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TRANSACTIONS

OF

THE CLINICAL SOCIETY.

VOL. XX.
TRANSACTIONS
OF
THE CLINICAL SOCIETY
OF
LONDON.

VOLUME THE TWENTIETH.

LONDON:
LONGMANS, GREEN, AND CO.
1887.
NOTICE.

The present Volume comprises the Proceedings of the Society during its Twentieth Session, October, 1886, to May, 1887.

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, 1887.
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1874 Charcot, J. M., M.D., Physician to the "Hôpital de la Salpêtrière."

1881 Esmarch, Friedrich, M.D., Professor of Surgery and Director of the Surgical Clinique in the University of Kiel; Surgeon to the University Hospital, Kiel, and Surgeon-General to the Prussian Army.

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1881 Mazzoni, Costanzo, Professor of Surgery at the Royal University of Rome, and Surgeon to the Hospital of San Giacomo at Rome.

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1881 Pasteur, L., Member of the Institute (Academy of Sciences) of Paris.

1874 Ricord, Philippe, M.D., Ex-Surgeon-in-Chief of the Hôpital du Midi, and late President of the Academy of Medicine, Paris.

1881 Verneuil, Aristide, Member of the Institute of Paris; Professor of Clinical Surgery at the Faculty of Medicine, Paris.

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The figures succeeding the word Trans. show the number of Papers contributed to the 'Transactions' by the Member to whose name they are annexed: C.S. refers to the specimens exhibited by card.

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1883 Adams, William Coode, M.B., 1, Elm Avenue, South Hampstead.

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Orig. Memb. Arnott, Henry. (C. 1871-5.) Trans. 3.

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1878 Ball, James Barry, M.D., 29, Belgrave Road, S.W.

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1882 Barker, Frederick Charles, M.D., Surgeon-Major, Bombay Medical Service, India [care of Arthur Barker, Esq., 87, Harley Street].

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, Wimpole Street, Cavendish Square, W.C. (C. 1880–82.) Trans. 9, C.S. 1.


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Orig. Memb.  Bryant, Thomas, Surgeon to, and Lecturer on Surgery at, Guy's Hospital; 65, Grosvenor Street, Grosvenor Square, W. (C. 1872, V.P. 1876-7, P. 1885-86.) Trans. 7.

Orig. Memb.  Buchanan, George, M.D., F.R.S., Medical Officer of the Local Government Board; 24, Nottingham Place, W. (C. 1877.)
1884  Buck, William Elgar, M.D., 5, Welford Road, Leicester.
1886  Bull, William C., M.B., 45, Curzon Street, May Fair, W.
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<tr>
<th>Year</th>
<th>Name</th>
<th>Address</th>
<th>Office Details</th>
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<td>1881</td>
<td>BURNET, ROBERT WILLIAM, M.D.</td>
<td>6, Upper Wimpole Street,</td>
<td>Elected 1881.</td>
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<td>1879</td>
<td>BURTON, WILLIAM EDWARD</td>
<td>24, Wimpole Street,</td>
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<td>1887</td>
<td>BUTLER-SMYTHE, ALBERT CHARLES</td>
<td>to the Grosvenor Hospital for</td>
<td>Senior Surgeon to the Grosvenor Hospital for Women and Children;</td>
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<tr>
<td></td>
<td></td>
<td>Women and Children;</td>
<td>35, Brook Street, Grosvenor Square, W.</td>
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<tr>
<td>1881</td>
<td>BUTLIN, HENRY TRENTHAM (C)</td>
<td>to, and Demonstrator of</td>
<td>Assistant Surgeon to, and Demonstrator of Practical Surgery and Diseases of</td>
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<tr>
<td></td>
<td></td>
<td>Practical Surgery and</td>
<td>the Larynx at, St. Bartholomew's Hospital; 82, Harley Street, Cavendish Square,</td>
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<td></td>
<td></td>
<td>Diseases of the Larynx at</td>
<td>W. (C. 1887.) Trans. 5.</td>
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<td>1871</td>
<td>BUTT, WILLIAM F.</td>
<td>48, Park Street, Park Lane,</td>
<td>Elected 1871.</td>
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<td>1884</td>
<td>BUXTON, DUDLEY WILMOT, M.D.,</td>
<td>W.</td>
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<td></td>
<td>B.S.</td>
<td></td>
<td>Administrator and Teacher of the Use of Anaesthetics in University College</td>
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<td>Hospital; Anaesthetist to the Hospital for Women, Soho Square, and to the</td>
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<td>London Dental Hospital; 82, Mortimer Street, Cavendish Square, W.</td>
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<td>Orig. Memb.</td>
<td>BUZZARD, THOMAS, M.D.</td>
<td></td>
<td>Physician to the National Hospital for the Paralysed and Epileptic; 56,</td>
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<td>1886</td>
<td>CAHILL, JOHN</td>
<td>12, Seville Street, Lowndes</td>
<td>Elected 1886.</td>
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<td>1883</td>
<td>CARTER, FREDERICK HEALES</td>
<td>Eaton Villa, Bellevue Road,</td>
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<td>1869</td>
<td>CARTER, ROBERT BRUDENELL</td>
<td>Ophthalmic Surgeon to, and</td>
<td>Ophthalmic Surgeon to, and Lecturer on Ophthalmology at, St. George's Hospital;</td>
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<tr>
<td></td>
<td></td>
<td>Lecturer on Ophthalmology at</td>
<td>Surgeon to the Royal South London Ophthalmic Hospital; 27, Queen Anne Street,</td>
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<td>St. George's Hospital;</td>
<td>Cavendish Square, W. (C. 1873-6, V.P. 1879-81.) Trans. 8.</td>
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<td>1885</td>
<td>CATON, RICHARD, M.D.</td>
<td>18, Croxteth Road, and 86,</td>
<td>Elected 1885.</td>
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<td></td>
<td></td>
<td>Rodney Street, Liverpool</td>
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<td>1868</td>
<td>CAVAFY, JOHN, M.D.</td>
<td>2, Upper Berkeley Street,</td>
<td>Elected 1868.</td>
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<td>Portman Square, W.</td>
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<td>Orig. Memb.</td>
<td>CAYLEY, WILLIAM, M.D.</td>
<td>Physician to, and Lecturer on</td>
<td>Physician to the Middlesex Hospital; Physician to the London Fever Hospital,</td>
</tr>
<tr>
<td></td>
<td></td>
<td>the Principles and Practice of</td>
<td>and to the North-Eastern Hospital for Children; 27, Wimpole Street, W. (C. 1874-5,</td>
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<td></td>
<td></td>
<td>Medicine at, the Middlesex</td>
<td>S. 1876-8, C. 1879-80, V.P. 1885-86.) Trans. 7, C.S. 1.</td>
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<td>Hospital; 2, Upper Berkeley</td>
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<td></td>
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<td>Street, Portman Square, W.</td>
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</table>
List of Members.

1885 Chalmers, John, M.D., 29, Keppel Street, Russell Square, W.C.

1884 Chapman, Paul M., M.D., Physician to the Hereford General Infirmary, 1, St. John Street, Hereford.

1885 Cheyne, W. Watson, M.B., C.M., Assistant Surgeon and Demonstrator of Surgery to King’s College Hospital; 59, Welbeck Street, Cavendish Square, W.

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


Orig. Memb. Church, William Selby, M.D., Physician to, and Lecturer on Clinical Medicine at, St. Bartholomew’s Hospital; 130, Harley Street, Cavendish Square, W. (C. 1874–6.)

1873 Churton, Thomas, M.D., Physician to the Leeds Infirmary, and Lecturer on Medicine in the Yorkshire College; 35, Park Square, Leeds. Trans. 2.

1882 Clapham, Edward, M.D., 29, Lingfield Road, Wimbledon.


Orig. Memb. Clark, Sir Andrew, Bart., M.D., LL.D., F.R.S., Physician to, and Emeritus Professor of Clinical Medicine at, the London Hospital; 16, Cavendish Square, W. (C. 1876–8, V.P. 1880–82, P. 1883–84.) Trans. 2.

1874 Clark, Andrew, Assistant Surgeon to, and Lecturer on Practical Surgery at, the Middlesex Hospital; 19, Cavendish Place, Cavendish Square, W.

1885 †Clarke, J. Michell, M.B., Assistant Physician to the Bristol General Hospital; 2, York Buildings, Clifton, Bristol.

1885 Clarke, William Bruce, M.B., Assistant Surgeon to, and Demonstrator of Anatomy at, St. Bartholomew’s Hospital; 46, Harley Street, Cavendish Square, W.

1877 †Clay, Robert Hogarth, M.D., 4, Windsor Villas, Plymouth.

List of Members.

Elected
1878 Collie, Alexander, M.D., Fever Hospital (Metropolitan Asylum District), The Grove, Homerton, E.
1882 Collier, Herbert, M.D., Marine Villa, Gorleston, Great Yarmouth, Norfolk.
1878 Collins, W. Maunsell, M.D., M.C., 10, Cadogan Place, S.W.
1882 Colquhoun, Daniel, M.D., Dunedin, New Zealand.
1872 Cooke, Thomas, Assistant Surgeon to the Westminster Hospital; 40, Brunswick Square, W.C.
1868 Cooper, Frank W., Leytonstone, Essex.
1880 Cottle, Wyndham, M.D., Senior Assistant Surgeon to the Hospital for Diseases of the Skin, Blackfriars; 3, Savile Row, W.

Orig. Memb. Cooper, John, Surgeon to the London Hospital and to the Royal London Ophthalmic Hospital; 80, Grosvenor Street, W. (C. 1874.)
1875 Coupland, Sidney, M.D. (C.), Physician to, and Lecturer on Practical Medicine at, the Middlesex Hospital; 14, Weymouth Street, Portland Place, W. (S. 1882–4, C. 1885–87.) Trans. 3.
1886 Cousins, John Ward, M.D., Riversdale, Kent Road, Southsea.
1881 Creighton, Charles, M.D., 11, New Cavendish Street, W.
1879 Cripps, William Harrison (C.), Assistant Surgeon to St. Bartholomew's Hospital; 2, Stratford Place, Oxford Street, W. (C. 1886–7.) Trans. 3.
1872 Critchett, Anderson, Ophthalmic Surgeon to St. Mary’s Hospital and to the Royal Free Hospital; 21, Harley Street, W.
1877 Crocker, Henry Radcliffe, M.D. Physician to the Skin Department, University College Hospital; and Physician to the East London Hospital for Children; 28, Welbeck Street, Cavendish Square, W. (C. 1884–5.) Trans. 14.

Orig. Memb. Croft, John, Surgeon to St. Thomas’s Hospital; 48, Brook Street, Grosvenor Square, W. (C. 1870–2, V.P. 1882–4.) Trans. 10.
1872 Dalby, Sir William Bartlett, M.B., Aural Surgeon to St. George’s Hospital; 18, Savile Row, W. (C. 1879–81.) Trans. 4.
List of Members.

Elected

1882 Dallaway, J. W. Dennis, 75, Park Street, Grosvenor Square, W.
1879 †Davy, Henry, M.D., 34, Southernhay, Exeter.
1868 Day, William Henry, M.D., Physician to the Samaritan Free Hospital for Women and Children; 10, Manchester Square, W. Trans. 5.
1872 De Castro, James Cato, M.B., Paris, France.
1879 †Dennis, Frederic S., M.D., 21, East 21st Street, New York, U.S.
1875 Dent, Clinton T., Assistant Surgeon to, and Lecturer on Practical Surgery at, St. George’s Hospital; 61, Brook Street, W. (C. 1884–6.) Trans. 2.

Orig. Memb. Dickinson, William Howship, M.D., Physician to, and Lecturer on Medicine at, St. George’s Hospital; Consulting Physician to the Hospital for Sick Children; Honorary Fellow of Caius College, Cambridge; 9, Chesterfield Street, Mayfair, W. (C. 1874–5.) Trans. 1.
1871 Diver, Ebenezer, M.D., Kenley, Caterham Valley, Surrey.

1874 Dowse, Thomas Stretch, M.D., 14, Welbeck Street, Cavendish Square, W. Trans. 5.
1868 Drage, Charles, M.D., Hatfield, Herts.
1879 Drewitt, F. G. Dawtrey, M.D. (C.), Physician to the West London Hospital, and to the Victoria Hospital for Children; 52, Brook Street, Grosvenor Square, W. (C. 1886–7.) Trans. 2.

Orig. Memb. Duckworth, Sir Dyce, M.D. (V.P.), Physician to, and Lecturer on Clinical Medicine at, St. Bartholomew’s Hospital; 11, Grafton Street, Bond Street, W. (C. 1875–7, V.P. 1887.) Trans. 14, C.S. 3.

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.
List of Members.

Elected
1884 Duke, Edgar, Locksley, Freshwater, Isle of Wight.
1869 Duke, Olliver Thomas, M.B., Surgeon, Bengal Army, India.

Orig. Memb. Durham, Arthur Edward, Surgeon to, and Lecturer on Surgery at, Guy's Hospital; 82, Brook Street, W. (C. 1867-9, V.P. 1884-5.) Trans. 5.

Orig. Memb. Edis, Arthur W., M.D., Obstetric Physician to, and Lecturer on Midwifery at, the Middlesex Hospital; 22, Wimpole Street, Cavendish Square, W. (C. 1884-6.) Trans. 1.

1884 Edmunds, Walter, M.C., 79, Lambeth Palace Road, S.E.
1882 Emond, Emile, M.D., Mont Dore, Auvergne, and 113, Boulevard Beaumarchais, Paris.

Orig. Memb. Eichsen, John E., LL.D., F.R.S., Surgeon Extraordinary to H.M. the Queen; Emeritus Professor of Surgery at University College, and Consulting Surgeon to University College Hospital; 6, Cavendish Place, Cavendish Square, W. (V.P. 1869-71.)

1868 Evans, Julian, M.B., Physician to the Victoria Hospital for Children; 123, Finborough Road, Redclyffe Square, S.W.
1877 Ewart, William, M.D., Assistant Physician to, and Lecturer on Physiology at, St. George's Hospital; 33, Curzon Street, Mayfair, W. (C. 1884-6.)

1868 Fairbank, Frederick Royston, M.D., 46, Hall Gate, Doncaster. Trans. 1.
1885 Fenn, Edward Liveing, M.D., 1, Portland Terrace, Richmond Green, Surrey.
1872 Fenwick, J. C. J., M.D., 25, North Road, Durham.
1878 Field, George P., Aural Surgeon to St. Mary's Hospital; 13, Wimpole Street, Cavendish 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. 1885-87.) Trans. 4.

1868 Fish, John Crockett, M.D., 92, Wimpole Street, Cavendish Square, W. (C. 1869-70.)
1885 Fitz-Patrick, Thomas, M.D., 30, Sussex Gardens, Hyde Park, W.
List of Members.

Elected

1878  *Fonmartin, Henry de, M.D., Parkhurst, Isle of Wight.

1881  Fowler, James Kingston, M.D. (C.), 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. (C. 1887.) Trans. 3, C.S. 3.

1886  Fox, R. Hingston, M.D., 43, Finsbury Circus, E.C.

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.

1868  Gant, Frederick James, Surgeon to the Royal Free Hospital; 16, Connaught Square, W. (C. 1877–9.) Trans. 3.

1887  Garrod, Archibald Edward, M.A., M.D., Casualty Physician to St. Bartholomew's Hospital and Physician to the Marylebone Dispensary; 9, Chandos Street, Cavendish Square, W.


1885  Gibbons, Robert Alexander, M.D., Physician to the Grosvenor Hospital for Women and Children; 29, Cadogan Place, S.W.


1882  Goddard, Eugene, M.D., 106, Highbury New Park, N.


1882  Goldie, Robert William, Medical Superintendent, Poplar and Stepney Sick Asylum; Devons Road, Bromley.

1878  Golding-Bird, C. H., M.B. (C.), Assistant Surgeon to, and Lecturer on Physiology at, Guy's Hospital; 13, St. Thomas's Street, Southwark, S.E. (C. 1887.) Trans. 10.
List of Members.

Elected

1875  GOODHART, JAMES FREDERIC, M.D., Physician to, and Curator of the Museum at, Guy's Hospital; 25, Weymouth Street, Portland Place, W. (C. 1880–2.) Trans. 11, 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., 26, Knightrider Street, E.C.

1877  GOULD, A. PEARCE, M.S. (C.), Assistant Surgeon to the Middlesex Hospital; 16, Queen Anne Street, Cavendish Square, W. (C. 1885–87.) Trans. 8, C.S. 2.

1871  GOVER, ROBERT M., M.B., 22, St. Mary's Road, Westbourne Park, W.

1875  GOWERS, WILLIAM RICHARD, M.D., F.R.S., 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 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.


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

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 in Ordinary to the Queen, and Physician in Ordinary to H.R.H. the Prince of Wales; Consulting Physician, Guy's Hospital; 74, Brook Street, W. (V.P. 1868–70, P. 1871–2.) Trans. 6.
**List of Members.**

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<td>1887</td>
<td>Habershon, Samuel Herbert, M.D.</td>
<td>2, Upper Wimpole Street, Cavendish Square, W.</td>
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<td>1882</td>
<td>Hadden, Walter Baugh, M.D. (C.), Assistant Physician to St. Thomas's Hospital, and to the Hospital for Sick Children; 21, Welbeck Street, Cavendish Square, W. (C. 1886–7.) Trans. 6, C.S. 1.</td>
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<td>1875</td>
<td>Hale, C. D. B., 8, Sussex Gardens, Hyde Park, W.</td>
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<td>1878</td>
<td>Hall, F. de Havilland, M.D. (C.), Assistant Physician to the Westminster Hospital; 47, Wimpole Street, Cavendish Square, W. (C. 1885–87.) Trans. 6.</td>
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<td>1885</td>
<td>Halliburton, William Dobinson, M.D., Assistant Professor of Physiology, University College, London; 25, Maitland Park Villas, Haverstock Hill, N.W.</td>
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<td>1886</td>
<td>†Handford, Henry, M.D., 8, Regent Street, Nottingham. Trans. 1.</td>
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<td>1886</td>
<td>Hardie, James, M.D., 6, St. Ann's Place, Manchester.</td>
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<td>1872</td>
<td>Harris, Henry, M.D., Trengweath, Redruth, Cornwall.</td>
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<td>1881</td>
<td>Harrison, Charles Edward, M.B., Grenadier Guards Hospital, Rochester Row, S.W.</td>
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<td>1886</td>
<td>Hawkins, Francis Henry, M.B., Physician to St. George's and St. James's Dispensary; 22, Henrietta Street, Cavendish Square, W.</td>
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<tr>
<td>Orig. Memb.</td>
<td>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. 1879–87.) Trans. 18.</td>
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</table>
List of Members.

Elected

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; 57, Harley Street, Cavendish Square, W.


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. Hicks, J. Braxton, M.D., F.R.S., F.L.S., Consulting Physician Accoucheur to Guy’s Hospital, and Physician-Acoucheur to St. Mary’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, C.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. 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.


1878  Hood, Donald William Charles, M.D., Senior Physician, North-West London Hospital; Physician to the West London Hospital; 43, Green Street, Park Lane, W. Trans. 1.
List of Members.

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. C.S. 1.

1884  Horsley, Victor, M.B., F.R.S., Assistant Surgeon, University College Hospital; Surgeon to the National Hospital for the Paralysed and Epileptic; Professor of Pathological Anatomy, University College, London; Superintendent of the Brown Institution, Wandsworth Road; 80, Park Street, Grosvenor Square, W. Trans. 1, C.S. 1.

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

1880  Howell, T. Mark, Junior Aural Surgeon to the London Hospital; Surgeon to the Hospital for Diseases of the Throat; 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; 59, Brook Street, Grosvenor Square, W. (C. 1881–3.) Trans. 3.


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.)


1879  Inkson, James, M.D., Brigade Surgeon, Army.

1883  Jackson, George Henry, Lansdowne House, Tottenham.

Orig. Memb. Jackson, J. Hughlings, M.D., F.R.S. (V.P.), Physician to the London Hospital; Physician to the National Hospital for the Paralysed and Epileptic; 3, Manchester Square, W. (C. 1872–3, V.P. 1887.) Trans. 2.
List of Members.

Elected

1877 Jacobson, Walter Hamilton Acland, M.B., M.S., Assistant Surgeon to Guy’s Hospital; 41, Finsbury Square, E.C.


1875 Jessett, Frederick Bowreman, Surgeon to the Royal General Dispensary; 16, Upper Wimpole Street, W.

Orig. Memb. Johnson, George, M.D., F.R.S., Consulting 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.

1887 Jones, Edward Ager, 413, Kingsland Road, E.


1872 Jones, Thomas Ridge, M.D., Physician to the Victoria Hospital for Children; 4, Chesham Place, Belgrave Square, S.W.

1876 Jordan, Furneaux, Consulting Surgeon to the Queen’s Hospital, Birmingham; Selly Hill, Birmingham. Trans. 1.

1886 Juler, Henry Edward, Assistant Surgeon to the Royal Westminster Ophthalmic Hospital; Junior Ophthalmic Surgeon to St. Mary’s Hospital; 77, Wimpole Street, Cavendish Square, W.

1878 Keeley, Charles Robert Bell, Senior Surgeon to the West London Hospital; Surgeon to the Surgical Aid Society; 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. 2, C.S. 1.
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<th>Year</th>
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<tr>
<td>1878</td>
<td>LACEY, THOMAS WARNER</td>
<td>196, Burrage Road, Plumstead, S.E.</td>
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<tr>
<td>1873</td>
<td>LACY, C. DE LACY</td>
<td>31, Grosvenor Street, W.</td>
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<tr>
<td>1883</td>
<td>LANE, WILLIAM ARBUTHNOT</td>
<td>14, St. Thomas's Street, Southwark.</td>
<td>Surgeon to the Hospital for Sick Children.  <em>Trans. 2, C.S. 2.</em></td>
</tr>
<tr>
<td></td>
<td>ORIG. MEMB. LANGTON, JOHN</td>
<td>2, Harley Street, W.</td>
<td>Surgeon to, and Lecturer on Anatomy at, St. Bartholomew's Hospital, and Surgeon to the City of London Truss Society.</td>
</tr>
<tr>
<td>1886</td>
<td>LANKESTER, H. H.</td>
<td>1, Elm Park Gardens, South Kensington, S.W.</td>
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<tr>
<td>1885</td>
<td>LARDER, HERBERT</td>
<td>Whitechapel Infirmary, E. C.S. 1.</td>
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<td></td>
<td>ORIG. MEMB. LAWSON, GEORGE</td>
<td>12, Harley Street, W. 1871-3, C. 1874-6, V.P. 1881-3</td>
<td>Surgeon Oculist to H.M. the Queen; Surgeon to the Middlesex Hospital, and to the Royal London Ophthalmic Hospital.</td>
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<tr>
<td>1877</td>
<td>LEDGARD, HENRY AMBROSE</td>
<td>41, Lowther Street, Carlisle. Trans. 4.</td>
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<td></td>
<td>ORIG. MEMB. LEE, HENRY</td>
<td>9, Savile Row, W. 1870-2. Trans. 7.</td>
<td>Consulting Surgeon to St. George's Hospital.</td>
</tr>
<tr>
<td>1877</td>
<td>LEES, DAVID B.</td>
<td>22, Weymouth Street, Portland Place, W.</td>
<td>Physician to, and Lecturer on Materia Medica at, St. Mary's Hospital, and Assistant Physician to the Hospital for Sick Children.</td>
</tr>
<tr>
<td>1879</td>
<td>LICHTENBERG, GEORGE</td>
<td>47, Finsbury Square, E.C.</td>
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<tr>
<td>1878</td>
<td>LISTER, SIR JOSEPH, BART.</td>
<td>12, Park Crescent, Regent's Park, W.</td>
<td>Professor of Clinical Surgery at King's College, and Surgeon to King's College Hospital.</td>
</tr>
<tr>
<td>1868</td>
<td>LITTLE, LOUIS STROMEYER</td>
<td>China.</td>
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<tr>
<td>1875</td>
<td>LIVING, EDWARD</td>
<td>52, Queen Anne Street, Cavendish Square, W.</td>
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<tr>
<td>1872</td>
<td>LIVING, ROBERT</td>
<td>11, Manchester Square, W.</td>
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</tr>
</tbody>
</table>
List of Members.

Elected
1885 Lockwood, Charles Barrett, Surgeon to the Great Northern Central Hospital, and Demonstrator of Anatomy at St. Bartholomew's Hospital; 19, Upper Berkeley Street, Portman Square, W.

1876 Longhurst, Arthur Edwin Temple, M.D., 22, Wilton Street, Grosvenor Place, S.W. Trans. 1.

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


1879 Lunn, John Reuben, Resident Medical Officer, New Marylebone Infirmary, Rackham Street; Ladbroke Grove Road, Notting Hill, W. Trans. 4, C.S. 7.

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., 6, Manchester Square.

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, and Professor of Ophthalmic Surgery in King's College, London; 5, Savile Row, W. Trans. 1.

1882 Mackenzie, Frederic Morell; 29, Hans Place, S.W.

Orig. Memb. Mackenzie, Sir Morell, M.D., Physician to the Hospital for Diseases of the Throat; 19, Harley Street, Cavendish Square, W. Trans. 4.


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.

1885 MacLaren, Roderick, M.D, Portland Square, Carlisle. Trans. 1.
List of Members.

Elected

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., Lecturer on Pathology at, and Assistant Physician to, St. Mary’s Hospital; 35, Westbourne Terrace, W. Trans. 1.

1881 Makins, George Henry, Assistant Surgeon to St. Thomas’s Hospital and to the Evelina Hospital for Children; 2, Queen Street, Mayfair, W. C.S. 2.


1875 Marshall, F. J., Resident Medical Officer, St. George’s Hospital, W.

1884 Maudsley, Henry Carr, M.D., Sydney, New South Wales.

1868 +May, Edward Hooper, M.D., High Cross, Tottenham, Middlesex, N.

1878 Meredith, William Appleton, M.B., C.M. (C.), Surgeon to the Samaritan Free Hospital for Women and Children; 6, Queen Anne Street, Cavendish Square, W. (C. 1887.) Trans. 2.

1873 Mickle, William Julius, M.D., Lecturer on Mental Diseases, University College, London; 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 University College Hospital, 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. 2.
<table>
<thead>
<tr>
<th>Year</th>
<th>Name</th>
<th>Position</th>
<th>Institution</th>
<th>Address</th>
<th>Notes</th>
</tr>
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<tr>
<td>1874</td>
<td>Morgan, John Hammond</td>
<td>Assistant Surgeon to</td>
<td>Charing Cross Hospital, and to the Hospital for Sick Children</td>
<td>63, Grosvenor Street, W.</td>
<td>(C. 1883–5.) Trans. 2, C.S. 3</td>
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<tr>
<td>1877</td>
<td>Morris, Henry, M.B.</td>
<td>Surgeon to, and Lecturer on Surgery at</td>
<td>Middlesex Hospital; 2, Mansfield Street, Portland Place, W.</td>
<td>(C. 1884–6.) Trans. 9.</td>
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<tr>
<td>1877</td>
<td>Morris, Malcolm Alex.</td>
<td>Surgeon to the Skin Department of, and Lecturer on Dermatology at</td>
<td>St. Mary's Hospital; 8, Harley Street, Cavendish Square, W.</td>
<td>Trans. 1.</td>
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<tr>
<td>1885</td>
<td>Mott, Frederick Walker, M.D., C.M.</td>
<td>Lecturer on Physiology, Charing Cross Hospital; Meadowlead, Gayton Road, Harrow.</td>
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<tr>
<td>1879</td>
<td>Moullin, Charles W. Mansell</td>
<td>Assistant Surgeon to the London Hospital; 69, Wimpole Street, Cavendish Square, W.</td>
<td>(C. 1877-9.) Trans. 1.</td>
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<tr>
<td>1875</td>
<td>Murphy, Shirley E.</td>
<td>Lecturer on Hygiene and Public Health</td>
<td>St. Mary's Hospital; 41, Queen Anne Street, Cavendish Square, W.</td>
<td>Trans. 2.</td>
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<td>1885</td>
<td>Murray, Alexander Dalton, M.B.</td>
<td></td>
<td>Rickmansworth, Herts.</td>
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<tr>
<td>1888</td>
<td>Murray, Hubert Montague, M.D.</td>
<td>Assistant Physician to, and Demonstrator of Pathology at, Charing Cross Hospital</td>
<td>27, Savile Row, W. Trans. 1.</td>
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<tr>
<td>1882</td>
<td>Myers, A. T., M.D.</td>
<td>Medical Registrar, St. George's Hospital</td>
<td>9, Lower Berkeley Street, Portman Square, W. (C. 1881–82.) Trans. 2.</td>
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<tr>
<td>1872</td>
<td>Myrtle, Andrew S., M.D.</td>
<td></td>
<td>8, Park Parade, Harrogate.</td>
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<tr>
<td>1874</td>
<td>Nankivell, Arthur Wolcot</td>
<td>Resident Surgeon, St. Bartholomew's Hospital, Chatham.</td>
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<tr>
<td>1875</td>
<td>Nettleship, Edward</td>
<td>Ophthalmic Surgeon to, and Lecturer on Ophthalmology at, St. Thomas's Hospital</td>
<td>5, Wimpole Street, Cavendish Square, W. (C. 1881–82.) Trans. 2.</td>
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</tbody>
</table>
List of Members.

