar, and sacral regions. The bodies of the vertebræ are all deficient, the spine being completely cleft, as high as the cervical region. The lower bodies of the undivided portion of the column have two centres, which serve as points of departure for the divided column below. There is no bony process crossing the vertebral canal at the point of division; below this point the canal on each side is unclosed on the inner aspect, there being apparently no formation of lamina in this situation.

No. 25.—*Royal College of Surgeons,* No. 271 A.—The bones from a case of lumbo-sacral spina bifida. The sacrum is twisted to the left side owing to a malformation of its upper three segments, consisting in a partial absence of the left

halves of the first and third segments; the left half of the second segment is produced upwards and downwards so as in some measure to compensate for the deficiency (Plate XX, and Fig. 7). The specimen was removed from the body of a boy æt. 12. The tumour was situated on the right of the median line; it had grown with the child's growth, but not otherwise. He had never walked without crutches, but he had complete control over bladder and rectum until he was turned eleven years; thin dribbling of urine commenced, and the bowel lost expulsive power (the rectum was distended with hard fæces, which had to be scooped away). An effort was made to remove the fluid as all the symptoms were becoming aggravated. He died of meningitis purulenta some weeks after the operation.

No. 26.—*Royal College of Surgeons,* No. 278.—The skeleton of a fœtus, showing deficient closure of the arches in the occipito-cervical region. Some of the centres of the cervical vertebræ are double. The column presents two lateral curvatures in the upper dorsal, one to the right the other to the left, owing to the absence of the opposite halves of the second and fourth of the bodies.

No. 27.—*Royal College of Surgeons,* No. 274.—The lower dorsal, lumbar, and sacral vertebræ of a newborn child. The lamina of the four lower dorsal vertebræ below are widely separated. The lumbar region is strongly concave anteriorly, the other spinal curves as far as shown being normal. Anteriorly the body of the vertebra at the bottom of the lumbar concavity is so reduced in thickness that the contiguous intervertebral discs lie almost in contact.

No. 28.—*Royal College of Surgeons,* No. 276 A.—The parts concerned in a lumbo-sacral spina bifida, which was cured by the injection of Morton's fluid. Externally there is a deep-puckered depression at the centre of the sac; the cuticle here is smoother and more shining than that around. On section the sac is represented by a mass of connective tissue sharply bounded in front by the dura mater; the spinal cord crosses the highest part of the sac through the fibrous tissue noted. In the lower part of the sac there are, anteriorly, sections of nerve-roots passing towards the intervertebral sacral foramina (Plate XIV, Fig. 2).

Part II.—Clinical.

Introductory.—In order to form a correct estimate of the value of the treatment of spina bifida by the injection of Dr. Morton's iodo-glycerine solution,* we have endeavoured to ascertain as far as possible what is the natural history of the deformity when untreated, and we have also prepared comparative tables of cases treated by injection of Morton's solution, by injection of simple solutions of iodine, by ligature, by excision, and by puncture and pressure.

Natural History of Spina Bifida.

From a study of all the cases tabulated at the end of this report, we have been able to gather certain facts in the natural history of spina bifida, but to show the natural course and termination of the deformity we have collected, in Table I, 60 cases, which have not been subjected to any operative interference. We have further appealed to the annual reports of the Registrar-General for England, and to a valuable report on Spina bifida by Professor Demme.†

Sex.—Our tables contain 245 cases; of 156 in whom the sex is stated; 82 were females, 74 were males. Of Demme's 57 personally observed cases, 31 were females and 26 were males. From Table A, page 376, kindly furnished us by the Registrar-General for England, we find that of 1768 cases of children with spina bifida dying in the first year of life, 989 were females and 779 were males. The deformity is therefore unquestionably more frequent in females than in males.

Position of Tumour.—The position of the tumour is stated in 236 of the cases collected by us. In the great majority of instances the statement rests upon clinical examination only, and as it is often difficult and may be impossible to determine with certainty the exact position of the spinal cleft without dissection, we would suggest that the subjoined figures should be corrected by those given in the first section of this report, and which we have added in a second column. Dr. Demme also gives the position of the tumour in his cases; these we have added in a third column.

* The following is Dr. Morton's *formula*:—℞. Iodi. gr. x, Potassii iodidi gr. xxx, Glycerini ʒj. M.

† *Zwanzigster Medicinscher Bericht des Jenner'schen Kinderspitals*, Bern., 1882. This report is specially valuable as it contains 57 cases observed by one Surgeon, and forming his entire experience.

Region.	Our cases.	Path. series.	Demme's cases.
Cervical . .	11	...1 case affecting...	5
Cervico-dorsal .	2	... whole of spine ...	2
Dorsal . . .	14	... 9 ...	13
Dorsi-lumbar .	8	... 3 ...	0
Lumbar . .	108	... 9 ...	17
Dorsi-lumbo-sacral	2	... 7 ...	0
Lumbo-sacral .	42	... 68 ...	11
Sacral . . .	42	... 21 ...	7
Coccygeal . .	7	... 1 (Sacro-coccyg.)	0

In two of Demme's cases there were two tumours; in one case over the third and fourth, and over the seventh and eighth dorsal vertebræ; in the other over the first and second, and over the eighth and ninth dorsal vertebræ. The above figures show a great preponderance of cases in the lower half of the spinal column.

Complications.—In many of the cases in our tables no mention is made of the presence or absence of complications, and the records are in several instances so brief that the absence of any mention of them cannot be taken as indicating the absence of such conditions. The following table is, therefore, probably of value as showing the *relative*, rather than the *absolute* frequency of the complications. Dr. Demme's cases having been all recorded by one careful observer are more exact in this as in some other particulars.

Complications.	No. of cases in our tables.	No. of cases in Demme's table.
Paralysis . .	53 ...	Not stated.
Talipes . . .	42 ...	13
Hydrocephalus .	30 ...	17
Idiocy . . .	2 ...	Not stated.
Cretinism . .	1 ...	„
Curvature of spine.	2 ...	„
Absence of cerebellum	1 ...	„
Harelip . .	0 ...	3
Cleft palate . .	1 ...	Not stated.
Imperforate anus .	1 ...	„
Ectopia vesicæ .	0 ..	1

General Nutrition.—In only 14 of our cases is there mention made of the state of the general nutrition of the patients; 7 are stated to have been marasmic, 7 quite healthy. Demme states that of 22 children brought to the hospital within the first three or four days of life,

16 weighed under 2800 grms. (6¼ lb.); the remaining 6 were well developed and weighed about 7 lb. He adds that all the cases that did not die early from rupture of the sac, or were not cured by an early operation, gradually lost weight and showed symptoms of intestinal catarrh.

HEREDITY.—In one instance recorded in our tables the deformity occurred in three successive children in a family, and in another in two successive children, the ninth and tenth. Demme mentions two cases, in each of which two previous children had the same deformity, and three cases in each of which one previous child of the same parents had spina bifida. In three cases the father had congenital club-foot, and in two the mother had harelip; the brother of one of the cases collected by us is stated to have had hydrocephalus.

Twenty-nine of Demme's children were first-born children, 17 were second-born, and 11 were later-born children.

FREQUENCY.—Dr. Demme found 57 cases of spina bifida in 36,148 children; Chaussier records 22 cases among 22,293 children in the Paris Maternité. The report of the Registrar-General for the year 1882, states that in England and Wales, 647 deaths were attributed to this cause alone.

NATURAL COURSE OF THE MALADY.

In Table I, we have placed 60 cases that had not been subjected to any operative treatment; of this number 25 (41·6 per cent.) are stated to have died, and 14 (23·3 per cent.) to have undergone spontaneous cure. Of the remaining 21, the termination of the case is not stated, although several of them were able to follow their ordinary occupations. These figures do not, however, represent the real mortality of the deformity when allowed to run its natural course, for cases of infants dying of this deformity without special treatment are not usually recorded in the medical journals, while such as recover, with or without treatment, or who live on in spite of their deformity, are almost certainly placed on record. A more correct estimate of the mortality is afforded by Demme, who gives the result in all the 57 cases observed by him. He states that of the 32 patients for whom no operation was undertaken, not one was alive at the end of two years.

11	died	between	the 8th and 14th day.
9	,,	,,	15th and 22nd day.
5	,,	,,	23rd and 30th day.
3	,,	,,	3rd and 4th month.

1 died at 5 months.
1 „ 8 months.
1 „ 12 months.
1 „ 2 years.

The Registrar-General of England has courteously favoured the Committee with the appended Table A, from which it is seen that the mortality among infants with spina bifida is very great in the early months of life. In the Report of the Registrar-General for 1882, 89 deaths are attributed to spina bifida in London, and of this number 86 died under the age of one year.

TABLE A.—*Deaths of males and females in England and Wales under one year of age from spina bifida in each of the three years* 1881-83.

Year and sex.	Age periods.			
	0—3 months.	3—6 months.	6—12 months.	Total under 1 year.
1881 { Males	188	29	26	243
1881 { Females	285	43	22	350
1882 { Males	216	39	22	277
1882 { Females	255	54	26	335
1883 { Males	202	32	25	259
1883 { Females	229	44	31	304
Total deaths in the three years 1881–83 { Males	606	100	73	779
Total deaths in the three years 1881–83 { Females	769	141	79	989
	1375	241	152	1768

CAUSE OF DEATH.—In only 17 of our cases in Table I is the cause of death sufficiently stated; of these

8 died from the effects of rupture of the tumour.
4 „ marasmus.
3 „ convulsions.
2 „ hydrocephalus.

Dr. Demme's figures are as follows:—

15 children died from rupture of the sac.
10 „ „ marasmus.
7 „ „ intercurrent disease.

32

From these facts it is evident that spina bifida is attended with great mortality at an early age. We regret that our statistics do not enable us to demonstrate the absolute mortality of the deformity when untreated, or the proportion in which death depends upon local and upon general conditions.

In a certain number of the cases recorded death has been caused by rupture of the sac, draining away of the cerebro-spinal fluid, and subsequent septic meningitis. But we believe that in a large proportion of cases death ensues from the marasmus and general defective nutrition, so often associated with this and other deformities, and which cannot be remedied by any local treatment of spina bifida.

Mode of Spontaneous Cure.—In 13 of our cases in Table I the mode of cure of the tumour is stated more or less fully. In 8 there was a gradual shrinking of the sac, in 4 the sac burst, and in 1 there was ulceration of the sac and general oozing. In view of the frequency with which rupture of the tumour leads to death, we are justified in speaking of the gradual shrinking of the sac of a spina bifida as its natural mode of cure.

Persistence of Tumour.—In our table are found the cases of patients who at the time of record were well and had attained the age of 9, 13, 13, 17, 23, 25, 25, 25, 32, 40 and 43. Two others were aged respectively 23 and 26 at the time of their death. In a certain minority of cases, therefore, the presence of spina bifida is not incompatible either with life, health, or general activity.

Treatment by Puncture.

In Table II, we have placed 46 cases treated by puncture or incision of the tumour with or without subsequent compression.

Results.—Of these, 30 died, 12 recovered, 2 were unrelieved, and in 2 cases the result is not stated. One case was submitted to tapping as many as seventy times.

Region affected.—The mortality in the different regions of the spine is shown by the following table:

Region of spine.	Recovered.	Unrelieved.	Died.
Cervical . . .	0	0	2
Dorsal . . .	0	0	1
Dorsi-lumbar . .	1	0	2
Lumbar (1 lost sight of)	5	1	16
Lumbo-sacral . .	2	1	2
Sacral . . .	4	0	6

In one case (fatal) the position of the tumour is not stated.

Nature of Tumour.—The only means we have of estimating the severity of these cases is to notice the nature of the sac-wall, the presence or absence of nerves in the tumour, and the complications mentioned. In a large number of instances no reference is made in the scanty record of the cases to any of these particulars.

The Sac-Wall.—The sac-wall was membranous in 13 cases, in 5 of which it was ulcerated. The sac-wall was covered with healthy skin in 5 cases. The sac-wall is undescribed in 27 cases.

Contents.—Nerves are stated to have been present in the sac in 11 cases and absent in 5 cases. No mention is made of the contents of the tumour in 29 cases.

The tumour is said to have been pedunculated in 2 cases.

Complications.—

Paralysis	10 cases.
Hydrocephalus	6 „
Talipes	1 „
Sloughing of sac	1 „
None	16 „
No mention of complications in	12 „

Cause of Death.—In the 30 fatal cases the cause of death was as follows:—

Meningitis	14 cases.
"Convulsions"	8 „
Marasmus	1 „
"Exhaustion"	1 „
Intercurrent disease	1 „
Not specified	5 „

Many of the cases described under the head of "convulsions" were probably really instances of meningitis. Whether this be so or not, it is obvious that meningitis is the great danger of this mode of treatment. This method of treatment is an imitation of that local change in the tumour (rupture) which most often ends fatally, and its disastrous results are therefore by no means surprising. While in many instances puncture of the sac of a spina bifida has proved entirely innocuous, it has been so much more often fatal that it should not be undertaken even as a palliative measure.

Treatment by Ligature.

In Table III, we have placed 16 cases which have been treated by ligature of the tumour.

Results.—Of these 6 died, and 10 recovered.

Region.—The result in the different regions of the spine was as follows:

Region of spine.	Recovered.	Died.
Cervical	2	0
Cervico-dorsal	1	0
Dorsal	0	1
Lumbar	4	4
Lumbo-sacral	1	1
Sacral	2	0

The Sac-wall.—The sac-wall is undescribed in 11 cases.
The sac-wall is described as healthy skin in . 1 „
The sac-wall is described as thin and membranous in . . . . . 4 „

In 3 cases the tumour was pedunculated; 2 recovered, and 1 died.

Contents.—Nerves are stated to have been absent from the sac in 3 cases. In no case are nerves stated to have been present in the tumour, although in one case two cords which resembled nerves passed through the sac.

This mode of treatment, like excision, permits of a ready and certain examination of the contents of the sac. In no case did persistent paralysis follow the operation; this result is incompatible with the presence of nerves in the sacs which were removed.

Complications.—Complications are recorded in 2 cases only, both of which recovered; there was hydrocephalus in 1 case, and deficient intellect in 1 case.

Cause of Death.—In the 6 fatal cases the cause of death was as follows:

Meningitis	3 cases.
Draining of cerebro-spinal fluid	1 „
Intestinal catarrh	1 „
Not specified	1 „

Ligature of the tumour is of course inapplicable to cases of myelocele, and there is no evidence that any of the 16 cases in this table were of such a nature; indeed the negative evidence is so strong as to amount to almost positive proof to the con-

trary. For cases of meningocele a mortality of 37·5 per cent. is higher than that obtained from other modes of treatment.