Elected


1880 O'Connor, Bernard, M.D., Physician to the North London Hospital for Consumption; 17, St. James' Place, S.W. Trans. 1.


1883 Oliver, George, M.D., West End Park, Harrogate.

1887 Oliver, Thomas, M.D., 12, Elden Square, Newcastle-on-Tyne.


1877 Ord, William Miller, M.D., Physician to, and Lecturer on Medicine at, St. Thomas's Hospital; 37, Upper Brook Street, Grosvenor Square, W. (C. 1882–4.) Trans. 5.

1887 Ormerod, Joseph Arderne, M.D., Assistant Physician to the National Hospital for the Paralysed and Epileptic, Queen Square, and to the City of London Hospital for Diseases of the Chest, Victoria Park; 25, Upper Wimpole Street, Cavendish Square, W.

1884 Ormsby, Lambert Hепенстал, 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, and Lecturer on Forensic Medicine at, 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.
**List of Members.**

<table>
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<tr>
<th>Year</th>
<th>Name</th>
<th>Title/Position</th>
<th>Address</th>
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<tr>
<td>1884</td>
<td>Paget, Stephen</td>
<td>57, Wimpole Street, Cavendish Square, W.</td>
<td><em>Trans. 1. C.S. 1.</em></td>
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<td>1873</td>
<td>Parker, Robert William</td>
<td>(Hon. Secretary), Surgeon to the East London Hospital for Children; 8, Old Cavendish Street, W. <em>(C. 1882-4, S. 1887.)</em></td>
<td><em>Trans. 6, C.S. 7.</em></td>
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<tr>
<td>1881</td>
<td>Parker, Rushton, M.B.</td>
<td>Professor of Surgery in University College, Liverpool, and Assistant Surgeon Liverpool Royal Infirmary; 59, Rodney Street, Liverpool</td>
<td><em>Trans. 1, C.S. 1.</em></td>
</tr>
<tr>
<td>1881</td>
<td>Pasteur, William, M.D.</td>
<td>Medical Registrar to the Middlesex Hospital; Physician to the North-Eastern Hospital for Children; 19, Queen Street, May Fair, W.</td>
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<tr>
<td>1883</td>
<td>Paul, John Liston</td>
<td>M.D., 43, Queensborough Terrace, W.</td>
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<td><strong>Orig. Memb.</strong> Pavv, Frederick William, M.D., F.R.S., Physician to Guy’s Hospital; 35, Grosvenor Street, W. <em>(C. 1869-71, V.P. 1882-4.)</em></td>
<td><em>Trans. 3.</em></td>
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<td>1886</td>
<td>Payne, Joseph Frank</td>
<td>M.D., Physician to, and Lecturer on Pathological Anatomy at, St. Thomas’s Hospital; 78, Wimpole Street, Cavendish Square, W.</td>
<td></td>
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<tr>
<td>1887</td>
<td>Pearse, Thomas Frederick</td>
<td>M.D., 6, Manchester Square, W.</td>
<td></td>
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<td>1879</td>
<td>Peel, Robert</td>
<td>130, Collins Street East, Melbourne, Victoria</td>
<td></td>
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<tr>
<td>1886</td>
<td>Penny, William John</td>
<td>42, Caledonia Place, Clifton</td>
<td></td>
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<td>1887</td>
<td>Penrose, Francis George</td>
<td>M.D., 50, Torrington Square, W.</td>
<td></td>
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<tr>
<td>1882</td>
<td>Pepper, Augustus Joseph</td>
<td>M.S., M.B., Surgeon to St. Mary’s Hospital; 122, Gower Street, W.C.</td>
<td><em>Trans. 1.</em></td>
</tr>
<tr>
<td>1874</td>
<td>Phillips, Charles Douglas</td>
<td>F., M.D., 10, Henrietta Street, Cavendish Square, W.</td>
<td></td>
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<tr>
<td>1884</td>
<td>Phillips, Sidney Philip</td>
<td>M.D., Physician to St. Mary’s Hospital; Assistant Physician to the London Fever Hospital; 21, Upper Berkeley Street, Portman Square, W.</td>
<td><em>Trans. 1.</em></td>
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</tbody>
</table>
Elected


1885 Pitt, George Newton, M.D., Assistant Physician to, and Pathologist at, Guy's Hospital; 9, St. Thomas's Street, Southwark, S.E.

1883 Pitts, Bernard, M.A., M.C., Assistant Surgeon, St. Thomas's Hospital; 31, Harley Street, W. Trans. 3.

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, Assistant Surgeon and Surgical Registrar to University College Hospital; Surgeon to the North-Eastern Hospital for Children; 24, Harley Street, Cavendish Square, W. Trans. 1.

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 Physician to University College Hospital; 30, Wimpole Street, W. (C. 1879–81.) Trans. 3.

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.

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, C.S. 1.

1884 Pye-Smith, Philip Henry, M.D., F.R.S., Physician to, and Lecturer on Medicine at, Guy's Hospital; Member of the Senate of the University of London; 54, Harley Street, Cavendish Square, W.
List of Members.

Elected

Orig. Memb. Quain, Richard, M.D., F.R.S., Consulting Physician to the Hospital for Consumption and Diseases of the Chest; Member of the Senate of the University of London; 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., 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, S.W.

1874 Ree, Frederick G., Lifford Lodge, Outram Road, Addiscombe, Surrey.

Orig. Memb. Rees, George Owen, M.D., F.R.S., Physician Extraordinary to H.M. the Queen; Consulting Physician to Guy's Hospital. (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, 55, New Bond Street, W.


1877 Rivington, Walter, M.S., M.B. (C.), Surgeon to, and Lecturer on Surgery at, the London Hospital; 22, Finsbury Square, E.C. (C. 1886–7.) Trans. 3.

1873 Roberts, David Lloyd, M.D., Physician to St. Mary's Hospital, Manchester; 11, St. John Street, Manchester,
List of Members.

**Elected**

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; 102, Harley Street, Cavendish Square, W.

1885  Robinson, Arthur Henry, M.D., Mile End Infirmary, Bancroft Road, E.  C.S. 3.

1885  Robson, A. W. Mayo, Surgeon to the Leeds General Infirmary, Hillary Place, Leeds.  Trans. 4.

1875  Rogers, William Richard, M.D., 56, Berners Street, Oxford Street, W.

1877  Roth, Bernard, 48, Wimpole Street, W., and Rossmore, Preston Road, Brighton.  Trans. 1, C.S. 3.

**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.

1887  Rutherfoord, H. T., M.B., 46, Queen Anne Street, Cavendish Square, W.

1885  Ryle, Reginald John, M.D., Green View, Hadley Green, Barnet.

1882  Sainsbury, Harrington, M.D., 63, Welbeck Street, W.

1888  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.

1873  Savage, George Henry, M.D., Medical Superintendent and Resident Physician to the Bethlem Royal Hospital, St. George's Road, S.E.  (C. 1882–3.)

1886  Savill, Thomas Dixon, M.D., Paddington Infirmary, Harrow Road, W.  C.S. 2.

1885  Sawtell, Tom Henry, M.B., 14, Stapleton Hall Road, Stroud Green, N.  Trans. 1.

1886  Scott, Alfred, 15, German Place, Brighton.

1877  Seaton, Edward, M.D., 35, George Street, Hanover Square, W.  Trans. 1.
List of Members.

Elected


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.


1886 Silcock, Arthur Quarry, M.D., M.S., Surgeon in charge of Out-Patients to St. Mary's Hospital, and Assistant Surgeon to the Royal London Ophthalmic Hospital; 101, Harley Street, Cavendish Square, W.

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 Orthopaedic Department, of the Farringdon Dispensary; 24, Queen Anne Street, Cavendish Square, W.  Trans. 1.

1868 Smith, Heywood, M.D., 18, Harley Street, Cavendish Square, W.

1868 Smith, Protheroe, M.D., Consulting 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.

List of Members.

Elected

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

1873  Smith, William Johnson, Surgeon to the Seamen's Hospital, Greenwich, S.E.

1872  Smith, William Wilberforce, M.D., 14, Stratford Place, Oxford Street, W.

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.D., 14, Carleton Road, Tufnell Park.

1882  Spooner, Frederick Henry, M.D., Maitland Lodge, Maitland Place, 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, Aldershot.

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, Sir 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, Assistant Surgeon to the Westminster Hospital, and to the Cancer Hospital, Brompton; 62, Welbeck Street, Cavendish Square, W. C.S. 3.

1878  Strugnell, Frederick William, 45, Highgate Road, Highgate, N. C.S. 1.
List of Members.

Elected

1878  †Sturte, 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.

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.

1887  Sutton, John Bland, Assistant Surgeon to the Middlesex Hospital; 22, Gordon Street, Gordon Square, W.

1882  Symonds, Charters James, M.S., Assistant Surgeon to Guy's Hospital; 26, Weymouth Street, Portland Place, W. Trans. 7, C.S. 1.


1885  Tait, Edward Sabine, M.B., 54, Highbury Park, N.

1885  Tait, Henry Brewer, 28, Hornsey Rise, N.

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

1886  Tay, Waren, Surgeon to the London Hospital and to the North-Eastern Hospital for Children, and the Hospital for Skin Diseases, Blackfriars; 4, Finsbury Square, E.C.

1878  Tayler, Francis Thomas, B.A., M.B., 224, Lewisham High Road, S.E.

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

1882  Taylor, Seymour, M.D., Physician to the North London Hospital for Consumption; 16, Seymour Street, Portman Square, W. Trans. 1.

1885  †Taylor, W. C. Everley, 34, Queen Street, Scarborough.

1886  Teale, Thomas Pridgin, M.B., 38, Cookridge Street, Leeds.


1882  Thin, George, M.D., 22, Queen Anne Street, Cavendish Square, W. Trans. 1.
List of Members.

Elected

1886 Thompson, Charles Herbert, M.D., Metropolitan Free Hospital, Kingsland Road, N.

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. (V.P.), Surgeon-Extra-ordinary to H.M. the King of the Belgians; Emeritus Professor of Clinical Surgery in University College; 35, Wimpole Street, W. (C. 1867-8, V.P. 1886-7.) Trans. 2.

1887 Thornton, John Knowsley, M.B., C.M., Surgeon to the Samaritan Free Hospital for Women and Children; 22, Portman Street, Portman Square, W.

1872 Thornton, William Pugin, 35, St. George's Road, Canterbury. Trans. 5.

1876 Thripp, James Godfrey, Fern House, Heston, Hounslow.

1885 Thursfield, Thomas William, M.D., 26, The Parade, Leamington.

1887 Totsuka, Kankai, 65, Lambeth Palace Road, S.E.

1874 Travers, William, M.D., 2, Phillimore Gardens, Kensington, W.

1884 Treves, Frederick, Surgeon to, and Lecturer on Anatomy at, the London Hospital; 6, Wimpole Street, Cavendish Square, W. Trans. 3.


1882 Turner, George Robertson, Visiting Surgeon, Seamen's Hospital, Greenwich; Demonstrator of Anatomy and 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 Ophthalmic Surgeon to University College Hospital; Surgeon to the Royal London Ophthalmic Hospital; 100, Harley Street, Cavendish Square, W.


1881 Uhthoff, John Caldwell, M.D., 46, Western Road, Hove, Brighton.

VOL. XX.
List of Members.

Elected

1868  Venning, Edgcome, 30, Cadogan Place, S.W. (C. 1876–8.) Trans. 2.

1886  Wade, Charles H., Scotleigh, Chudleigh, Devon.

1868  Wagstaffe, William Warwick, Purleigh, St. John’s Hill, Sevenoaks. (C. 1878.)

1886  *Wailléwright, Benjamin, M.B., C.M., 6, Harley Street, Cavendish Square, W. Trans. 1, C.S. 1.

1885  Wakley, Thomas, Jun., 96, Redcliffe Gardens, S.W.

1885  Walker, Charles Rotherham, M.D., 7, Grove Road, Leytonstone, E.

1875  Walsham, William J., Assistant Surgeon to, and Demonstrator of Practical and Orthopaedic Surgery at, St. Bartholomew’s Hospital; Surgeon to the Metropolitan Free Hospital; 27, Weymouth Street, Portland Place, W. (C. 1882–4.) Trans. 4. C.S. 1.

1876  Waters, John H., M.D., 101, Jermyn Street, St. James’s, S.W.

1886  Watkins, Edwin T., M.D., 61, Guilford Street, W.C. (C. 1881–83.)


1879  de Watteville, Armand, M.A., M.D., B.Sc., Physician in Charge of the Electro-Therapeutical Department, St Mary’s Hospital; 30, Welbeck Street, W.


1876  Weir, Archibald, M.D., St. Mungho’s, Great Malvern.

1868  Wells, Sir Thomas Spencer, Bart., Surgeon in Ordinary to H.M.’s Household; Consulting Surgeon to the Samaritan Free Hospital; 3, Upper Grosvenor Street, W. (C. 1873.)

1885  West, Charles, M.D., Corresponding Member of the Academy of Medicine of Paris; 55, Harley Street, Cavendish Square, W.
List of Members.

**Elected**

1882 West, Samuel, M.D. (C.), Assistant Physician to St. Bartholomew's Hospital; Physician to the City of London Hospital for Diseases of the Chest, Victoria Park, and to the Royal Free Hospital; 15, Wimpole Street, Cavendish Square, W. (C. 1887.) *Trans.* 8, *C.S.* 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.* 11.

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

1882 White, Edwin Francis, 7, Dealtry Road, Putney.


1883 White, William Henry, M.D., Assistant Physician to the Royal Hospital for Diseases of the Chest; 43, Weymouth Street, W.

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. (V.P.), Member of the Senate of the University of London; Consulting Physician to Guy's Hospital; 72, Grosvenor Street, W. (C. 1871–2. V.P. 1886–7.) *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.

List of Members.

Elected

**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., Professor of Midwifery, University College, London; Obstetric Physician to University College Hospital; 11, Queen Anne Street, Cavendish Square, W. (C. 1885–86.)

1870 Williams, William Rhys, M.D., Commissioner in Lunacy; 13, Gloucester Street, Warwick Square, S.W.

1876 Williamson, James Mann, M.D.; Ventnor, Isle of Wight.

**Orig. Memb.** Willis, Francis, M.D., The Spa, Braceborough, Stamford.

1886 Wilson, Albert, M.D., Leytonstone.

1880 Wood, John, F.R.S. (C.), Professor of Clinical Surgery in King's College, London, and Senior Surgeon to King's College Hospital; 61, Wimpole Street, Cavendish Square, W. (C. 1886–7.)

1883 Woodcock, John Rostron, 263, 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.
LIST OF MEMBERS

ARRANGED ACCORDING TO DATE OF ELECTION.

ORIGINAL MEMBERS.

Sir Henry Acland, M.D., F.R.S.
James Andrew, M.D.
Henry Arnott.
W. Morrant Baker.
Richard Barwell.
Henry Charlton Bastian, M.D., F.R.S.
John Syer Bristowe, M.D., F.R.S.
William Henry Broadbent, M.D.
Bernard Edward Brodhurst.
Thomas Bryant.
George Buchanan, M.D., F.R.S.
Thomas Buzzard, M.D.
William Cayley, M.D.
William Selby Church, M.D.
Edward Clapton, M.D.
Sir Andrew Clark, Bart., M.D., F.R.S.
John Couper.
John Croft.
William Howship Dickinson, M.D.
John Langdon Down, M.D.
Sir Dyce Duckworth, M.D.
Alfred B. Duffin, M.D.
Arthur Edward Durham.
Arthur W. Edis, M.D.
John Eric Erichsen, F.R.S.
E. Headlam Greenhow, M.D., F.R.S.
Sir Wm. Withney Gull, Bart., M.D., F.R.S.
Samuel Osborne Habershon, M.D.
John Harley, M.D.
Ernest Hart.
Christopher Heath.
Sir Prescott Gardner Hewett, Bt., F.R.S.
Graily Hewitt, M.D.
J. Braxton Hicks, M.D., F.R.S.
Timothy Holmes.
Barnard Wight Holt.
Carsten Holthouse.

John Whitaker Hulke, F.R.S.
George Murray Humphry, M.D., F.R.S.
Jonathan Hutchinson, F.R.S.
J. Hughlings Jackson, M.D., F.R.S.
Sir William Jenner, Bart., M.D., F.R.S.
George Johnson, M.D., F.R.S.
Sydney Jones.
Charles Kelly, M.D.
John Langton.
George Lawson.
Henry Lee.
Sir Morell Mackenzie, M.D.
William Mareet, M.D., F.R.S.
Arthur Treherne Norton.
Thomas William Nunn.
John William Ogle, M.D.
Frederick William Payy, M.D., F.R.S.
Thomas Pickering Pick.
Richard Douglas Powell, M.D.
Richard Quain, M.D., F.R.S.
J. Spence Ramskill, M.D.
George Owen Rees, M.D., F.R.S.
John Russell Reynolds, M.D., F.R.S.
Sydney Ringer, M.D., F.R.S.
James Rouse.
John Burdon Sanderson, M.D., F.R.S.
Septimus William Sibley.
Thomas Smith.
Reginald Southey, M.D.
Henry Gawen Sutton, M.B.
William F. Teevan.
Edward Symes Thompson, M.D.
Sir Henry Thompson.
William Spencer Watson.
Hermann Weber, M.D.
Samuel Wilks, M.D., F.R.S.
Alfred Willett.
Charles Theodore Williams, M.D.
Francis Willis, M.D.
List of Members arranged according to Date of Election.

1868
William Cholmeley, M.D.
Constantine Holman, M.D.
Thomas Tillyer Whipham, M.B.
Christian G. H. Baümler, M.D.
John Cavafy, M.D.
Frederick James Gant.
James Grey Glover.
T. Henry Green, M.D.
Howard Marsh.
Arthur Bowen Richards Myers.
Charles Prentis.
Adolphus A. Rasch, M.D.
Hugh James Sanderson, M.D.
Edgcombe Venning.
Sir Thomas Spencer Wells, Bart.
John Ford Anderson, M.D.
George Granville Bantock, M.D.
William H. Brace, M.D.
George Charles Bright, M.B.
Frank W. Cooper.
Julian Evans, M.B.
Edward Hooper May, M.D.
Henri Gueneau de Mussy, M.D.
William Warwick Wagstaffe.
Edwin T. Watkins, M.D.
William Ogle, M.D.
Protheroe Smith, M.D.
James Edward Pollock, M.D.
Franz Oppert, M.D.
William Henry Holman, M.B.
William V. Snow, M.D.
Charles Drage, M.D.
Heywood Smith, M.D.
John Tatham, M.D.
Frederick Royston Fairbank, M.D.
Henry A. Reeves.
Michael W. Rice, M.D.
William Henry Day, M.D.
John Meaburn Bright, M.D.
Berkeley Hill.
Louis Stromeyer Little.
John Crockett Fish, M.D.

1869
Robert Brudenell Carter.
Leonard William Sedgwick, M.D.
J. Warrington Haward.
Henry Frederick Augustus Goodridge, M.D.
Olliver Thomas Duke, M.B.

1870
William Rhys Williams, M.D.
Thos. Clifford Allbutt, M.D., F.R.S.

1871
William Althaus, M.D.
Robert M. Gover, M.B.
Sir William Mac Cormac.
Alfred Playne, M.B.

1871
William F. Butt.
George Wight, M.B.
Ebenezer Diver, M.D.
George Vivian Poore, M.D.
William Edward Stewart.

1872
Thomas Cooke.
J. Burney Yeo, M.D.
James Cato De Castro, M.B.
Henry Harris, M.D.
William Pugin Thornton.
Robert Liveing, M.D.
Anderson Critchett.
J. C. J. Fenwick, M.D.
Andrew J. Myrtle, M.D.
Sir William Bartlett Dalby.
Thomas Ridge Jones, M.D.
George Slight, M.D.
Henry Sutherland, M.D.
Thomas Stretch Dowse, M.D.
William Wilberforce Smith, M.D.
Gifford Ransford, M.D.

1873
William Julius Mickle, M.D.
Robert William Parker.
David Lloyd Roberts, M.D.
George Henry Savage, M.D.
Heinrich Port, M.D.
Edwin Chisholm, M.D.
Thomas Churton, M.D.
C. de Lacy Lacey, M.B.
William Johnson Smith.
William Hope, M.D.

1874
John Hammond Morgan.
Edward R. Rowland.
Claudius Galen Wheelhouse.
Charles Douglas F. Phillips, M.D.
W. M. Whistler, M.D.
Edward C. Stirling, M.D.
William Henry Bennett.
Frederick G. Ree.
William Travers, M.D.
William Brown Holderness.
Andrew Clark.
Arthur Wolcot Nankivell.

1875
Thomas Barlow, M.D.
Marcus Beck.
Sidney Coupland, M.D.
Edward Bellamy.
Clinton T. Dent.
C. D. B. Hale.
Frederick Bowrman Jessett.
Edward Liveing, M.D.
Edward Nettleship.
William J. Walsham.
<table>
<thead>
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<th>Date</th>
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</table>
| 1875 | Rickman John Godlee, M.B.  
William Richard Rogers, M.D.  
Arthur Paul Sherwood.  
T. Gilbart Smith, M.D.  
James Frederic Goodhart, M.D.  
William Richard Gowers, M.D., F.R.S.  
William Smith Greenfield, M.D.  
Charles Macnamara.  
Shirley F. Murphy.  
Herbert W. Page.  
Frederick Taylor, M.D.  
Arthur E. J. Barker.  
Horatio Percy Symonds.  
James Godfrey Thrupp.  
A. Balmainno Squire, M.B.  
Archibald Weir, M.D.  
David White Finlay, M.D.  
Henry Greenway Howse.  
John H. Waters, M.D.  
Furneaux Jordan.  
R. Clement Lucas, M.B.  
James Mann Williamson, M.D.  
George Buckston Browne.  
Arthur Edwin Temple Longhurst, M.D. |
| 1877 | Robert Hogarth Clay, M.D.  
A. Pearce Gould.  
Edward Milner.  
Henry Radcliffe Crocker, M.D.  
David B. Lees, M.D.  
Walter Hamilton Acland Jacob-son, M.S.  
Isambard Owen, M.D.  
William Ewart, M.D.  
Henry Morris, M.B.  
William Miller Ord, M.D.  
Walter Rivington, M.B.  
Henry Rayner, M.D.  
Edward Seaton, M.D.  
Henry Ambrose Lediard, M.D.  
Bernard Roth.  
William A. Skinner.  
John Tweedy.  
Henry Hugh Clutton.  
Malcolm Alex. Morris.  
Alexander Collie, M.D.  
George P. Field.  
Thomas Warner Lacy.  
Thomas Colcott Fox, M.B.  
Felix Semon, M.D.  
Henry de Fonmartin, M.D.  
C. H. Golding-Bird, M.B.  
Donald Wm. Charles Hood, M.D. |
| 1878 | Sir Joseph Lister, Bart., F.R.S.  
Francis Thomas Tayler, M.B.  
F. de Havilland Hall, M.D.  
Storer Bennett.  
Walter B. Houghton, M.D.  
Sir William Stokes, M.D.  
William Allen Sturge, M.D.  
William Joseph Tyson, M.D.  
W. Maunsell Collins, M.D.  
James Barry Ball, M.D.  
William Johnston, M.D.  
Charles Robert Bell Keetley.  
William Appleton Meredith, M.B.  
Frederick William Strugnell.  
William Adams.  
William Edward Burton.  
James Magill, M.D.  
Wm. John Vereker Bindon, M.D.  
Edward Markham Skerritt, M.D.  
Henry Wilcox, M.B.  
James Inkson, M.D.  
John Abercrombie, M.D.  
F. G. Dawtry Drewitt, M.D.  
Stephen Mackenzie, M.D.  
William Harrison Cripps.  
Francis Patrick Staples.  
Geo. Courteney Henderson, M.D.  
Thomas John Maelagan, M.D.  
James Edward Adams.  
Henry Davy.  
Thos. Walter Harropp Garstang.  
George Lichtenberg, M.D.  
Charles W. Mansell Moullin.  
John Reuben Lunn.  
Armand de Watteville, M.D.  
George P. M. Woodward, M.D.  
J. Neville Davies-Colley, M.B.  
Robert Peel.  
Frederic S. Dennis, M.D. |
| 1879 | John Wood, F.R.S.  
T. Mark Hovell.  
Wyndham Cottle, M.D.  
B. Ball, M.D.  
Henry Francis Baker.  
Bernard O'Connor, M.D.  
Charles Edward Beevor, M.D.  
George Henry Makins.  
Robert William Burnet, M.D.  
James Kingston Fowler, M.D.  
Charles Edward Harrison, M.B.  
Malcolm Macdonald McHardy.  
Hugh Alex. Gordon, M.D.  
Rushton Parker.  
John Williams, M.D. |
List of Members arranged according to Date of Election.

1881 Montagu Lubbock, M.D.
James Black.
Charles Creighton, M.D.
William Pasteur, M.D.
Henry Fraser Stokes.
John Caldwell Uhthoff, M.D.
Henry Treatham Butlin.
H. A. Powell.

1882 George Robertson Turner.
E. Noble Smith.
Robert William Goldie.
Walter Bangh Haden, M.D.
Frederick Charles Barker, M.D.
William Henry Kesteven.
Frederic Morell Mackenzie.
A. T. Myers, M.D.
Daniel Colquhoun, M.D.
Seymour Taylor, M.D.
Francis Charlewood Turner, M.D.
Philip Henry Bindley, M.B.
Edward George Whittle, M.D.
D. H. Goodsell.
Frederick Henry Spooner, M.D.
J. W. Dennis Dallaway.
Frederick Haycraft Berry, M.D.
Herbert Collier, M.D.
Samuel West, M.D.
Emile Emond, M.D.
Eugene Goddard, M.D.
Charters James Symonds.
Angel Money, M.D.
Alfred G. Bateman, M.B.
C. F. Coxwell, M.B.
George Allan Hayon, M.D.
Augustus Joseph Pepper, M.B.
Edward Clapham, M.D.
Harrington Sainsbury, M.D.
George Thin, M.D.
Edwin Francis White.
Charles Gross.
Anthony A. Bowlby.
James Anderson, M.D.
Cecil Yates Biss, M.D.
Percy Kidd, M.D.
William Henry White, M.D.
George Oliver, M.D.
Hubert Montague Murray, M.D.
Robert Fitzroy Benham.
William Henry Allchin, M.B.
John Mitchell Bruce, M.D.
William Arbuthnot Lane, M.S.
Bernard Pitts.
Winckworth Tonge Smith, M.D.
William Hale White, M.D.

1883 William Coode Adams, M.B.
William Anderson.
Robert Leamon Bowles, M.D.
James Dixon Bradshaw, M.D.
George Henry Jackson.
George Hunt Orton, M.B.
John Liston Paul, M.D.
Thomas Laurence Read.
Frederick Thomas Roberts, M.D.
Charles Alfred Ballance, M.B.
Frederick Heales Carter.
John Hopkins.
John Rostron Woodcock.
Alexander Wm. Macfarlane, M.D.
Edward Cuthbert Ring

1884 Frederick Willcocks, M.D.
R. Percy Smith, M.D.
Edgar Duke.
John Mackern, M.B.
Paul M. Chapman, M.D.
Wilmot Parker Herringham, M.B.
Philip Henry Pye-Smith, M.D., F.R.S.
Charles Stonham.
Dudley Wilmot Buxton, M.D.
Edwin Worts.
Seymour J. Sharkey, M.B.
Frederick Treves.
William Elgar Buck, M.D.
John James Pringle, M.B.
Frederick Lucas Benham, M.D.
Walter Edmunds, M.D.
Arthur Fergusson McGill.
Stephen Paget.
Lambert Hепенстал Ormsby, M.D.
John Poland.
Edwin Leonard Adeney, M.D.
Victor Horsley, F.R.S.
Henry Carr Maudsley, M.D.
Bilton Pollard.

1885 Frederick Spicer, M.B.
Herbert Larder.
A. Hughes Bennett.
James Berry.
Sydney H. Henty.
Frederick Walker Mott, M.D.
George Newton Pitt, M.D.
John Chalmers, M.D.
W. C. Everley Taylor.
Sidney Philip Phillips, M.D.
A. W. Mayo Robson.
Thomas Wakley, jun.
Herbert William Allingham.
Thomas William Thursfield, M.D.
List of Members arranged according to Date of Election.

1885
Alexander Dalton Murray, M.B.
Robert Maguire, M.D.
Robert Alexander Gibbons, M.D.
Thomas Fitz-Patrick, M.D.
Tom Henry Sawtell, M.B.
Wm. Dobinson Halliburton, M.D.
Henry Brewer Tait.
Charles Rotherham Walker, M.D.
Richard Caton, M.D.
Arthur Henry Robinson, M.D.
Edward Sabine Tait, M.B.
William Bruce Clarke.
Charles Barrett Lockwood.
Charles West, M.D.
Reginald J. Ryle, M.D.
J. Michell Clarke, M.B.
Henry George Armstrong.
Roderick Maclaren, M.D.
W. Watson Cheyne.
Edward Liveing Fenn, M.D.

1886
Francis Henry Hawkins, M.B.
R. Hingston Fox, M.D.
Henry Edward Juler.
John Ward Cousins, M.D.
Joseph Frank Payne, M.D.
T. Prudgin Teale.
H. H. Lankester.
Arthur T. Davies, M.B.
William C. Bull, M.B.
Charles Herbert Thompson, M.D.
Arthur Quarry Silkock.
Henry Handford, M.D.
Alfred Scott.
Albert Wilson, M.D.

1887
Archibald E. Garrod, M.D.
H. T. Rutherfoord, M.B.
Kankai Totsuka.
Thomas Frederick Pearse, M.D.
Thomas Oliver, M.D.
Francis George Penrose, M.D.
Edward Ager Jones.
Samuel Herbert Habershon, M.D.
John Knowsley Thornton.
John Bland Sutton.
Oswald Auchinleck Browne, M.B.
Albert C. Butler-Smythe.
Joseph Arderne Ormerod, M.D.
REPORT
OF THE
COUNCIL OF THE CLINICAL SOCIETY.
DECEMBER, 1886.

THE activity of the Clinical Society during the Session 1885—86, is shown by the size of vol. xix of the Transactions, which is occupied by no less than forty-nine ordinary communications, and the accounts of twenty-six specimens described by card, while it contains only one report. This, the report of the Urinary Tests Committee, is a very short one, though of great value. The Society is, however, more to be congratulated upon the importance of these communications than upon their mere number; any great increase in the latter direction is indeed undesirable, as it would almost necessarily lead to the curtailment of debate; a result which, in the opinion of the Council, would be much to be deprecated.

So much has the Council been impressed with the fact that this, perhaps the most important record of clinical work in England escapes, for the most part, the attention of the profession in the colonies and abroad, that it has been determined to print annually fifty more copies of the Transactions than heretofore, with the view of presenting them to about forty of the more important foreign and colonial seats of learning.

Our numbers are increasing; there are now 317 resident and 110 non-resident members, of whom 19 resident and 9 non-resident have been elected during the past year. One resident member has become non-resident. Five members have resigned, and we have to deplore the loss by death of one honorary member, Dr. Austin Flint, and four ordinary members, Mr. John Burton, Dr. Sutro, Dr. Wiltshire and Dr. Moxon.
Dr. Moxon's loss to our Society, as to the other circles in which he occupied such a brilliant position, is felt to be especially great.

The finances of the Society are in a satisfactory condition. The balance at our bankers is £94 6s. 6d., notwithstanding that the first instalment of a somewhat heavy account which has been incurred in connection with the labours of the Myxœdema Committee has been paid. These labours, which are likely to result in what is hoped will prove to be the classical account of this important subject, are now approaching conclusion. The report of the Committee on Charcot's joint disease will probably appear in the next volume of the Transactions.

More need not be said in order to justify the Council in congratulating the Society upon its continued prosperity, and the promise of increased usefulness.
<table>
<thead>
<tr>
<th>1885-6</th>
<th>£ s. d.</th>
<th>1886</th>
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<td>Sale of <em>Transactions</em> by Messrs. Longmans at Berners Street</td>
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Amount of Stock standing in the names of the Trustees, January 1, 1887 | £600.

Examined and found correct | MONTAGUE MURRAY, M.D.,

CHRISTOPHER HEATH, Treasurer | FREDERICK TREVES,

R. J. GODLEE, Hon. Sec.
GENTLEMEN,—I should be little worthy of the honour which you have conferred upon me in electing me president of this Society, did I not feel grateful for the confidence you have placed in me, and take pride in the high position to which your kindness has raised me. It has always impressed me to run down the simple list of presidents of the great medical societies, a place in which is an unimpeachable title of honour; and if I have had any ambition beyond that of doing my duty, it has been that I might some day leave my name on one or other of those honoured rolls. But however great my pleasure in realising this ambition, it is with no feeling of elation that I take this chair; rather is it with a sense of responsibility almost oppressive in degree, and a fear lest I should fail adequately to fill the position, and to represent the Society.

But I have already said more than enough of myself, and, after all, the progress and fortune of this great Society do not depend upon the president of the hour. Its basis is sound, its work is substantial, and it has an inherent vitality which will carry it over any stumbling block created by my shortcomings.

It is unnecessary in this, its twentieth year, to explain the objects or justify the existence of the Society as a separate organisation. It is equally unnecessary to vaunt its useful-
ness, which is acknowledged on all hands, but I may for a moment foreshadow the further service it is destined to render to medicine.

This Society represents the medical man at work in the relief of suffering and the cure of disease. Whatever, then, may advance our knowledge of disease; whatever may assist us in the recognition of all its forms and stages when present; whatever may enable us to forsee its approach and detect its causes—these, and all that may aid us in preventing, resisting, and curing disease, are the subjects which engage our attention.

Now it seems to me that we are at a very interesting and important period in the development of medical knowledge, and that the immediate future is pregnant with discoveries bearing on the treatment of disease. Pathology, for a long time occupied mainly in the examination and record of results, has long ceased to be content with this, and has advanced far in the study of processes. But these processes are none other than the diseases which we watch at the bedside. While morbid anatomy was the goal of the pathologist, pathology and therapeutics advanced on parallel lines, and seemed as if they could never meet; indeed, the post-mortem table appeared only to demonstrate the absurdity of the attempt to combat disease. Now, however, pathology and therapeutics join hands. But morbid processes have beginnings and causes, or, if we are not allowed to use this word, antecedents. Some of these can only be ascertained by observation and inquiry, the results of which must often take the form of statistics, as, for example, with regard to the influence of heredity, of climates, of habits, of food; but many morbid processes can be started at will, and this permits of exact knowledge of the initial causes, and minute investigation of the succession of changes from beginning to end. Experiment, then, the chief instrument of exact science, enters the service of medicine. Experimental pathology has, in effect, never been much behind experimental physiology, and both are actively contributing to our knowledge. Experimental inquiry, however, not only occupies itself in the investigation of physiological processes and in the production and elucidation of morbid phenomena, but also in the examination and determination of influences which check, control, and counteract disease. All these lines of research are being enthusiastically pursued by zealous and able men, especially young men; knowledge comes crowding in from every side in perplexing abundance; and, sensibly or insen-
Address by the President.

sibly, practical medicine is coming more under the influence of science and of scientific methods.

But no idea can be formed from the separate and isolated contributions by physical, chemical, physiological, pathological, and experimental science, which help medicine, now here, now there, now in the comprehension of morbid processes, now in the application of remedial measures—no idea can be formed of the flood of light which will be thrown upon the theory and practice of medicine when all these sciences have completed their survey of the body in health and disease, and meet in the explanation of the operations of which it is the seat; when, say, the chemistry of nutrition and of the secretions is as well known as the rate of travel of nervous impulses; when the mode of action of drugs is as clear to our minds as the interchange of gases in respiration. We are very far from any such ideal as yet; but we have instances in which the convergence on one point of knowledge from various sources apparently irreconcilably apart, is beautifully illustrated. What can be more fascinating, for example, than the combination, for a given end, of the considerations brought to bear in recent cerebral surgery, of which such brilliant examples have recently been furnished by Hughes Bennett and Godlee in cooperation, and others? We have symptoms which the physician, largely through the insight and work of Hughlings Jackson, refers with confidence to a cerebral tumour. This is localised with precision through the experimental investigations of Hitzig and Ferrier. These and earlier investigations, again, having taught that portions of brain can be removed without danger to life; Lister's antiseptic precautions—themselves the outcome of scientific experiment in a totally different field—make the trephining of the skull and the excision of the growth comparatively safe; and, by a last refinement introduced by Victor Horsley, advantage is taken of the anaemia of the cortex during morphine sleep, found years ago by Durham, to diminish the haemorrhage during the operation.

Whatever the knowledge of the day may be, and whether it has been reached by scientific investigation or has as yet only the sanction of experience, it is through the physician or surgeon that it is brought to bear on individual cases. And here let me repudiate and protest against the idea that there is any antagonism between scientific and practical medicine, or any incompatibility between practical skill in the treatment of disease and love of knowledge for its own sake. It is contradicted throughout the history of medicine. The medical
man ought to be animated by the scientific spirit, and for the
most part, is. It is true that a knowledge of men and women
may lead to success in practice, as well as a knowledge of
disease, and that the weakness, frailty, and folly of mankind
constitute a mine of wealth to the unscrupulous man who
obtains power and opportunity to exploit them by a medical
degree; but so it is in all professions, and it need not make us
doubt that the practice of medicine is fundamentally scientific.
Nor need we concern ourselves with such instances; they are
unworthy of our attention as individuals, and we may be sure
that the very last place to which the triumphs of the charlatan
will be brought for criticism will be this Society.

The medical man, as I was about to say, is the intermediary
who gathers up and applies for the benefit of the patient all
the knowledge available at any given period, and whatever is
most essential to individual members of the profession in the
exercise of this function is the most worthy of the attention of
this Society. This, unquestionably, is diagnosis. I have
heard distinguished lawyers in confidential after-dinner chat
parody Danton's noted phrase about andacity, and say that the
first condition of success at the Bar is good animal spirits, the
second good animal spirits, and the third good animal spirits,
adding that a little knowledge of the law is not amiss. In
more serious mood, I should reply to the question, "What is
most important at the bedside"—diagnosis! diagnosis! dia-
gnosis! But diagnosis is a word of deep meaning—thorough
knowledge. It is no diagnosis at all to say that such and such
is a case of valvular disease of the heart, or even that it is a
case of aortic or mitral disease, obstructive or regurgitant.
We must be prepared to estimate the degree of obstruction or
the amount of regurgitation to say whether the valvular change
is probably stationary or likely to be progressive, to measure
the effects on the cardiac muscle and cavities, and to judge in
what direction and in what degree lethal tendencies are
developing themselves. It is no diagnosis to say that a man
has hemiplegia; we must localise and specify the lesion which
has given rise to the paralysis. But the lesion itself again is
only an incident in some general morbid tragedy which is
being acted in the body, and this must be read from the
beginning.

Diagnosis, moreover, embraces a knowledge, not only of
the seat and character of the disease, but also an appreciation
of the condition of the patient in relation to the attack. Is
the disease merely the winding up of a bankrupt constitution?
—a mode of dying rather than a cause of death? or is it, on the other hand, a necessary though violent readjustment of the organism to the environment with which it had been long getting out of harmony, or a defensive reaction against some powerful disturbing influence? The problems presented are often of the highest degree of complexity, and we cannot too diligently cultivate our powers of observation, aiding the senses with the instruments with which science has furnished us, and bringing everything, as far as possible, to the test of measurement and the balance. With regard to this, however, I need say nothing; every meeting of the Society gives proof of the zeal with which morbid phenomena are watched and recorded, and of the acumen with which they are discussed and criticised. It is sometimes objected that the cases read here are usually such as are rare and exceptional, and therefore of little practical use; but the exceptional cases of to-day are the common ones of to-morrow. There is no one who has for many years been in the habit of noting all he sees, who cannot look back to cases which he did not at all understand at the time, but which are now perfectly clear. And I would remind my hearers of the profound saying of Sir James Paget, "That an exception to one rule is only an example of another rule as yet unknown." The explanation of an exceptional case may thus be the revelation of a new law.

It is not so easy to deliver my mind on the subject of treatment. On looking through our Transactions the reproach that we give attention to everything but treatment might seem to be well founded. This reproach, it is true, could not be addressed to the surgeons. Surgery is treatment. Medicine may be or may not; and as a physician, I am tempted to envy my distinguished surgical predecessor the splendid record of improvements and advances which have marked his term of office. I cannot hope that my reign will compare with his in this respect, although I trust the physicians and general practitioners will be stimulated to a generous rivalry with the surgeons. One reason why we seem to lag behind them is that our results are less tangible than theirs. We may, and we do, check the ravages of phthisis in the individual as in the community; we may beat back for years the advance of renal disease; we may deprive locomotor ataxy of its qualifying adjective "progressive"; but these and like instances, which are our most genuine triumph, cannot well be put into the form of cases. Many such diseases, indeed, deadly as they are, get a worse character than they deserve through our
Address by the President.

reticence; and young practitioners are often astonished to find the fulfilment of their gloomy prognostications so long deferred. I wish it were more easy to exemplify the results of successful resistance to disease, and it would be a great advantage if we could have brought within the ken of the Society some of the minor ailments which so constantly come before us. A certain proportion of them are early stages or forerunners of serious disease, and others, which we can only look upon as functional derangements, inflict more misery upon the patient than cancer itself. They are, therefore, supremely worthy of our attention. It would be difficult, I admit, to convey the fine distinctions, on which so much depends in giving an account of such affections; but well-observed individual cases would be far more instructive than the generalised descriptions which are given in treatises. An opening would be afforded also for the discussion of many unsettled questions. The mental depression, for example, which is often clearly traceable to functional derangement of the liver (to employ a term sanctioned by the authority of Murchison)—is it a reflex disturbance or inhibition of some nerve centre? or is it the effect of a blood-impurity acting as a poison to the brain like alcohol or opium? or is it a local or general derangement of the cerebro-spinal circulation? It is an every-day experience that a dose of blue pill will relieve the mind, clear the complexioin, and relax the arterioles. Now, in many cases of obstinate and severe melancholia there is every reason to believe that the cause is outside the nervous system; and if we could, in the familiar examples of low spirits and irritable temper, trace accurately and minutely the way in which the initial gastro-hepatic disorder produces its effects on the nervous system, and the exact process by which these effects are reversed, we might understand and prevent or remedy the more serious overthrow of the mind seen in melancholia. It is not a final explanation, discharging us of all responsibility in searching out the cause, and rendering futile all efforts in the direction of cure, to find a family history of insanity. The hereditary tendency to melancholia may be indirect, and capable of defeat, just as hereditary tendency to apoplexy may be traceable to family gout, which can be prevented from developing its evil effects in individual members.

Whether we find such an opportunity of considering the forms and phases of diseases to which I have just alluded, or not, I hope we shall not lack cases illustrating the effects of remedies, and by remedies I here mean drugs.
Address by the President.

Nursing, care, appropriate food and stimulants, do much for our acute cases; diet, clothing, climate, hygiene, are powerful remedial agencies in chronic disease, and we cannot too closely study the surroundings of the patient, or direct too minutely the management of the sick room; we cannot inquire too carefully into the habits and mode of life which have led to chronic ailments, or give instructions too detailed as to the means by which the injurious influence of past errors may be counteracted in the future. Were we to neglect these particulars, we should in effect ignore the first principle of rational treatment—"the removal of the cause"—and wilfully leave an insuperable obstacle in the path of recovery. But when we have attended to everything of this kind, we should be shorn of our strength were we debarred from bringing to the aid of sufferers the powerful remedies we hold in our hands. In acute disease they often turn the scale in favour of the patient, as, for example, when death is averted in enteric fever by a timely dose of opium or quinine, or when the distended right heart is relieved by a calomel purge. In chronic affections, again, even if the patient is prepared to renounce his self-indulgence and forego his pleasures, or to neglect his business and abandon his ambitious schemes—and how often is he unwilling to do the one, or unable to do the other?—the long chain of evil consequences would be very slowly unwound, whereas we can often snap it almost at a stroke.

I shall venture to assume that scepticism, in the evil sense of disbelief in medicinal agencies, is extinct. I shall not, at any rate, argue the question, but take it for granted that we have confidence in the drugs we employ, and believe we have abundant evidence of their good effects. Were this not so, few of the men now before me would consider the medical profession worthy of their adoption as the work of their lives. We look to quinine in ague, to mercury and iodide of potassium in syphilis, to iron in most forms of anaemia, for effects as certain and as constant as chemical reactions. These and similar examples, of themselves, or the single instance of the multifarious benefits obtainable from the administration of opium and its alkaloids, would justify us in asserting that we are able to modify profoundly and beneficially the course of disease. It is only want of knowledge of the morbid process on the one hand, and of the mode of action of the drug on the other, which prevents our having the same certainty in a thousand other instances. In proportion as our investigation of the cases which come before us is thorough, and our aim
and object in the employment of medicines are clear, definite, and precise, in that proportion will our confidence in the uses of drugs be firm and will continue to increase.

With regard to the effects of remedies, however, we have, for the most part, only the testimony of experience pure and simple, and our experience is a very insecure anchorage for belief, and a very feeble agency in carrying conviction to the minds of others. A man's own experience will, for himself, outweigh that of any number of others, and a single instance in which his own observation contradicts, or appears to contradict, the concurrent testimony of the rest of the world, will overthrow his belief in that testimony. In the complicated phenomena of disease, again, it must often happen that all the facts are not known, and the experience of one case may not apply in another apparently similar, and this unsettles confidence. When, however, we have an explanation which commends itself to our judgment, adverse facts often make little impression. Hence the sway of theory; whether we will or no, we refer the action of remedies to some theoretical cause, and it is because we have not always confidence in our theory, and not because we have any doubt about our facts, that we sometimes appear to falter in our testimony in regard of therapeutics.

The remedy for this is a knowledge of the mode of action of drugs, and it is to discoveries in this direction that I alluded when, in an earlier part of this address, I said that we stand at an important period in the development of medical science. Already we employ digitalis with greater confidence and discrimination from the light which experiment has thrown upon its action on the arteries and heart, and nitro-glycerine, amyl nitrite and the nitrites have actually come into use on the ground of their experimentally demonstrated effects in relaxing the arterioles, and have proved to be of immense benefit. We have also introduced into practice, on experimental evidence, substances which directly and constantly bring down the temperature of the body. Enormous importance attaches to the investigations which are in progress on all hands, and I would refer particularly to Dr. Sydney Ringer's experiments, showing the influence of minute proportions of soda, potash, and lime salts, on the action of the heart, which bring into evidence once more the radical differences which exist between substances commonly regarded as all but identical. Soda and potash, for example, are, for the most part, looked upon simply as two alkalies, which may be employed almost indifferently,
the only exception recognised being, perhaps, that potash is the better solvent for uric acid and more of a diuretic, whereas their alkalinity is almost the only property they have in common; when regarded as medicines their relations with the organic constituents of the body and their influence on muscular contractility are totally different; they are found in different structures, and a minute proportion of any potash salt injected into a vein paralyses the heart, while soda salts have no such effects.

But while the mode of action of drugs is engaging attention, and great discoveries are bound to come before long, the progress made as yet is only small; and although the eagerness with which new remedies, which come accredited by science, are seized upon by the profession is a hopeful sign—there is danger of present injury from their employment. It is a tremendous piece of knowledge that a given drug will certainly lower the temperature, but this does not by any means warrant its indiscriminate administration in pyrexia. There is the further question, by what processes the temperature is reduced, and what concomitant effects are produced. Quinine, salicine, salicylic acid and its compounds, antipyrin and like substances, aconite, and many other drugs, agree in diminishing the body-heat, but this is the only effect they have in common. There is positive peril in acting simply on the knowledge that the temperature can be lowered. Are we certain, to begin with, that a high temperature is always mischievous? May we not, in checking it, be interfering with defensive or readjusting processes? Are we to assume that Nature does not know what she is about when she sets the heat-regulating mechanism for a higher level whenever things go wrong in the economy? To take a practical example. In enteric fever, in which the long-protracted pyrexia becomes in itself a distinct danger by its effects on the nervous system, by disintegration of the muscular fibres of the heart and the glandular structures, and by the strain on nutrition, I am convinced of the utility of systematic cold bathing, by my own observation as by the testimony of such men as Brand, Liebermeister, Cayley, and Triper (of Lyons); but I think, from what I have seen, that the repression of body-heat by means of salicylates, antipyrin, and the like in this disease, is very dearly bought. Nor can I see what we have to gain by suppressing the short sharp fever, say, of pneumonia. Even with regard to the employment of salicine and the salicylates in rheumatism—remedies which came to us straight from the
laboratory, and whose mode of action is expounded in chemical formulae by Dr. Latham—I think it well to raise a warning note. Properly employed, salicine and the salicylates almost rob acute rheumatism of its terrors and dangers, but given in routine fashion, as we should give an effervescing mixture, they have seemed to me capable of doing serious harm. At any rate, I have seen deaths in rheumatic fever, of a kind quite new and strange to me, after prolonged administration of salicylate of soda every four or six hours. The pain and fever had never yielded, the drug had been continued in hope of this result, till, in course of time, warning was given by delirium, the pulse became more frequent and extremely weak, what has looked like a modified hyperpyrexia has set in, with flushed face and hot perspiring skin, and the patient has rapidly sunk, the temperature, however, never rising above 105° Fahr.

I come back, then, to my point that, in order that we may employ antipyretics, or remedies of any kind, with confidence, we must know exactly how the effects produced are brought about. With regard to this we are, however, only on the threshold of an inquiry which must go more deeply into the relations between physiological processes and the substances which modify them than, as yet, we have any conception of, for our information is superficial. Now, it is an axiom in science that for every action there is an equivalent expenditure of energy. It is not by its mere presence in the blood or in the nervous centres, or in the gland structures that a poison or remedy produces its effects; there is some dynamic agency at work. In the case of such bodies as the powerful organic alkaloids this can scarcely have any other source than chemical change in the substance itself, while in the case of inorganic salts, it would seem that they condition reactions between other substances. We know that carbonic oxide proves fatal by combining with the haemoglobin to the exclusion of oxygen, so that the blood-corpuscles are no longer available as carriers of oxygen to the central nervous system, and the anaesthetic gases and vapours act much in the same way, their safety consisting in the fact that the higher centres concerned in the mental operations and sensation succumb to the privation of oxygen before the lower centres, which are the seats of the vital reflexes, and that these substances are easily displaced by oxygen when this is again freely supplied. Here the proportion of the anaesthetic agent is comparatively large, and the mode of operation is physical rather than chemical, the vapour-
density actually counting in the comparative effects of different anaesthetics.

I might name other actions more or less capable of explanation, but it is such effects as those produced by aconite, atropine, morphine, and prussic acid, in which a fraction of a grain will give rise to profound disturbance or destroy life, that we need to understand. Twenty years ago, or more, when science was not prepared to answer, nor I qualified to put, such questions as those relating to the action of poisons and remedies, looking at the fact that all powerful alkaloids contained nitrogen, and that all, or nearly all, of which the constitution as well as the composition was known, belonged to the class, so named at that time, of amides, imides, and nitriles, in which an ammonium radicle had been robbed of successive atoms of hydrogen, I conceived the idea that this departure from a stable type gave rise to a chemical tension capable of producing effects when brought into presence of the chemical tension which seemed to me to be required for the explosions of nerve-force.

It occurred to me that prussic acid—the most deadly of poisons, and, at the same time, almost the simplest of organic substances—offered the best possible chance of ascertaining the exact mode of action of, at any rate, one drug.

Hydrocyanic acid is composed simply of a single molecule each of carbon, hydrogen, and nitrogen, and there is no room for complex changes. The hypothesis I entertained was that, when it reached the nerve-centres, the dislocating influence of nitrogen found its opportunity, under the influence of the chemical operations concerned in the evolution of nerve-force, and the nascent carbon and hydrogen flying apart, appropriated the oxygen needed by the nerve-cells, death resulting from the consequent arrest of activity of the centres in the medulla necessary to life. I cannot say that I proved my point but I have met with no more satisfactory explanation since, and I met with facts which have never failed to interest men when I have related them. For instance, prussic acid is not a deadly poison to frogs. Many times I injected as much as ten minims of the pharmacopœial solution (one minim of which was fatal to a rat) under the skin of a frog, and as long as he was allowed to hop about in the room, he appeared to be no worse for it. If, however, even at the end of half an hour, I put him under a bell jar with other frogs, the prussic acid he exhaled sent the whole lot into a state of torpor. My interpretation was that the evolution of nerve-force in the frog was...
not attended with sufficient chemical energy to explode a HCN molecule. Another curious fact which turned up was that nitro-glycerine, which proved fatal to rats, apparently by paralysing the nerve-endings like curare, was a convulsant of terrible energy when administered to frogs.

For a full comprehension of the action of remedies we must know and understand the chemical actions and reactions which are taking place in nutrition, secretion, and disintegration; the chemical changes which attend and condition the evolution of nerve-force and muscular action; the modification of the chemical processes which result from the administration of a drug, and the change in the composition of the drug itself, through which the energy is evolved by means of which it produces its effects. The future of therapeutics is thus in the womb of chemistry. What microscopy has been to anatomy and pathology, chemistry will be to physiology and therapeutics. Anatomy and pathology are the statics of medical science, physiology and therapeutics the dynamics; microscopy, therefore, represents the statical, chemistry the dynamical department of medical investigation. The microscope reveals the ultimate structure of tissues, the methods by which they are developed, and the changes which take place in them in disease; chemistry deals with the forces and operations by which the structures are built up or destroyed, and with the actions and reactions of the atoms and molecules which enter into their composition, and will ultimately extend our ideas as much beyond their present scope as the microscope has enabled us to advance beyond the knowledge of the old anatomist.

I cannot invite the relation of researches and the discussion of speculations on the mode of action of remedies in our meetings here, but I do not think I have gone beyond the scope of the Society's aims in speaking of them, since the moment views and investigations are brought to the test of observation at the bedside and can be embodied in cases, they may be brought before us. This, I acknowledge, I should earnestly desire to encourage.

I can, indeed, refer to contributions of my own in the Transactions of this Society, insignificant it is true, in which I tested the hypothesis that substances closely allied chemically ought to have a similar action on the system, or that any diversity in their operation should be capable of explanation on chemical principles, by experiment with the iron group of metals and with the arsenic group of bodies. I may perhaps
be allowed to say, incidentally, that if the medical men and pharmacists, who devoted so much time and pains a while ago to devising methods for administering that intractable body, permanganate of potash, had been familiar with my paper and with observations long antecedent to mine, they might have been spared much trouble by the information that it is the manganese itself, and not the latent oxygen they vainly thought they were getting into the system, which acts as an emmenagogue, and that the chloride and the sulphate of the metal can be administered as easily and as freely as the corresponding salts of iron.

But I must recall myself and you from the contemplation of future powers to the consideration of our present duties. What we have to do is to observe and record; to watch to-day with a mind instructed and guided, but not biassed, by the results of yesterday; to bring the experience we gather, without reserve before the Society for the information of others, submitting our views and conclusions to the judgment of our colleagues, and bringing an open mind to the appreciation or criticism of cases which they contribute in their turn. As iron sharpeneth iron, we shall put a finer edge on each others' faculties, and render our individual and collective diagnosis more certain, clear, and deep.

Diagnosis will not lose in importance as knowledge increases or become more easy. If we knew all that is to be known of the action of remedies, it would only render diagnosis more important than ever, just as every improvement in the accuracy and power of artillery makes the aim of the gunner of greater consequence. A bad gun—to continue a simile employed before—badly directed may hit the mark; a good one could not by any possibility; so precision in the employment of remedies would only make failure more certain and disastrous if the diagnosis were wrong.

I have been brought irresistibly back to diagnosis, since from it all treatment must flow; and now I conclude, as I began, by thanking you for the honour you have done me in entrusting to me the duties of President of this Society, assuring you that to them will be devoted my best energies.
COMMUNICATIONS.

I.—A Case of congenital Malformation of the Heart, with systolic and prediastolic basic Bruit; with observations on the causation of Bruits preceding the cardiac Sounds. By F. Charlewood Turner, M.D. Read October 8, 1886.

The case of a child, the subject of congenital malformation of the heart with cyanosis, who was exhibited to the members of this Society at a former meeting, is of interest on account of the unusual character of the cardiac murmur audible in the region of the pulmonary valve, and especially in relation to the question of the physical causation of cardiac murmurs immediately preceding and running up to the sounds of the heart.

Excepting in regard to these points there appears to be nothing of particular interest in the case.

The child, who is now six years old, has been under observation at the North-Eastern Hospital for Children off and on since the age of two, and during all that time the auscultatory signs and general symptoms of the cardiac lesion have continued much the same.

He is small for his age, fairly nourished, and markedly cyanosed, with clubbing of the fingers. The cyanosis has been noticed by the mother ever since an attack of "croup" at the age of ten months, during dentition. He then used to have attacks of difficulty of breathing, attended with much lividity, on being put to bed, the attacks lasting about an hour. Cyanosis, with shortness of breath on any exertion,
Dr. Turner's Case of Congenital Malformation of the Heart.

has been noticed ever since this time, especially with cough, to which he has been more or less subject, and on going out of doors.

At the end of 1882 he had an attack of measles, which was followed by scarlatina in the early part of 1883, from both of which illnesses he recovered satisfactorily. He has been subject to bronchitis, especially in cold weather, when the cyanosis is greater, and to occasional attacks of croup of the character above described.

The mother states that she never had acute rheumatism, and has not been subject to rheumatism. She had no illness during pregnancy with this child. She has three other healthy children.

On examination of the child's chest the sternal region is seen to be prominent. Partial cardiac dulness extends far to the right, nearly to the nipple line, and there is visible impulse to the right of the sternum. The absolute cardiac dulness is of limited extent, and to the left of the sternum. The heart's apex is in the left nipple line. On auscultation there is heard, in the region of the pulmonary valves, a loud and harsh systolic bruit, which appears continuous with a loud and rough bruit of short duration, which immediately precedes and runs up to an accentuated second sound, by which it is abruptly terminated; the prolonged basic bruits exactly resembling a prolonged diastolic and "presystolic" apical bruit running up to an accentuated first sound. On palpation a slight thrill is felt at the base of the heart during the systolic period, and continued up to the diastolic valvular impact (or tension). The systolic bruit is audible over the chest anteriorly and posteriorly. It is loudly heard under the left clavicle, less loudly under the right clavicle. In the back it has a softer blowing character. The "prediastolic" bruit is very distinctly heard down the sternum, the systolic bruit being there diminished in intensity.