Treatment by Excision.

In Table IV, we have placed 23 cases treated by excision of the sac.

Results.—Of these 16 have recovered, 7 have died.

Regions.—The mortality in the different regions of the spine was as follows:

Region of spine.	Recovered.	Died.
Cervical	2	0
Dorsal	2	1
Lumbar	9	4
Sacral	2	1

In two cases, one of which was fatal, the region of the spine is not stated.

Nature of Tumour.—The sac-wall is undescribed in . . . . . 12 cases.
The sac-wall is described as membranous in 3 „
(In two of these it was ulcerated.)
The sac-wall is described as covered with healthy skin in . . . . . 3 „

Contents.—Nerves were certainly absent from the sac in . . . . . 16 „
Nerves were certainly present in the sac in 1 „
(This case was fatal)
No mention of contents of sac in . . 6 „

Complications.
None present . . . . . 5 cases.
Talipes . . . . . 3 „
Suppuration of tumour . . . 1 „
No mention of complications . . 14 „

Cause of Death.—In the 7 fatal cases, the cause of death was—
Meningitis . . . . . 5 cases.
Marasmus . . . . . 1 „
Not specified . . . . . 1 „

Plastic Operation.—Appended to Table IV, are two cases in which a plastic operation was performed, the inner sac of

the tumour being preserved, while flaps of skin reflected from its base were united over it; one of these cases recovered, the other died.

For reasons which are stated lower down we are compelled to regard excision of the tumour as an inappropriate treatment of spina bifida.

Treatment by Injection of Simple Iodine Solution.

In Table V, we have placed 26 cases of spina bifida which have been treated by injection into the tumour of aqueous or spirituous solutions of iodine.

Result.—Of these cases 20 recovered, 5 died (19·2 per cent.), 1 was unrelieved. The results in the different regions of the spine were as follows:

Region of spine.	Recovered.	Unrelieved.	Died.
Cervical . .	1 ...	0 ...	0
Lumbar . .	7 ...	1 ...	3
Lumbo-sacral .	3 ...	0 ...	2
Sacral . .	6 ...	0 ...	0
Not stated .	3 ...	0 ...	0

The Sac.—The sac is described as membranous in (1 ulcerated, 1 nævoid, 1 very thin) . . . . . 3 cases.
The sac is described as covered by healthy skin in . . . . . 1 „
The sac is not described in the remaining cases. 1 „

Complications.

Paralysis	5 cases.
Hydrocephalus	3 „
Idiocy	1 „
None	15 „
None specified	2 „

Cause of Death.—In all the 5 fatal cases convulsions preceded death, and in 2 of these 5 cases the sac burst. In 1 case as many as fourteen injections were made.

Treatment by Injection of Dr. Morton's Fluid.

In Table VI, we have placed 71 cases treated by the injection of Dr. Morton's iodo-glycerine solution. Forty-one of these cases have been specially reported to the Committee, and have

not been previously published; the remaining 30 cases have been collected from various medical journals.

Result.—Of the 71 cases, 35 recovered, 27 died, 5 were unrelieved, 4 were relieved.

The 35 cases numbered as recoveries, recovered as regards the sac only, the complications associated with the deformity remaining unaltered. The 27 fatal cases died at various periods after the operation, and not all as a result of the operation. The cases described as "relieved" are those in which as a sequel to the injection the tumour became smaller or partially solidified.

Region.—The results in the different regions of the spine were as follows:

Region of spine.	Recovered.	Relieved.	Unrelieved.	Died.
Cervical	2	0	0	0
Dorsal	4	0	0	3
Lumbar	15	2	1	6
Lumbo-sacral	8	1	1	13
Sacral	5	0	1	3
Coccygeal	0	1	0	1
Not stated	1	0	2	1

The Sac-Wall.—The sac-wall is described as membranous in (in 14 of these it was also ulcerated) . . . . 43 cases.
The sac-wall is described as covered with normal skin in . . . . . 3 „
The sac-wall is not described in . . . 25 „

Complications.

Paralysis	22	„
Talipes	20	„
Hydrocephalus	7	„
Rupture of the sac at birth	2	„
None	17	„
None mentioned	9	„

Causes of Death.—In the fatal cases the causes of death were as follows:

Meningitis	7	cases.
Shock	5	„
Marasmus	7	„
Hydrocephalus	2	„
Convulsions	2	„
Diarrhœa	1	„
Not specified	3	„

In a letter (dated May 11, 1885) addressed to the Committee Dr. Morton says:— . . . "Including those published in my small book in 1877, I am able to refer to 50 cases which have been treated by tapping and injection of the iodo-glycerine solution. Of these 41 have been regarded as successful by those who saw them. Thus 9 have been unsuccessful. This is about the proportion which has been noted ever since the adoption of this method of treatment."

Remarks.

In the earlier part of this report we have adduced evidence which shows conclusively that in a large proportion of specimens of spina bifida the spinal cord is in the sac, and this evidence cannot be gainsaid by reference to any cases observed in which no nerve-elements have been present in the sac. We are not acquainted with any means by which it is possible to determine in the living subject that the spinal cord is not in the sac of a spina bifida. For this reason we believe that any operation involving interference with or removal of the *median vertical portion* of the tumour should be entirely abandoned. We are quite conscious of the large measure of success that has attended the operations of ligature and excision, but we cannot lose sight of the fact that these operations always expose the patient to the grave dangers attending removal of the expanded spinal cord and attached nerves.

The treatment by repeated tapping has proved far too fatal to warrant its repetition, and it does not rest upon any scientific basis.

We believe that success in the treatment of spina bifida is to be best obtained by following closely the process of nature when spontaneous cure of these tumours occurs. This natural process of cure appears to consist in a gradual shrinking of the tumour, and the treatment by injection of iodine commends itself to us as the best hitherto introduced, not only on account of its great success, but also because its mode of action most nearly resembles the natural mode of cure.

A comparison of Tables V and VI does not show any advantage derived from the use of Dr. Morton's iodo-glycerine solution. We are of opinion that this comparison is probably deceptive, for we believe that Dr. Morton's treatment has been adopted more widely than any other plan of treatment and, as our table shows, in a number of quite hopeless cases, in which other methods of treatment would not have been entertained.

Although only nine years have elapsed since the publication of Dr. Morton's first case, the number of cases so treated already largely exceeds that treated by any other means, a fact largely due to the great success of the earlier cases.

Dangers of the Operation.—The first immediate danger of the operation is shock, which seems to have been the cause of death in five instances. Meningitis, with or without a continuous drain of fluid, has been still more fatal. In one case, at least, paralysis of limbs supervened and persisted after cure of the tumour and appeared to be due to the treatment adopted. In four or five other cases paralysis of the lower limbs has occurred but has passed off after a variable time; complete paraplegia is reported to have occurred once, and to have subsequently disappeared. In three cases hydrocephalus set in subsequently to the cure of the spina bifida, and in other cases the cure of the latter had no beneficial effect in delaying the steady progress of the hydrocephalus. It is not to be expected that any treatment of spina bifida will influence this cranial condition. In one case the injection of a drachm of the iodo-glycerine solution appeared to cause a profuse secretion of saliva.

Selection of Cases.—We believe that the best results of Dr. Morton's treatment are only to be obtained by a somewhat careful selection of cases. The circumstances which contraindicate it are advanced marasmus, great and increasing hydrocephalus, and intercurrent disease. As a rule, it is well to wait until the child is at least two months old before submitting it to operation, but where the sac-wall is threatening to burst treatment may be carried out earlier. Where paralysis or talipes complicate spina bifida the cure of the latter must not be regarded as offering any prospect of improving the former.

Method of Operating.—Some importance has been attached to the position of the child during the injection. While we regard the fears that have been expressed on this point as exaggerated and due to a misconception of the usual anatomy of the tumour, we would recommend that the child should be laid upon its side. The puncture into the tumour should be made at one side of the base, obliquely through healthy skin, and not through membranous sac-wall, the objects being to avoid wounding the expanded spinal cord, and subsequent leakage of the cerebro-spinal fluid. It is not necessary to withdraw any of the fluid contents of the tumour before injecting the solution of iodine. From half a drachm to

two drachms of Dr. Morton's iodo-glycerine solution have been generally injected, and a drachm of the fluid commends itself to us as a suitable quantity in the majority of cases. Should the injection fail altogether, or only a part of the cavity be obliterated, the operation may be repeated at intervals of about a fortnight.

Conclusions.

1. Notwithstanding many failures, the plan of treatment by injection is the best with which we are acquainted, and the only one which we feel justified in recommending.
2. A more careful selection of cases than has hitherto been made is necessary.
3. Marasmus, hydrocephalus, and intercurrent disease contraindicate the operation.
4. In cases in which the operation may, nevertheless, be legitimately performed, we should consider the following as unfavorable circumstances :—
   - α. Distinct evidence of the cord being in the sac, as shown by umbilication or a longitudinal furrow.
   - β. A very thin membranous or ulcerated sac.
   - γ. Previous rupture of the sac.
   - δ. The occurrence of a distinct impulse between the tumour and the anterior fontanelle; or a sac, the contents of which are easily returned into the spinal canal.
   - ϵ. A very early age of the patient.
5. The best result is to be hoped for in children, who have reached the age of two months, in whom there is no paralysis or hydrocephalus, and when the sac is covered by healthy skin.

We cannot close this Report without acknowledging the indispensable assistance of Mr. S. G. Shattock, Curator of the Museum, St. Thomas's Hospital, in analysing the pathological specimens, in making dissections, as well as the drawings with which the Report is illustrated.

(Signed) Howard Marsh,
A. Pearce Gould,
H. H. Clutton,
Robert Wm. Parker, *Hon. Sec.*

TABLE I.—*Containing 60 Cases, not subjected to any Operative Treatment.*

No.	By whom reported and where recorded.	Sex and age.	Character of tumour, position, size.	Complications, ulceration, paralysis, oozing.	Treatment.	Result.	Remarks.
1	Sir A. Cooper, Med.-Chir. Trans., vol. ii	—	Lumbar, size of a walnut at birth, enlarged to small orange	None	Pressure, first by bandage, then plaster of Paris, and finally by a truss	Recovery	Occasional convulsions. Child was dull and heavy for a short time after complete reduction of tumour.
2	Mr. (now Sir) P. Hewett, Lond. Med. Gaz., 1844	—	Lumbar, size of a large egg; skin at apex thin and transparent	None	Support	Death	Inflammation of skin, rupture of tumour, suppuration; right laminæ of middle lumbar vertebræ deficient; left well developed; nerves crossed the sac.
3	Ditto	M. 17 yrs.	Sacral, size of adult head	Dribbling of urine; partial control over rectum	—	Nil	—
4	Ditto	F. 25 yrs.	Sacral, size of a child's head; skin thick	No paralysis; when sitting could not retain urine	—	Nil	—
5	Mr. Lawrence, Med. Times, 1858, ii	F. 2 mos.	Lumbar, size of a child's fist; skin thin	Talipes varus; hydrocephalus	Protection	Recovery	Gradual shrinking and cure of spina bifida. Death from hydrocephalus at the age of 9 months.
6	Mr. Wormold, Med. Times, 1858, ii	M. Infant	Lumbar, "very small"	—	Moderate pressure	Recovery	Tumour gradually shrunk away.
7	Dr. Moore, Med. Times, 1858, ii	—	Lumbar, size of a large egg	—	Moderate pressure	Recovery	The sac ulcerated, fluid oozed from it, and afterwards the tumour disappeared.
8	Mr. Hutchinson, Med. Times, 1858	M. 32 yrs.	Sacral, size of a duck's egg	—	None	Nil	Not noticed till 12 years of age.

9	Mr. Valentine, Med. Times, 1858, ii	M. 13 yrs.	Sacrum, 6 in. in circumference at the base	—	No treatment	Nil	When tumour was pressed upon there was giddiness and a sensation of fulness of the head. After falling on the tumour was unable to walk for a week, lower limbs feeling numbed.
10	Dr. Butler, Med. Times, 1858, ii	1 day	Dorso-lumbar, size of a large orange	Impaired motion and sensation in lower limbs; pressure on tumour caused convulsions	—	Death	On 8th day rupture of sac, and one long convulsion, ending in death.
11	Mr. W. Carr, Med. Times, 1858, ii	F. 26 yrs.	Sacral, size of a quart basin	—	Protection	Death	Tumour was first noticed after a difficult labour, the size of a walnut. Pressure upon it caused confusion in the head, and a sense of impending unconsciousness. The withdrawal of a small quantity of fluid caused "uneasy sensations about the brain."
12	Mr. Stafford, Injuries and Diseases of Spine, 1826	M. 2½ yrs.	Sacral	Hydrocephalus, paralysis, imperforate anus	None	Recovery	The tumour burst and then sloughed. Fissure of bodies of the sacral vertebræ.
13	Ditto	M. 9 yrs.	Lumbar	None	None	Recovery	At birth tumour was like a bladder; it burst and healed up, leaving a solid tumour which constantly discharged watery fluid.
14	Ditto	3 wks.	—	Paraplegic hydrocephalus	—	Death	The bladder-like tumour burst, and never healed; extreme hydrocephalus.
15	Mr. Wardrop, Lancet, xiii	F.	Sacral	—	None	Nil	—
16	Mr. D. Richards, Lancet, 1862, ii	F.	Dorsal; skin absent over tumour	Talipes varus, cleft palate, angular curvature	—	Recovery	Opening in spine gradually and completely closed. Death subsequently from marasmus.
17	Dr. Madge, Lancet, 1867, ii	—	Lumbar	Talipes varus	None	Death	At 7 months tumour became opaque and mottled; the child very ill and died.

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2	Mr. (now Sir) P. Hewett, Lond. Med. Gaz., 1844	—	Lumbar, size of a large egg; skin at apex thin and transparent	None	Support	Death	Inflammation of skin, rupture of tumour, suppuration; right laminæ of middle lumbar vertebræ deficient; left well developed; nerves crossed the sac.
3	Ditto	M. 17 yrs.	Sacral, size of adult head	Dribbling of urine; partial control over rectum	—	Nil	—
4	Ditto	F. 25 yrs.	Sacral, size of a child's head; skin thick	No paralysis; when sitting could not retain urine	—	Nil	—
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7	Dr. Moore, Med. Times, 1858, ii	—	Lumbar, size of a large egg	—	Moderate pressure	Recovery	The sac ulcerated, fluid oozed from it, and afterwards the tumour disappeared.
8	Mr. Hutchinson, Med. Times, 1858	M. 32 yrs.	Sacral, size of a duck's egg	—	None	Nil	Not noticed till 12 years of age.