The auscultatory phenomena indicate a stenosed and thickened pulmonary valve, and the short "prediastolic" (i.e. pre-diastolic-sound) bruit must, I think, be regarded as due to reflux into the ventricle arrested by delayed closure of a defective but not incompetent valve, as pointed out by Walshe, who recognised bruits of this character, and described them in his work on diseases of the chest.

A bruit of this character produced at the semilunar valves admits of no other explanation, and an exocardial origin of the bruit in this case is excluded by the phenomenon accompanying
Dr. Turner's *Case of Congenital Malformation of the Heart.* 3

it. Stretching of pericardial adhesions, if capable of producing a sound resembling this bruit, could not account for the conduction of the bruit down the sternum, nor for the thrill attending it, nor for the accentuation of the second sound.

A consideration of the mechanism of the closure of the valve, moreover, shows that with a certain degree of rigidity of the valvular curtains there must be limited reflux, which, if sufficiently prolonged, would produce a bruit having all the distinctive characters of the "prediastolic" bruit. If the semilunar curtains are too rigid to be brought together by the comparatively low pressure upon them at the conclusion of the ventricular systole, but not too rigid to admit of effective closure by the higher pressure of arterial recoil, there will necessarily be reflux, to a greater or less extent, during the brief interval of the diastolic period preceding the closure of the valve, and second sound thus delayed. A bruit produced by this reflux would have the progressive intensification, and abrupt termination by an accentuated second sound, characteristic of the "prediastolic" bruit heard in this case, and it would have also a rough character and accompanying thrill, like the systolic bruit preceding it, from a combination of vibrations of the freely hanging curtain, of the patent valve, with those produced in the blood by its passage through the contracted aperture. The occurrence of a "prediastolic" valvular bruit shows that the interval of the diastolic period preceding the second sound may be sufficiently prolonged for the production of a distinct bruit. Its rarity shows that, owing to the highly impulsive character of the recoil of the elastic arterial walls distended by the force of ventricular systole, this interval can be appreciable only under conditions especially favorable to its prolongation.

Amongst such conditions are especially (1) a low mean arterial tension, and (2) forcible discharge of blood from the ventricle; these conditions tending to the formation of the initial pulse-wave at a greater distance from the heart.

These conditions are presented in a high degree in cases like the present by great stenosis of the orifice of the pulmonary artery and hypertrophy of the right ventricle, which sufficiently account for the occurrence of the bruit in this instance.

Thus regarded the occurrence of this bruit supplies a feature in the clinical picture of lesions of the semilunar valves which would naturally be looked for.

But the occurrence is, I think, of more particular interest
in connection with the question as to the causation of the more familiar "presystolic" bruit, as corroborating the views of those who, with the late Dr. Barclay, believe this bruit to be similarly due to regurgitation at the commencement of the ventricular contraction arrested by the delayed closure of rigid and defective, but not incompetent, mitral curtains, similarly filling up what would otherwise appear to be wanting in the clinical picture of lesions of the auriculo-ventricular valves.

The "prediastolic" bruit is not mentioned in medical works for the most part, and this may be accounted for by its exceptional occurrence, and by the absence of any diagnostic or prognostic importance attached to it. This bruit has, however, been recognised and described by Dr. Walshe,* by Dr. Hayden,† and by Dr. Augustus Waller,‡ and in vol. vii of the second series of the *St. Thomas's Hospital Reports* I have recorded two cases in which it was observed by myself. Dr. Walshe refers it to regurgitation through a defective, but not incompetent valve, as above explained.

In the case of mitral curtains of a certain degree of rigidity there will be delayed closure, with reflux arrested by contact of the flaps. And this arrested reflux, if sufficiently prolonged, and if the position of the valvular curtains and the force of the blood stream were suitable—and the free edges of the mitral curtains held out from the ventricular wall by adhesions would be most favorably placed—would produce a bruit with the distinctive character of the presystolic murmur. It would have, in common with the diastolic mitral bruit, those characters which are dependent upon patency of the valve, and are referable to predominance of valvular over fluid vibrations in the production of the sound,—the former comparable with the sounds produced in a wind instrument by vibrations of the musician's lips; the latter to the whistling noise produced in the instrument by an inexpert person blowing forcibly through compressed lips. It would on this account be a rough and harsh bruit, with sensible thrill conducted to the apex of the ventricle and to the chest wall in contact with it, by the chorda tendinea and papillary muscles, and would be limited to that region. And by these characters it would be distinguished from the blowing systolic bruit of regurgitation after contact of the curtains.

It would necessarily have also the progressive intensifica-

† Hayden, *Heart and Aorta*, p. 885.
‡ Waller, *Practitioner*, i, 80, p. 344.
tion and abrupt termination in an accentuated first sound so characteristic of the "presystolic" bruit, and so suggestive of reflux arrested by closure of the valves at a later and more active period of the ventricular systole.

Barclay's interpretation thus affords a fully sufficient explanation of the clinical phenomena in relation to the anatomical lesion with which they are associated. If it could be shown that in cases of mitral stenosis the period of the systole preceding the closure of the valve and first sound is liable to be sufficiently prolonged for the production of an audible bruit, this would suffice to prove that Barclay's explanation of the bruit is the true one.

That this interval of the systole is prolonged in cases of mitral stenosis is indicated by the retardation of the heart's action observable in such cases when quiescent, a retardation adapted to the effective use of the force expended in presence of this impediment to the circulation, and also by the relatively short duration of the interval between the first and second sounds, facts especially noticed by Barclay. That this interval is, more or less often, sufficiently prolonged for the production of an audible bruit is, I think, proved by cases such as that above detailed.

For the conditions are, on two accounts, much more favorable to the occurrence of a "presystolic" than of a "prediastolic" bruit by reflux before the closure of rigid valvular curtains.

1. The muscular contraction of the ventricle commencing from a state of relaxation cannot have the impulsive character of the arterial recoil. And that this is so, is shown by the difference in the character of the sounds. The sharpness of the second sound shows, that the vibrations of the semilunar valves caused by the impact of the recoil wave are promptly arrested by the rapid rise of pressure upon them. The comparative flatness of the first sound shows, that the vibrations of the mitral curtains are less quickly arrested by a more gradual rise in the intraventricular pressure. There must then be, ceteris paribus, a longer period of reflux through a rigid and defective mitral, than through a similarly defective semilunar valve. But if the ventricular systole is slower than normal, its "presystolic" portion may be increased to an extent limited only by the requirements of the maintenance of the circulation.

2. But the conditions favorable to the occurrence of a "presystolic" regurgitant bruit contrast with those so unfavorable to the prediastolic bruit in another important circumstance; and
this is that there is a certain interval between the first act of ventricular contraction and the commencement of the general expulsive contraction of its walls.

The contraction of the muscular fibres which encircle the mitral orifice, is at once the completion of the auricular systole and the first step in the contraction of the ventricle, and it serves an important purpose in each of these acts in the cycle.

As the completion of auricular systole, its effect is to prepare the mitral valve to resist, and to support it under, the strain of the ventricular systole, and is comparable with the resistance to reflux by full contraction of this part of the original vascular loop, of which the heart is a development, in the vermicular propulsions of the blood through it.

On the other hand, the effect of this basal contraction as the preliminary step in the ventricular contraction, closing up and drawing aside the interpapillary portion of the cavity, and propelling its contents into the apex and infundibulum—thus placing the whole of the ventricular contents in a position favorable for propulsion into the aorta—is the same as the effect of the passage of the ventricular contraction along the first limb of the original vascular loop, which is thus represented in the fully developed organ; the succeeding propulsive ventricular systole, from apex to base, representing, and taking the place of, the vermicular contraction of the second limb of the loop.

Thus there is a certain interval between the first act of ventricular contraction—with arrest of influx from the auricle, and closure of the mitral valve under normal conditions—and the commencement of the general systole, to which this preliminary contraction is a necessary preparation. And on this account, as well as on account of the less degree of rapidity of the muscular contraction, the physical conditions of closure of the mitral valve are much more favorable to the occurrence of a "presystolic" bruit, by checked or arrested reflux, than those of the semilunar valves to the production of a "pre-diastolic" bruit.

These considerations, with the clinical facts adduced, seem to afford the fullest corroboration of Dr. Barclay's views of the causation of the "presystolic" bruit, and to take away the basis on which the alternative auriculo-systolic hypothesis rests, viz. the assumption that there is in these cases no appreciable interval between the completion of auricular systole and the first sound.
Professor Gairdner* says that when the first sound is heard in the course of an apparently continuous bruit we shall "know absolutely that the first part, preceding and running up to the first sound, must be auriculo-systolic, and that the part succeeding the first sound must be ventriculo-systolic.†

To this assumption Barclay demurs, and what has been said above will, I think, suffice to show that such a statement could not be admitted without proof.

The supposition that a bruit running up to—or appearing to run up to—an accentuated first sound, is produced by auricular systole, involves the assumption that this, ceasing at the acme of its propulsive effect, is immediately followed by a forcible and impulsive ventricular contraction, by which the valve is closed and made tense within an inappreciable period of time from the acme of influx through it. Of such a rapid and impulsive ventricular systole in cases of mitral stenosis there would appear to be no evidence or probability, the conditions, on the contrary, being such as to necessitate and produce a retardation of the heart's action.

The direct evidence of the cardiograph, moreover, is, I think, inconsistent with the supposition that this bruit can be due to the auricular systole. For the tracings obtained in such cases, under the skilful hands of Dr. Galabin and the late Dr. Mahomed, show a well-defined interval between the ventricular systole and the preceding auriculo-systolic elevations.‡

Supposing this elevation to represent the force of the auriculo-ventricular influx, it would be scarcely conceivable that a bruit with a maximum intensity so indicated could appear to run up to the first sound, or accentuated termination of the first sound.

But it may be questioned whether this auriculo-systolic elevation is an indication of the force of influx into the ventricle.

It is, I think, more probable that this elevation, and the corresponding elevations in Marey's simultaneous tracings from auricle, ventricle, aorta, and heart's impulse, indicate the completion of the auricular systole and the contraction of muscular fibres surrounding the mitral orifice.

† One would say, from Dr. Barclay's point of view, that when the first sound is heard in the course of a continuous systolic bruit, it shows that the valvular curtains are rigid and resistant to closure, as well as unable to sustain the strain upon them.
‡ See Guy's Hosp. Rep., New Series, vol. xx, plate iii, figs. 9, 10, 15, 19. This feature is pointed out both by Dr. Galabin and Dr. Mahomed, as especially characteristic of cardiograms from cases of presystolic bruit, being indicative of an earlier and more powerful contraction of the auricle than normal.
In the absence of any observations to show that this elevation in the cardiographic tracing does precede the completion of the auricular systole, it appears to me almost certain that pressure upon Marey's sound by the walls of the auricle when fully contracted (i. e. at the moment corresponding with full contraction of this part in the vermicular contractions of the original vascular loop), must have been greater than at any previous moment, while the blood was flowing freely into the relaxed ventricle; and I think that the initial tension of the ventricle, with the movement of its contents and alteration of form, by this preparatory basal contraction, would be more conspicuously represented in the impulse tracing and in the ventricular and aortic pressure curves, than an auriculo-systolic increase of influx preceding it. The notable duration of the "presystolic" bruit heard in some cases seems to me to point to such an interpretation of this feature of the cardiographic tracing.

Another clinical fact connected with the occurrence of the "presystolic" bruit, which, while according with Barclay's views, appears scarcely consistent with the alternative hypothesis, is the observed occurrence of a "presystolic" bruit in cases of cardiac dilatation, without stenosis of the mitral valve. A prolongation and modification of the first sounds, with the distinctive character of the presystolic bruit, is, I think, not uncommon; and I may here mention an observation of the late Dr. Mahomed,* quoted in Fagge's Medicine, that in cases of Bright's disease with cardiac hypertrophy the first sound is sometimes preceded by a short sound having precisely the character of a presystolic bruit of mitral stenosis. Cases in which a distinct bruit of this character has been heard during life, and in which an autopsy has shown that there was no mitral stenosis, have been recorded by Dr. Flint† and by myself in the paper before referred to.‡

The facts, thus considered individually and collectively, appear to me to present great difficulties in the way of the auriculo-systolic hypothesis of the causation of the presystolic bruit, and, notwithstanding the sanction of high authority and the wide acceptance of that view, I think that they will confirm the conviction of the late Dr. Barclay that this cardiac murmur is systolic and regurgitant, the clinical evidence of a defectively acting, but not incompetent, mitral valve.

* Fagge, ii, p. 449.
† Austin Flint, Amer. Journ. of Science, vol. 44.

The man who is the subject of this short paper is now twenty-seven years of age, and first came under my notice in March of this year, on account of a left oblique inguinal hernia. The man is just over six feet high and weighs eleven stone two and a half pounds. He is of slender frame, with light auburn hair, none of which has grown on the face. The complexion is delicate, his skin soft and smooth, and his muscular power is less than that of men of his age and height. His voice is boy-like.

The genital organs may be described in one word as undeveloped. The penis is small, about the size of a boy’s of ten years old; the preputial orifice is too small to allow of the exposure of the glans. Both testicles are small; the body of the organ on each side is about the size of a linnet’s egg, smooth and tense. The left epididymis is of proportionate size, but the right is a little larger and indurated. The scrotum is small, and thinly scattered over it and the mons veneris is light hair, which does not extend at all up towards the umbilicus in the middle line.

Examination per rectum shows that the prostate is very small, so small and soft, indeed, that its exact outline cannot be traced. Nor can the finger feel with any clearness the vesiculae seminales.

As already mentioned, there is a reducible oblique inguinal hernia on the left side, which he has been conscious of for eight or nine years, and for which he now wears a truss.

On examining his throat in the usual way, nothing is seen of either tonsil, and the pillars of the fauces are unusually close together; during an effort of deglutition, or by external pressure, the fossa between the pillars can be made to project inwards, and then a very small tonsil is seen on either side, the left being smaller than the right. With the finger, too, small tonsils can be felt; there is nothing abnormal—no pitting nor scars—about the fauces or tonsils.

The man is innocent of sexual desire, and the only sign of
any functional activity of his sexual organs is occasional slight priapism under the stimulus of lascivious reading.

As regards his history, he states that he had a blow on the scrotum when eleven years of age, which was followed by "inflammation," for which he had to lay up for three weeks. To this attack I presume we may attribute the thickening of the right epididymis. He has not had any bad attacks of sore-throat; he is not "subject to cold," but at times he has had slight catarrhs. He is quite clear that he has never had any long-continued and severe affection of the throat; indeed, he speaks of himself as the strongest of his family. He has not had mumps.

His family consists of four brothers and three sisters. All the sisters are married and fruitful. One of his brothers is married, but his wife has not had a child; he does not know that there is anything wrong with either of his brothers. I have not been able to see them.

Of the fact of the want of development of the sexual organs, I presume there can be no question, and that it affects the entire sexual apparatus. In this there is nothing very remarkable. The tonsils vary in size within somewhat wide limits, and some may question whether in this case they are too small to be called normal. In my opinion they are, and Dr. de Havilland Hall, who kindly examined the man some time ago, reported that the tonsils are very decidedly below what may be called the normal standard. Assuming this to be so, I have thought the case of sufficient interest to bring before this Society, particularly in view of the prevalent popular notion that excision of the tonsils during childhood endangers the individual's virility. There are, however, two distinct questions that may be asked. The first is, whether there is any functional or developmental connection between the sexual organs and the tonsils. Such a case as that now presented suggests that there may be, and that the tonsils may sustain a relation to the testicles similar to that of the larynx. This view is supported by the apparently marked effect upon hypertrophy of the tonsils which the arrival of puberty sometimes produces. Thus, I know of a family in which the father and each one of six sons has suffered from very troublesome hypertrophy of the tonsils during boyhood, that has ceased to trouble and considerably diminished at and after puberty. This must, however, be regarded as quite an exceptional occurrence. On the other hand, I have to mention the cases of two women whom I have seen by the kind-
ness of Dr. John Williams, in whom the ovaries are believed to be entirely absent or quite rudimentary; in each of these women the tonsils are of the usual size. Dr. Langdon Down informs me that he has never noticed absence or defect in the tonsils in any of the many idiots and cretins that he has examined.

The second question, which is quite distinct, and which admits, I believe, of a definite negative answer, is whether excision of the tonsils causes atrophy or non-development of the testicles. Dr. Shorthouse, who is quoted by Dr. Ogle, speaks of wasting of the testicle following excision of the tonsils as a "familiar fact." In addition to the large opportunities of observation afforded by the frequency of this operation in this country, there is a piece of evidence which appears to be absolutely conclusive; it is, that in Zanzibar all boys have their tonsils excised before puberty, and that atrophy of the testicles is quite unknown there.*

III.—A Case of Pulsating Tumour of the Head with Raynaud's Disease. By Frederick Treves. Read October 8, 1886.

The patient, a printer, aged 17, was admitted into the London Hospital in June, 1886. He was of fair complexion, was much below the average height, was thin and of feeble muscular development. He looked about fourteen years of age. He was well educated and intelligent, and was stated to be a very efficient workman. His family history revealed nothing of note. His father, who died of smallpox, was subject to rheumatism. His mother and his three brothers enjoy perfect health. There is no evidence of inherited syphilis. The lad's personal history is as follows: He has always been delicate. In infancy he suffered from whooping-cough, and later on from measles. When one and a half years old he had a "fit," which lasted thirty minutes. It was followed by a stiff neck that lasted for three weeks, the head being rigidly turned all that time to the left side. He had two more "fits" of a precisely similar character at intervals of six or eight months; on each occasion the fit was followed by a rigid turning of the head to the left side. Since this time the attacks have never recurred. At the age of eleven he had what was termed "low fever." He was confined to his bed for a month. The only symptoms appear to have been great prostration with constipation, occasional sickness, and intense headache.

Two years ago he received a cut above the right mastoid process from a stone. The wound bled profusely; three arteries were ligatured, but no symptoms attended the accident other than the local symptoms of the injury. Eighteen months ago he had a very notable illness. He was suddenly seized with severe pain in the upper part of the dorsal spine. The pain was intense and extended down both upper limbs to the wrist. There was tenderness over the spine, the head was poked forwards, the neck was quite stiff, and the patient was confined to the house for fourteen days. When the attack passed off he noticed that his hands and the lower parts of both forearms were bluish and cold. They have remained in this condition ever since.

Present condition.—He states that he has always been
Mr. Treves's Case of Pulsating Tumour of the Head. 13

liable to headache from time to time. For the last twelve months he has presented the following symptoms, which, he says, are increasing in degree. Almost every morning he awakes with a headache. This is severe and frontal. It extends over the whole of the forehead, and disappears as he moves about. He rarely has headache at other times. Nearly every morning also on getting up he is troubled with retching or vomiting. This symptom he presented during his stay in the hospital. He is not sick at other times. He is often giddy, and then has to lie down. He cannot say that he is giddy in any direction. If he looks up suddenly from his work he is apt to suffer from transient vertigo and to see double. He has never fainted. He frequently "comes over in a hot sweat," especially when dressing in the morning. The sweating is mostly of the head and neck. He eats and sleeps well. All the functions of the body are normally performed.

Head and neck.—Pulsation is visible on the left side of the neck. The carotid is greatly and evenly enlarged, and appears to be about the size of the adult forefinger. The right carotid is enlarged in like manner, but to a less degree. Its pulsations also are visible. Over both vessels is to be heard a very loud booming bruit; this is louder and harsher on the left side than on the right, and in both parts is increased in intensity by pressure upon the vessel. The temporo-facial and internal jugular veins are very large on both sides; they are rendered quite evident by pressure, and the latter is the width of the thumb. They are free from pulsation. Over the great vessels is to be felt a thrill that is continuous, but increased in intensity during each systole. This thrill depends evidently upon the presence of the large veins in front of the dilated arteries. It is more marked on the right side than on the left, and at the lower part of the neck than the upper; it ceases to be felt at a point a little below the angle of the jaw, and can be increased by pressure upon the great veins at the root of the neck. (Pressure here always causes immediate distress; the face becomes congested, the patient feels suffocated and complains of his head being "too full.") The bruit, it is evident, depends also in the main upon the fact that the carotids lie between the dilated veins in front and the spine behind. The lad's neck is very thin. The dilatation of the carotids can only be traced about one inch beyond the bifurcation of the common trunk. With the exception of the left posterior auricular and occipital, no branches of the external carotid arteries on either side are dilated. None of the scalp arteries
can be readily made out with the exception named. Pulsation can be felt but not seen in the temporals. The bruit is most intense at the root of the neck; it becomes much less marked about the angle of the jaw. It can be followed nearly all over the head by keeping to the lines of known arteries. In the skull, however, it is very faint, and on the right side is barely audible. It is evidently conducted, and certainly extracranial. There is no reason to suppose that the dilatation affects the internal carotids.

The face is of normal colour, and the lips show no evidences of anaemia. If he perspires he perspires equally on both sides of the face. The face is not symmetrical, the right side being a little larger than the left. The muscles of the right side act also with greater vigour, as can be seen in frowning, and in screwing up the mouth. The left facial muscles now and then seem to be slow or uncertain in their action, and an occasional movement of the face may be non-symmetrical in consequence. The general expression is peculiar. The lad looks less intelligent than he is, and his face often bears an appearance of distress. The face feels to him in all respects normal, and he says that his cheeks never become flushed.

The right eye is more prominent than the left, and the right palpebral fissure is the larger. The pupils on the first examination were found to be of normal size and equal, and to remain equal when the eyes were shaded. On subsequent occasions the right pupil was found to be a little larger than the left, but this difference in size never became constant. Some very tortuous varicose veins are to be seen on the conjunctiva, and especially in the palpebral folds. The sclerotic is bluish, and its veins large and evident. Mr. Waren Tay, who kindly examined the boy, states that his vision is normal. The vessels of the retinae are somewhat unduly numerous. There is a slight venous pulse in the left eye, and a like pulse occasionally in the right. Apart from this the discs are normal. There is slight nystagmus of both eyes. The eyes do not always move in concert; the left is apt to lag behind. This is especially to be noticed when the left external rectus is used, that muscle being distinctly sluggish in its action. To this uncertain or slow muscular movement in the left eye the occasional transient diplopia may be ascribed.

The left side of the tongue is smaller than the right. When the organ is protruded the tip usually turns to the left. It is equally furred. Palate, pharynx, and tonsils normal. Perfect teeth. He does not complain of dryness of the mouth.
He speaks in a slovenly way, and with the lack of precision often noticed in patients with sore tongues that cannot be freely moved. There are no enlarged glands in the neck or elsewhere.

The pulsating tumours.—There are two of these tumours on the head. No. 1 is situated on the left side just behind the posterior edge of the mastoid process. It is round and flat, and about the size of a half-crown piece. It does not reach to the level of the tip of the pinna, nor extend below the line of the hair. The hair and skin that cover it are normal. Into it runs a large dilated artery that pulsates vigorously. This vessel comes from the external carotid, is thin walled, a little tortuous, and apparently about the size of the common carotid of the adult. It probably represents the posterior auricular. The pulsations in the tumour are very energetic and synchronous with the systole. The tumour is clearly defined, and is obviously made up of tortuous arteries or a single tortuous artery. With it large veins are apparently associated. Over the mass a distinct thrill is to be felt similar to that noticed at the root of the neck. A very loud harsh bruit is also to be heard. This bruit is louder and more rasping than that heard in the common carotid. The tumour can be emptied, and when compressed it is made evident that the bone beneath is marked by a number of irregular channels. These felt like open sutures, but further examination showed that they did not correspond to the normal divisions of the skull. They had no doubt been formed by the long dilated vessels.

Below the posterior auricular artery a largely dilated occipital artery was discovered. It gave a branch to the tumour. The trunk was lost behind the mastoid process. No pulsation could be detected in the terminal branches of the artery, nor, in fact, could those branches be made out.

From the tumour ran one small, tortuous, and pulsating vessel. After a course of about one inch it ceased to be recognisable. It was moving in the direction of the occipital protuberance. It had not grooved the bone, nor had the posterior auricular artery. I believe the grooves in the skull to have been produced by veins congenitally dilated. Pressure around the lower margin of the tumour at once entirely arrested all pulsation, thrill, and bruit.

I consider this tumour to be a cirsoid aneurism, about which are some dilated veins. There is no evidence of arteriovenous communication.
The second pulsating tumour is of a totally different character. It is exactly in the median line, and lies at the back of the head. It is of oval shape; its long axis corresponding with the median line. It measures 3½ inches vertically and about 1½ inches transversely. It is covered by normal scalp. It extends from the occipital protuberance to a point 1½ inches behind the bregma. The tumour is soft and feels like a thin-walled cyst. It presents a feeble pulsation, but no bruit and no thrill. It can be entirely reduced. Its margins are very well defined. Pressure carefully applied all round the edge of the tumour has no influence upon its pulsation. Pressure over the left carotid diminishes the pulse and the bruit in the cirsoid aneurism, but such pressure has no effect upon the median tumour. When the tumour has been emptied a median slit can be felt corresponding to the patent interparietal suture. Anteriorly this ends in a circular gap or hole in the bone, which coincides with the position of the parietal foramen. The lambda can be defined; about 1 inch of the lambdoid suture can be made out on either side of the median line. The tumour extends below the lambda, but no median gap in the occipital line can be defined. That bone appears to be quite normal. The interparietal suture is open from the lambda to the site of the parietal foramen. No arteries can be made out in the vicinity of the tumour.

I consider this median swelling to be a venous tumour communicating with the superior longitudinal sinus and receiving its pulsations from the brain.

The sphygmographic tracings of the two tumours obtained by Mr. Perry, of Cambridge, reveal remarkable differences. To the touch the pulsations in the two tumours did not appear to be synchronous. The tracings show that they are. The sphygmogram of the cirsoid aneurism shows a vertical ascending stroke; the summit of the wave being directly reached. The sphygmogram of the venous tumour shows a feeble wave. The ascending stroke is oblique, and the summit of the curve is reached later than in the case of the arterial tumour. Synchronous tracings show that the summits of the two curves do not correspond in point of time, and it is this fact that explains the apparent lack of correspondence in the two pulses. The tracing of the venous tumour shows the respiratory curve, that of the cirsoid aneurism does not. The sphygmograms of the arterial tumour and of the carotid arteries are very similar.

Just to the right of the median line of the forehead is a
PLATE I, ILLUSTRATING MR. TREVES'S CASE OF PULSAT-ING TUMOUR OF THE HEAD AND RAYNAUD'S DISEASE.
Cirrhotic Aneurism.
1 oz.

Venous Tumour.
1 oz.

Left Radial.
1/2 oz.

Right Radial.
1/2 oz.

Tracings from Mr. Treves' case of Pulsating Tumours of the Head.

Danielsson & Co., lith.
large frontal vein. It is not tortuous, and is lodged in a deep suture-like groove in the bone. There is no left frontal vein. Certain veins behind the external angular processes of the frontal bones have led to distinct irregular grooves on the bone. No grooves can be felt elsewhere. No sutures can be detected other than those named. The sutures at the bregma and pterion cannot be made out. The skull is of perfectly normal outline, and exhibits no abnormalities other than those described. The temperature in the external auditory meatus is the same on the two sides.

The patient hears a blowing noise in the head, but only when lying down and when lying upon one or other ear.

*The upper limbs.*—Both hands are cold and purple. There is no oedema and no thickening of the tissues. The nails are of normal outline and well shaped. The forearms are purplish, but the discolouration is more in the form of mottling. The limbs become of normal aspect as the shoulders are approached. The lower parts of the arms are a little dusky, but the upper parts are of normal appearance. Both limbs are exactly alike. The lad has had chilblains upon the fingers every winter since he can remember. He has never had chilblains on his feet. He says his hands are always cold, and that they perspire a great deal.

The temperature of the hands varied from 85·5° to 92·5°. The right hand usually showed a lower temperature than the left. The main vessels of the upper limbs appear to be in every respect perfectly normal. The sphygmograms of the two radials are identical. The lower limbs are of normal aspect.

*Chest,* &c.—Dr. James Anderson kindly examined the boy's chest, and made this note: "There is a faint apex systolic murmur with reduplicated pulmonary sound, but otherwise there is no abnormality in the heart signs. A diffuse venous hum is heard over the manubrium and neighbouring ends of the clavicle."

All the vessels of the body that were accessible to examination were examined. They all appeared to be normal. The temperature, as taken in the axilla, was always normal.

The dates given for the commencement of the various local symptoms are as follows:

So long as the boy can remember he has been aware of a throbhing in the neck. It has become worse during the last few years. A blowing noise in the head was first heard by the patient about a year ago. The median venous tumour was discovered by accident when the lad was eleven years of age.
The cirrhotic aneurism was first noticed twelve months ago. The blueness of the hands appeared about eighteen months before admission.

The boy improved during his stay in the hospital, probably benefiting more from the rest involved than from the treatment; galvanism and faradisation produced no change in the condition of the upper limbs; of the many drugs administered, one only appeared to be attended with the least benefit—digitalis. The boy remained in the wards about two months, and when he left had had neither headache nor sickness for some weeks.

The interpretation of this case must be a matter of speculation. There is every reason to believe that there must have been a congenital enlargement of certain of the veins about the skull. The deep grooves that these veins had made in the cranial bones support this view, and lead to the supposition that they must have existed as large, tortuous, varicose trunks at a period when the skull was comparatively soft. The median venous tumour is also probably congenital. The lad did not notice it until he was eleven years of age, but its manifestations are even now so little marked that it may well have escaped observation. A dilated condition of the emissary veins connected with the superior longitudinal sinus would account for the breach in the skull near the lambda, and for the communication that now exists between the extra- and intracranial venous channels. A like communication may possibly exist over the mastoid process and be obscured by the violent pulsations of the enlarged artery in that site.