59	Mr. Lunn, specially reported	F., 6 mos.	Dorsi-lumbar, size of an orange; covered with skin	Talipes varus; paraplegia	None	Death	Death at 11 months; the tumour had solidified to a large extent; there was some hydrocephalus.
60	Mr. Clutton, specially reported	M., 2 wks.	Cervico-dorsal; sac wall translucent; no ulceration; sac measures 5½ in. from above downwards	Well-nourished child; no paralysis	—	—	Broad-based tumour, rather tapering. Undergoing spontaneous cure; is still under observation. This child is brother to No. 27 of Table VI.

TABLE II.—*Containing 46 Cases treated by tapping.*

1	Sir A. Cooper, Med.-Chir. Trans., vol. ii	M. 10 weeks	Lumbo-sacral, size of half a billiard ball	None	Tapped on 8 occasions; pressure	Recovery	After 5th tapping fluid was sanious; after 7th it was mixed with coagulable lymph. No paralysis.
2	Ditto	F. 11 days	Lumbar; covering of tumour ulcerated	—	Tapped once	Tumour cured; child died	On 4th day tumour was filled with coagulable lymph; convulsions 6th—10th day; death. P.M.—Brain soft, 6 oz. of fluid in ventricles.
3	Ditto	M.	Sacral, "very large"	None	Tapped over 30 times, and pressure	Recovery	Convulsive twitchings on one occasion.
4	Mr. (now Sir) P. Hewett, Lond. Med. Gaz., 1844	5 mos.	Sacral, size of turkey's egg; skin natural	Hydrocephalus—"not to any great degree"	Tapped 3 times	Death	Child took measles and died. Spinal cord crossed the cavity and was blended with the sac.
5	Mr. Tatum, Med. Times, 1858, ii	M. 20 yrs.	Sacral, pedunculated, larger than man's head; skin ulcerated two or three times	Occasional involuntary movements of legs and passage of fæces	Tapped twice, half a basin of fluid removed	Death	Erysipelas, meningitis. No nerves in sac.
6	Dr. W. Ogle, Med. Times, 1858, ii	1 week, infant	Sacral, considerable size	Right leg atrophied; feet incurved	Tapped 2 or 3 times	Death	Convulsions preceded death in 9 weeks. See also 'Path. Soc. Trans.,' vol. viii.

No.	By whom reported and where recorded.	Sex and age.	Character of tumour, position, size.	Complications, ulceration, paralysis, oozing.	Treatment.	Result.	Remarks.
18	Mr. F. T. Coates, Lancet, 1871, ii	F. Infant	Lumbar	Partial paraplegia	None	Death	On 3rd day it burst, and again in the 5th week and the 12th week; convulsions, coma, death 13th week.
19	Mr. Tom Guy, Prov. Med. and Surg. Journ., 1850	25 yrs.	Lumbar, size of fœtal head	Awkward gait; diminished sensibility in legs	None	Recovery	Tumour burst, fluid escaped from time to time.
20	Dr. Hance, Prov. Med. and Surg. Journ., 1857; Amer. Journ., 1850	F.	Lumbar, would hold a quantity of fluid	—	—	Recovery	After measles the sac sloughed; partial paraplegia, followed by complete recovery.
21	Dr. E.T. Hughes, Prov. Med. and Surg. Journ., 1851	M.	Sacral, size of adult head	—	—	Death	Spontaneous rupture, meningitis. Sac found to project the lower spines of the spinal canal. When a boy he had headache on stooping, and always had severe pain in the head when the tumour was struck.
22	Dr. Behrend, Archiv. f. Kinderkrankheiten, Brit. Med. Journ., 1860	—	—	—	Collodion and protection	Recovery	—
23	Dr. G. Hewitt, Lancet, 1862, i	F.	Sacral; 1½ in. diameter	—	Nothing	Death	On 12th day, from convulsions.
24	M. Blot, Lancet, 1863, i, Gaz. Méd., 1863, i	—	Cervical. No skin over it except at margin	—	Protection	Nil	—

25	Dr. Barnes, Brit. Med. Journ., 1863, ii	—	—	Hydrocephalus at 9th month	—	Nil	—
26	Mr. R. Duck, Brit. Med. Journ., 1869, ii	F.	Dorsi-lumbar, 3½ in. in circumference	Spinal cord plainly seen in the sac; complete paralysis before death	Support	Death	—
27	Mr. Hilton, Med. Times, 1858, ii	F., 23 yrs.	Sacral, size of two fists	—	None	Nil	Married, 2 children. Pressure upon tumour caused fulness of the head and thighs. After a fall on the tumour was at one time unable to stand for 14 days, and at first could not move her arms at all.
28	Dr. Behrend, Med. Times and Gaz., 1859, ii	—	Lumbar, size of a small orange, transparent, skin over it very delicate	Pressure on the tumour made it disappear, and caused pain and contraction of features	Collodion	Recovery	The tumour was reduced to the size of a filbert in a week, and subsequently only skin and subcutaneous tissue was left.
29	Mr. F. T. Coates, Lancet, 1881, i	M., infant	Lumbar, size of small orange, discharging straw-coloured fluid	Talipes	Protection by a gutta-percha cone	Nil	Child was larger than most children of his age.
30	Mr. E. E. Abbott, Lancet, 1881, i	F.	Dorsi - lumbar, 4 × 3 in., covered in part by thin membrane	Double talipes; hydrocephalus	Protection by a gutta-percha shield	Nil	—
31	Mr. H. Thompson, Brit. Med. Journ., 1884, i	M.	Lumbar, size of a walnut, covered with thin bluish skin	Ulceration of sac	None	Death	The child was well nourished; convulsions preceded death.
32	Mr. H. G. Dyer, Brit. Med. Journ., 1879, i	M.	—	—	—	Stillborn	Three successive children of the same parents, each with a large spina bifida; the first was stillborn, the other two died in a "few hours," and "36 hours" respectively.
33	Ditto	F.	—	—	—	Death	
34	Ditto	M.	—	—	—	Death	

TABLE I.

No.	By whom reported and where recorded.	Sex and age.	Character of tumour, position, size.	Complications, ulceration, paralysis, oozing.	Treatment.	Result.	Remarks.
34A	Mr. T. Smith, Lancet, 1878, i	M., 40 yrs.	Dorsi-lumbar, size of a hen's egg	None	None	Nil	No ill effects observed.
35	Mr. J. Hutchinson, Brit. Med. Journ., 1877, i	F., 13 yrs.	Lumbar, "like a mamma"	Incontinence of urine and fæces	—	Nil	—
36	Ditto	M.	Lumbar, size of a duck's egg	Incontinence of urine and fæces; tendency to hydrocephalus	—	Nil	—
37	Mr. J. S. Steele, Med. Times and Gaz., xxxvii	—	—	Tumour ulcerated; it ruptured at birth	None	Death	Child gradually gradually wasted and died in convulsions on the 18th day.
38	Dr. Kennedy, Brit. Med. Journ., 1872, ii	F.	Lumbar, 2 inches across, no skin over sac	Slight hydrocephalus; dislocation of ankles forward	—	Nil	—
39	Dr. J. Morton, Brit. Med. Journ., 1874, ii	—	Dorsal	—	None	Death	Spontaneous draining away of the fluid; spinal cord was found in the sac.
40	Mr. F. Monser, Brit. Med. Journ., 1875, i	F., 11 weeks	Lumbar	Paraplegia; talipes varus	None	Nil	—
41	Dr. Drummond, Brit. Med. Journ., 1875, i	25 yrs.	Lumbar, "large"	Cretinism; paraplegia	—	Nil	—
42	Mr. Pearce Gould	M., 10 days	Sacral, size of a fig, sac very thin and ulcerated	Paralysis of rectum and bladder; legs always drawn up, if drawn down and	A tannin lotion	Death	The tumour diminished in size, but the child wasted and died.

				feet are tickled legs at once drawn up; no talipes			
43	Mr. W. Adams, Path. Soc. Trans., iii	—	Lumbo-sacral, small, skin over it imperfect	Extreme talipes; equino-varus	None	Death	The child lived only a few hours; the spinal cord and nerves were in the sac.
44	Mr. Amyot, Med. Times and Gaz., 1869, i	Infant	Lumbar, size of hen's egg; sac very thin, semi-transparent	Slight convulsive attacks; slight talipes varus after 8 months; hydrocephalus	None	Recovery	At 1 month hydrocephalus developed and steadily increased. Death from bursting of the head at 9 months. By the 8th month the spinal tumour had disappeared, and the skin over it was sound.
45	Mr. J. Hutchinson, Path. Soc. Trans., viii	M.	Sacral, size of fœtal head	Hydrocephalus; talipes calcaneus; legs wasted	None	Death	Sac ruptured; death from "exhaustion;" dilatation of central canal of cord
46	Mr. Coghlan, Path. Soc. Trans., ix	M., 4 days	Lumbo-sacral	Sac gangrenous at birth; extreme talipes varus	None	Death	Sac ruptured during birth; death 4th day; spinal cord in sac.
47	Ditto	19 days	Lumbar	Continuous oozing from sac; "feet appeared compressed laterally, and more pliant from side to side than natural"	None	Death	Death on 19th day; the child gradually wasted.
48	Dr. G. Ogle, Path. Soc. Trans., xi	F.	Sacral	Right talipes varus	—	Stillborn	A large bundle of nerves crossed the sac; one other child had hydrocephalus
49	Ditto	—	Lumbar	Double talipes varus	—	Death	Nerves in the sac; great kyphosis from non-development of anterior part of 2nd lumbar vertebra, and growth of this bone backwards.
50	Ditto	—	Dorsi-lumbo-sacral	—	—	Death	Nerves traversed sac.
51	Mr. Callender, Path. Soc. Trans., xv	F., 12 mos.	Lumbo-sacral	Talipes calcaneo-varus; hydrocephalus; convulsions	None	Death	Hydrocephalus became extreme; nerves traversed sac, which was completely shut off from spinal canal by firm fibrous adhesions.

TABLE I.

No.	By whom reported and where recorded.	Sex and age.	Character of tumour, position, size.	Complications, ulceration, paralysis, oozing.	Treatment.	Result.	Remarks.
52	Dr. Playfair, Path. Soc. Trans., xvi	F., 12 mos.	Lumbo-sacral, size of child's head; coverings very thin and vascular	—	—	Recovery	Spontaneous cure of the spina bifida at 12 months.
53	M. Boinet, Gaz. des Hôp., 1859	5 wks.	Size of half a small orange; coverings very thin	—	—	—	—
54	M. Laborie, Annales de la Chir., 1845	—	Dorsal. Skin thin and injected	—	None	Death	Death almost at once after birth; spinal cord adherent to sac.
55	M. Broca, Bull. de la Soc. de Chir.	F., 3 mos.	Lumbar, size of an orange; sac very thick	Paraplegia at birth; this passed off	—	—	In middle line of sac were three depressions, the lowest one discharging pus
56	Ditto	M., 43 years	Lumbar, size of adult head	Incontinence of urine up to 12 years of age; pressure on sac caused vertigo, pain in spine and forehead	None	—	There was a median longitudinal depressed band in sac; death from stone in the bladder; 6 years before had had lithotomy performed. P.M.—Spinal cord not in sac.
57	Ditto	F., 23 years	Lumbar; skin healthy, except over greatest prominence, where it was thin and stretched	Pressure on tumour caused frontal headache	—	Death	Cause of death unknown.
58	M. Marjolin, Bull. de la Soc. de Chir.	F., 3½ years	Lumbo-sacral, size of a fist; skin thick; no impulse on crying; a small depression below centre	Incontinence of urine and fæces; double talipes calcaneus, valgus on left side, varus on right; no paraplegia, but is weak on her legs	—	—	—

59	Mr. Lunn, specially reported	F., 6 mos.	Dorsi-lumbar, size of an orange; covered with skin	Talipes varus; paraplegia	None	Death	Death at 11 months; the tumour had solidified to a large extent; there was some hydrocephalus.
60	Mr. Clutton, specially reported	M., 2 wks.	Cervico-dorsal; sac wall translucent; no ulceration; sac measures 5½ in. from above downwards	Well-nourished child; no paraly sis	—	—	Broad-based tumour, rather tapering. Undergoing spontaneous cure; is still under observation. This child is brother to No. 27 of Table VI.

TABLE II.—*Containing 46 Cases treated by tapping.*

1	Sir A. Cooper, Med.-Chir. Trans., vol. ii	M. 10 weeks	Lumbo-sacral, size of half a billiard ball	None	Tapped on 8 occasions; pressure	Recovery	After 5th tapping fluid was sanious; after 7th it was mixed with coagulable lymph. No paralysis.
2	Ditto	F. 11 days	Lumbar; covering of tumour ulcerated	—	Tapped once	Tumour cured; child died	On 4th day tumour was filled with coagulable lymph; convulsions 6th—10th day; death. P.M.—Brain soft, 6 oz. of fluid in ventricles.
3	Ditto	M.	Sacral, "very large"	None	Tapped over 30 times, and pressure	Recovery	Convulsive twitchings on one occasion.
4	Mr. (now Sir) P. Hewett, Lond. Med. Gaz., 1844	5 mos.	Sacral, size of turkey's egg; skin natural	Hydrocephalus—"not to any great degree"	Tapped 3 times	Death	Child took measles and died. Spinal cord crossed the cavity and was blended with the sac.
5	Mr. Tatum, Med. Times, 1858, ii	M. 20 yrs.	Sacral, pedunculated, larger than man's head; skin ulcerated two or three times	Occasional involuntary movements of legs and passage of fæces	Tapped twice, half a basin of fluid removed	Death	Erysipelas, meningitis. No nerves in sac.
6	Dr. W. Ogle, Med. Times, 1858, ii	1 week, infant	Sacral, considerable size	Right leg atrophied; feet incurved	Tapped 2 or 3 times	Death	Convulsions preceded death in 9 weeks. See also 'Path. Soc. Trans.,' vol. viii.

TABLE II.