The dilatation of the carotids is not easily accounted for, especially as the dilatation did not extend to the branches of those arteries, except in the two instances named.

Evidences of some paralysis of the left cervical sympathetic are afforded by the narrowing of the palpebral fissure, the retraction of the globe, the occasional contraction of the left pupil, the wasting of the left side of the face. There has been no unequal congestion of the face, no unilateral sweating, and no difference was ever noticed in the surface temperature of the two sides.

The local asphyxia in the upper limbs may have some connection with the vascular changes in the head and neck, or the relation between these two phenomena may be purely casual. The veins of the upper limbs were in no way dilated, and the blueness of the parts appeared to be due simply to a loss in the vis a tergo. It is significant that the lad had been
liable to chilblains of the hands practically all his life, but that
the permanent blueness of the upper extremities was a recent
condition. This symptom followed an attack of severe pain in
the upper dorsal and lower cervical spine, associated with
pain in the arms. The latter symptom has accompanied
certain cases of Raynaud's disease, and at least one instance
has been recorded (Vailland) in which the trouble in the
extremities was ushered in with tenderness and pain in the
lower cervical spine. It would be difficult to assume on anato-
mical grounds that the narrowing of the arteries in the
upper limbs was compensatory to the enlargement of vessels
in the head and neck. If there were a left as well as a right
innominate artery, the assumption could be more reasonably
advanced. It may be noted, however, in this connection, that
the right hand was generally colder than the left.

In the place of further speculation, it may be well to call to
mind the anatomical conditions of some parts of the sympathetic
as they have been laid down by the most recent writers. The
vaso-constrictors of the head arise from the first three dorsal
nerves, and pass through the inferior and superior cervical
ganglia, and the cervical sympathetic cord. The vaso-con-
strictors of the arm are derived from the first seven dorsal
nerves, and can be traced to the inferior cervical and upper
thoracic ganglia, and to the thoracic sympathetic cord.

The vaso-dilators of the bucco-facial region come from the
second to the fifth dorsal nerves, and pass by way of the first
thoracic ganglion and the cervical sympathetic cord. Those
of the head, eye, and ear come from the eighth cervical and
first dorsal nerves, and pass through the same ganglion. The
vaso-dilators of the arm are traced into the upper thoracic
sympathetic cord, which they reach from the eight cervical and
five upper dorsal nerves. Upon anatomical grounds, therefore,
it may not be difficult to connect together the symptoms pre-
sented in the case, viz. vascular disturbances in the head,
neck, and upper limbs—lesion of the cervical sympathetic—
pain in the lower cervical and upper dorsal spine.

The chief surgical interest in the case concerns the venous
tumour. So far as I can ascertain, that variety of tumour was
first described by Percival Pott in 1808.* The case was that
of a boy who received a blow upon the head, that, however,
produced no wound. In time a soft tumour formed about the
size of a walnut, and presented "a dull kind of pulsation." Pott
cut down upon it—although it appears to have occasioned

no symptoms—and discovered a cyst filled with venous blood communicating with the superior longitudinal sinus. "It was found that the sagittal suture was broken, and that a portion of the fracture was forced into the sinus." The skull at the site of the injury was trephined, and the fragment removed. The lad made a perfect recovery. Many years later Dufour* and Hutin† published like cases with autopsies. Both the patients were soldiers who had received injuries to the head in battle. Subcutaneous venous tumours resulted, but caused no trouble, and the patients, after many years, died of other causes. In both cases holes in the skull were found at the seat of injury that communicated with the superior longitudinal sinus.

In 1858 Dupont‡ published the first systematic account of this species of tumour. He recognised that the majority were due to injury, but that some were of spontaneous origin. He considered that the communication between the sinus and the external tumour may be due to a varicose emissary vein.

It appears that some years previously Chassaingac had cited the case of a child who presented this latter condition, with the result that a tumour existed which swelled when the patient cried.

Since Dupont's time many examples of this affection have been published, but mainly by French surgeons. The tumour appears to have been overlooked by the writers of English text-books.

Certain of the reported cases were congenital, while many occurred in children. In not a few instances the nature of the case is open to considerable doubt.§ The best modern account of the condition is given by Gayraud in his article "Crane" in the Dictionnaire Encyclopédique, 1879.

Taking the cases generally, the following facts may be noted in connection with them. In the majority of cases the tumour followed an injury. In the non-traumatic cases some were congenital while in some the tumour appears to have developed upon congenital holes and fissures in the skull or over imperfectly closed sutures. In many instances the communication appears to have been brought about by atrophy, and possibly through the agency of an emissary vein. In other examples a "rarefying osteitis" is reported to be the

† Mém. de Méd. Militaire, Paris, 1854.
‡ Thèse de Paris, 1858.
cause of the opening in the skull, and the bone affection considered to be akin to that met with in cases of pneumatocele. (This affection of the bone is probably that now better known as caries sicca.) In every example the communication was with the superior longitudinal sinus. The gap in the bone was sometimes single, sometimes multiple, or the communication was effected through many minute holes. The larger apertures were round or irregular, some appeared as suture-like gaps, and others as foramina. Dr. Ogle* records a case in which the abnormal channel appeared as a hole in the occipital protuberance that carried an emissary vein leading from the torcular Herophili to the subcutaneous cyst. Some patients suffered from headache or vertigo, or experienced distress when the swelling was reduced. In the majority the tumour caused no trouble even when handled. The frontal region was most commonly involved, next the parietal, and only in a very few cases the occiput. The tumours were small, rounded, flattened, soft and fluctuating. Some exhibited a feeble pulsation. Many increased in size during expiratory efforts, and when the head was dependent. In none was any bruit observed. All were reducible either completely or in part. In the present case it is probable that certain of the cranial veins were congenitally dilated, that this condition affected the emissary veins about the posterior part of the superior longitudinal sinus, and that as a consequence the parietal foramen was enlarged, while the sutures about the lambda were unable to close.

[Since this paper was written a very exhaustive account of these venous tumours has been written by Dr. Wm. Mastin. The paper is entitled "Venous Blood Tumours of the Cranium in communication with the Intracranial Venous Circulation." The paper appeared in the Journal of the American Medical Association (Sept. 25, 1886), and concludes with a very full bibliography.]


The following notes will serve to make the account of this case as complete as can be desired.

On the 3rd of February I received a letter from the patient's father, who, it will be remembered, was a medical man, which contained the following passage: "About a fortnight ago he had a good deal of pain in the left loin, very much as he used to have in the right: I found he was passing small pieces of calculus, soft and friable, and that the urine was alkaline. I kept him very quiet for a few days and gave him nitro-hydrochloric acid. The wound is now entirely healed, the urine normal, except a little mucus occasionally, the pain in the loin quite gone, and I am happy to say he is progressing most satisfactorily."

In December, 1882, I received a note from the patient himself to say that he felt impelled to write to tell me that at the end of a year after the day he left the hospital he was quite sound and in excellent health.

Again, on the 15th of September of the present year, 1886, I received a letter from the father, saying, "You will be pleased to hear that my son, who was operated on for calculus in the kidney, has remained very well ever since he left the hospital in 1881. He is now in Africa, and writes to say how well the hot climate suits him."

The case is thus complete for five years after the operation, and it is satisfactory to be able to report the good health of the patient during these five years. But there are other reasons which render this note important. It has been more than once proposed that, in cases similar to this, the entire kidney should be removed, instead of limiting the operation to the extraction of the stone. The history of the case shows, not only that the right kidney, from which the calculus was removed, has exhibited no signs of disease since the recovery from the operation, but that there were for a short period distinct symptoms of affection of the left kidney. Supposing this patient had been deprived of the right kidney at the time
of the operation, he would therefore not merely have been deprived of an organ which has been probably ever since a useful and working organ, but might have been exposed to the gravest danger during the attack of calculous disease of the left kidney.

My own feeling has for a long time past been in favour of making a more determined effort in cases of calculous disease to save the organ, even when it has been the seat of dilatation, and when the dilated parts have been partly filled with tiny calculi contained in soft, pulpy, green material, such as I have several times seen. It is quite possible that such a kidney may so far recover as to be a useful organ, although perhaps not such an efficient organ as the other kidney. In case of affection of the other kidney, which has been noted in several instances, this partly effective organ may serve to excrete a sufficient quantity of urine to enable the patient to tide over what would otherwise be a fatal period.
V.—A Case of Removal of the Scapula for Osteo-sarcoma.

By CHARTERS J. SYMONDS, M.S. Read October 22, 1886.

ROBERT W., æt. 34, carpenter, was admitted into Guy's Hospital March 26th, 1884, under my care, with a tumour of the scapula. The man came of a healthy stock in which there was no history of cancer. Five months before admission he fell a distance of eight feet on to his right shoulder, the skin was grazed and a little swelling followed, but there was no discolouration. He attended the Croydon Hospital for a month, where he was told that he had ruptured a muscle. A month after the accident an enlargement appeared at the seat of injury, and has steadily advanced to the present dimensions. With regard to this history, it appeared that the injury was not a severe one, and it seems probable that the swelling detected at Croydon was really the commencing sarcoma. The man gave but few details, and seemed to take but little notice of his complaint.

On admission he was a healthy looking man, but states that he had, during the last month, lost flesh. Connected with the posterior surface of the right scapula was a hard growth with a smooth though somewhat lumpy outline. It measured nine inches from side to side, ten inches from above downwards, and projected five inches from the surrounding surface. It did not appear quite to reach the glenoid cavity, and moved with the scapula freely over the chest wall. The spine of the scapula could just be traced and the acromion was free, while the inferior angle of the bone was concealed.

On April 1, the entire scapula with the exception of the acromion and coracoid processes was removed. Mr. Poland compressed the subclavian. An incision was made across the top of the scapula from its posterior border over the acromion process, and from the centre of this a vertical one was carried downwards to the inferior angle. The inner triangular flap was reflected, and the posterior border of the scapula freed by dividing the muscles. Here the posterior scapular artery was seized with forceps and divided; the lower angle was next raised up, and an attachment of the latissimus dorsi severed. The hand was passed under the bone so as to separate it as far as possible. By another vertical incision a wedge-shaped
piece of skin one inch wide above was left attached to the growth. The outer flap was now turned outwards and the axillary border freed; the dissection here was conducted entirely by the fingers until the subscapular artery was reached. This vessel was seized and divided between two pairs of forceps. The teres major was divided close to the bone, the spine and acromion process now were freed and the latter sawn off, the deltoid and trapezius being first detached. The upper border was cleared, and the supra-scapular artery taken up with forceps. It was now found that the growth reached the glenoid cavity, the exact position of the shoulder-joint was ascertained by rotating the humerus, and then the muscles passing from the scapula with the capsular ligament were divided. Next the coracoid process was sawn off, with a small piece of the superior border of the bone; the scapula was then raised up, the serratus magnus divided, and the entire mass removed. The vessels now in the grasp of forceps (together with a few muscular branches) were secured with catgut. The parts were brought together by wire sutures. The entire operation was conducted under a carbolic spray and the usual antiseptic dressings applied.

The man lost very little blood, as all the vessels were taken up before being divided, the compression of the subclavian, moreover, added to this result.

After being put to bed he became restless and was unable to lie down, the pulse was strong, at 100—104. This feeling of oppression lasted during the next day, but no explanation was found for it. A considerable serous oozing took place, and the next day he was feverish.

To summarise the report of the case it may be noted first that a rise of temperature took place almost immediately, and lasted some time, gradually assuming hectic characters. That with this there was no tension in the parts, no tenderness but only free suppuration and oedema of the arm. On April 24, I made a counter-incision in front, as the pus seemed to collect in that part of the capsule. This was followed by so little relief that on May 9 I cut down and removed the whole of the articular surface of the bone, which appeared to be the cause of the prolonged suppuration; and so it proved to be, for the cartilage was gradually suppurating away. The improvement was immediate, he was up in a few days, and on May 31 it is noted, "he is quite fat, and the wounds just healed, and there is no sign of secondary deposit."

The leading points calling for attention in the above case
are, first, the operation. The scapula has been removed now several times, and most frequently from young people. The incisions selected in the present case being those usually adopted, seem to give free access to the scapula, and by proceeding to clear first the posterior border, then the inferior angle, next the axillary border, and finally the upper, securing the vessels with forceps before their division but little blood is lost and the time is shortened as much as possible. Compression of the subclavian artery materially assists in checking the haemorrhage. It is to be noted also, that the head of the humerus was left; and it proved afterwards that this gave rise to prolonged suppuration. While admitting that articular surfaces may frequently be left, the cartilage becoming closely connected with the fibrous structure, and primary union result, still in such patients as this it proved an unwise proceeding, and for reasons given later on I think it would have been better to have removed all the cartilage, and this I have done in all subsequent amputations involving the joints.

The pyrexia next calls for attention. The temperature rose the same evening, and, with remissions, continued till the head of the bone was removed. The following is the record:

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<td>April 1</td>
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The temperature never fell to normal till after the second operation on May 9th, which point it reached in three days.

With this fever there was profuse suppuration, the pus thin and watery. There was free sweating also, and once or twice diarrhoea.

This fever may, I think, be called traumatic at first, passing later into hectic. It is not usual, as is well known, under the antiseptic methods to see this fever, and from its occurrence in two other cases, it would seem that it is due to some peculiarity on the part of the patient, and that in them suppuration is unavoidable. The man's urine was healthy, and there was no evidence of carbolic absorption.

In connection with this fever were the suppuration and the oedema of the arm. The oedema was noticed about the elbow on April 8th, and continued till the removal of the head of the bone. The swelling seemed entirely passive, and was un-
attended with redness, but the enlargement was great. Thrombosis of the axillary vein seemed the most probable cause of the swelling, but it was difficult to see why this should occur, especially as there was no subclavicular swelling, no redness round the shoulder-joint, and no oedema here. The pus I thought came from the shoulder-joint structure, and so it proved in the end. Finding that no relief followed an incision in front down to the head of the bone I finally excised this part of the humerus. On exposing it through a vertical incision nearly all the cartilage had disappeared, the articular lamina was hard and bare, and was beginning to be separated by granulation tissue, which was creeping under its edges, and had in a few places perforated it. The articular surface was removed by Gowan's osteotome, an instrument which cut through the bone in a few seconds without inflicting the slightest injury on surrounding parts. Rapid improvement followed, and in a short time the wounds were all healed.

There seems no doubt that to the presence of the articular surface and the synovial membrane was due the prolonged suppuration, and, as suggested above, I am inclined to think that had it not been for the traumatic fever suppuration might have been absent. Still, I would ask, is it not wiser to remove all cartilage in such operations together with the articular lamella? I have had the opportunity of dissecting more than one stump after the operation known as Stokes-Gritti, in which prolonged suppuration existed, and I have found that the pus was derived from a small part of the articular surface, more or less denuded of cartilage, the articular lamella itself having necrosed.

The tumour on section showed that it grew from the infra-spinous fossa. The bone was nearly destroyed, the growth appearing on both aspects. Spicules of new bone radiated from the centre. The chief components are fibrous tissue and cartilage. The cells of the latter are round and rapidly proliferating. The softer parts show round sarcoma cells. It is therefore one of the slow growing and least malignant forms.

Result.—When seen in December, 1884, the man remained in good health, and there was no sign of recurrence. He possessed, unimpaired, all the movements of the elbow, and could carry his arm forwards and backwards from the shoulder, but could not raise it at right angles from the trunk. He could raise the shoulder, and carry it back, the rhomboidei acting well, having obtained apparently an attachment to the fascia and skin. The humerus could be fully rotated without giving
him pain. The man had done some work since his discharge, but has made no special effort to develop his muscles, and therefore had not obtained the full usefulness of his arm. He could drive screws and nails, and do light work, but found himself unable to use a plane.

Further Note, October 22, 1886.—The man was exhibited to the Society, it being now rather more than two and a half years since the operation. He was in good health and had a useful arm, being able to do all the lighter work of a carpenter, including the use of a plane. Overhead work he could not do.
VI.—Ligature of the Common Iliac Artery with Kangaroo Tendon; yielding of the Knot; Re-ligature with Silk; Gangrene of the Limb; Death. By Thomas Smith. Read October 22, 1886.

G., aged 52, was admitted under Mr. Smith's care into St. Bartholomew's Hospital, October 14, 1885. He was suffering from an aneurism of the external iliac artery on the right side, which extended from a point midway between the umbilicus and crural arch into the upper part of the thigh; it pulsated very strongly; there was no bruit, and the pulsation could be commanded by firm pressure on the vessel above the aneurism.

The venous circulation on the same side was much obstructed, the right thigh and leg measuring respectively 22 and 15 inches, while on the left side they measured 19 and 15 inches.

The patient was a soldier and had served in India; he had syphilis thirty years ago; he had suffered no injury. He has been aware of some troubles about his groin for two years, and for the last three months his thigh and leg had been getting larger. In other respects he was a healthy man; the urine was free from albumen and sugar.

On October 22 the artery just above the aneurism was ligatured at one spot with a kangaroo tendon, which was very firmly tied; no room could be got for a second ligature or one would have been applied and the vessel divided. The vessel at the point of ligature was very large, but appeared healthy. It was erroneously supposed to be the external iliac. Pulsation in the aneurism ceased at once; the ligature was cut short, a drainage established, and the rest of the wound closed.

October 23.—Pulsation had returned in the aneurism, and was as forcible as before the operation.

A consultation was held, and it was agreed to reopen the wound and religature the vessel. This was done, carbolised silk being used, and two ligatures were applied, one just above and the other just below the site of the former ligature. The knot of the first ligature was found in situ and not obviously loose, but allowing the point of an aneurism needle to be
Mr. Smith’s Case of Ligature of the Common Iliac Artery.

hitched between it and the artery. The silk ligatures were cut short and the wound dressed as before. Pulsation in the aneurism at once ceased.

October 24.—No pulsation in the sac, leg warm but darkish in colour.

October 25.—Pulsation evident in sac, leg mottled and purple, pain in foot; wound doing well; later in the day the leg became colder and insensible; thigh discoloured.

October 26.—Pulsation in the sac increasing; condition of the whole limb much improved as regards colour and temperature.

October 27.—Pulsation in the sac continues, a slough forming in the calf.

October 28.—Pulsation continued in sac, leg and foot becoming blue. Pulse 120, temp. 103°.

October 29.—Much the same.

October 30.—Gangrene seems limited to foot and front part of leg; sac still pulsates.

October 31.—Pulsation in sac less, and signs of a line of demarcation; patient’s general condition a little better; temp. 102°.

During the next two days the line of demarcation became evident, and on November 2nd amputation was performed just above the condyles.

The sac had now ceased to pulsate; at first he seemed to rally after the operation, but next day he began to sink, and he died November 3rd.

Post-mortem Examination.—No general peritonitis, but some matting of the coils of intestines in the immediate neighbourhood of the wound. The common iliac artery was found to have been tied just above its bifurcation with two silk ligatures; the vessel was closed by clot for half an inch above the upper ligature. The external iliac artery was dilated in the upper third; its lower two thirds formed a large fusiform aneurism, which extended beneath Poupart’s ligament and involved the upper part of the common femoral. The internal iliac was closed by clot for 1 inch below the ligature. The common femoral was filled with dark, adherent clot, and the upper two thirds of the superficial femoral was in the same condition; the profunda was filled with clot. There was half an inch of clot in the popliteal just above its ligature in the stump; above this point it was pervious.

The sac of the aneurism was quite filled with firm clot, which was laminated in parts.
Veins.—No vein had been injured in the operation. The common iliac was in a normal condition. The external iliac was natural down to Poupart’s ligament; at this level it seemed to have been compressed by the sac, and here it was occluded by a dark adherent clot, which filled the common femoral and the profund. The superficial femoral vein was plugged in places; here and there its canal was free. The small venous radicles of the main veins were generally plugged. The internal saphena was empty, except where it joined the femoral. Here it was plugged.

The fatal issue of this case was due primarily to compression of the iliac vein beneath Poupart’s ligament by the sac of the aneurism, leading to gangrene of the limb—one of the most common causes of death in this form of aneurism.

I have ventured to bring the case before the Society as being, so far as I know, the first instance recorded of the yielding of a tendon ligature when applied to a large artery.

It is certain that at the time of its application the ligature completely arrested the circulation. It is almost certain that within twenty-four hours it had so far relaxed its grip of the vessel as to permit pulsation in the aneurism; at all events the aneurism did pulsate freely, and on examination, though there was no appearance of a loosened knot or relaxed ligature yet the point of an aneurism needle could be passed between the ligature and vessel.

Though we were probably correct in assuming that so speedy a return of pulsation indicated that there was some failure of the ligature, yet I would point out that in two days after the second operation, when silk ligatures were used, the pulsation had returned in the sac; and in this instance it is known that the ligatures did not yield.

Granting that the kangaroo-tendon ligature did in some way cease to constrict the vessel, the question arises whether the material of the ligature was in fault or the method of its application. It was reasonably suggested by someone present at the operation that the ligature may have been applied obliquely to the course of the vessel, and that it subsequently assumed a transverse direction and thus lost some of its constricting power. So far as I am a judge the ligature was not passed in this manner, but I believe that had it been so applied it would, on the tightening of the ligature, at once have assumed a direction transverse to the course of the vessel.

The ligature was tied, as I believe, in a reef-knot, and a
third knot was added for security. It certainly was tied very tightly, but it is quite likely that at the bottom of so deep a wound, as there was in this case, there might have been some springing open of the first knot before the second could be tied; still, even if this did occur, it was not sufficient to prevent the ligature from immediately arresting all pulsation in the sac.

I believe the explanation of the accident must be sought chiefly in the slippery nature of the ligature. The one used was a perfect specimen of a kangaroo tendon, highly polished, glistening, and absolutely free from any fraying of its surface; it had, as I believe, no sufficient roughness to be held by the ordinary knot, when considerable constriction was required. The artery tied was large and needed considerable compression to obliterate its lumen and it is more than probable that the knot gradually yielded to a small extent after it had become soaked in the fluids of the part; as a knot it certainly neither came undone nor showed any external signs of loosening.

I take this opportunity to suggest for the consideration of the Society that the ordinary method of applying a ligature to a large artery is not all that could be desired. The object should be to constrict the vessel evenly, at all points of its circumference, by a ligature which should be a perfect circle and should not compress the arterial coats more at one point than another. I submit that the ordinary single ligature tied in a reef-knot does not accomplish this. The ligature thus applied is not a perfect circle and it does not press evenly on the coats of the vessel. On the contrary, the knot dints into the arterial coats, and should the ligature divide the vessel the knot is generally the first part to penetrate the canal of the artery, and in cases of secondary haemorrhage the situation of the knot is often the point from which the blood issues.

I venture to think that these considerations derive additional importance from the publication of Dr. Edmunds’ and Mr. Ballance’s paper,* in which it is proved that injury to the arterial coats is neither necessary nor desirable in order to secure a final, complete obliteration of the lumen of the vessel.

With the object of securing uniform constriction of the artery by a ligature, which will lie perfectly evenly on the arterial wall, I beg leave to recommend that the ligature be passed twice round the artery and be drawn tight as a clove-

* "The Ligature of the Larger Arteries in their Continuity."
Mr. Smith's Case of Ligature of the Common Iliac Artery. 33

hitch or knotted so that the knot falls on the first circle of the ligature and not on the vessel itself.

A clove-hitch will be found to answer perfectly without any knot at all, and this has the great advantage of being easily drawn tight even at the bottom of a deep wound. It is an exceedingly tenacious form of ligature, there is no knot, and to fasten it one only needs to draw the opposite ends of the thread away from one another as in fastening a knot.

It may be objected that to pass a ligature twice round needs a second introduction of the aneurism needle, but it is a matter of no difficulty. Of course, to pass a ligature twice round an artery the aneurism needle must be introduced a second time, unthreaded, and be again threaded with the end of the ligature which lies furthest from its point. This is a matter of no difficulty with a ligature lying beneath the vessel.

A clove-hitch fastened in this way forms a complete circle, exercising uniform compression on all parts of the circumference of the vessel.

Notwithstanding the accident which occurred in this case I still believe antiseptic kangaroo tendon to be the best ligature we possess at present for the tying of large arteries in continuity, and I believe that there is no chance of its slipping if it be secured in the manner I have recommended. I believe also that it may be safely trusted to hold as a single ligature secured by the reef knot, even in a very large artery, if the tendon be roughened on its surface by lightly scraping with a blunt knife.

It is beyond the scope of this paper to discuss questions of treatment, or to enter fully into the comparative merits of silk, catgut, and tendon as ligatures for large arteries.*

But on the question of treatment I may be allowed to say that I should have hesitated to reopen the wound to tie the artery a second time had I been in possession of the information contained in Dr. Edmunds' paper, namely, that when an artery is nearly occluded by a ligature plastic processes supervene which permanently block the lumen of the vessel.

* This is exhaustively dealt with in Dr. Edmunds' and Mr. Ballance's paper.
VII.—A Case of Axillary Aneurism, for which the Subclavian Artery was ligatured in the third part of its course; Slipping of the Ligature shortly after the Operation; Re-ligature; Collapse; Death. By H. T. Butlin. Read October 22, 1886.

W. M., set. 52, a distiller's carman, was admitted into St. Bartholomew's Hospital under my care on January 25, 1885. He had been in his usual state of health and activity until three months previously, when he first noticed a painful swelling in the left axilla. He attributed it to his habit of driving a pair of hard-mouthed horses about thirty miles daily, always with the left hand, and to the use of the left hand in rolling heavy casks and performing the most laborious parts of his duties. He was considered a sober man, but was in the habit of drinking a great quantity of beer. He had suffered from syphilis about thirty years ago, and from rheumatic fever six years ago.

No treatment had been adopted for the swelling, which had become larger and more painful.

On admission, there was a pulsating circumscribed swelling in the situation of the third part of the axillary artery, with a bruit, but without thrill. Although the swelling was circumscribed, the whole length of the axillary artery, as well as could be distinguished beneath the pectoral muscles, appeared to be larger than natural, and the subclavian artery was thought to be larger and to pulsate more forcibly than the artery on the right side. The arteries at the wrist appeared more tortuous than natural, but not rigid. There was no obvious sign of affection of the heart or of any of the internal organs.

A consultation was held, and the opinion was general in favour of ligature of the subclavian artery in the third part of its course.

The operation was performed on February 6. The artery was easily and quickly exposed, but there was difficulty in separating it from its sheath, which led to the belief that it was not a very healthy vessel. It was of very large size. It was ligatured with what appeared to be a very good piece of kangaroo tendon. A second ligature of the same material
was placed around the artery, about two thirds of an inch or more below the first, and the vessel was divided between the ligatures. Retraction of the divided ends took place to the extent of about half an inch. The retracted ends appeared, not only to myself, but to Mr. Marsh and Mr. Walsham, who were kind enough to assist me, to be thoroughly secured by the ligatures. The wound was closed, and the patient sent back to bed.

About half an hour after the operation I was summoned in a great hurry to the ward from the committee-room. I hastened across the hospital square, and found the patient bleeding from the wound, but the bleeding was partly controlled by the pressure of the thumbs of the house surgeons. With their help, and also assisted by Mr. Bowlby and Mr. Morrant Baker, who came from the opposite ward, the haemorrhage was got under control. The ligature of the proximal end of the artery was found loose in the wound, but with the knot still tied, as if it had slipped over the end of the divided vessel. The ligature of the distal end was in its place and secure. A silk ligature was applied on the proximal end; and, for greater safety, a second silk ligature was placed on the distal end. In spite of the rapidity with which aid was forthcoming, the loss of blood was extremely large before the ligatures were applied. The patient was anæmic and collapsed, his temperature fell to 95°, and he died about six hours after the operation.

I learned that the haemorrhage had followed immediately on a violent fit of coughing.

At the autopsy, it was found that both the subclavian and carotid arteries were very atheromatous and somewhat dilated. The ends of the divided subclavian were firmly secured by the ligatures. The aneurism of the axillary artery was fusiform and extended from immediately below the seat of ligature for a distance of 5 inches along the vessel. It contained a very small quantity of old, as well as some soft recent clot.

The heart was hypertrophied and its walls were fatty; the aorta was much dilated and atheromatous. There was no other organic disease.

Remarks.—Two reasons have induced me to bring before the Society this unsuccessful ligature of a large artery: first, the use of kangaroo tendon; second, the division of the artery. As these questions are still sub iudice, every circumstance, which can throw light on them, is deserving of attentive consideration.
The piece of tendon with which the artery was tied was flattened, but so little flattened that it might be described as almost round. It was thicker than thick silk. The force with which it was applied was not sufficient to divide the middle and internal coats of the vessel, yet it appeared to be very firmly fixed. My impression is that it did not merely slip off the end of the vessel, but was pushed off in the following manner:—In the retraction of the upper end of the divided artery, the knot of the ligature rested against the outer border of the scalenus anticus muscle, so that it is possible that very slight pressure was exerted in such a manner as to edge it over the end of the artery after every pulsation of the vessel and during the movements of respiration. Then there occurred a violent paroxysm of cough, during which still more forcible pressure of the same kind was exercised. Had a silk ligature been used so as to divide the middle and internal coats of the artery it is probable it would have resisted this pressure, and the catastrophe might not have happened.