No.	By whom reported and where recorded.	Sex and age.	Character of tumour, position, size.	Complications, ulceration, paralysis, oozing.	Treatment.	Result.	Remarks.
7	Dr. Barnes, Med. Times, 1858, ii	M. 4 days	Lumbar, 2½ in. in diameter, irregular; covered with thin membrane	Mother thinks legs were paralysed	Tapped once	Death	Sac burst 2nd day; convulsions in head, trunk, and arms. Death 4th day.
8	Dr. G. McOscar, Med. Times, 1858, ii	M. 14 days	Lumbar, size of orange, rapidly increasing; covered with thin membrane	No motor palsy; sensation diminished	Tapped twice	Death	Child in constant pain; convulsions. Death at 6 weeks.
9	Mr. R. A. Stafford, Injuries and Dis. of Spine, 1826	F. 11 days	Lumbar, large; rapidly increasing; integuments ulcerated	—	Repeated tapping	Tumour consolidated; child died	No paralysis. Death in convulsions. Two nerves seen crossing sac.
10	Mr. F. L. Probart, Lancet, xi	M. 10 wks.	Lumbar, slowly increasing, readily reducible	Complete paralysis of lower limbs; convulsions every day	Tapped 3 times, then pressure	Recovery	Use of limbs gradually regained.
11	Mr. Wardrop, Lancet, xviii	—	Dorsal, very large	None	Repeated tappings	Death	A sudden, severe, and fatal convulsion. P.M.—Spinal cord "sound."
12	Mr. W. Law, Lancet, xvi	M. 2 mos.	Lumbar, 2 in. in diameter; covering very thin	"Too frequent discharge of fæces; constant discharge of urine"	Repeated tapping, with firm pressure	Death	The tumour became small and firm, apparently cured; the fæces and urine were discharged normally; then the tumour suppurated, and the child died.
13	Mr. Walker, Lancet, 1830-31, i	F.	Lumbar, skin normal	None	Tapped	Death	Two cysts opened into the spine; half the cord was in the sacrum; half, which was "ligamentous," passed into the sac.
14	Mr. Dawson, Trans. Prov. Med. Surg. Ass., i	F.	? Small bone	—	Tapped 4 or 5 times	Recovery	Became a strong young woman.
15	Dr. Verrall, Lancet, 1840-41, i	M. 13 yrs.	Lumbar, size of a man's head	None	Tapped and incised	—	—

16	Mr. W. T. Stiff, Lancet, 1850, i	F. 1 mo.	Lumbar, size of a half a small apple; skin over it thin, shiny	None	Tapped 8 or 9 times	Recovery	—
17	Ditto	1 mo.	Lumbar	Defective motor power in legs	Tapped 3 or 4 times	?	Lost sight of.
18	Ditto	F. 3 wks.	Lumbo-sacral	Legs always drawn up	Tapped 6 times	Death	Spinal cord and nerves were spread out over posterior part of sac.
19	Lancet, 1846, ii	F.	Lumbo-sacral, size of a plum	None	Tapped 7 times, then pressure	Recovery	—
20	Mr. H. Smith, Lancet, 1869, ii	M. 32 yrs.	Sacral	None	Tapped, 22 oz. of fluid removed	Recovery	Acute inflammation of walls of sac. Had been often tapped before without effect.
21	Dr. Stevens, Prov. Med. Surg. Journ., 1843-44	F. 8 mos.	Sacral; covering very thin and membranous; tumour was "indented along the middle vertically by a thick irregular band of integument"	None	Tapped 4 times	Recovery	After last tapping, oozing of fluid for 24 hours, and then inflammation of the sac.
22	Mr. Porter, Prov. Med. Surg. Journ., 1844	6 mos.	Lumbar, size of a cricket ball	None	Tapped twice	Death	The sac thickened, skin over it inflamed and ulcerated, and the sac burst.
23	von Langenbeck, Brit. Med. Journ., 1871, ii	F.	Cervical; size of ostrich's egg	Hydrocephalus	Tapped	Death	Continuous draining of fluid; convulsions 3rd day. Neural arch of 5th cervical vertebra unclosed; a fissure one cm. long led into the central canal of the cord expanded to ½-¾ cm.; posterior pillars of cord thinned to 1 mm. Absence of cerebellum.
24	Dr. Camara Cabral, Brit. Med. Journ., 1872, i	25 days	Lumbar	None	Tapped 5 times	Recovery	Very albuminou fluid removed; after the last two tappings signs of meningitis.

TABLE II.

No.	By whom reported and where recorded.	Sex and age.	Character of tumour, position, size.	Complications, ulceration, paralysis, oozing.	Treatment.	Result.	Remarks.
25	Dr. J. Morton, Brit. Med. Journ., 1872, i	—	Lumbar	—	Tapped	Death	Continual leaking of fluid.
26	Mr. Porter, Med. Times and Gaz., xi	—	Sacral; size of a cricket ball	None	Tapped twice	Death	The cord was found spread out in the sac.
27	Mr. Hutchinson, Med. Times and Gaz., 1834	M., 7 mos.	Lumbo-sacral	Hydrocephalus	Tapping	Death	At first treated with a shield; then head enlarged and symptoms of cerebral irritation come on. P.M.—Thick pus in sac.
28	Mr. Hulke, Med. Times and Gaz., 1863, i	—	Lumbar	—	Tapped 5 times	Death	—
29	Mr. W. Campbell, Med. Times and Gaz., 1870, i	—	Dorsi-lumbar	Hydrocephalus; paralysis of left leg	Tapped occasionally to relieve pain	Death	Continuous enlargement of sac; death from "exhaustion." Spinal cord below 8th dorsal vertebra consisted of two unequal divisions.
30	Mr. Hacon, Path. Soc. Trans., xv	F.	Lumbar; size of small orange	Paraplegia; incontinence of urine	Tapped repeatedly	Death	Suppuration in sac and up the cord. Cord adherent to middle of sac and nerves radiated from it.
31	Mr. T. Smith, Path. Soc. Trans., xxi	14 mos.	Lumbar; size of a cricket ball at birth	None	Tapped	Death	Spinal meningitis, death in two days. Two cysts were found, one which was tapped opened into the arachnoid cavity, a second smaller one opened into subarachnoid space.
32	M. Dubois, Ann. de la Chir., 1845	F., infant	Lumbo-sacral; skin thin, injected, at two places leaking	None	Tapped 5 times, then base constricted and transfixed by needles	Death	Meningitis; spinal cord lost in sac.

33	Hoffmann, Boyer's Mat. Chir.	6 wks.	Lumbar, size of orange	None	Tapped and wound plugged with charpie	Recovery	—
34	Camper, Boyer's Mal. Chir.	—	Lumbo-sacral	Lower limbs small, not paralysed	Tapped	Unrelieved	Tumour refilled and steadily grew to size of a man's head. At age of 20 patient had fever, the tumour inflamed, and underwent spontaneous cure
35	Mr. Whitehead, Med.-Chir. Trans., 1884	F., 27	Lumbo-sacral, measured 12 × 10 in., covered by healthy but attenuated skin	—	Tapping, drainage, actual cautery, and free incision	Recovery	Drainage caused severe symptoms.
36	Dr. G. B. Barron, Lancet, 1882, ii	3 yrs.	Lumbar, 17 in. in circumference; coverings thin	Straddling gait	Repeated tapping	Unrelieved	No change.
37	Mr. Jefferson, Lancet, 1883, ii	F., 4 yrs.	Sacral, size of a pigeon's egg, covered by a congenital lipoma	Incontinence of urine and fæces	Incision	Death	Spinal cord in the sac; cerebral sinuses and veins enormously engorged.
38	New York Jour. of Med., quoted in Lond. Med. Gaz., new ser., i	?	Sacral, 3½ in. in diameter; covering of thin red membrane	—	Tapped 4 times	Recovery	Seen two years later quite well.
39	Mr. Hutchinson, Med. Times, 1858, ii	7 mos.	Lumbar, sike of a fist; skin thin	Hydrocephalus	Tapped	Death	At 7 months the tumour and the head rapidly enlarged; 8 oz. of fluid removed; 10th day convulsions and death; pus in the sac, which was crossed by nerves.
40	Dr. G. Ogle, Path. Soc. Trans., xi	M., 15 mos.	Dorso-lumbar	Sloughing of sac	Tapped several times	Death	The tapping caused temporary benefit; the child was quite well until 2 days before death, when the sac sloughed and convulsions occurred. P.M.—Nerves crossed the sac, which was filled with soft fibrinous material.

	No.	By whom reported and where recorded.	Sex and age.	Character of tumour, position, size.	Complications, ulceration, paralysis, oozing.	Treatment.	Result.	Remarks.
Table II.	41	Th. Skinner, Gaz. Méd., 1837	17 mos.	Lumbar	None	Puncture 70 times	Death	Finally the tumour became inflamed. P.M.—No meningitis; no nerves in the sac.
	42	M. Ritter, Gaz. Méd., 1845	—	Lumbar; tumour pedunculated; commencing gangrene of sac	—	Puncture	Death	P.M.—Myelitis.
	43	M. Page, L'Union Méd., 1847	3 mos.	Lumbar; skin inflamed	—	Puncture	Death	Child well for 8 days; death in convulsions on 12th day.
	44	M. Robert, Soc. de Chir., 1847	15 mos.	Sacral	Inability to walk	Puncture 3 times at 10 days' interval	Death	Convulsions; spinal cord in the sac.
	45	M. Pooley, Gaz. Méd., 1848	—	Cervical	Hydrocephalus	Puncture twice at a month's interval	Death	Child wasted; died at 2 months; no nerves in sac.
	46	M. Velpeau, Méd. Opérat., t. iii	—	Lumbar; thin rose-coloured sac	Paralysis	Puncture 4 times	Death	Meningitis.

Table III.—*Containing* 16 *Cases treated by Ligature of the Sac.*

1	Mr. (now Sir James) Paget, Med. Times, 1858, vol. ii	F., 4 mos.	Lumbar; size of her head	Slight talipes varus (?); no paralysis, no hydrocephalus	Subcutaneous ligature of neck of sac	Death	Dribbling of the fluid to a small quantity noted; no convulsions; death 8th day. P.M.—No inflammation; one sac the size of an orange opening by a small aperture into a second the size of a walnut which was crossed by nerves, and

							communicated freely with the spinal canal. Two plates of cartilage in the gap over the sac.
2	Mr. Erichsen, Med. Times and Gaz., 1858, ii	6 wks.	Lumbar; sac size of a small orange with a pedicle the size of a finger	None	Ligature	Death	Sac was not materially diminished by pressure. Child did well till the 8th day; the sac was then sloughed; death in a fortnight from "irritation."
3	Dr. J. G. Wilson, Med. Times and Gaz., 1858, ii	M., few days	Lumbar; size of a small orange; pedicle 1 in. in diameter. Coverings very thin	Morbid tenderness; little, if any, impairment of voluntary motion	Ligature-tightened every day, and puncture	Recovery	Child quite healthy three months after operation. No mention of spinal cord in the sac; opening into spinal sac seen.
4	Mr. Erichsen, Lancet, 1852, ii	Infant	Lumbar; size of a pomegranate	—	Ligature	Death	Tumour not altered by compression. A double ligature applied, as for nævus. Child was well for some days, then died from "constitutional irritation and the disease."
5	Mr. E. Sidebottom, Lancet, 1869, ii	M., 2 days	Lumbar; size of an orange	—	Ligature	Recovery	Ligature was not drawn very tight; two others subsequently applied. Clonic convulsions for several hours after the second ligature. Sac separated in 3 weeks; wound soon healed, leaving lower limbs partially paralysed; this disappeared at the end of 3 months (? from use of cold to spine). At 3 years boy was quite well and strong.
6	Dr. Schindler, Brit. Med. Journ., 1853 (Deutsche Klinik, May 7, 1853)	F., 2 yrs.	Sacral; size of an egg	Intellect defective	Ligature	Recovery	Punctured three times and once injected with iodine without benefit. Two large nerves (?) passed through the sac. No nervous symptoms.
7	Mr. E. Atkinson, Brit. Med. Journ., 1875, i	11 weeks	Cervical; size of a tennis ball; pedicle size of a man's thumb. Coverings thin and transparent	Hydrocephalus, no paralysis	Ligature elastic	Recovery	No nerves in the sac.

TABLE III.

No.	By whom reported and where recorded.	Sex and age.	Character of tumour, position, size.	Complications, ulceration, paralysis, oozing.	Treatment.	Result.	Remarks.
8	M. Laroyenne, L'Union Médicale, 1875	—	Cervico-dorsal	—	Ligature elastic	Recovery	—
9	Dr. A. Turretta, Lancet, 1884, i	F., 2 mos.	Cervical; skin very thin; size of small apple	None	Ligature elastic	Recovery	Covered with thin bluish-red skin. Twitchings of the upper limbs, and a tendency to coma followed upon the ligature. Puncture was tied before the ligature.
10	M. Dubourg, Gaz. Méd., 1841, p. 483	1 week	Lumbo-sacral; very large, reaching to heels	None	Ligature	Death	Death on second day. The ligature was intended to be preliminary to excision of sac.
11	M. Mouchet, L'Union Médicale, 1876	1 day	Sacral; reaching to ham	—	Ligature elastic	Recovery	Death 6 months later from an independent disease.
12	Ditto, ditto	?	Lumbar; 10 × 6 centimetres	—	Ligature elastic	Death	No nervous symptoms; death on 8th day attributed to gastro-enteritis.
13	M. Beynard, Gaz. Méd.	—	Lumbar; size of a hen's egg; skin thin	—	Ligature, gradually tightened	Recovery	By gradual compression of the base of the tumour adhesion was obtained, and the sac was then cut away.
14	M. Latil de Thimécourt, Gaz. Méd., 1845	F., 2½ mos.	Lumbo-sacral; size of a fœtal head; sac thin and membranous	Convulsions when tumour was reduced	Compression of base of tumour	Recovery	The gangrenous sac was cut away.
15	Mr. Vincent Jackson, reported to Committee by Dr. Batterham	F., 3 yrs.	Dorsal; size of an orange; covered by skin. Child well nourished	None	Ligature elastic gradually tightened	Death	Died on 8th day; convulsions. (No. 22 on List, Plate XIX and Fig. 12, p. 370.)
16	Dr. Cavagnis, Annali Universali, July, 1878, quoted in Med. Times and Gaz., 1878, ii	24 dys.	Lumbar; size of two thirds of an egg	—	Ligature elastic	Recovery	Ligature tightened 2nd day, removed 4th day. Fifteen months later child quite well.

TABLE IV.—*Containing 23 Cases treated by Excision of the Sac.*

1	Mr. Dawson, Trans. Prov. Med. Surg. Assoc., vol. i	F., 38 years	Sacral, size of a pomegranate; at birth size of a filbert; had been enlarging for 18 months; covered with healthy skin	—	Excision	Death	Incision was made into tumour by mistake; an attempt was then made to remove the sac; sloughing of wound and sac; suppuration all up the spine outside dura mater; meningitis; no paralysis.
2	M. Royer, Lancet, 1856, i	3 days	Sacral	—	Excision	Recovery	No bad symptoms. Death from diarrhœa 9 months afterwards. P.M.—Cicatrix firm; aperture in 4th and 5th sacral vertebræ; 4th posterior sacral nerves united and passed through the aperture; no 5th pair found. Wound was united by twisted suture.
3	M. Gigon, Lancet, 1861, i	14 years	Position not mentioned; size of fist; pediculated	At 4 years of age there was double talipes valgus and difficulty in walking; this was gone at 14	Removal with écraseur	Recovery	No bad symptoms; twisted suture employed; the sac "distinctly communicated with the spinal canal."
4	M. Dubourg, Prov. Med. Surg. Journ., 1841; Gaz. Méd. de Paris, 1841	F., 8 days	Lumbar, size of apple; very thick walls; opaque; pedicle 5½ in. in diameter	None	Excision	Recovery	No nerves in sac; spinal cord felt in canal; twisted suture employed; cure perfect.
5	Ditto, ditto	F., 11 days	Cervical, size of an orange	None	Excision	Recovery	No nerves seen in the sac; opening into the spine the size of the thumb.
6	Mr. T. Dorrington, Prov. Med. Surg. Journ., 1844	F., 6 days	Dorsal (4th); tumour large, pediculated; sac thin and membranous	None	Excision	Death	Aperture into spine the size of a pin's head; death from suppurative meningitis; puncture of tumour performed twice.

TABLE IV.

No.	By whom reported and where recorded.	Sex and age.	Character of tumour, position, size.	Complications, ulceration, paralysis, oozing.	Treatment.	Result.	Remarks.
7	Mr. Page, Month. Journ. of Med. Sci., 1847	F., 2½ yrs.	Lumbar, 7 in. in circumference; thick integuments	None	Excision	Recovery	Elastic band was applied twice to base of tumour to narrow it; then excision and ligature of the neck of the sac, which was exposed by dissecting up flap; the sac was lined by the dura mater and arachnoid; no nerves in sac.
8	Dr. Tavignot, Med. Times, x	6 days	Lumbar, size of egg; sac very thin	—	Excision	Death	Nerves in sac; death on 8th day, attributed to draining of the fluid.
9	Dr. J. B. Moxon, Lancet, 1850, i	F.	Lumbar, size of a small egg, with a narrow base	None	Excision	Recovery	The base was transfixed with a double ligature and tied, and the sac cut off; child was subsequently seen "running about the house."
10	Dr. Wilson, Path. Soc. Trans., xiv	M., 33 days	Upper dorsal; "integuments thin and translucent," and at time of operation ulcerated	None	Excision	Recovery	Sac at birth was flaccid; it was tapped 5 times to relieve tension; no nerves in sac; only loose adhesions between dura mater and skin; the fluid was in the arachnoid cavity.
11	Hamilton, Annales de Chirurgie	?	?	—	Excision	Recovery	Sac removed included parietes down to parietal arachnoid.
12	Mr. W. Page, Brit. Med. Journ., 1881, ii	F., 2 mos.	Lumbar, size of infant's head; pedicle size of thumb; skin very thin and translucent in two places	Slight talipes calcaneus	Excision	Recovery	Tumour contained two sacs, each lined with membrane like arachnoid, and continued into pedicle; much fibrous tissue and no nerves in the sac; clamp and cautery to pedicle.
13	Dr. T. Joyce, Brit. Med. Journ., 1881, ii	M., 1 day	Dorsal, diameters 5 and 3½ in., circumference of pedicle 3 in.;	None	Excision	Recovery	Subcutaneous ligature of the sac was followed by its excision; convulsions occurred after the 2nd ligature.

14	Mr. A. W. M. Robson, Brit. Med. Journ., 1883, ii	7 dys.	Lumbar, size of tennis ball	None	Excision	Recovery	Cauda equina was seen lying in the spinal canal; separate suture of arachnoid by skin; a piece of rabbit's skin was laid between the two.
15	Mr. Jessop, Brit. Med. Journ., 1883, ii	3 wks.	Lumbar, size of a tangerine orange	—	Excision	Recovery	Flaps of skin were raised over the pedicle; the neck of sac was tied with catgut, and the rest was cut off; careful suture of sac and skin.
16	Mr. Hilton, Lancet, 1850, i	1 year	Lumbar, size of a small Dutch cheese	None	Excision	Death	Skin was dissected up, the neck ligatured, and the sac cut off; no nerves in the sac; continuous oozing of cerebro-spinal fluid; death in about a week.
17	M. Dubourg, Gaz. des Hôpitaux, 1849	—	Cervical, size of hen's egg; skin thin and transparent	Reduction of tumour caused pain and violent distress	Excision	Recovery	Wound united with 4 pins and twisted suture.
18	Gaz. des Hôp., 1846	—	Lumbar, size of an apple	—	Excision	Death	No nerves in sac; death in 40 hours after the operation.
19	M. Courtin, Bull. de la Soc. Anat., 1849	—	Sacral, 1 decimetre in circumference	—	Excision	Death	No nerves in sac; meningitis; death 3rd day.
20	M. Nott, Gaz. Méd., 1856	—	Lumbar, 1½ in. across	—	Excision	Recovery	No nerves in sac.
21	Mr. A. W. M. Robson, Lancet, 1885, i	F., 18 days	Lumbar, sac thin and translucent; child puny, ill-developed	—	Excision	Death	Wound healed by first intention; death from marasmus.
22	Ditto	F., 16 years	Lumbar, size of a fœtal head	Talipes, suppuration of sac	Excision	Recovery	Tumour aspirated 8 times before excision
23	Ditto	7 yrs.	Lumbar, 7½ in. in circumference; sac thin and inflamed	—	Excision	Recovery	Mr. Robson's four cases are fully reported in this volume of the Clinical Society's Transactions.

TABLE IVB.—*Two Cases treated by Plastic Operation.*

No.	By whom reported and where recorded.	Age and sex.	Character of tumours, position, size.	Complications, ulceration, paralysis, oozing.	Treatment.	Result.	Remarks.
1	Mr. Borlase Childs, Med. Times and Gaz., xxxvi	1 mo.	Lumbo-sacral, size of pigeon's egg	None	Skin dissected off sac, latter folded into spinal canal, and the skin united over it by hare-lip pins	Death	During the operation the sac was pricked in two or three places; several times during operation child became very rigid, afterwards was very restless and ill, and died next morning.
2	Dr. W. Koch, Mittheil. über Fragen der Wiss. Med., 1881	F.	Dorsi-lumbo-sacral	Hydrocephalus, paraplegia	Two flaps of skin raised and united over the tumour	Recovery	Child died 8 weeks afterwards from extensive hydrocephalus without signs of meningitis.

TABLE V.—*Containing* 26 *Cases treated by the Injection of Simple Solution of Iodine.*

1	Mr. W. M. Coates, Lancet, 1866, vol. i	F. 3 mos.	Lumbo-sacral, size of a walnut; skin very thin	—	Injection: Iodine gr. iiss, iod. of pot. gr. v, in water ʒij	Recovery	"Child runs about as other children."
2	M. Rouse, Lancet, 1869, i (Bull. de Thérap.)	F. 6 wks.	Sacral, very large, hanging down to lower third of thighs	—	Tapped, iodine injected, sac kneaded, all fluid withdrawn by exhausting syringe	Recovery	Canal was occluded during operation.

3	Dr. Gross (N. Amer. Med.-Chir. Rev., Nov., 1860), Brit. Med. Journ., 1861, i	M. 11 weeks	Lumbar, size of orange	None	Injection of iodine, gr. ½, and iod. of pot. gr. ¼	Death	Sac burst on 9th day; child died in convulsions. No nerves in sac, which was injected and coated with a layer of lymph.
4	Ditto	M. 3 mos.	Lumbo-sacral, size of billiard ball	None	Injected 14 times as above, iodine increased to gr. ⅛	Death	One-third cavity obliterated by coagulated lymph. Rupture of sac, and death in convulsions.
5	Dr. Léyerie, Brit. Med. Journ., 1862, i	F. 9 mos.	Lumbar, size of orange	Complete paralysis of lower limbs, partial of upper	Injection of equal parts of tincture of iodine and water	Recovery	Paralysis passed off, leaving only incontinence of urine. Two years later the tumour was the size of a walnut.
6	Dr. Brainard, Med. Times and Gaz., xviii (Illinois & Ind. Med. Surg. Journ.)	F. 13 yrs.	Sacral	Incontinence of urine and fæces, partial paraplegia, idiocy	Injection of iodine gr. ss in a drachm of water	Recovery	Tumour much shrunk.
7	Dr. Brainard, Amer. Med. Jour. Med. Soc., 1861	F. 3 yrs.	Sacral; skin healthy, except at one spot, where it is nævoid	None	Injection: Iodine gr. iiss, iod. of pot. gr. viiss, water ℥ss	Recovery	—
8	Dr. Brainard, Bull. de la Soc. de Chir.	F. 13 yrs.	Sacral	None	Injection with diluted iodine	Recovery	—
9	Ditto	F. 8 days	Lumbar	Hydrocephalus	Ditto	Recovery	—
10	Ditto	F. 3 mos.	Lumbar	Hydrocephalus	Ditto	Recovery	—
11	Ditto	M.	?	None	Ditto (several times)	Recovery	Suppuration of sac.

TABLE IVB.—*Two Cases treated by Plastic Operation.*

No.	By whom reported and where recorded.	Age and sex.	Character of tumours, position, size.	Complications, ulceration, paralysis, oozing.	Treatment.	Result.	Remarks.
1	Mr. Borlase Childs, Med. Times and Gaz., xxxvi	1 mo.	Lumbo-sacral, size of pigeon's egg	None	Skin dissected off sac, latter folded into spinal canal, and the skin united over it by hare-lip pins	Death	During the operation the sac was pricked in two or three places; several times during operation child became very rigid, afterwards was very restless and ill, and died next morning.
2	Dr. W. Koch, Mittheil. über Fragen der Wiss. Med., 1881	F.	Dorsi-lumbo-sacral	Hydrocephalus, paraplegia	Two flaps of skin raised and united over the tumour	Recovery	Child died 8 weeks afterwards from extensive hydrocephalus without signs of meningitis.

TABLE V.—*Containing* 26 *Cases treated by the Injection of Simple Solution of Iodine.*

1	Mr. W. M. Coates, Lancet, 1866, vol. i	F. 3 mos.	Lumbo-sacral, size of a walnut; skin very thin	—	Injection: Iodine gr. iiss, iod. of pot. gr. v, in water ʒij	Recovery	"Child runs about as other children."
2	M. Rouse, Lancet, 1869, i (Bull. de Thérap.)	F. 6 wks.	Sacral, very large, hanging down to lower third of thighs	—	Tapped, iodine injected, sac kneaded, all fluid withdrawn by exhausting syringe	Recovery	Canal was occluded during operation.

3	Dr. Gross (N. Amer. Med.-Chir. Rev., Nov., 1860), Brit. Med. Journ., 1861, i	M. 11 weeks	Lumbar, size of orange	None	Injection of iodine, gr. ½, and iod. of pot. gr. ¼	Death	convulsions. No nerves in sac, which was injected and coated with a layer of lymph.
4	Ditto	M. 3 mos.	Lumbo-sacral, size of billiard ball	None	Injected 14 times as above, iodine increased to gr. ½	Death	One-third cavity obliterated by coagulated lymph. Rupture of sac, and death in convulsions.
5	Dr. Léyerie, Brit. Med. Journ., 1862, i	F. 9 mos.	Lumbar, size of orange	Complete paralysis of lower limbs, partial of upper	Injection of equal parts of tincture of iodine and water	Recovery	Paralysis passed off, leaving only incontinence of urine. Two years later the tumour was the size of a walnut.
6	Dr. Brainard, Med. Times and Gaz., xviii (Illinois & Ind. Med. Surg. Journ.)	F. 13 yrs.	Sacral	Incontinence of urine and fæces, partial paraplegia, idiocy	Injection of iodine gr. ss in a drachm of water	Recovery	Tumour much shrunk.
7	Dr. Brainard, Amer. Med. Jour. Med. Soc., 1861	F. 3 yrs.	Sacral; skin healthy, except at one spot, where it is nævoid	None	Injection: Iodine gr. iiss, iod. of pot. gr. viiss, water ℥ss	Recovery	—
8	Dr. Brainard, Bull. de la Soc. de Chir.	F. 13 yrs.	Sacral	None	Injection with diluted iodine	Recovery	—
9	Ditto	F. 8 days	Lumbar	Hydrocephalus	Ditto	Recovery	—
10	Ditto	F. 3 mos.	Lumbar	Hydrocephalus	Ditto	Recovery	—
11	Ditto	M.	?	None	Ditto (several times)	Recovery	Suppuration of sac.

TABLE V.

No.	By whom reported and where recorded.	Age and sex.	Characters of tumour, position, size.	Complications, ulceration, paralysis, oozing.	Treatment.	Result.	Remarks.
12	Dr. Brainard, Bull. de la Soc. de Chir.	?	?	—	Injection with diluted iodine twice	Recovery	—
13	Ditto	?	?	—	Ditto	Recovery	—
14	M. du Tremblay, Bull. de la Soc. de Chir.	M. 14 mos.	Lumbar	None	Injection of a strong solution of iodine	Recovery	—
15	M. de Chassaignac, Bull. de la Soc. de Chir.	M. 2 mos.	Sacral	None	Injection of iodine	Recovery	Convulsions.
16	M. Velpeau, Bull. de la Soc. de Chir.	F., 4 mos.	Lumbo-sacral	None	Injection of iodine 6 times	Recovery	Slight convulsions.
17	M. Maisonneuve, Bull. de la Soc. de Chir.	M., 4 days	Lumbar	Paraplegia	Injection of iodine	Recovery	—
18	M. Viard, Bull. de la Soc. de Chir.	M., 2 mos.	Lumbo-sacral	Paraplegia	Ditto	Recovery	—
19	M. Nélaton, Bull. de la Soc. de Chir.	M., 8 days	Lumbar	None	Ditto	Unrelieved	—
20	M. Robert, Bull. de la Soc. de Chir.	M., 3 mos.	Lumbar	None	Ditto	Recovery	Convulsions.