I do not think this case can be used as an argument against the division of the artery. The accident of the slipping of the ligature might certainly not have occurred, and could not have occurred in the same manner, if the artery had not been divided. But the character of the material employed for the ligature had undoubtedly so much to do with the slipping that the blame must be thrown on the use of the tendon.

It is probable that the question of the best material for the ligation of large arteries will remain still for a long period unsettled, and that the second question of the desirability of dividing the artery when it has been tied in its continuity will also find warm advocates and as warm opponents. But I think the lesson taught by this case is clear,—namely, that the two things, the tendon ligature and the division of the artery, should not be used together.
VIII.—A Case of Total Excision of the Larynx for Epithelioma; Death from Exhaustion on the eighth day after Operation. By Henry Morris, M.B.

Read November 12, 1886.

Charles Henry L., æt. 59, a judge's clerk, began in June, 1882, to lose his voice. For eleven months he was under Dr. Habershon's treatment and subsequently was seen by Mr. Durham, who on laryngeal examination detected two small abnormal "specks" in the right vocal cord and proposed an operation for their removal; but it was not allowed by the patient to be carried out. After this he was under the care of Dr. Morell Mackenzie for fourteen months. In February, 1885, he was admitted into the Throat Hospital, Golden Square, for tracheotomy, as his breathing had become most difficult. After the operation he had bronchitis, and remained in the hospital altogether two months. For a time he felt relieved by the tracheotomy, but before many weeks paroxysms of dyspnœa occurred, and rapidly increased both in severity and frequency.

In July, 1885, five months after tracheotomy had been performed, I was asked to see him in consultation with Dr. Walker, of Putney. He was then confined to his room, in a very feeble condition, looking much older than his years, with a very livid face, the lividity being increased from time to time by a bronchitic cough attended with troublesome expectoration. He was wearing a tracheotomy tube which, though requisite for respiration, was itself a source of serious irritation. It had of late become tilted by the pressure of the downwardly increasing growth within the larynx, so that the upper part of the flange pressed deeply against the skin, and the lower border projected far away from the surface. On trying to correct this displacement a distinct resistance could be felt against the upper surface of the tube.

The paroxysms of dyspnœa and coughing were so severe that he seemed in imminent danger of death at any moment. He lived, indeed, alternating between the dread of these attacks and a state of impending suffocation while they lasted. On examination with the laryngoscope the nodular outline of a growth filling the whole of the upper aperture of the larynx was seen. In considering what could be done for his relief
there appeared to be two courses open: one was to prolong the tracheotomy incision downwards so as to put the tracheal tube beyond the reach of pressure by the growth; the other was to lay open the larynx and, if possible, to remove the growth from within, and if not to excise the larynx in part or entirely. Even if the first plan could give relief for a time, it seemed more than probable that by extension of the growth a similar interference with the tube would soon again occur; and as the patient had been prepared for the second alternative I decided on adopting it. For this purpose he was transferred to the Middlesex Hospital on July 8, 1885. Mr. Hensman, who kindly assisted me at the operation, now examined the larynx and made the drawing of its appearance. On July 11, ether was administered by Mr. Davis, at first by the mouth and tracheotomy tube, and afterwards through Trendelenburg's tampon tube. An incision 3 inches in length was made commencing just above the upper border of the thyroid cartilage and continued to within a few lines of the tracheotomy orifice. A few small vessels were secured by pressure forceps; and then the calcified thyroid cartilage was split with Salmon's shears. The halves of the thyroid were held aside with retractors, and the interior of the larynx was found to be stuffed with a very vascular papillomatous-looking growth which in places had eroded the thyroid cartilage, and which could not be removed from within the larynx. Excision of the larynx was therefore proceeded with by prolonging the skin incision into the tracheotomy opening and then peeling off the perichondrium and overlying fascia and muscles from the front and sides of the thyroid and cricoid cartilages, by means of the scalpel and a raspatory. This was accomplished without any bleeding. Before commencing to divide the trachea from the cricoid cartilage I distended the tampon surrounding Trendelenburg's tube just sufficiently, as I thought, to render the lungs secure against the entrance of blood. Instantly thereupon a violent expiratory spasm was excited which lasted several seconds and would, I feel sure, have proved fatal had I not released the air from the tampon. A second attempt to employ the tampon was followed by precisely the same effect. I therefore resolved on trusting to the careful use of fragments of sponge to suck
up any blood which flowed on section of the tissues between the first ring of the trachea and the cricoid. By the time this division was completed a good deal of air had entered the lungs and the patient was commencing to regain consciousness; but by inserting into the top of the trachea a piece of large-bore gutta-percha tubing fitted to the end of a bent leaden pipe the anaesthetic could be freely administered at a considerable distance from the field of our manipulations. A stout ligature was passed on each side between the first and second rings of the trachea by which we had complete control of the upper end of the trachea. In this way the operation was continued and completed without any important escape of blood into the lungs or any further difficulty in the administration of the anaesthetic; indeed, the facility with which the rest of the steps of the operation were effected was due to this arrangement of the leaden pipe which the perusal of the late Dr. Foulis's paper had led me to have in readiness.

By keeping close in contact with the cricoid and thyroid cartilages the soft structures were entirely peeled off from them, and then the oesophageal mucous membranes and submucous tissue were separated from the larynx, whilst the cartilages were pulled forwards and partly rotated from side to side. Finally, the thyro-hyoid membrane was divided and the whole of the diseased mass removed.

The oesophageal wall was nowhere button-holed. At one spot on the right side the disease was found to have honey-combed the entire thickness of the thyroid cartilage, so that it was thought best to thoroughly scrape away the perichondrium of this part from the adjoining tissues.

No tube was left in the trachea after the operation, but the silk ligatures which had been used for steadying the upper end of the trachea were fixed by strapping to the skin of the neck. Iodoform was puffed into the wound, and a nurse was specially charged to remove from the wound with antiseptic sponges the mucus as it was coughed up from the bronchi or collected from the mouth. Microscopical examination showed the growth to be a typical epithelioma, the bird-nest bodies being specially numerous and large, as were also the ingrowths of columns of epithelial cells.

For two hours after the operation sharp pain on coughing was complained of by the patient; then he had a very severe paroxysm of coughing, and became quite blue in the face, and cold. Mr. Paul, the house surgeon, thereupon introduced an ordinary silver tracheotomy tube, and the attack, which had
lasted from ten to fifteen minutes, ceased immediately. From this time a tube was always kept in the trachea. The patient was fed by nutrient enemata, and his mouth was moistened from time to time with lemon-water.

July 12.—Passed a comfortable night. There have been a few slight paroxysms of cough, and patient has brought up a good deal of mucus, some of it slightly blood-stained. There has been no dyspnœa and no sickness. The feeding enemata are retained. The wound is kept sweet and clear, though a great deal of mucus and saliva gets into it. Iodoform is insufflated into the wound occasionally. The temperature throughout the first twenty-four hours varied from 100° to 101.8°. Pulse 94; respirations 36.

July 13.—Another good night, breathing quite easy; some coughing in paroxysms during the night. Complains of feeling very weak. One of the nutritive enemata returned—on trying to swallow a little water it escaped through the wound. Both ligatures removed from the trachea. During the day more of the enemata failed, and the bowels acted. He sweated much, and a large quantity of mucus and saliva had to be removed from the wound—in fact, incessant attention to this point was requisite. The wound, however, was quite sweet and healthy, and was contracting. The oesophageal tube was used for feeding during the latter part of this day, and was regularly employed from this time.

July 14.—Seems better and less weak. Coughed very little during the night, and is bringing up much less mucus. Iodoform to wound from time to time. A large india-rubber tracheal tube made to fit the upper end of the trachea precisely was substituted for the silver tube. After this was done he had a sharp attack of coughing, and brought up a large quantity of mucus. This was followed by many hours of great comfort and entire freedom from coughing.

July 15.—Passed a fairly comfortable night, but at times was restless, and complained of great thirst. Bowels relaxed twice. Feeding almost entirely accomplished by the oesophageal tube, but enemata continued at long intervals.

July 16.—About the same as yesterday, but enemata not retained and bowels again relaxed two or three times. An attempt was made to swallow water, but nearly the whole of it passed through the wound, and provoked coughing. Otherwise cough is much improved, and is now slight.

July 17.—Another good night. Very little cough. Wound looking well and much contracted. In the evening of this day
Mr. Morris’s Case of Total Excision of the Larynx.

complained of intense weakness; his bowels were very irritable, and the enemata were entirely omitted. He was fed by the oesophageal tube every six hours, all the food being peptonised. Sometimes after the withdrawal of the oesophageal tube he coughed and brought up a quantity of mucus. He had at times been slightly delirious, and very restless.

July 18.—Bowels still irritable. Patient much weaker, and very restless.

July 19.—During the night the patient had an alarming attack of syncope, from which he only rallied after the subcutaneous injection of ether. From time to time he relapses into a state of semi-delirium and drowsiness, and resists the introduction of the oesophageal tube. He passes his urine unconsciously into the bed. His breathing is regular and comfortable, and there is no sign of any acute lung mischief.

July 20.—At 2.30 A.M. he vomited ten minutes after being fed by the oesophageal tube, having struggled violently against the introduction of the tube. Again at 6.30 he vomited immediately after the withdrawal of the oesophageal tube. This much exhausted him, and within a few seconds more he was dead.

Post-mortem examination, made six hours after death, by Dr. Fowler.—An incision in the middle of the neck, over site of larynx, about 2½ in. in length; blood-stained frothy fluid was issuing from the opening in considerable quantity. The tongue, trachea, and oesophagus having been removed, the following conditions were noted:—The epiglottis was intact. The cricoid and thyroid cartilages had been entirely removed and the two upper rings of the trachea divided; the ends of these latter were felt at the lower part of the wound. The wound appeared healthy. There was no part of the original growth found, and no secondary deposit. On laying open the upper end of the oesophagus a small abscess the size of a pea was seen beneath the mucous membrane of its anterior wall; it occupied the middle line immediately below the operation wound. The mucous membrane surrounding this small abscess was swollen and oedematous. The trachea and bronchi were filled with frothy mucus. The lungs were emphysematous and oedematous throughout. There were some catarrhal pneumatic areas in the lower lobe of the right lung. The pneumogastric and recurrent laryngeal nerves were dissected out, the latter in their whole course. They were quite normal. The stomach was somewhat dilated, and contained about four
ounces of fluid. It was in places slightly ecchymosed; there was no ulceration.

The rectus abdominis on each side was ruptured about 2 inches above the pubis; blood was effused around the broken ends. The tissue of these and other abdominal muscles was soft and fatty.

Remarks.—For the first four or five days our hopes were encouraged by the great improvement which followed the operation, but at length the patient succumbed from exhaustion precipitated by the struggling and vomiting excited by the oesophageal tube.

It was against the success of the operation that he had been for some time before, and was at the time of the operation, suffering from severe chronic bronchitis; moreover, his strength had been much reduced by several months of sleeplessness and exhausting paroxysms of dyspnœa. But he was a man of great courage and patience, indeed, mentally an excellent subject for an operation, so that I had hoped, if the operation was well borne, that he might be brought through the after-dangers by rectal alimentation and careful attention to the cleanliness of the wound.

I did not so much dread further pulmonary complications because the previous tracheotomy had inured his lungs to the direct ingress of air; and the freer egress of the bronchial sputa which the operation afforded was a distinct gain.

Unfortunately, however, the nutritive enemata began to fail on the second day, and the use of the oesophageal tube became necessary. To this latter mode of feeding he soon took a strong aversion and I do not think I can exaggerate the depressing influence, which the loss of power of swallowing and the realization that he would have to submit to the use of the tube for some time, had upon the course of the case.

This thought seemed to take away all hope and all wish to recover; and I was much impressed at the time that it greatly contributed to the fatal result.

The healthiness of the wound throughout left nothing to desire, but I am not sure that the insufflation of iodoform was an altogether unmixed good. I think its absorption may have contributed if it did not altogether induce the restlessness and partial delirium.

The temperature after the operation was never high; only once did it reach 101°, and after the fifth day it never reached 100°.

He had one severe paroxysm of dyspnœa after the opera-
tion, and that was before a tracheotomy tube was introduced. I thought, having fixed the trachea by ligatures, I need not retain a tube, but this was a mistake, for owing to the cut section of the trachea being horizontal and at the lower end of the wound the mucus in the end of the wound blocked it; whereas with a tracheotomy tube retained, the breathing orifice was at the surface of the neck, and thus, in spite of mucus in the wound, air could enter freely into the trachea. I should not in another case try to do without a tube but would at once plug the cut end of the trachea with an india-rubber tube of large size and ordinary curve.

I do not doubt that a preliminary tracheotomy does away with some of the after-risks of the operation but I am not prepared to say how far, if at all, it facilitates the operation itself. I would especially draw attention to the advantages, whether in total or partial excision of the larynx, of the method which I adopted of detaching all the soft parts with a raspatory from the cartilages. It entirely prevents hæmorrhage.

From my experience of this case I am led to the conclusion that excision of the larynx ought to be very rarely undertaken, that all other measures should have been tried and failed before resorting to it, and that the subsequent difficulties of feeding are for a considerable time very trying and obnoxious to the patient.

There seem to be two sets of conditions, however, which indicate and warrant the operation, and these both existed in the case just related. The one is where a malignant disease, filling or nearly filling the larynx, is confined within it, and therefore where, though surrounding parts are threatened with infiltration, the whole of the disease is nevertheless capable of removal. In such a case the operation affords a chance of cure. The other is where, tracheotomy for intralaryngeal growth having failed to afford relief, the patient is deprived of rest and sleep and his life is incessantly imperilled by paroxysms of dyspnœa and cough. Under such circumstances (even if the disease had encroached upon surrounding parts) the comfort derived from the operation, judging by this case, would be great, and if in the end death ensues the excision will have provided a far less terrible mode of dying.
IX.—A Case of Partial Extirpation of the Larynx for Epithelioma of the Left Ventricle of Morgagni; Recovery. By Felix Semon, M.D. Read November 12, 1886.

Mr. M. W., æt. 52, barrister, a spare-built man of middle height, Sallow complexion, and nervous temperament, consulted me on April 15, 1886, on account of almost complete aphonia. His voice had become, according to his statements, slightly hoarse about a year previously without any cause known to him, and the hoarseness had gradually increased until, a few weeks before he consulted me, the voice had become almost extinct. Nevertheless he had practised at the Bar until the first week of April. He had sought various medical aid, and had, amongst other things, been treated for a considerable period with intralaryngeal faradisation for what was supposed to be paralysis of the left vocal cord. No improvement, however, had resulted from this or any other of the medications to which he had been subjected. The loss of voice was his only complaint; there was neither pain nor dyspnœa, no dysphagia, no emaciation, or loss of appetite, and the general state of nutrition was excellent. No hereditary taint of cancer or tuberculosis could be made out, and the patient had never suffered from syphilis. His general health had always been remarkably good. The thoracic and abdominal organs were perfectly healthy.

On laryngoscopic examination it was seen that almost the whole of the left vocal cord appeared to be embedded in an irregularly longitudinal, greyish white, warty tumour of about the size and form of a small bean, which seemed to be strictly limited to the vocal cord itself. The mobility of the tumefied part, however, and of the left arytenoid cartilage was well preserved. The rest of the larynx appeared to be healthy. Externally the larynx was unaltered in shape, and not tender on pressure. Enlargement of cervical lymphatic glands was carefully sought for but not discovered.

The laryngeal tumefaction at first sight appeared to be a simple papilloma, and this view seemed to be further corrobo-rated by the absence of several of the symptoms which are held to be characteristic of malignant neoplasms, such as pain,
cachexia, swelling of neighbouring lymphatic glands, &c. But apart from the fact that from a large experience concerning malignant neoplasms of the larynx, I have learned that there is not one of the symptoms, usually supposed to be characteristic of these tumours, which may not be occasionally absent, I must say that I always entertain a suspicion of malignancy if I find in an individual, who has passed the age of thirty-five, an extensive warty tumefaction strictly limited to one side of the larynx. Besides, in this case, the neoplasm, though mammillated, had not the well-developed cauliflower appearance of genuine papilloma.

On the other hand there was one circumstance which induced me to incline to a somewhat more hopeful view, viz. the well-preserved mobility of the affected cord. I have stated elsewhere,* that it is, in my experience, one of the most constant and valuable points of differential diagnosis between innocent and malignant tumours of the vocal cords, that the latter usually cause a considerable impairment of the mobility of the affected cord at a very early period, i.e. when only a small projection can be seen on the cord. This phenomenon, which is entirely absent in cases of benign growths of similar size, is no doubt due to the infiltrating character of malignant tumours. Its absence in the present case, in which the tumour had already attained a comparatively considerable size, will be explained further on.

In view of the doubtful diagnosis I told the patient that there could be no doubt as to the existence of a tumour in his larynx, but that only intralaryngeal removal and subsequent microscopic examination of a small fragment of the new growth could give a clue as to its nature, and consequently as to the treatment which should be adopted.

The patient placed himself entirely in my hands, and I at once proceeded to the extirpation by means of cutting forceps of a small piece from within. The tumour, however, so little projected into the interior of the larynx that I only succeeded, in spite of previous applications of a 20 per cent. solution of cocaine, in removing a very small piece after repeated trials. This fragment, when examined by myself, and later on by Mr. Shattock, exhibited the characters of a simple papilloma; it was, however, so small that it did not seem justifiable to draw from its examination too absolute a conclusion as to the nature of the whole growth. I never could get rid of a certain feeling that the neoplasm was malignant after all, and I determined

Dr. Semon's Case of Partial Exirpation of the Larynx.

to have another piece microscopically examined before I proceeded with the intralaryngeal extirpation of the whole mass. The events proved how justified this precaution was. On April 23 I succeeded in removing a piece about the size of a pea from the upper surface of the growth, and this showed on microscopic examination, kindly made for me by Mr. Shattock, the undoubted characters of squamous carcinoma, cell-nests, giant-cells, and epithelial proliferations. The growth in parts was undergoing cornification.

I was apprised of this result on April 28, and on the same morning laid, as gently as I could, before my patient—who, I may say, had all through shown that curious presentation of the seriousness of his affection which so markedly differentiates patients suffering from laryngeal cancer from those suffering from laryngeal phthisis—the sad alternatives between which he had to choose, viz. on the one hand palliative treatment with the certainty of an agonising death after, at the utmost, according to average experience, four years from the first commencement of the disease; whilst the dyspnoea which was sure to arise would necessitate tracheotomy in about a year's time, and the dysphagia which would very probably occur would very likely demand other surgical interference or artificial alimentation; on the other hand, radical treatment, i.e. partial, or, if the disease should be more extensive than laryngoscopic inspection seemed to show, even total, extirpation of the larynx, an operation very serious in itself, often fatal, possibly leading to permanent loss of voice, and difficult alimentation per os, and even if successful affording no guarantee against recurrence of the affection; but, after all, giving the only chance of a real cure.

Abiding by the rules which I have recently ventured to lay down concerning this most disagreeable and responsible duty,* I explained the whole matter as fully as I could to the patient, but left the decision to him, and only urged that, in case he thought of the radical operation, he ought to come to a decision as soon as possible, inasmuch as the chances of a lasting cure were the better the sooner the operation was performed.

Without a moment's hesitation the patient decided in favour of the radical operation, and selected as operator, from amongst those I named to him as having had special experience concerning this operation, Dr. Eugen Hahn, chief surgeon of the Town Hospital of Berlin, this gentleman not

only having performed, according to the then published statistics, extirpation of the larynx more frequently than any other surgeon, viz. in ten out of ninety published cases, but being also the author of a very excellent monograph on the subject,* in which he describes some important modifications concerning the technique of the operation, of which he had, as his results show, successfully availed himself.

Dr. Hahn was immediately communicated with, consented to come, and arrived here with a former assistant of his, Dr. Borchert, of Berlin, on May 1. On the following day a consultation took place between Sir James Paget, Dr. Hahn, Dr. Borchert, and myself, in which it was decided that the operation should be performed, Dr. Hahn expressing a confident belief that it would be possible, as I had hoped, to limit the extirpation to the left half of the larynx, and thus to dispense with the permanent wearing of a cannula.

The operation was performed the next day, May 3, at 2.30 P.M., at Fitzroy House, by Dr. Hahn, in the presence of Sir James Paget. Dr. Borchert and I acted as assistants, and Mr. Mills gave chloroform.

Tracheotomy having been performed, Dr. Hahn’s “compressed sponge cannula” was introduced. Of this cannula Dr. Hahn gives the following description in his monograph:

“It consists of a common outer cannula terminating in a small movable shield, at the lower end of which there is, in its entire circumference, an elevation about 1 cm. long and about 2 mm. thick. The internal cannula runs immediately in front of the shield vertically downwards for a distance of 2 cm., and then in a right angle forwards. The purpose of this curvature is not to narrow the field of operation, and especially to avoid that the operator be interfered with by the india-rubber tube, through which the chloroform is administered. The outer cannula is surrounded from behind the shield down to the elevation by compressed sponge about 2 mm. in thickness, which is fixed by a contiguous longitudinal suture running along the concave aspect of the tube. In order to secure the sponge even more firmly, it must be wrapped up above and below circularly with silk threads.

“The compressed sponge is prepared from soft, spongy sponges in the following manner:—First the sponges are laid into a solution of iodoform and ether (1 in 7), and this may be done several times in order to saturate them thoroughly

* * * Ueber Kehlkopfextirpation, Volkmann’s Sammlung klinischer Vorträge, Nr. 260.
with iodoform. Next the sponge is moistened with warm water and submitted to powerful compression. In order to become well compressed moistening is necessary, and water is used because it does not dissolve iodoform and does not remove the quantity of this drug with which the sponge has been previously impregnated. If the sponge, merely moistened by means of the iodoform ether solution, is at once compressed, too little iodoform remains in the compressed sponge. Previous to all this the sponges must be disinfected by means of sublimate or carbolic acid.

"The sponge must not project above the lower thicker end of the cannula, in order not to impede the introduction of the latter. Previous to introduction the lower end of the cannula ought to be slightly oiled. Already after a short time the sponge, admitting moisture, expands and presses so firmly against the mucous membrane of the trachea that an absolutely secure occlusion is obtained. I have never seen an untoward accident arise when it was thus used. As soon as the sponge had become expanded not a drop of blood ran down into the bronchi, even if there was blood above the sponge throughout the operation. Slipping of the sponge from the cannula never occurred, nor was any unfavorable influence seen from the pressure of the sponge upon the mucous membrane.

"The cannula is removed after twenty-four hours, and replaced, according to the emergencies of the case, either by another compressed sponge cannula, or by one wrapped up in iodoform gauze, or by a common one, above which the cavity of the wound is plugged."

A cannula of this description having been introduced in the present case Dr. Hahn almost immediately proceeded with the operation by performing thyrotyotomy. On the two halves of the thyroid cartilage being held asunder with blunt retractors, it was seen that there was indeed a tumour in the left half of the larynx, but that it did not originate, as had seemed on laryngoscopic examination, from the left vocal cord itself, but from the left ventricle of Morgagni, and that it merely rested on and covered the left vocal cord, which was apparently quite healthy. The free mobility of the left cord and left arytenoid cartilage, which had given rise to so much doubt about the nature of the neoplasm, was thus satisfactorily explained. The tumour, the appearance of which quite corresponded with the above description, originated in a broad but distinct pedicle from the posterior part of the ventricle. The neighbouring structures were apparently quite free from infil-
and the original plan, viz. to extirpate only the left half of the larynx, could thus be adhered to.

Dr. Hahn now made two oval incisions, which met in front and behind, round the soft parts of the left half of the larynx, at some distance from the tumour, down to the cartilage, freed the left half of the thyroid cartilage by means of an elevator from its perichondrium and the surrounding soft parts, and removed this half, as well as the left arytenoid cartilage and the soft parts previously isolated, by means of bistoury and scissors. The cricoid cartilage was left intact. The haemorrhage was comparatively slight, and no vessel of any importance had to be ligatured, but the occlusion of the lower air passages seemed to have been not quite complete, for the patient, whose lungs had been examined very carefully the day previous to the operation and had been found quite healthy, repeatedly coughed during the latter part of the operation, and expectorated some blood through the tube. The whole wound was plugged with iodoform and bismuth gauze. The duration of the operation was forty-five minutes.

No nourishment was administered during the rest of the day. In the evening, temp. 100.2°, pulse 96. (It may be stated here at once, that the temperature only once again rose to 100° on the sixth day after operation, and usually varied between 98° in the morning and 99° in the evening until the eighth day after operation, when it became quite normal; during the same period the pulse varied between 72 and 96.) There was some cough with expectoration of blood-tinged sputum during the first night, but the patient had some sleep and looked remarkably well next morning. During the first three days the patient was twice daily fed with strong beef-tea, milk, and port wine, by means of a soft oesophageal feeding-tube; on the second day, however, Dr. Hahn allowed him to drink some water, and the experiment was perfectly successful, the patient swallowing with ease, and nothing going "the wrong way." The compressed sponge cannula was replaced on the next day after operation by one covered with india rubber. The wound was plugged twice daily, from the fifth day once daily, with iodoform gauze. On the eighth day after the operation the cannula was entirely removed. Both the internal and the external wound were then painted once or twice daily with a weak solution of nitrate of silver (1:50). The patient was permitted to quit his bed for an hour on the fifth day after the operation, and from that day he got up daily for gradually longer periods. From the fourth day he
took all nourishment, solid as well as fluid by the mouth. He never had any pain on swallowing, and nothing ever went the wrong way.

Whilst thus, on the whole, matters progressed very satisfactorily, two circumstances retarded to some extent convalescence. The cough, which had been present from the moment of the operation, and which had troubled the first night's rest, increased in severity and got most troublesome towards the fifth day, by which time an extensive bronchial catarrh had developed. This cough, accompanied by expectoration of enormous quantities of at first slightly blood-tinged, later on purely catarrhal, frothy sputa, gave the patient hardly a moment's rest—especially when the dressing was changed—and greatly disturbed his sleep. When, after administration of different expectorant and sedative remedies, it somewhat subsided on the eighth day, the patient began to complain of a pain in the lower parts of the right half of the chest, both in front and posteriorly. The pain was most marked on coughing and taking a deep inspiration. There was no dulness to be made out, both halves of the thorax moved equally well, the temperature did not rise, and the breathing was neither superficial nor hurried, but on May 10, 11, and 12 very fine friction was heard over a circumscribed spot in the axillary line in the seventh and eighth intercostal spaces, normal vesicular breathing sounds being heard simultaneously.

The pain was relieved by means of poultices and cold-water compresses. On the 13th it had almost entirely subsided, and no friction could be made out over the spot where it had formerly been heard. The cough also was much better, and the wound was closing rapidly. (On the afternoon of this day Sir James Paget saw the patient again and declared himself much pleased with his progress.)

May 17.—Patient feels quite well. Has been up to-day seven hours, and does not feel at all fatigued. Expectorates already through the mouth. Speech quite distinct, voice loud whisper. Departure of Drs. Hahn and Borchert.

May 19.—External wound cauterised a few times during last few days with nitrate of silver on account of luxuriant granulations. To-day wound entirely closed.

May 22.—Patient has been driving and going out during last few days and feels perfectly well. Laryngoscopic examination: The whole left side is changed into a concave smooth surface. The right vocal cord crosses the middle line on phonation. The voice improves daily in strength. In the
morning it is sometimes almost natural. The patient goes into the country.

Since then I have seen the patient at intervals, and examined him regularly, the last time on October 20, i.e. four and a half months after the performance of the operation. His general health is excellent, there is no trace of a recurrence to be made out, and his voice was so surprisingly improved, that he came on the 20th of last month in order to ask me whether he might not now resume his practice at the Bar. In consideration, however, of the fact that his particular duties would at once have necessitated prolonged and excessive use of voice, and thus have led to overworking of the remaining cord, I explained to him the objections to such a course, and he acquiesced in my advice.

The case is placed on record for the following reasons:

In the first place, the operation of extirpation of the larynx for malignant disease is still so much on its trial that it appears desirable that we should publish every case, whether successful or unsuccessful, in which that operation has been performed. Only by doing this and also by not losing, if we can help it, the apparently "cured" cases from view, can we hope, I believe, to come to a greater consensus of opinion concerning the value of the operation for carcinoma of the larynx, than exists at present. I shall consider it a duty to report again to the Society the further progress of this case, and I trust that the report may be favorable.

Secondly, I wish to draw special attention to a very important point concerning the technique of the operation. I refer to the necessity of securing a perfect occlusion of the lower air passages from the field of operation, before the second step of the operation, viz. thyrotomy, is proceeded with. The great stress I lay upon this point will be easily understood if it be remembered that according to the careful calculations of the latest writer on the subject, M. Schwartz, of Paris,* death took place in no less than twenty cases, or 21 per cent. of the whole number of extirpation for carcinoma, during the first fortnight after the operation from pneumonia, broncho-pneumonia, pleurisy or gangrene of the lungs, i.e. from respiratory complications. These are indeed, as M. Schwartz justly observes, the most redoubtable of all possible complications so far as both frequency and severity are concerned. My patient had, it would seem, a narrow escape from a pleuritic complication, and his recovery was certainly re-

tarded by the troublesome bronchial catarrh I have dwelt upon. As his air passages had been found to be quite healthy before the operation, we are driven to the conclusion that these complications were due to some blood having found its way into the lower air passages before the compressed sponge had had time to expand properly, and I would fain draw the lesson from this case, that, if Hahn’s excellent compressed sponge cannula be used, a little time, say ten minutes, should be allowed to elapse after its introduction before the thyroid cartilage is split. I urged the desirability of this precaution upon my friend Mr. Butlin, when I had the privilege of assisting him at an operation, the report of which will immediately follow the reading of this paper, and to a great extent I attribute the quick and excellent recovery his patient made to his having complied with this suggestion.