21	M. Serres, Bull. de la Soc. de Chir.	F., 8 days	Lumbar	None	Injection of iodine twice	Death	Convulsions.
22	M. Jobert, Bull. de la Soc. de Chir.	1 month	Lumbo-sacral	None	Ditto	Death	Convulsions.
23	M. Texier, Bull. de la Soc. de Chir.	F., 3 mos.	Lumbar	None	Ditto	Death	Convulsions.
24	M. Viard, Bull. de la Soc. de Chir.	M., 2½ mos.	Sacral; skin ulcerated	Hydrocephalus; paraplegia and paralysis of bladder	Injected with strong iodine, left in 10 minutes	Recovery	Fluid markedly albuminous; death from diphtheria æt. 4. years. Sensation normal; incontinence of urine; feet paralysed.
25	Mr. R. Howson, Lancet, 1884, ii	2 wks.	Lumbar, size of pigeon's egg, covered by normal integuments	None	Injection 3 times *a.* Tinct. Iodi mxv Aq. mxl *b.* Tinct. Iodi Aq. partes equales *c.* Tinct. Iodi ʒj	Recovery	After third injection the tumour was contracted to the size of a cherry; it was then cut off.
26	Dr. Demme, 20ter Jahresbericht des Jenner'schen Kinderspitals in Bern	F., 3 yrs ?	Cervical	None	Injection with 2·5 grammes of tinct. of iodine	Recovery	—

TABLE VI.—*Containing 71 Cases treated by the Injection of Dr. Morton's Iodo-Glycerine Solution.*

No.	By whom reported and where recorded.	Sex and age.	Character of tumour, position, size.	Complications, ulceration, paralysis, oozing.	Treatment.	Result.	Remarks.
1	Dr. Morton, Lancet, 1876, vol. ii	14 dys.	Lumbar; size of a peach	—	Injection of solution of iodine and iod. of pot. in glycerine	Recovery	—
2	Mr. W. Berry, Lancet, 1876, ii	M., 3 mos.	Lumbar	Hydrocephalus	Ditto, 4 times	Unrelieved	After each injection some improvement was noticed. Death from marasmus 4 months later
3	Dr. Morton, Brit. Med. Journ., 1872, i	M., 2 mos.	Lumbar; size of orange	None	Ditto, twice	Recovery	Simple tapping twice previously.
4	Ditto, do.	F., 2 mos.	Lumbar; size of orange; several striæ seen on its internal surface	None	Ditto, once	Recovery	—
5	Dr. Morton, Brit. Med. Journ., 1874, ii	7 wks.	Upper dorsal; size of peach	None	Ditto, twice	Recovery	—
6	Mr. J. E. Burton, Brit. Med. Journ., 1875, i	—	Lumbar	Talipes varus; paralysis of legs and sphincters	Ditto	Death	Convulsions and rigidity of arms and hands.
7	Mr. J. A. Angus, Brit. Med. Journ., 1875, i	M.	Lumbar; size of peach	—	Ditto, twice	Recovery	—
8	Dr. Long, Brit. Med. Journ., 1876, i	—	Sacral; size of small orange	Talipes varus	Ditto	Death	From shock.

9	Mr. Eate, Med. Times and Gaz., 1875, ii	M.	Lumbar; size of large orange; skin ulcerated at apex	Paraplegia; sphincter ani "enfeebled"	Ditto	Recovery	Rupture of sac day after injection, slow draining of fluid, no ill effect noticed.
10	Mr. Ewart, Liverpool and Manchester Med. and Surg. Rep.	—	Lumbar; surface ulcerated	Slight talipes varus	Ditto, 5 times	Recovery	—
11	Ditto	F.	Lumbar; tumour soft, fluid highly albuminous	—	Ditto, 4 times	Recovery	Death 3½ months after last tapping; hydrocephalus developed just before death.
12	Ditto	F.	Lumbar	Sac ruptured at birth; reflex action wanting	Ditto, once	Recovery	Death suddenly 6 weeks later in convulsions. "Very little movement in legs."
13	Mr. Berry, (private letter)	F., 6 wks.	Lower dorsal; size of orange; sac thin and membranous, had doubled in size since birth	Child puny; hydrocephalus	Ditto	Death	Tumour swollen, its base appeared solid; death 20th day.
14	Mr. Pearce Gould, Clin. Soc. Trans.	M., 18 mos.	Lumbo-sacral	None	Ditto, 3 times	Recovery	—
15	Mr. Pearce Gould, (not published)	M., infant	Sacral; sac thin	Talipes	Ditto, twice	Death	Suppurative meningitis.
16	Mr. F. H. Davis, Lancet, 1877, i	F., 6½ mos.	Lumbar; 8½ in. in circumference	Hydrocephalus; double talipes varus; knee-joints imperfect	Ditto, 3 times	Recovery	Spina bifida cured; death 10 days later from hydrocephalus.
17	Dr. Cormack, Brit. Med. Journ., 1877, i	4 days	—	Perforated by midwife in mistake for the membranes	Ditto	Recovery	Pad was applied to stop leakage, and 4 days after it was injected with one drachm of a mixture of Lin. Iodi ℥j with Glycerine ℥iij.

TABLE VI.

No.	By whom reported and where recorded.	Sex and Age.	Character of tumour, position, size.	Complications, ulceration, paralysis, oozing.	Treatment.	Result	Remarks.
18	Dr. Cormack, Brit. Med. Journ., 1877, i	2 days	Lumbo-sacral; membranous sac	Paraplegia	Injection of solution of iodine & iod. of pot. in glycerine twice	Death	Morton's solution used 2nd day and 14th day. Death half hour after 2nd injection. P.M.—Only a small central cavity unobliterated; sac filled with venous blood which welled up from the spine; cause of death shock and hæmorrhage. Cauda equina in sac.
19	Dr. St. George, Brit. Med. Journ., 1877, ii	14 dys.	Lower dorsal	None	Ditto	Recovery	Subsequently died from bronchitis (?)
20	Dr. Thomson, Brit. Med. Journ., 1878, ii	M., 1 month	Upper lumbar; large, 12 in. in circumference; covered in part by thin purple ulcerated membrane	Paralysis of anus, not of legs. Talipes	Ditto, 3 times	Recovery	Anus recovered power. Strands of nerves (?) were crossing sac. Severe shock after 1st operation. Child strong and healthy.
21	Mr. W. Berry, Brit. Med. Journ., 1881, i	F., 5 mos.	Dorsal, size of small orange; partly covered by thin transparent membrane	Complete paraplegia	Ditto	Death	Draining of fluid for 6 days; death on 7th day.
22	Mr. Bryant, Brit. Med. Journ., 1884, i	6 mos.	Lumbar, 2¾ × 3½ in.; skin very thin, ruptured at one point	—	Ditto	Death	Death 5th day, "apparently from subsequent draining of fluid from the sac."
23	Mr. Noble Smith, Lancet, 1883, ii	2 mos.	Lumbo-sacral, circumference at base 8½ in.; centre of sac ulcerated	Right leg small, weak, talipes valgus	Ditto	Relieved	Tumour was reduced to rough thickened skin except at one small area; hydrocephalus came on 6 months later and caused death in 2 years; no nerves in sac.

	24	Dr. Marshall, Lancet, 1883, ii	6 wks.	Lumbar, 3½ × 2½ in.	Paraplegia; hydrocephalus	Ditto, 4 times	Recovered	Child could move his legs and feet a little after the 1st injection; hydrocephalus continued to increase.
	25	Mr. P. Gould, Clin. Soc. Trans.	—	Lumbo-sacral	None	Ditto	Recovery	—
	26	K. McLeod, Operative Surgery	M., 1 year	Sacral, size of fœtal head	—	Ditto, 8 times	Unrelieved	No effect.
	27	Mr. Clutton, Clin. Soc. Trans., 1883	4 wks.	Lumbar, small, very thin walls	No paralysis; child quite healthy	Ditto, once	Recovery	—
	28	Ditto	2 wks.	Lumbar, surface of sac, in part ulcerated	—	Ditto, once	Death	Sudden death immediately on making the injection.
	29	Dr. McWatt, Edin. Med. Journ., 1880	F., 3 wks.	Cervical, size of small orange; skin thin and livid	Hydrocephalus	Ditto, twice	Relieved	Death soon after from hydrocephalus; tumour was beginning to enlarge again when last seen.
	30	Ditto	M., 3 wks.	Size of Mandarin orange; skin nævoid	—	Tapped, and 2 days later mxx Morton's fluid injected; operation repeated 3 months later and 3ss injected	Recovery	—
Cases not hitherto published.	31	Mr. Thomas Smith	F., 8 mos.	"Large translucent sac" in coccygeal region	Paralysis of lower extremities and club-feet	Injected on 3 occasions	Death	There were no ill consequences after the first two injections practised at an interval of 1 month. On the 3rd occasion the child became very blue, and breathed with difficulty; she died comatose a few hours later. The autopsy recalled an almost obliterated coccygeal cyst lying superficial to a spina bifida, but having no connection with it. The lining membrane of the latter was stained with iodine.

TABLE VI.

No.	By whom reported and where recorded.	Sex and age.	Character of tumour, position, size.	Complications, ulceration, paralysis, oozing.	Treatment.	Result.	Remarks.
32	Mr. Thos. Smith	F., 8 wks.	Sac size of half an orange over sacral region; skin over tumour thick	No paralysis or deformities	Injected twice	Recovery	The injections were practised at an interval of 2 weeks; no immediate effects. "Discharged; no further troubles; sac diminished."
33	Mr. Callender (reported by Mr. A. Bowlby)	F., 3 wks.	Sac size of an orange in lumbo-sacral region; skin thin	Paralysis of sphincters and of lower extremities	Injected once	Death	Blanched and collapsed day after injection. Tumour diminished, and child improved 5 days after injection. Died 8 days after injection from sloughing of sac wall and asthenia; no meningitis.
34	Mr. Walsham	F., 9 mos.	"Large sac, translucent, evidently containing nerve-trunks"	Hydrocephalic when first seen	Injected once	Nil	Removed from the hospital by parents and lost sight of; no symptoms produced by the treatment.
35	Mr. M. Baker	M., 5 mos.	A healthy child in other respects, and able to move its limbs. "Sac had thin bluish walls, mottled with thicker skin texture"	—	Injected twice	Recovery	Soon after the first injection the urine dribbled away; soon after 2nd injection the sac became thickened; two or three weeks later there was loss of motion and of sensation in the lower extremities, which persisted.
36	Mr. Thomas Smith (reported by Mr. Eve)	M., 7 wks.	Rapidly increasing sac, in lumbo-sacral region, of large size	Oozing	Injected twice, sac painted with collodion	Death	No convulsions; child died with the head retracted and marked pyrexia. Complete paralysis of the upper extremities came on; death occurred 21 days after 1st injection; usual anatomy.

37	Mr. Clutton	M., 11 days	Lumbar region; skin margin; membranous centre	Paralysis of sphincters; ulceration on surface of sac; very slight talipes calcaneus	Injected once	Doubtful	—
38	Mr. B. Pitts	M., 2 mos.	Lumbar, skin around base; membranous centre	Paralysis of limbs; talipes varus	Injected twice	Recovery	Very restless for 24 hours. Cyst gradually shrank. Injected at intervals of 14 days. Sac sloughed subsequently and discharged pus. Paralysis of limbs remained unaffected.
39	Ditto	M., 3½ wks.	Lumbar, skin margin; membranous centre	Ulceration of sac wall; oozing	Injected once (3ij)	Recovery partial	Some convulsions followed, and subsequently paralysis of limbs, bladder, and rectum, *which completely passed off*. Died of hydrocephalus one month subsequently.
40	Ditto (Mr. Ballance)	M., 4 mos.	Lumbar, skin margin; membranous centre	Talipes calcaneus	Injected once (3ij)	Recovery	Two weeks after the injection, the tumour was reduced almost to the level of the skin; complete paraplegia supervened, but passed off again. No convulsions.
41	Mr. Godlee	M., 9 wks.	High dorsal (2nd to 6th); skin margin, membranous centre	None mentioned	Injected twice	Recovery	Injections practised with an interval of 14 days. Child was fretful for a few days after the first injection; after the second there was slight paraplegia and retraction of the head, which passed off. The child died one month after discharge from the hospital "emaciated and having had some slight convulsion."
42	Ditto	M., aged about 6 wks.	Lumbo-sacral, skin margin; membranous centre	None mentioned	Injected twice	Recovery	The tumour gradually shrivelled up. No ill effects produced at the operation.
43	Mr. Haward	M., 4½ mos.	Lumbar, skin margin; membranous centre	Talipes equinus, both feet; paralysis of sphincters	Injected once	Death	The child at once became very pale; death took place two days subsequently, without convulsion; the child never recovered from the shock.
44	Mr. Heath	Sex not specified	Lumbo-sacral	Hydrocephalus	Injected once	Recovery partial	A very marasmic child; the hydrocephalus continued to develop. When seen by the committee, it was thought that the spina bifida, though much reduced in size, was only partially cured.

	No.	By whom reported and where recorded.	Sex and Age.	Character of tumour, position, size.	Complications, ulceration, paralysis, oozing.	Treatment.	Result.	Remarks.
TABLE VI.	45	Mr. Morgan	M., 8 mos.	Lumbo-sacral, skin covered in greater part	None	Injected 4 times	Recovery	No symptoms after the two first injections; after the third there was some temporary retention of urine, and cystitis, which passed off. The sac gradually shrank.
	46	Ditto	M., 8 mos.	Lumbo-sacral, healthy skin over the whole tumour	Slight talipes calcaneus	Injected 4 times	Recovery	No immediate symptoms followed the operations. The left lower limb became paralysed, but recovered again perfectly.
	47	Ditto	No details	Mid-dorsal	None	Injected	Death	No details.
	48	Ditto	Ditto	Lumbar	No paralysis	Injected	Death	Sac was heart-shaped and very considerable in size.
	49	Ditto	Ditto	Lumbo-sacral	Paraplegia; constant oozing; centre of sac presented a mass of granulations	Injected	Death	Tumour slowly increased in size. Hydrocephalus came on, and became very marked. Death occurred at about two years of age, after two severe convulsions.
	50	Ditto	Ditto	Lumbo-sacral, sac rather to the left of the median line	Paralysis of lower limbs	Injected once	Death	This and the three preceding cases all died. In two, the large size of the communication with the spinal canal made the operation from the first almost hopeless. Mr. Morgan was unable to give further details.
	51	Mr. Thomson Haywood (Lanark)	M., 5 wks.	Lumbar region (1st and 2nd vert.). Sac, size of an orange; skin margin, membranous centre. Well nourished	No paralysis of limbs, bladder, or rectum	Tapped once and painted with collodion; 3 days later tapped & injected	Recovery	A little puckered skin is all that remains. Slight collapse after the injection; under stimulants it soon passed off.

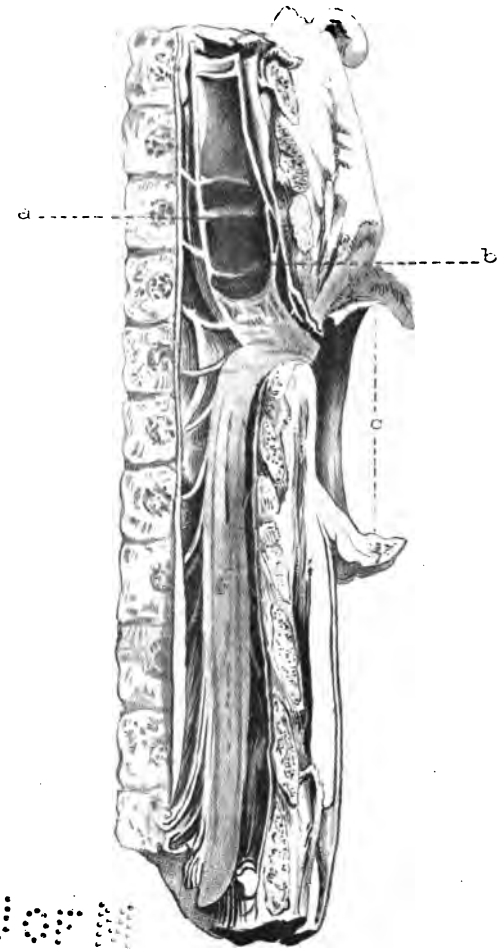
52	Dr. Oxley (Liverpool)	M., 2 days	Sacral, skin margin; membranous centre	Paralysis of limbs; a marasmic child; ulceration on summit of sac	Injected twice	Death	Sac became almost solid, and rapidly diminished in size, then suppurated and discharged pus; some convulsions at this time. The child died in convulsions two months subsequently.
53	Mr. Rawdon (Liverpool)	F., 2 wks.	Sacral, thick skin around base; membrane elsewhere	Paralysis of limbs	Injected twice, collodion afterwards	Recovery	Rapid diminution in size. The child was in good health one year later; paralysis of limbs unchanged.
54	Mr. Pughe (Liverpool)	F., 6 mos.	Lumbo-sacral	Limbs well developed	Injected 7 times within 1 month, then twice subsequently	Recovery	Ten minims of Morton's fluid only were injected. "Some slight signs of spinal irritation" were caused by the two last injections.
55	Dr. Davidson (Liverpool)	M., 10 dys.	Sacral, everywhere covered with skin, which was "thin in the centre"	Movements of the limbs not impaired; well developed; sphincters good	Injected 4 times	Recovery	In 3 months the tumour was "pretty solid and contracted;" 14 months later child was in good health.
56	Mr. R. W. Parker	M., 2 wks.	Lumbo-sacral; skin margins; membranous centre	Ulceration; talipes calcaneus	Injected once	Death	The injection produced no sensible effect, death taking place from general marasmus.
57	Ditto	F., 5 wks.	Lumbo-sacral; skin margins; membranous centre; sac size of orange	Ulceration over apex; talipes calcaneus; paralysis of limbs and sphincters	Injected twice	Recovery	The sac became very much swollen and solid. Some hydrocephalus set in, but did not proceed to any great extent, and then remained stationary. Paralysis remained as before. A deep well-marked "post-anal dimple." Child is still alive, and well in other respects.
58	Ditto	F., 2 dys.	Lumbo-sacral; skin margins; membranous centre; size of small orange	Ulceration on surface; oozing	Injected once	Death	On the day following operation child became very blue and cold; refused food. Some sloughing of surface of sac, and more oozing took place; died on 4th day. Excess of fluid found in the lateral ventricles. The child was well nourished.

TABLE VI.

No.	By whom reported and where recorded.	Sex and Age.	Character of tumour, position, size.	Complications, ulceration, paralysis, oozing.	Treatment.	Result.	Remarks.
59	Mr. R. W. Parker	M., 12 years	Lumbo-sacral; well-formed skin, and a thick layer of fat covered the sac, which was about as large as a fist	Sac was placed to the left of the median line; at the time of operation there were incontinence of urine and loss of walking power, which had lately supervened	Injected 3 times	Nil	The 2nd injection was followed by a rise in temperature and frontal headache, which quickly passed off. The other injections produced no visible effects whatever. After several months the condition remained unchanged. This boy had done well, and had not experienced any ill effects up till about his 10th year. The sac then began to enlarge, incontinence of urine came on gradually, loss of muscular power in the lower limbs, and loss of expulsive power in the rectum, the sphincter being powerless also. Fæces collected in the lower bowel, and towards the latter end of life had to be scooped out. Subsequently the sac was opened and emptied under antiseptic precautions; the boy died some 3 or 4 months afterwards. (Plate XX and Fig. 7, p. 359.)
60	Ditto	M., 14 days	Lumbo-sacral; skin margins; membranous centre; size of small orange	Talipes calcaneus	Injected once	Death	The injection produced no marked effect at the time. Four days subsequently the child became very dusky, and died on the 6th day of general marasmus.
61	Ditto	F., 2 wks.	Lumbar; sac wall membranous for most part with scattered patches of skin; sac size of an orange	Talipes calcaneus	Injected once	Death	The injection did not produce any palpable immediate effects. The child died of marasmus.
62	Ditto	M., 5 days	Lumbo-sacral; skin round margins, sac elsewhere memb.; size of an orange	Double talipes varus; paralysis of limbs and sphincters	Injected once	Death	The serum which was drawn previous to the injection was turbid, but not offensive. Death took place in convulsions 36 hours after the injection.

63	Ditto	M., 6 wks.	Sacral; skin around base; sac wall elsewhere membranous	No paralysis; slight ulceration over apex of sac	Injected twice	Recovery	The injections produced no appreciable immediate effects. The sac gradually increased in size, and finally nothing but a little puckered skin remained. The child was well a year afterwards.
64	Ditto	F., 3 days	Lumbo-sacral; sac size of a small orange	No paralysis; sac growing rapidly	Injected once	Death	No immediate effects produced by the injection. The child died 1 month later of diarrhœa.
65	Ditto	F., 6 wks.	Lumbo-sacral; skin base, membranous centre; sac heart-shaped	No paralysis	Injected 4 times	Recovery	The first 3 injections were practised within a month, at the end of which time no alteration had taken place. The 4th injection was followed by a rise of temperature and some retraction of the head. Without altering much in size, the tumour became quite solid, a little spontaneous oozing taking place previously. Seven weeks from the 1st injection, and 3 weeks after the 4th, the child one day became collapsed, but rallied under the administration of stimulants. Was discharged from hospital with the tumour much shrunken. A month later it was found dead in bed one morning, having probably been overlaid. The condition of the tumour is shown in Plate XIV, fig. 2.
66	Ditto	M., 2 wks., a 7 mos. child	Lumbo-sacral; sac size of a small orange; skin margins; membranous centre	Ulceration of apex; paralysis of limbs and bladder; talipes; slight prolapse of rectum	Injected 5 times	Death	No immediate effects produced by the injections, nor did any change take place in the sac itself. The child died some weeks after the last injection of marasmus. The condition of the tumour is shown in fig. 6.
67	Ditto	F., 3 days, an 8 mos. child, well nourished	Lumbo-sacral; skin margins; membranous centre. Much constricted at base; small sac	Slight ulceration of apex of sac; talipes calcaneus	Injected once	Death	The child died aged 12 days; the injection produced no visible effect.

	No.	By whom reported and where recorded.	Sex and Age.	Character of tumour, position, size.	Complications, ulceration, paralysis, oozing.	Treatment.	Result.	Remarks.
TABLE VI.	68	Mr. T. Holmes	M., 5 wks.	Coccygeal; sac size of a small orange	None mentioned	Injected once	Recovery	After a portion of the fluid had been withdrawn another cyst became obvious, which did not empty with the former, and into which the injected fluid did not penetrate. A few days later a small piece of shrivelled skin sloughed. The second cyst suppurated 13 weeks later; it was treated like a common abscess. The child got quite well. The sac was thought to be multilocular.
	69	Mr. Golding-Bird	M., 6 mos., well nourished	Lumbo-sacral; skin margin; membranous centre; sac size of small orange	No paralysis; talipes calcaneus	Injected once	Death	Four ounces of fluid withdrawn previous to the injection. Twenty minims of iodo-glycerine injected. Next day the child was ill, had "crowing inspiration;" temp. 102° F. It died on the 3rd day quietly, never having been convulsed. At the autopsy the sac contained sero-purulent fluid, the lateral ventricles much dilated.
	70	Mr. Knaggs (Huddersfield)	F., 6 mos.	Not stated	—	Injected once	Death	Death occurred 1 week subsequently, and was "preceded by convulsions." No further particulars obtainable.
	71	Mr. Thomas Smith (Dr. Sloman)	F., 5 wks.	Mid-dorsal	Some paralysis of lower limbs, especially of right side	Injected once	Recovery	The child is now 9½ years old. The tumour is now "a flattened protrusion, about 2 × 2 inches, roughly circular, and only stands about ¼ inch from the surrounding skin. Her paralysis continued, but "for nearly two years she has been getting about without any support at all." There is still some incontinence of urine.



Stephen Miller (Glasgow) del.

Danielsson & Co. lith.

DESCRIPTION OF PLATE XIII.

Vertical section of the parts concerned in a spina bifida of the mid-dorsal region; the chief part of sac has been removed.

a.—Dilated central canal in the portion of spinal cord above the protrusion.

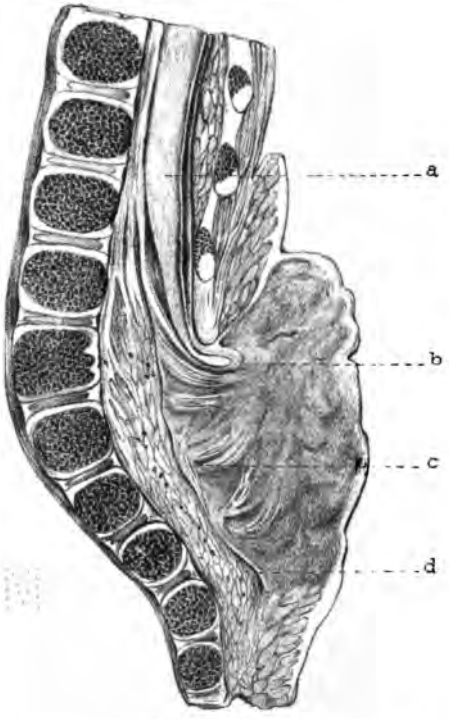
b.—Incomplete septa crossing the dilated canal.

c.—Remains of sac-wall.

There is a slight prolapse of the cord into the mouth of the sac. *Vide* p. 365, No. 3.

Stephen Miller (Glasgow) del.

Fig. 1.



S.G.Shattock del.

Fig. 2.

Danielsson & Co lith.

DESCRIPTION OF PLATE XIV.

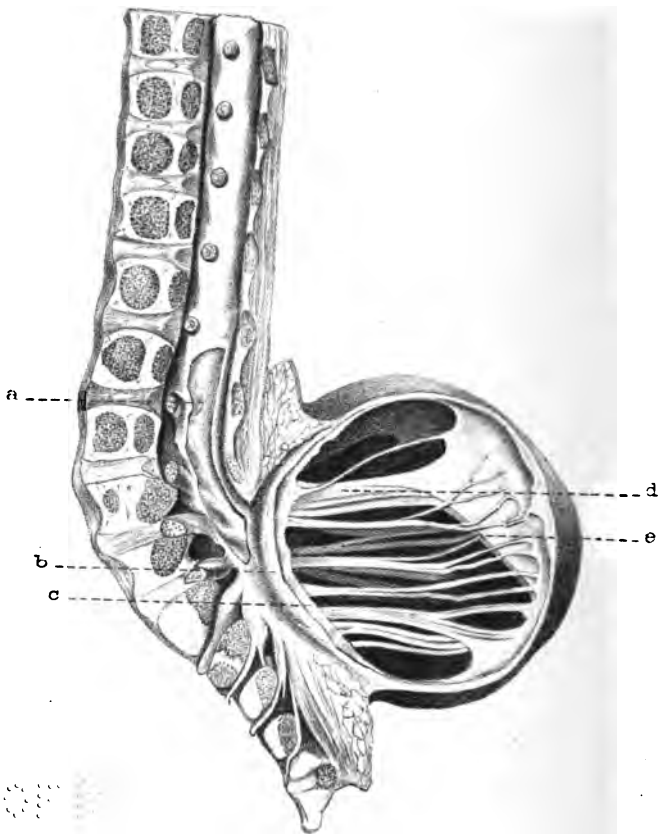
Fig. 1.—Portion of spinal column from the dorsal region; the laminæ of the right side have been removed to show the spinal cord.

a.—Outer margin of the aperture in the neural arch through which the protrusion has occurred.
b.—Neck of the consolidated protrusion.
c, *d.*—Reflected portions of the dura mater.

The line of origin of the nerve-roots is quite normal. There is slight prolapse of the cord into the mouth of the sac, as in the preceding specimen. *Vide* p. 365, No. 4.

Fig. 2.—Vertical section of lower part of the spinal column after the cure of a spina bifida by the injection of Dr. Morton's iodo-glycerine solution.

a.—Lower portion of the spinal cord above the sac.
b.—Young connective tissue produced after the injection and filling the sac; into this the spinal cord is traceable for a short distance.
c.—Nerve-roots lying in the new connective tissue, and passing forwards to the intervertebral foramina.
d.—Anterior divided edge of the dura mater. *Vide* p. 372, No. 28.



S. G. Shattock del.

Danielsson & Co lith.

DESCRIPTION OF PLATE XV.

Dissection of the parts concerned in a lumbo-sacral spina bifida, showing *the typical anatomical disposition* in cases of meningo-myelocele. A portion of the sac-wall has been cut away to show the interior.