Finally, I wish to correct a misapprehension of what I said on extirpation of the larynx in cases of carcinoma, in the discussion on that subject on the occasion of the Seventh International Medical Congress.* I find that in the three most recent and important contributions to the subject, viz. in the papers of Hahn,† Lublinski,‡ and Schwartz,§ I am more or less represented as being an opponent a principio to the operation in cases of carcinoma. I find it somewhat difficult to understand how such an erroneous interpretation of what I said could have arisen, considering that I wound up verbatim as follows:—“In conclusion I desire to say that I have no theoretical objections against the operation as such, and that I agree with many of the indications laid down by my friend Schech, but that I consider it a duty to protest against its indiscriminate recommendation in all (this word is also in the original printed in italics) cases of carcinoma, a recommendation which I think is with difficulty theoretically justifiable, and which is certainly practically not justified.”

From this quotation and, indeed, from the whole gist of my argument, it appears to me to follow with sufficient clearness that I did not protest in a general manner against extirpation of the larynx in cases of malignant disease, but that I protested against the formulation of the indication laid

† L. c., p. 2261.
§ L. c., p. 207.
Dr. Semon's *Case of Partial Exirpation of the Larynx.* 53

down by the late lamented Dr. Foulis, and maintained by Prof. Czerny, viz. that the operation "was required as soon as the diagnosis was clearly made."

This is not a distinction without a difference, but a distinction with a very great difference indeed, and one that goes to the root of the whole question. If in all cases of malignant disease of the larynx the course of events was such as I have described in the present instance, *i.e.* if a well-defined neoplasm, a piece of which could be removed for microscopic examination with comparative ease, made its appearance within the precincts of the larynx proper, and came under competent observation shortly afterwards, no valid objection could, I think, be entertained against Dr. Foulis's and Prof. Czerny's indication. Unfortunately, however, this is not the case. Only in a comparatively small number of cases does the disease start with the formation of a well-defined, easily accessible neoplasm within the larynx itself, which allows of a certain diagnosis at an early stage of the whole process, and even in such cases the patients unfortunately come but rarely at an *early* period under competent observation. Much more frequently, according to my experience, one of the following contingencies occurs,—either the disease is already too much advanced, when the patient comes under observation, to permit a reasonable hope of success to be entertained, or the new growth, though perhaps not very extensive, has already invaded the food-passages, or the cancer commences in the infiltrating form, and breaks down quickly, when the diagnosis may remain so long doubtful between carcinoma and tertiary syphilis or other affections, that when it is finally made with certainty, the time for operation has passed away; or, though its first manifestations are laryngeal, the carcinoma has in reality started from the neighbouring lymphatics, which become engorged, in such cases, at an apparently very early period; or the general health of the patient is very unsatisfactory, so that death from collapse is to be feared after the operation; or there is already, as frequently in these cases, extensive pulmonary catarrh, justifying the fear of pulmonary complications after the operation.

Of all these and other contingencies I could bring forward illustrative cases, but the limited time does not allow me to further extend this paper. I must content myself, therefore,

* L. c., p. 60. Dr. Foulis only thus far narrows his indication by adding: "When the cervical glands are involved, these may be a barrier to the operation;" and "very old people, *e.g.* above seventy years of age, are not fit subjects for this operation."
by saying that in view of all the contingencies just named, I entirely maintain the protest I raised in 1881 against the dogmatic rule that the larynx must be excised as soon as the diagnosis of carcinoma is clearly made, because I find that in many instances a certain diagnosis can only be made at a time when in my opinion the operation is no longer justifiable. If no account be taken of all the contra-indications and the operation be proceeded with simply on the basis of Dr. Foulis's indication, whatever the accompanying circumstances be, the result will be in many instances disastrous. In some cases it will be impossible to finish the operation, and parts of the new growth will have to be left behind; in other cases the patient will die from shock or collapse, or hæmorrhage or pulmonary complications or early recurrence, &c. The published records of the operation give but too many illustrations of all these contingencies, and it is, in my opinion, due in no small degree to the non-observance of the contra-indications that the statistics of the operation show so unfavorable a general aspect. That is why I maintain my protest. But this protest does not imply that I should be a blind opponent to extirpation in really suitable cases of carcinoma. It is perhaps at present not yet possible to lay down absolute indications, but my ideas on this point go entirely with those of Butlin,* when he says that in the immediate future extirpation of the larynx for carcinoma should be practised only for intrinsic carcinoma which is still limited to the larynx. Hahn† expresses similar views. Though such cases will perhaps always remain rather the exception than the rule, because, as stated before, the disease only in a minority of cases allows of a really early recognition of intrinsic laryngeal cancer, it ought I think to be the ambition of every surgeon to increase their number by aiming at an early and certain diagnosis. Such diagnosis, in this respect almost all authors on the subject are agreed, can only be arrived at by intralaryngeal removal of a small piece for the purpose of microscopic examination. The present case, however, shows that this proceeding may have to be repeated before a reliable result is arrived at, and teaches the lesson, that even a negative result of the examination of a small part of the growth does not entitle us to exclude the idea that a suspected laryngeal tumour is malignant.

The importance of an early diagnosis can hardly be over-

† L. c., p. 2302.
rated. Not only will the operation, if decided upon, have a better chance, because the general health of the patient has probably not yet suffered; not only are the chances better with regard to the recurrence, because there is greater probability of the affection being still purely local; but in many instances it will be possible to avoid the more dangerous total extirpation with all its subsequent drawbacks, and to substitute for it partial extirpation with its infinitely superior functional results and greater comfort of subsequent life.

Note (May 22, 1887).—Since this paper was read I have seen the patient at regular intervals, the last time on April 15, 1887, i. e. almost a year after the operation. There is no trace of recurrence, his general health is excellent, and his voice is so strong that he can easily fulfil the duties of a police-magistracy, to which he was appointed half a year ago.
X.—On a Case of Epithelioma of the Vocal Cords, for which Partial Excision of the Larynx was performed.

By H. T. BUTLIN. Read November 12, 1886.

In November of last year (1885) I was consulted by a gentleman, fifty years of age, on account of extreme hoarseness, which had commenced about two years previously and had slowly increased in spite of the general improvement which his health had manifested of late. On examination, a warty growth was apparent on the front part of the left vocal cord, prominent, not ulcerated, partly pedunculated, and only differing from most papillary growths of the larynx in its very deep colour, which amounted almost to complete blackness here and there. The cord moved freely, and there was no enlargement of the larynx or of the glands in the neck. I advised that it should be removed with forceps.

In December, at several sittings, I removed portions of the growth, which was soft and vascular. It was cut in sections and examined, and although there was nothing to lead to the belief that it was epitheliomatous, the irregular character of the epithelium and the large number of connective-tissue cells in the central part of the sections led me to believe that it was not an instance of ordinary papilloma. The use of the forceps set up so much irritation in a mucous membrane which he said was always prone to inflame that no further attempt at operation could be made until the end of January (1886).

At that time I had not seen him for a month previously. There was a small portion of warty growth remaining, and there was red thickening of the cord at the point where the first portions had been attached.

Not liking the appearance, I held a consultation with Dr. Semon, who thought the disease was certainly papillomatous on account of the free mobility of the cord and the long duration of the case.

During the next few days I removed all that I could see of the growth, and with much less irritation than before. The cord remained thickened, and the voice did not recover as I hoped it would.

Again in May I removed several small fragments of growth and one large fragment, which I thought I must have overlooked.
Mr. Butlin's *Case of Epithelioma of the Vocal Cords.* 57

on account of the fact that the disease lay partly beneath the cord and under the shelter of the epiglottis.

On June 21, after three weeks in the country, he returned with his voice weaker than ever. Examination showed a fragment of growth as large as had been removed, and it was quite certain this was a recurrence. The cord was red and its mobility was decidedly impaired. There was not any enlargement of the larynx or of the lymphatic glands.

A second consultation was held with Dr. Semon, who agreed with me that the aspect of the disease was much more like that of a malignant disease. It was decided to remove a fragment of the growth for microscopical examination, and if it proved to be malignant that the question of removal of half the larynx should be submitted to Mr. X.

Examination of sections of a fragment proved that the disease was epithelioma, not sarcoma, as I had at one time been led to suspect. The opinion of Dr. Heneage Gibbes and of Mr. Marcus Beck completely coincided with my own on the nature of the growth.

In laying the question of excision of a portion of the larynx before Mr. X., although I could not strongly recommend him to undergo the operation, I could say that his case was peculiarly suited for such an operation. The disease appeared to be very limited in extent; it was of steady but not of very rapid growth. It had only quite lately been associated with immobility of the cord; it was decidedly intrinsic (i.e. originating in the interior of the larynx); it did not appear to be associated with affection of the framework of the larynx, and there was no evident affection of the lymphatic glands. Mr. X. was also in very good condition, and although not a strong man was one who seemed likely to bear a serious operation well.

The operation was performed on June 26, Mr. Mills giving chloroform and Dr. Semon assisting me. A longitudinal incision was made from the hyoid bone to the level of the third tracheal ring, the trachea was opened below the cricoid cartilage and Hahn's tube was introduced. Nearly a quarter of an hour was allowed to elapse, in order to ensure the swelling of the compressed sponge and consequent obstruction of the trachea. Then the thyroid cartilage, which was ossified, was divided in the middle line, and the interior of the larynx was examined. The left cord was fixed to the cartilage by a small firm warty mass, which extended back almost to the arytenoid cartilage. The disease entered the ventricle and
passed down beneath the cord, but did not reach more than a short distance below, or so far upward as to affect the ventricular band (false cord). Unfortunately, it extended from the left cord round the angle to the right cord, which was affected on its under aspect almost back to the arytenoid cartilage. The disease was quite limited to the right cord on the right side. I cut away the left ala of the thyroid cartilage, leaving the upper and lower cornua. In doing this a little slit was made with the scissors into the pharynx, owing to the drawing up of the soft parts. The opening was sewn up with a continuous silk suture, and gave no further trouble. On the right side, instead of removing the ala, the soft parts were raised off it, the false and true cords and the tissues for a short distance below the true cord were cut away as far back as the anterior angle of the arytenoid cartilage. This was done without difficulty. In the removal of the left ala only one vessel bled freely, apparently the superior thyroid artery, and was tied.

The tracheotomy tube answered admirably, so that no blood passed down the trachea and we were not interrupted by cough. The wound was sponged thoroughly with carbolic lotion and plugged with strips of iodoform gauze rubbed over with powdered borax.

Mr. X. was allowed to suck ice through the night, the operation having been performed late in the afternoon.

June 27.—A sleepless night on account of feeling very hot, although his temperature was only 99·4°. There has been no sickness. He now complains of feeling very empty, of being very hot, and of being continually choked with blood and phlegm. He appeared very well in spite of his discomfort. A fire had been kept up during the night and a steam kettle set at work, but the heat was so great that the fire was obliged to be extinguished. A large quantity of blood-stained saliva had run away and had been sopped up with cotton wool. I administered his first food with a Higginson’s syringe through a soft catheter, half a pint of beef-tea, half a pint of milk, an ounce of port wine, and 15 minims of laudanum (because he had had no sleep). The wound was cleansed, dusted with iodoform, and the gauze replaced. The pulse was 80, and remained at 80 or 84 during the entire treatment. Mr. X. was propped up on a bed-rest. In the afternoon, and again at night, the feeding was repeated, first without the wine and laudanum, then with both (laudanum 20 min.). The dressing required to be changed at night, as it was soaked with saliva.
He seemed well, and complained of want of food for two hours before his evening feed.

June 28.—Pulse and temperature natural. Slept fairly. There had been no cough since the operation. Feeding and dressing of the wound as on the previous day. In the afternoon Hahn's tube was taken out and replaced by a smaller silver tube, surrounded by a piece of india-rubber tubing. In the evening he had a violent attack of cough, which was relieved by turning on his side. Finding that saliva was coming through the tube I replugged the wound above it.

June 29.—Had one or two attacks of cough during the night. Is not so heavy this morning (the laudanum was omitted last night). With his evening feed an ounce of castor-oil was mixed.

June 30.—Disturbed during the night by the action of the bowels and by cough, but is well this morning, with normal temperature and pulse. Feeding and dressing as before. In the course of the day several fragments of ice were swallowed, and the flow of saliva from the wound was noticeably less than it had been. In spite of the most careful plugging of the wound above the cannula and the covering of the cannula with gauze, saliva made its way into the trachea and produced a great many attacks of cough.

July 1.—Still much cough. In the afternoon swallowed some jelly well. In the evening, as he was fatigued with the frequency of the cough, and the plugging did not appear to relieve it, the fire was relit, for the weather had become very cold and the wind was east to north-east.

July 2.—Passed the best night since the operation, coughing scarcely at all. Only two feeds daily, for he is able to take the midday allowance by the mouth. In the evening very much cough, which produced very troublesome diaphragmatic spasm.

July 3.—This morning able to drink milk. I took the tracheotomy tube out, thinking it might irritate the trachea and be itself a cause of cough. After this the discharge had to be frequently cleansed from the trachea with pledgets of cotton wool. In the evening was as comfortable without the tube, and not more annoyed by secretion than he had been when it was in the trachea.

July 5.—Less cough. Takes solid and fluid food without difficulty. It has not been necessary to re-introduce the tracheotomy tube.

From this time he made rapid progress towards recovery,
suffering only from indigestion, to which he has always been subject. The cough gradually ceased, unless a collection of discharge obliged him to cough to clear his throat. The edges of the wound came together, and were only supported with strapping.

On July 17 Mr. X. was able to leave London and drive down to Wimbledon, and a week later he returned to his home in the Midland Counties.

On September 28 he came to see me. The wound was completely and well healed, with the exception of a tiny opening at the lower part, which was the entrance of a small sinus running upwards for more than half an inch, probably due to the presence of a tiny fragment of necrosed thyroid cartilage. Examination of the outside of the throat led me to believe that it was perfectly healthy and that there was no sign of glandular disease. Examination with the laryngoscope showed the open larynx without any re-appearance of the disease. The movements of the arytenoid cartilages were well performed. Mr. X. was able to speak in a gruff whisper, which could be easily heard and understood at some distance from him. He complained of the formation of a quantity of secretion in the back of the throat, which was probably due in large measure to the granular pharyngitis from which he suffered. And he also told me that although he had no difficulty in breathing when on flat ground or in moving at an ordinary pace, he was a little troubled with shortness of breath on exertion or walking uphill. Nevertheless, he appeared in excellent health and was able to walk and ride.

Remarks.—Several reasons have induced me to bring this case before the Society. They are the following:

First, the operation of removal of the larynx either in whole or part has been very seldom performed in England, and still more seldom successfully performed. This circumstance determined Dr. Semon’s patient to select a German surgeon.

Second, the reasons which led me to consider the present case suitable for operation. Some time ago I expressed the opinion that want of care in the selection of cases for operation was one of the causes of the large mortality and unfortunate results which have attended excision of the larynx. I then suggested "that in the immediate future extirpation of the larynx for carcinoma should be practised only for intrinsic carcinoma which is still limited to the larynx, and that tracheotomy should be performed at least a fortnight or three
weeks before the extirpation, in order to give the patient a
good opportunity of gaining strength to bear a larger opera-
tion" (Malignant Disease of the Larynx, 1883, p. 62). In
the case of Mr. X. the conditions were such as have been
described; the disease was of intrinsic origin and was limited
to the interior of the larynx. Further, the general condition
of the patient enabled me to dispense with the preliminary
tracheotomy, and to complete the operation at one sitting.
Experience has proved that removal of half the larynx is far
less dangerous to life than removal of the entire larynx, and
the appearance of the growth led both Dr. Semon and myself
to hope that the operation might be limited to excision of
half the organ. When first we came to examine the interior
of the larynx we were disappointed at finding that the disease
had spread to the right cord. Happily, as I think, I adopted
the expedient of removing the affected soft parts very freely,
and leaving the framework. There was not the least sign of
affection of the ala of the cartilage; indeed, the disease had
not penetrated through the soft parts even to the calcified
cartilage. Examination of the parts after removal leads me
to form the opinion that this conservative surgery might have
been carried further with benefit to the patient. Although the
small mass of disease on the left cord sits as it were close
upon the ala of the thyroid cartilage, and appears to be
attached to it, it can nevertheless be moved on the cartilage,
and might therefore have been raised off it and removed. At
the time of the operation I did not discover this relation of
the disease to the thyroid ala; the sensation communicated
to the tip of the finger introduced into the interior of the larynx
through the divided angle was that the mass of disease was
actually adherent to the cartilage. If I am called on to per-
form an operation for disease of similar nature and situation
on a future occasion, I shall certainly make the attempt to
remove it without removing the framework of the larynx.
Even if malignant disease extends into the substance of the
cartilage there is no reason why the affected area of the cartilage
should not be scraped away or treated with the cautery.
Experience shows that such a disease as epithelioma affecting
the soft parts over a bone and spreading to the superficial
layers of the bone, is far less likely to recur in the bone after
removal than in the soft parts. A familiar example may be
found in disease of the floor of the mouth affecting the super-
ficial layers of the lower jaw.

Third, Hahn's tube proved a thorough success, not only
during, but after, the operation for the first forty-eight hours. After it was removed Mr. X. was much troubled by cough owing to the passage of liquids down the trachea. This might, I think, have been obviated by the use of a larger tube than that which was used to replace Hahn's tube (which is of exceedingly large size). My reason for not using a larger-sized tube was that I could not obtain one, although I made an endeavour to do so at several instrument makers. The gauze which was wrapped round it was soon soaked by the discharges, and appeared of itself to irritate the interior of the trachea.

Fourth, it is scarcely necessary to draw attention pointedly to the manner of feeding. It is possible that Mr. X. might have been able to swallow within twenty-four hours after the operation, and that the use of the tube and syringe was not absolutely needful. But I think that most surgeons are now of opinion that, even if it is not absolutely necessary to feed such patients artificially, it is yet decidedly desirable to do so. Quite apart from the fact that food is administered regularly and in sufficient quantity, the parts are maintained in a much more satisfactory condition of rest, and the patient is not forced to make painful efforts to obtain needful nourishment.

Fifth, a very interesting circumstance in connection with this case is the present condition of the patient in reference to speech. Both cords have been removed from both sides of the larynx as far back as their attachment to the arytaenoid cartilages, therefore wholly. Yet Mr. X. can produce so loud and distinct a whisper that he can be easily heard and understood at a distance of several, if not many, feet. The whisper is evidently, from its gruffness and general character, formed in the larynx, but I cannot tell what structures take part in its formation.
XI.—Six Cases of Tumour of the Bladder removed during the last twelve months, with a Brief Sketch of their Histories and the Results. By Sir H. Thompson. Read November 26, 1886.

I propose to give a brief sketch of six cases of tumour of the bladder, in which I have operated during the last twelve months, showing the parts removed in each case. In three the supra-pubic operation was employed; in two the perineal incision only, and in one, that of a female, urethral dilatation was sufficient for the purpose.

Case 1.—A lady, æt. 73, sent by Dr. Edis, of Gloucester, August 11, 1885, was first seen by me. Marked symptoms for about eighteen months. During the last six months there has been much bleeding; her sufferings have been very severe. No trace of tumour débris could be found in the urine. The progress of her case made digital exploration necessary, and it was performed on October 17, 1885. Having dilated the urethra, and introduced my finger, I found a large growth springing from the top of the bladder. I at once performed the supra-pubic operation, my friend, Mr. Buckston Browne, assisting me. The tissues were divided chiefly by the ivory separator as usual, and I removed a large growth from a rather wide base in the situation described. No vessel was tied. Injection of the bladder being impossible, the groove-ended staff was used. She made a slow recovery, and in seven or eight weeks returned to the country, the hæmaturia having ceased, but the wound remained fistulous for some time, and micturition was frequent and painful. She lived nine months after, her sister writing me that she believes “her symptoms were mitigated and her life prolonged by the operation.” The tumour examined by Mr. Shatlock is a fibrous papilloma; it is at the museum of St. Thomas’s Hospital.

Case 2.—A gentleman, æt. 34, sent by Dr. Stevenson, Camberwell Grove. October 25, 1885, I first saw him. Symptoms commenced about seven years ago. Not much pain or frequency of micturition but attacks of severe bleeding from time to time. On washing out the bladder, good specimens of
papilloma were seen under the microscope. Consequently, I advised digital exploration without delay. It was performed on November 12, Mr. Buckston Browne and Mr. T. Hart-Smith, of University College Hospital, assisting me. I removed without difficulty, through the perineal wound, a single polypoid growth, and could feel no others present. The bleeding was profuse, unusually so, during five or six days. He made a good recovery, the wound healing on the last day of November, soon after which he left town, quite free from symptoms. The growth was a fimbriated papilloma. The patient has enjoyed good health ever since. I have seen him very recently; he has long been actively engaged in his business, and no sign of bleeding or of urinary trouble has been observed. I have no doubt he is now safe. The first case also I operated on thus, now five years ago, was a very similar one, and he also has continued perfectly well to the present date.

Case 3.—A gentleman, æt. 55. November 11, 1885, I saw him for the first time; sent to me by Dr. Lloyd, of Bloomsbury. Symptoms of four or five years' standing; now extremely severe. He passed urine with great pain every half hour day and night, and was much reduced and very weak. Bleeding only during the last year and a half. No evidence from urinary débris as to the nature of the disease. Rectal examination showed absence of scirrhus, but revealed a soft full bladder beyond the prostate. His case was most unpromising, but I advised him, considering the severity of his sufferings, which at all events would be diminished, to try the slender chance which a digital examination offered him.

I did this on November 16, finding a condition almost identical with that of Case 1, opened the bladder above the pubes and removed a large mass of soft growth springing from a thickened stratum involving the upper part of the vesical wall. He left town at the end of December, the wound not having quite closed. In the February following I heard from Mr. Tytheridge, of Epsom, that two masses of growth had appeared in the situation of the wound. Sometime after this he died. The tumour, which was examined by Mr. Stonham, of University College, is now in the museum there; it is reported by him to be a round-celled sarcoma.

Case 4.—A lady, æt. 59. March 3, 1886, I first saw her, meeting Sir William Roberts and Mr. Whitehead, of Man-
chester, at Bolton, where I had been summoned for consultation. She had been the subject of severe hæmaturia for six or seven years, the extent being remarkable, and the persistence equally so. She was almost bloodless in appearance, and was moved and managed with difficulty on account of her feeble condition and liability to faint. We decided to explore at once, and I did so, finding a very small, soft, floating, polypoid growth on the right side (patient's right) of the bladder, and nothing else. I removed it easily with one application of the forceps, feeling very strongly that there must be some further source for the profuse hæmorrhage, but I could not detect any, nor could Mr. Whitehead, whom I desired to examine carefully after me. This little growth proved, however, to be the whole of the disease, for she made an excellent recovery, soon resuming her active life, previously long laid aside, and has never bled since, now nine months ago. She is therefore safe. Fimbriated papilloma, examined by Mr. Shattock.

Case 5.—C. W., æt. 55, sent by Dr. Percy Reynolds, Stamford Hill. On August 10, 1886, I saw him for the first time. Earliest symptoms, bleeding chiefly, were observed about four years ago. During last year, the urine has been persistently bloody. Nothing was discovered by two examinations of the urine. Advised digital exploration, and performed it on August 18; finding a large growth fairly accessible, I decided to remove it by forceps from the perineal opening, and did so, leaving a thickened base. Hæmorrhage very free; the tube was retained in consequence three or four days. The wound was closed on September 3; all urine passed naturally, a little blood appearing occasionally, and he left at his particular request on the 13th for the country. I have recently heard that bleeding has never quite ceased, and that micturition is frequent and painful.

The tumour was chiefly fibrous papilloma, but one portion of it presents a formation which must be regarded with extreme interest, and is thus described by Mr. Shattock, "I find a sessile solid growth of cartilaginous firmness; this under the microscope is a chondrifying sarcoma, the transition from the sarcoma tissue to cartilage being readily traceable."

Case 6.—J. A., æt. 44, brought to consult me by Mr. Maurice, of Reading, who was present at the operation. Saw him for the vol. xx.
first time on September 23, 1886. Symptoms commenced about four years ago, and have been very severe during the last year. Micturition is now difficult; there are always about six ounces of residual, very offensive urine. Washing-out the bladder produced no characteristic débris. Advised digital examination and performed it on October 8. Mr. Maurice, Dr. George Buchanan, of Glasgow, and Mr. Rice Ord, of Clapham, were present. When my finger entered, it encountered a large growth springing from the upper part and right (patient's right) side of a distended bladder, and I decided at once to open it above the pubes, using the groove-ended staff, as well as the rectal bag. I removed at first several large masses, finally leaving only a thick base from which they arose. The bleeding was profuse during the proceeding, but almost ceased on the removal of the rectal bag; no vessels were tied. Two stitches brought the parietes of the abdomen together above the tube. He made rapid progress, both wounds appearing nearly healed by first intention at the end of the week, the urine issuing through a small opening at the lower angle of the supra-pubic wound.

The patient was presented at the last meeting of the Society, a month having elapsed since the operation, the wound being healed and no symptoms present. The tumour was a fibrous papilloma.

I think it will be admitted that the results of operation in these cases are encouraging. Thus in two of them without doubt a complete and permanent cure is effected. In two others the disease, although known at the time to be considerable, was more so than might have been expected from preliminary examinations, and death, in each case imminent, was in one certainly, perhaps in both, postponed for a short time. The remaining two are of recent date, and it is therefore only possible at present to speak with uncertainty as to their future. One of them was present at the last meeting, and was seen by several of the members. A month had elapsed since the supra-pubic operation had been performed; the wound had closed, and the patient was absolutely free from symptoms. The other case, of nearly the same date, that in which the tumour partly consisted of a chondrifying sarcoma, is not progressing satisfactorily, and cannot be said yet to have shown improvement of symptoms, although the bleeding has been checked.

In endeavouring to estimate the true value of operations
for removing tumour of the bladder, it should be remembered that a bleeding growth there is invariably fatal, sooner or later. I am not aware of any instance of cure, either by spontaneous action or by medication, local or constitutional. Hence whatever success is obtained by operating is so much clear gain to patients thus affected. Then besides the moderate proportion permanently cured, there is no question that prolongation of life is often ensured by operating.

This, indeed, is the most frequent result. I have now operated in all on thirty-three individuals for removal of vesical tumour, and on two of these twice. In the great majority of these cases the fatal event was postponed, for a few of them; during a considerable term. Some who had been unfitted for active life resumed it, and are thus now engaged after the lapse of two or three years since the date at which the tumour was removed. One even married a year and a half after the operation, and continues the exercise of his business pursuits at the present time. Signs of reappearance of the growth are obvious to some of them, and three patients have themselves proposed and are looking forward to a repetition of the operation.

I shall only add two practical remarks suggested by my experience up to the present date.

The first relates to operative treatment, my earliest views of which have been somewhat modified by that experience; the second relates to diagnosis of the tumour.

In regard to the first, I would never operate with a view to removal when a mass of irregular hard deposit is felt in the situation of the prostate and bladder by rectal examination; although for the purpose of removing the urine direct through a perineal opening, the proceeding may be desirable to relieve extremely difficult or painful micturition. The physical condition described being absent, I should prefer in most cases of suspected vesical tumour to open the membranous urethra, by a small incision, so as to admit the finger to explore the interior of the bladder in the manner I have now for some years largely practised and advised. If a polypoid growth is present, which the operator feels he can separate at its neck without much difficulty by the forceps, more need not be done, as in two cases of this series. But if there is a large mass of growth, soft or firm, springing from a broad base or from separate parts of the bladder, the most efficient procedure will almost certainly be supra-pubic cystotomy. There is, I have observed, still another advantage by this method, viz. if the patient has
already lost much blood, he may subsequently be liable to bleed less by this operation than by the perineal one, because haemorrhage is more easily controlled. With only a perineal opening there are no means of controlling subsequent haemorrhage.

Secondly, and this has regard to the subject of diagnosis, it is worthy of remark that when the history of a suspected case of vesical tumour extends over three or four years or more, and the earliest symptom was bleeding, painful micturition, except in the result of obstruction by clots, being inconsiderable, the cause is almost certainly papilloma. On the other hand, when painful and frequent micturition have been the earliest and most manifest symptoms, and bleeding has appeared at a later stage, the disease is likely to be more or less malignant in its nature, and to be either epithelioma, sarcoma, or carcinoma.
Mr. Pitts's Case of Supra-pubic Cystotomy for Tumour. 69

XII.—Supra-pubic Cystotomy for Tumour, two years after Removal of Growth by the Perineal Operation.
By Bernard Pitts, M.C. Read November 26, 1886.

In May, 1885, I brought before this Society a case of tumour of bladder. A villous growth had been removed in September, 1884, by the perineal operation, and I expressed my hope that the cure would be permanent, since the growth was simple in character, had a definite pedicle, was apparently solitary, and the pedicle had been divided close to its origin by means of an écraseur. Having recently had to operate a second time on this patient, I am glad of this opportunity to give the further history of the case.