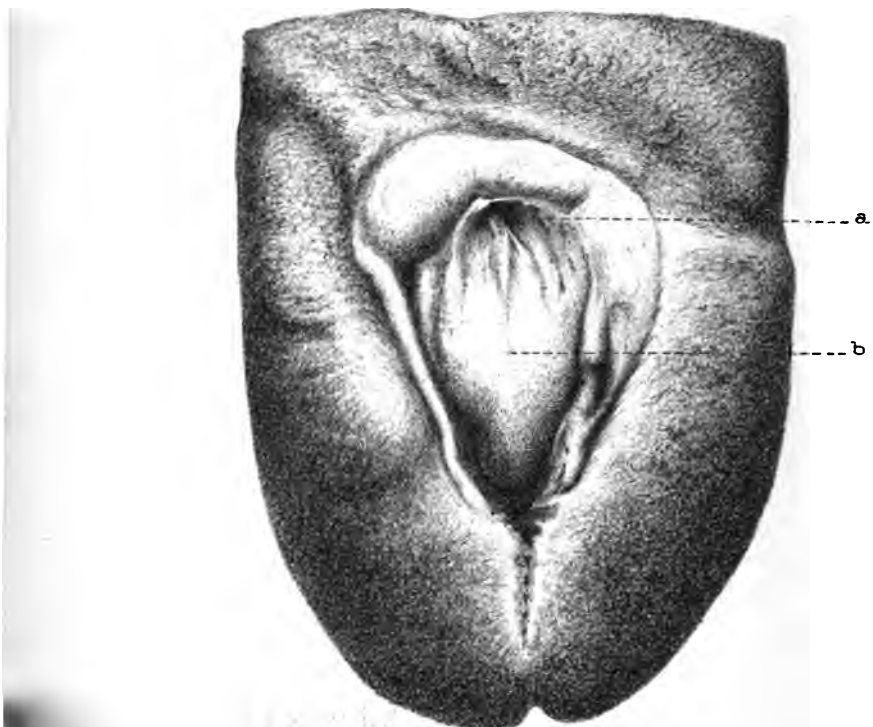
a.—Surface of cord, covered with arachnoid, exposed by the removal of portion of dura mater.

b.—Dura mater entering into the formation of the sac-wall.

c.—Arachnoid lining the sac.

d.—Lower portion of the spinal cord crossing the interior of sac; some of the nerve-roots pass forwards upon it, towards the intervertebral foramina. The other nerve-roots arise from the posterior wall of the sac in a vertical series and traverse the space horizontally.

e.—Falciform process continuous with the pia mater, separating the anterior and posterior roots of the nerves of the left side; there is a corresponding process on the right side. *Vide* p. 342-3.



S. G. Shattock del.

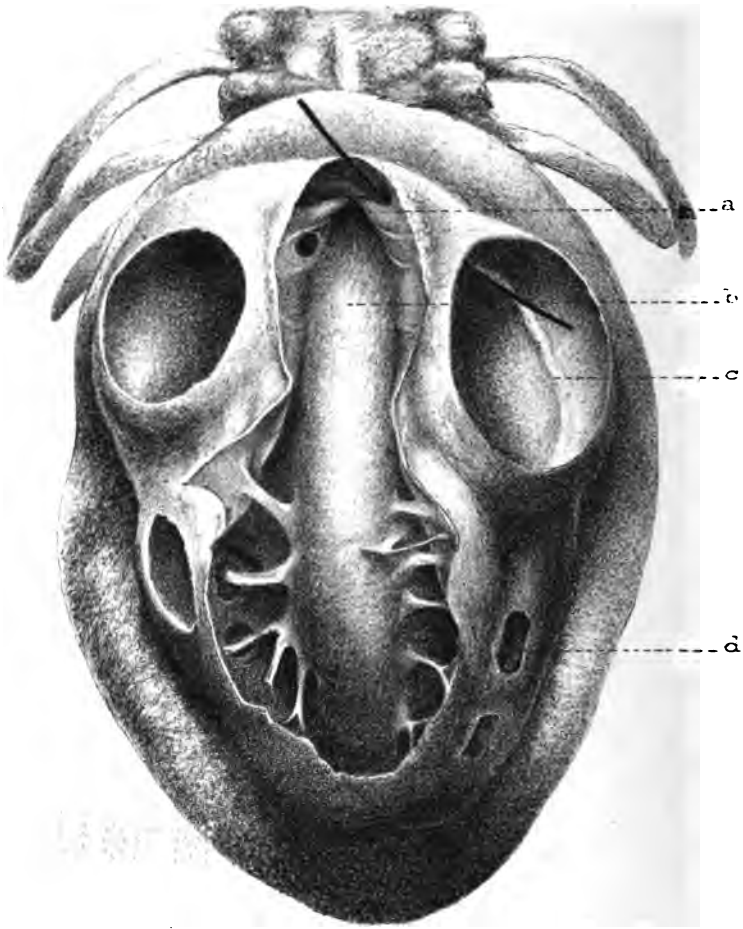
Danielsson & Co. lith.

DESCRIPTION OF PLATE XVI.

Posterior view of the lower part of the trunk of a fœtus at term affected with spina bifida.

a.—Foramen leading from the outer surface of the protrusion to the cental canal of the cord.

b.—Central or chief part of the protrusion; around this is an eminence caused by a series of secondary cavities. *Vide* p. 367, No. 13.



S. G. Shattock del.

Danielsson & Co lith.

DESCRIPTION OF PLATE XVII.

Posterior view of a large multilocular spina bifida involving the lowest dorsal and the lumbo-sacral regions. The sac-wall has been cut away in various situations to show the different divisions of the interior.

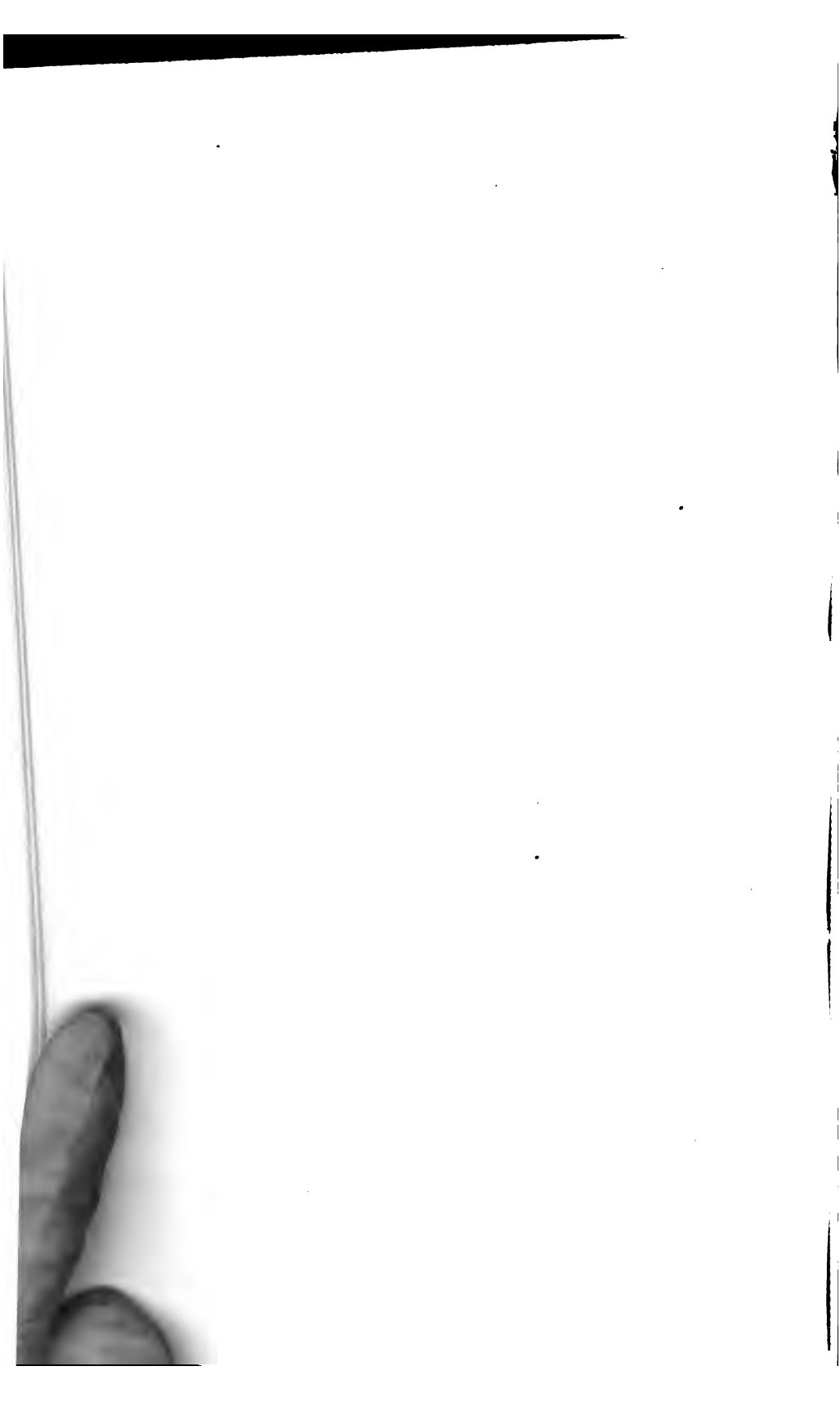
a.—The right half of the emerging portion of the spinal cord, which is divided by a process of bone, as it enters the sac. A bristle has been passed from above this into a lateral sac on the right side.

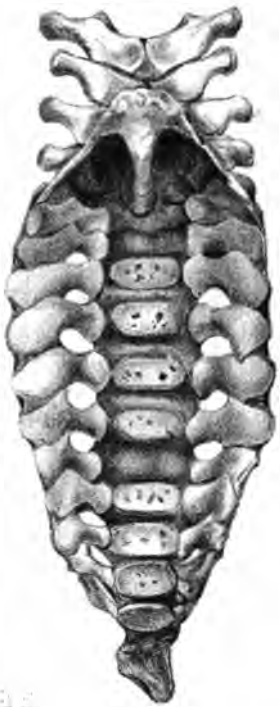
b.—Posterior surface of the bodies of the vertebræ; the lower part of the column generally is convex.

c.—The highest of the lateral sacs of the right side.

d.—An opening made into one of the lower sacs.

The nerve-roots arise from the sac-wall and pass forwards to the intervertebral foramina, which they enter in a normal manner. *Vide* p. 369, No. 20.





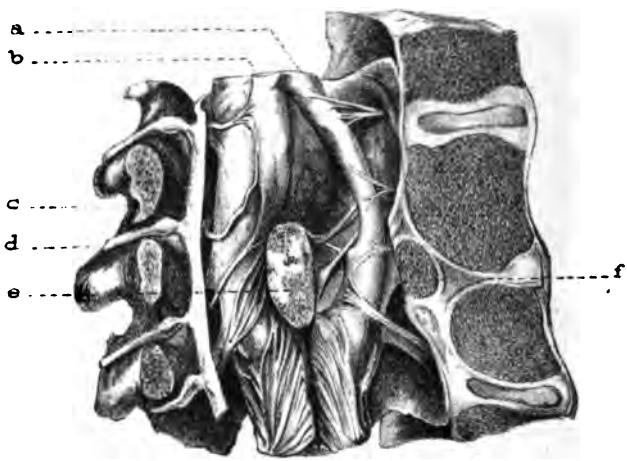
S.G.Shattock del.

Danielsson & Co lith.

DESCRIPTION OF PLATE XVIII.

Part of a spinal column viewed from behind. There is a long fissure in the vertebral canal extending from the tenth dorsal vertebra to the lower end of the column.

There is a slender process of bone crossing the vertebral canal at the highest part of the fissure; anteriorly this abuts against the posterior surface of the vertebral bodies, posteriorly it is expanded, and completes the arch of the tenth dorsal vertebra. *Vide* p. 369, No. 21.



S.G. Shattock del.

Danielsson & Co lith.

DESCRIPTION OF PLATE XIX.

The last two dorsal with the upper two lumbar vertebræ. The bodies of the vertebræ have been divided vertically, as well as the pedicles of the arches on its right side; the right halves of the bodies have been entirely removed, so as to expose the spinal cord; the cord has been more fully exposed by turning back the laminæ of the right side. The cord is perforated by a bony process which crosses the canal.

a.—The left division of the cord above the perforation, consisting in its upper part chiefly of the anterior median column; in its lower part it is almost of the same size as the right.

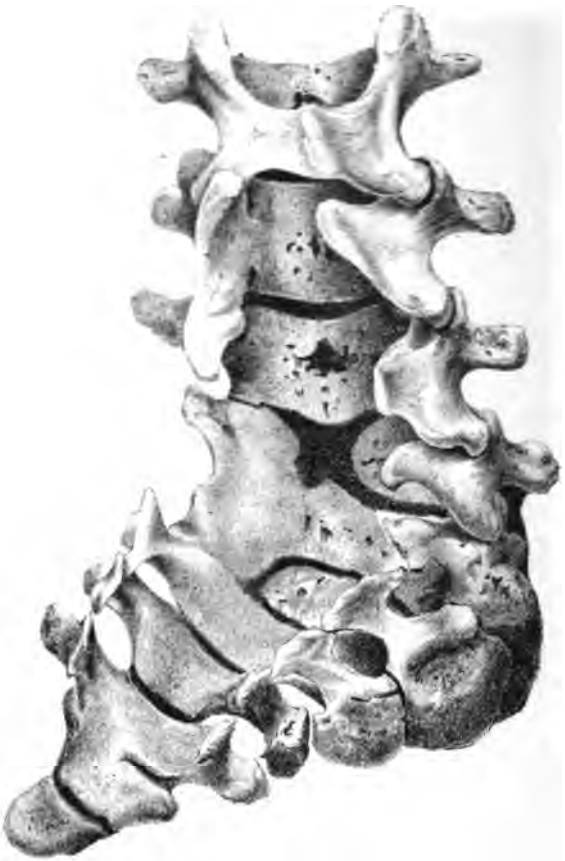
b.—The right division of the cord.

c.—Sheath of dura mater.

d.—The last dorsal nerve.

e.—Divided surface of the osseo-cartilaginous process (by which the cord is perforated) turned forwards.

f.—Anterior portion of the same process connected with the bodies of the vertebræ. *Vide* p. 370, No. 22.



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DESCRIPTION OF PLATE XX.

Lower part of the spinal column from a case of spina bifida viewed from behind. There is a fissure involving the arches of the fourth lumbar and succeeding vertebræ. The sacrum is sharply inclined to the left side. *Vide* pp. 371-2, No. 25.

INDEX.

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