The patient is a man aged forty-five. He began to have haemorrhage from the bladder about three years before the first operation, or five years ago. When he came under my care he was losing a large quantity of blood, his water was very offensive, he was cachectic and unable to follow his occupation. After operation he regained his health, his water became quite clear, and he had no sign of haemorrhage in it for a year and a half.

In March last he first noticed an occasional trace of blood in his water, and consulted me about this in July last. He was in good health, and actively engaged in his occupation as a watchmaker, but he had then a constant deposit of blood and mucus in his urine; he was occasionally passing small clots; micturition was frequent and attended with some pain. His symptoms during the next few weeks of rest and treatment remained unaltered, and he was re-admitted under my care in St. Thomas's Hospital.

On September 22 I opened the bladder above the pubes, and found that at the site of the old pedicle, viz. just below the right ureter, there was a widely diffused recurrence; the growth was for the most part but slightly elevated, but there were several cone-like projections of the size of the top of one's finger.

These projecting buds were nipped off with forceps, and the broad base of the growth was scraped with a sharp spoon. Scattered over the base and sides of the bladder numerous
small sessile villous tufts could be seen, and, so far as possible, were scraped away, but the bleeding rendered the rest of the operation obscure, and one had to trust more to the sense of touch than to sight.

The rectum was dilated by a bag in the usual manner, but after the bladder was opened it was found more convenient to withdraw the bag, and trust to the finger of an assistant in the rectum to bring the deeper parts of the bladder into view. No attempt was made to close the opening, a soft rubber tube was introduced into it, and the upper part of the external wound was brought together by a silk suture.

The patient made a good recovery. On October 12 he passed four ounces of water by the urethra, and a week later, or one month after the operation, the bladder opening had entirely closed. He is now in good health, the urine is free from any trace of blood, and he has lost all pain on passing it.

The supra-pubic method was selected on the second occasion to enable me to more efficiently examine the bladder, and I would certainly in any future case of exploration of bladder prefer this method to the perineal operation, since it avoids all the possible difficulties of the latter, more especially the risk of accidental haemorrhage. It allows of a very complete examination, and is suitable for any age or altered condition of neck of bladder.

The symptoms at the time of operation were comparatively slight, the man was in good general health, and in a very different condition to his state prior to the first exploration. I expected the growth to be limited and hoped to be able to suture the bladder opening and obtain a more rapid recovery. I confess I was disappointed, and learnt that there may be considerable changes in the bladder with very few symptoms present.

The main growth was found at the seat of the old pedicle. Knowing the position of this to be just below the right ureter, one naturally, after opening the bladder, at once examined this spot for recurrence.

The portions of growth removed were firm, and quite different in naked appearance to what one found on the first occasion. The surface was slightly villous, but on immersion in water there were none of the long fimbriated processes that were so marked a feature of the primary growth.

My impression at the time of the examination was that the growth had undergone a malignant change. On the microscopical examination, however, which was kindly made for me
DESCRIPTION OF PLATE II, ILLUSTRATING MR. PITTS' CASE OF RECURRENT TUMOUR OF THE BLADDER.

Fig. 1.—Represents microscopical appearance of the primary growth.

Fig. 2.—The appearance of the recurrent growth.
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by Mr. Shattock, the recurrent growth proved to be a papilloma. [Sections were shown under the microscope when the paper was read.] The drawings (Plate II) show the difference in the two specimens. It will be seen in the last case that the villous processes are short, the cellular elements more numerous, the cells themselves are oval instead of pear-shaped as in the first specimen, and the connective tissue of the base is less mature and much richer in cells. In fact the tumour presents many suspicious appearances. How far these changes may be due to inflammation, or whether the growth is not now altogether of a simple nature, may be open to doubt. I fear myself that it will recur before long in a more decided malignant form. Naturally the future of this patient gives one great anxiety, since it is only too probable that further operative interference will some time be necessary, and I shall be glad to have suggestions as to the proper course to pursue. I will, in conclusion, briefly state what appears to me to be the line of treatment to adopt.

First, not to be in a hurry to interfere because of slight return of symptoms, but to wait until the patient is rendered unfit by hemorrhage or exhaustion to follow his occupation. Then to again open his bladder above the pubes, and to remove the growth as freely as possible. If on examination the growth presents no further evidence of a malignant change, to be content with this, and trust for a renewed lease of life and comfort to the patient. But in case there be distinct evidence of change in the character of the growth, or if the growth be so luxuriant that successive operations fail to keep it in subjection, then it would seem one's duty to give the patient a chance of life by diverting the ureters and subsequently removing such parts of the bladder as necessity compels, and, provided that the kidneys are healthy and the patient's general condition permit, I believe this may be done without unwarrantable risk. The method that suggests itself is as follows:

First, to ligature the ureter on one side by a lumbar incision, and to open the pelvis of the kidney. So soon as the patient has become accustomed to this alteration in his urinary apparatus, to do the same operation on the opposite side. By preference (as suggested to me by Mr. Ballance) the ureter might be reached by making an incision as for ligature of the external iliac artery, and after reflecting the peritoneum, ligation could be done as near the bladder as possible, and the end brought to the surface at a separate opening.
The diversion of the ureters into the rectum would seem too dangerous, and even if successful there would be great risk of a septic change subsequently taking place in the kidneys.

If one succeeded in diverting the ureters and thus rendering the bladder a passive organ it would be possible to deal effectively with the growth through the supra-pubic opening, either by application of caustics or by stripping back the peritoneum as far as the attachment of the ureters and then removing the greater part of the organ, taking away the mucous coat from such part of the bladder as from the connections it might be unadvisable to otherwise meddle with.

The operation, or rather series of operations, that I have ventured to suggest, will, I fear, almost seem to involve too much risk to entertain. The same, however, may be said of many surgical procedures that have proved successful, and it must be right to offer a patient the chance of prolonging his life, provided he be fully informed of the risks and the subsequent inconveniences.

June, 1887.—Since writing this paper the patient has again come under observation in the hospital—he has on one occasion had sharp hæmorrhage—but the urine is now free from blood. There has been for several months severe pain in the left thigh, and there is marked wasting of the left leg, and a growth can now be felt deep in the pelvis on the same side. There can be but little doubt therefore of the malignant nature of the case, which, on examination three years ago, was ascertained to be a simple pedunculated tumour. A report will be made of the subsequent history.

In July, 1885, I was asked by the house surgeon to see a case which had just come in to the casualty department of the West London Hospital. The patient, David B., aged 22, a riveter by trade, had just fallen from a ladder, the height of which he estimated roughly at 20 ft., upon his right hand.

There was much pain in the right elbow-joint and effusion had already set in. All the movements of the joint were limited. There was no dislocation. The shafts of the radius and ulna could be felt to be intact; but owing to the effusion it was difficult to be sure whether the head of the radius rotated with the shaft.

On making pressure over the head of the radius and rotating the arm distinct crepitus could be felt and considerable pain was experienced. At this time I was not aware that the head of the radius could be fractured vertically, and I diagnosed a fracture of the neck. The arm was accordingly fixed on a rectangular splint with a pad in front of the head of the radius and kept so for three weeks. At the end of this time the splint was removed and passive movement and rubbing commenced.

I saw the patient again, thirteen weeks after the accident, during which time two attempts to move the joint under an anaesthetic had been made without any good result.

The condition was then as follows:

The ulna is apparently intact. The measurement between the two condyles of the humerus is about half an inch more on the right side than the left. The external condyle is ill defined. The head of the radius rotates with pronation and supination, but appears not to be in a line with the shaft of the radius but to be set on at an angle with it. The length of the two radii is the same. Flexion and extension can only be performed through an angle of 30°, and pronation and supination are reduced to about half.
He is quite unable to work.

The patient, being anxious to have something done to relieve him, I determined to make another trial to move the arm under an anaesthetic and, should that fail, to make an exploratory incision and remove any portion of bone that was found to be limiting movement.

Accordingly, on October 16 (thirteen weeks after the accident), he was put under ether and a steady but somewhat forcible attempt made to flex the forearm. As this was being done something was felt to give way with a grating like bone. No increased movement followed, however. I accordingly proceeded to make an incision about 3 inches long, over the posterior part of the head of the radius in the groove between the supinator longus and radial extensors of the carpus and the other extensors.

I at once disclosed the head of the radius and perceived that it was divided by a vertical cleft, now filled with fibrous tissue, in two nearly equal halves. As the head, thus enlarged by one third of its normal width, seemed quite enough to limit movement to the extent described, I determined to remove it, which was easily accomplished by dividing the neck with a narrow saw. I next attempted to flex the joint but found that some obstacle still remained.

On examination with the finger, a piece of bone could be felt broken off but still adherent to the periosteum; this was removed after some trouble with sequestum forceps in two pieces and proved to be the greater part of the coronoid process of the ulna. The arm could now be flexed until the hand touched the shoulder and pronation and supination were quite free.

I did not feel any split in the humerus, but the cleft might have been filled, like that in the radius, with fibrous tissue. The coronoid showed a recently fractured surface; and this was, no doubt, what was felt to give way when forcible flexion was attempted before the operation; but, I think, one cannot doubt that there must have been a previous fracture which had united imperfectly and in bad position or it could not have prevented flexion nor have broken so easily. The operation had been performed throughout with the strictest antiseptic precautions.

I inserted a deep chromic-acid catgut stitch to keep the muscles in place, and put a medium-sized drainage-tube into the joint; the rest of the wound was closed with chromic-acid catgut stitches. The dressing was sero-sublimate.
The operation was followed by no rise of temperature or other constitutional disturbance, there was no discharge after the first dressing, except a small quantity of serum. The skin wound united by first intention, except where the drainage-tube had been. The track of the tube was prevented from closing for some time by the knot of the deep catgut stitch which acted as a foreign body and at last came away, after which the minute opening closed at once.

He was discharged on November 16, just a month after the operation, with the wound entirely healed, but no movement.

Soon after his discharge he fell whilst attempting to get into a tramcar and split open part of the cicatrix; but, fortunately, did not open the joint. I had to wait for this to heal, and so it was not till a month after the first healing of the incision that I made an attempt to break down the adhesions which had occurred in the parts about the joint. On attempting to move the joint under ether, these adhesions at once gave way and full movements could be made in all directions. Since this, however, there has been some return of the stiffness or, rather, I should say, some limitation of flexion which seems to be due to a production of bone by the periosteum of the coronoid process.

The patient soon after this returned to his work and has gone on steadily improving. Pronation and supination are almost perfect, and the elbow can be flexed to a right angle.

I showed the patient, and the pieces of bone removed, to this Society in May last, and his condition was then as above stated. I will now briefly describe the condition of the head.

It is split by a vertical transverse fracture into an anterior and a posterior portion. The posterior part measures ½ in. from before backwards and is continuous with the shaft; the anterior part, ⅜ in. from before back and is displaced downwards to the extent of about ½ in. and its surface is tilted forward so that it joins the neck below to which it is united by bone, but its cartilaginous surface forming the anterior part of the cup is separated from the posterior by a gap of ⅜ of an inch which is filled with fibrous tissue.

There has been a complete fracture, as shown by the crepitus, which has imperfectly united in a bad position. The two portions of the coronoid are irregular in shape and show clear signs of fibrous union.

That the form of injury above described is of some rarity seems evident from the fact that its existence is ignored in most of our text-books of surgery.
Mr. Bryant had a case in which the coronoid was also fractured and the injury compound.

Malgaigne mentions two cases, in both of which amputation was performed. The coronoid was also fractured, and there was the complication of dislocation backwards.

Ashhurst states (International Encyclopædia of Surgery, p. 155) that there are two such specimens in the Warren Museum, both of which are complicated by fractures of the coronoid, one of which was comminuted. He also quotes Stimson as having seen a fracture of the outer half of the head of the radius, produced by direct violence, and followed by suppurrative arthritis in a boy of thirteen years. Excision of the joint established the diagnosis.

Adams exhibited at the Pathological Society (Trans., vol. xxii, 1871, p. 205) a specimen in which several fissures radiated from a point just below the head of the radius, up towards the articular surface, caused by a fall from a height.

Ashhurst adds that other cases followed by recovery, and hence open to doubt, have been reported.

Mr. Gant, in his Science and Practice of Surgery (p. 642), says, "Fracture through the head of the radius occurring alone, has, I believe, never been met with. In conjunction with fracture of the coronoid process of the ulna and dislocation of both bones backwards, this form of injury has occurred in two instances, both in the same subject."

But most of our information on this subject is due to a paper by Prof. Bruns, of Tubingen (Centralblatt für Chirurgie, No. 22, 1880), which has been reported by Mr. Johnson Smith in the London Medical Record for 1880. In this paper Prof. Bruns gives twenty-one cases, proved by dissection. He considers that these numbers give no idea of the frequency of the injury, as many are very difficult, and some impossible to diagnose. He describes two forms (one incomplete, two complete).

Bruns has produced both forms experimentally—the incomplete twice, and the complete once.

"The incomplete form commences at the discoidal articular surface, and passes down the neck, ending in a blind extremity. The fragments may remain in close apposition, or be more or less widely separated. This form may also be fissured in several directions.

"In the complete form a fragment is detached completely, usually from the anterior part of the head, but is kept in
position by the ligaments. The neck may or may not be involved."

In five out of his twenty-one cases the fracture of the head was uncomplicated. The most frequent complication is fracture of the coronoid.

The most frequent causes are a fall on the flexed elbow or extended hand, and direct injuries from fall of heavy body, machinery, or gunshot wound. Fracture from indirect violence is probably commonest, caused by the impact of the head on the capitellum of the humerus. It is more likely to occur in extension than in flexion. Hence the anterior portion is usually broken off, and hence also the fracture of the coronoid.

Bruns mentions the possibility of diagnosis by crepitus during pronation and supination, and by the broadening of the head felt posteriorly in cases where there is considerable separation of the fragments. In his collected cases there are three with bony union, and some displacement (one upwards, one downwards, one outwards). In one case the fragment had united with the coronoid. In three other cases the fragment had been converted into a free articular body.

Dr. O. Pinner,* of Freiburg, reports two interesting cases, of which I add an abridged account. He considers that Bruns has established this injury as one of the definite and typical fractures. He thinks the paucity of reported cases is due to the extreme difficulty in many instances of making a diagnosis.

One of his two cases is that of a colleague of his own, who tripped and fell upon both palms with the arms extended. The accident was at once followed by weakness in the right hand and forearm, but no impairment of movement was noticed. In an hour severe pain and swelling occurred in the elbow-joint. Dr. Pinner examined the arm shortly after, and could not discover any displacement of bones, as the arm was fat and there was much pain and swelling. The localised tenderness at the upper third of the radius led him to think a fracture in that position possible, and the arm was accordingly put up in full supination. The next day the patient could not extend his fingers.

The following day, the swelling having subsided, acute pain and crepitus were elicited by pressure on the outer and fore part of the head of the radius. The head of the radius rotated with the shaft, but there was limitation of pronation and supination. There was paralysis of all the muscles supplied by

* Zeitschrift für Chirurgie, 1884.
the posterior interosseous nerve. The joint was fixed for three weeks, after which electricity was employed, and finally full movement of the hand was restored. (N.B.—It is not stated whether the full movements at the elbow were restored.) A thickening could be observed over the outer and fore part of the head of the radius, and a kind of crackling could be felt as if the smoothness of the surface was broken at some points.

Dr. Pinner's other example of this injury, accompanied by illustrations, was taken from an anatomical specimen (of which he could obtain no history) belonging to the collection of the Pathological Institute at Freiburg. The bones are those of an adult. In this case one half of the head of the radius has been broken off, and remained adherent to the capsule, and the fractured portions, instead of uniting, have formed a false joint by eburnation of their surfaces.

In conclusion, I may say that the special interest of the case which I have just related to the Society is that, as it would appear, contrary to the general rule, the injury in my case caused so much loss of power in the arm that the man was completely incapacitated from following his business, and that by the removal of the fractured portions of bone he has been restored to a life of useful activity.

This case agrees with those reported by Prof. Bruns in all respects, and I cannot but think with him that, when this form of fracture is more generally recognised, it will not be found to be so very infrequent.
ON February 18, 1886, I admitted Emma C., æt. 9, into the West London Hospital.

**History.**—A week before Christmas she was taken ill with sore-throat. She was in bed three weeks at home. During convalescence food used to return partly through her nose. She also spoke through her nose, and when she left her bed found that she had partly lost the use of her legs.

Her illness was not known to be diphtheria. There were five other children in the family. Only one, a boy of three, was ill at the same time. He was only poorly for three days, and had no paralysis afterwards. It was not known that his throat was sore. Several other children, however, had diphtheria at the same time in the same street.

On admission, she was a ruddy stout girl; her voice was nasal, her palate drooped a little more than natural, but food no longer came through the nose. Her gait was so tottering and unsteady that she could neither walk nor stand alone.

Examining the legs further I found no wasting or flabbiness; the power of resistance I could not well determine but believed it to be diminished, the knee-jerks were absent, and there was no ankle-clonus. There was no anaesthesia, and she could give no account of subjective sensations. The superficial reflexes were natural. The muscles contracted easily under faradic currents.*

The heart's apex was just within the nipple line, in the fourth space, its beat feeble, and its sounds clear.

The urine was not albuminous.

The nasal voice vanished in a few days, but otherwise she remained the same for about three weeks until, on March 4, I found a soft systolic murmur and occasional irregularity of the heart's action while lying in bed. After some slight exertion the irregularity was very considerable. On the 16th the heart was regular. On the 24th, the apex was in the nipple line,

* No history or sign of paralysis within or without the eye; no loss of sight. The arms were never affected, nor the sphincters.
the action regular even after exertion, and a systolic murmur was heard at the apex. The murmur and the enlargement of the heart persisted from that time to the present (September 23, 1886), and the murmur was traceable slightly outwards, though not round to the back.

The gait had meanwhile been improving. On April 8 there was still no knee-jerk. In voluntary movement I noticed that the muscles did not contract like those of health, but with visible intermissions forming a clonus. There was, however, no ankle-clonus made by stretching the calf-muscles.

Reaction to faradism was more difficult than before, as compared with my own muscles. On April 15 these clonic contractions were the same, but the knee-jerk had returned, and was somewhat excessive, and there was ankle-clonus on both sides. These signs continued until on May 13 the knee- jerks were about natural, less than they had been up to that time, and ankle-clonus was only obtained on the right side. On May 20 no ankle-clonus was found, but voluntary movement was still clonic. On May 27 the muscles reacted easily to faradism. On June 3 movement was performed without any clonus.

On March 4, while the girl was still an in-patient, I admitted John B., æt. 4, to the same ward for diphtheritic paralysis.

_History._—Early in February he could not walk or stand. In the middle of February he squinted for three or four days. He came for the weakness of the legs.

He had not been noticed to be in any way ill before the weakness began. At no time had he any sore-throat, nasal voice, or difficulty in swallowing. His sister Florence, æt. 8, had a bad sore-throat in Christmas week, with a swelling of the neck which kept her in bed a fortnight. A short time afterward his mother had a sore-throat, and in the beginning of January a child living in the next house died of diphtheria in the West London Hospital.

On admission, there was no paralysis except in the legs. He could not stand or walk alone. The condition of the legs was exactly that of Emma C., no wasting, no loss of sensation, and natural superficial reflexes, with no knee-jerk, and easy reaction to faradism. There was no albuminuria.

The heart’s apex was in the fourth space, half an inch inside the nipple line, with clear sounds and regular beats.

On March 21 the heart-beat was rather irregular, and the first sound indistinct at the end. On April 8 the apex was in the nipple line, but there was no murmur, and it remained
so all the time he was in the hospital. He was sent out on May 8. On June 3 the apex was in the nipple line, and a systolic murmur was heard all over the heart in front. This remains until the present time (July 22).

The legs had meanwhile improved. On April 8 he could walk alone, and on this day movement, whether voluntary or produced by pricking the feet, was clonic. There was no knee-jerk or ankle-clonus. The muscles did not react so easily as before to faradism. The clonic character of the contraction lasted about two months, and gradually faded until on June 3 the muscles contracted as in health, and on June 17 the knee-jerks appeared. He never had ankle-clonus or excessive knee-jerk.

There will be no doubt that the first of these cases is one of diphtheritic paralysis. The second is more open to question, but, when considered, not more doubtful. The paralysis is like that of diphtheria alone, the surroundings are those of diphtheria, and it is not hard to suppose that the sore-throat was unnoticed, or even that he never had any. An epidemic has been described in France where, in many cases, paralysis was the first sign of illness, and no sore-throat occurred.*

The cardiac murmurs are interesting. I do not think them hæmio, because the children were not anæmic themselves, and because the murmurs lasted so long. This persistence, together with the fact that the pulse was regular and fairly strong, led me to believe that there was valvular disease. Supposing the affection valvular, is it due to diphtheria? I do not think that rheumatism can be alleged in the case of the girl. There was no history of it in herself or in her family, and the murmur developed while she was lying in bed under our eyes. In the case of the boy this is not so clear. He has never had rheumatism himself that I can learn, but a sister has had rheumatic fever, and another, aged 8, who has never had rheumatism has a distinct mitral bruit.† Again in his case the murmur appeared only after he had left the hospital and was walking about. It is therefore doubtful whether the affection is rheumatic or not in his case.

I do not know that any accurate observations have been made of hearts from cases of diphtheritic paralysis. That diphtheria affects the heart has been both stated and denied. Labadie Lagrave in 1873 stated that twenty-two cases out of

* Boissarie, Gaz. Hebdom., 81, Nos. 20, 21.
† This sister had, however, a severe sore-throat—probably diphtheritic—at Christmas.
forty had vegetating endocarditis, and the lungs sometimes
infarcts. His vegetations, however, were extremely small, and
were denied by other observers to be vegetations at all, while
his infarcts are not at all convincing. Bouchut taught the
same doctrine.

During eleven years, October, 1867, to October, 1878, there
were examined at St. Bartholomew's twenty-two cases of
diphtheria, of which notes remain. In six cases the heart was
altered.

M. 33. Pericarditis.
M. 3. Pericarditis and recent mitral granulations.
M. 1. Minute granulations on mitral.

It is curious that though there were eleven of each sex, the
hearts of the females were all untouched.

These deaths, however, occurred while the diphtheria was
attacking the throat. In the case I have just related the
heart disease came on long after this had ceased. I can no-
where find a similar case described.

The state of the leg-muscles is also interesting, and here
again I have been unable to find any cases watched all through
the paralysis, until the actions and reactions were again
healthy.

Déjerine, Abercrombie, and Kidd have found changes in
the spinal cord. Up to that time they had not been discovered
and since then Dr. Hebb was unable to find them. That they
are at any rate not systematic is shown by the great variety
of symptoms which the disease possesses. I expect that there
is some alteration in the minute part of the circulatory system
which affects at one time the sensory, at another the motor
parts, and at another both together, and while it lasts perhaps
visibly alters the nervous structures.

The excessive knee-jerk and the ankle-clonus, developed
in the girl, were not present in the boy, and are therefore
evidently not necessary stages in recovery. The question
arises, What produced them? Hitherto the presence of these
two symptoms has been held to mean disease of the lateral
columns, and two theories have been formed to account for
them,—one that the inhibitory power of the brain was inter-
rupted, the other that a structural alteration of the grey matter,
spreading from the lateral tracts, caused an increase of the
normal myotatic irritability.
I am unable to understand how they were caused in this case, but at least there is no symptom pointing to sudden involvement of the lateral columns; they occurred during convalescence from what has been supposed to be an affection of the grey matter. If, as I venture to guess, the paralysis was the result of some change in the blood supply, say diminution, the jerks and clonus may mean the return of the blood-flow to grey matter for some time unaccustomed to such good feeding.

Note as to subsequent condition:
April 28, 1887.—Emma C. Heart's apex in the fifth space, nipple line; impulse rather stronger than normal. No definite murmur; first sound rather prolonged; second sound accentuated at base.
June 6.—Ditto.

April 28.—John B. Heart's apex in the fifth space, just within nipple line; impulse not increased; sounds natural.
June 6.—Ditto.
Mr. Treves's Case of Acute Myositis.


A CARMAN, set. 22, was admitted into the London Hospital on December 20, 1884, with an "inflamed arm."

The patient was a dark-complexioned man, spare, vigorous, and muscular. His account of himself was as follows: three weeks before admission he was seized with a severe shivering fit while driving his van. It was a rainy morning in mid-winter and very cold, and the patient's clothes were entirely wet through. He managed to drive for another hour, feeling exceedingly ill, but when he came to his journey's end he was quite unable to unload his van. After sitting before a large fire for two hours he felt better and resumed his work. He now found that he was unable to grasp the reins, because his hands were weak and felt cramped. The sense of weakness was apparent in both hands, but more especially in the left wrist. About each wrist there was a vague pain like that of cramp. The patient remained at home next day, feeling ill in himself and unable to eat. He did not leave the house again until he came to the hospital. For three days there was pain with stiffness about the right wrist, but the part was never perceptibly swollen, and soon regained its normal condition. On the third day after the rigor severe cramp-like pains appeared in the calves of the legs, but especially in the left calf. This part was swollen and tender, and he was unable to move the leg or foot without much pain. These symptoms persisted only for three or four days, when they all passed away. The left hand and wrist, however, still remained painful, the pain increasing rather than diminishing. On the third day after the commencement of his illness a swelling appeared in the front of the left forearm, about its middle. This swelling gradually spread up the limb until it reached the shoulder, and within nine days of the initial chill the whole of the left upper limb was swollen, tender, painful, and stiff. The swelling appears to have been limited to the front of the arm and forearm, and the limb became stiff with the elbow flexed. The patient ascribed this condition to rheumatism, and did not consult any medical man. As the arm did not improve he came to the hospital twenty-one days after the
rigor that marked the onset of the trouble. There had been no subsequent rigors, and the patient was emphatic in asserting that he had enjoyed excellent health up to the date of the chill.

On examination a considerable swelling of the left arm was evident. The whole of the deltoid muscle was enlarged, was very hard, tender, and rigid. The upper half of the biceps was occupied by a like hard swelling, that clearly brought out the details of the muscle. In the forearm an induration of identical character occupied the upper part of the muscles arising from the outer condyle of the humerus. The hardness of the swelling in each case was pronounced. The parts were exceedingly tender, but not painful unless the limb was moved. The skin was of normal appearance, but there was some trifling subcutaneous oedema over the affected muscles. There was no oedema elsewhere. There was a fair degree of active movement in the hand and wrist. A slight rotation of the radius was possible, but no active movements could be effected either at the elbow or shoulder.

All attempts at passive movement at the shoulder were resisted, and caused considerable pain. A moderate degree of flexion at the elbow was well borne, but any attempt at extension caused much distress. The lower half of the biceps muscle appeared to be normal. It was a little tender. The line of demarcation between the upper and lower segments of the muscle was well marked. The elbow was fixed at a right angle. The patient asserts that the swelling in the forearm had been much more extensive, but was subsiding. No other muscles of the body were affected, and no effusion could be detected in any of the articulations. The patient's temperature on the evening of December 20 was 103°. He had lost flesh, and felt weak and ill. His tongue was clean, his bowels regular, and his appetite fair. Urine normal.

The patient was kept in bed. An ice-bag was placed on the shoulder, and evaporating lead lotion on the arm and forearm. Iron and quinine were administered. The temperature soon sank to between 99° and 101°, and after some fluctuations became normal on January 3rd.

The improvement in the limb was slow but continuous. The swelling became less hard and less tender. The oedema disappeared. Within seven days of admission the induration in the forearm had quite gone. Within three weeks of admission the induration in the deltoid muscle had disappeared, but there was still a firm hard swelling in the upper part of
the biceps muscle. This swelling had ceased to be tender. The patient was up and in excellent health, but still carried his arm in a sling. The active movements in the hand and wrist, together with pronation and supination, were perfect. The elbow and shoulder were still very stiff, the latter especially. Massage and friction with liniment were now employed, and active and passive movements encouraged. Six weeks from the date of his admission, and nine weeks after the commencement of the attack, the patient was discharged. Nothing abnormal could now be detected in the muscles, and, apart from some weakness in the deltoid and biceps, the use of the limb was unimpaired.

That this was a case of acute or subacute myositis there can be, I imagine, no doubt, but the cause of the trouble and the character of the inflammation must be points open to question. Myositis, in the sense in which the term is usually employed, is an uncommon affection. Primary myositis independent of actual and direct lesion, of diathesis and of parasites, is certainly very rare, and the existence of such an affection is doubted by more than one author. Before discussing the forms of myositis with which this case might claim connection it will be well to dispose of the question of rheumatism in connection therewith. I imagine that at the present day it is allowed that the local changes in so-called muscular rheumatism are independent of a true inflammatory process. Such, however, has not always been the state of the scanty pathology of this affection. In the early part of the century the present
case might well have been described as acute muscular rheumatism since that term covered nearly all painful affections of muscles. Ozanam,* writing in 1814, describes a case of acute muscular rheumatism ending in suppuration and death, and in other examples of disease similarly named the muscles were obviously the seat of considerable inflammation. In the case now brought forward the patient himself had never had any form of rheumatism nor was any member of his family liable to that affection. It may here be observed that seven weeks before the date of his illness the patient had a slight attack of gonorrhoea. The discharge ceased without treatment in about a fortnight. There was no trace of urethral trouble when the patient was admitted.

An examination of the literature of myositis, to which, by-the-bye, the text-books contribute singularly little, makes clear the following fairly definite varieties of the affection:

1. Simple myositis.
2. Myositis from cold.
3. Infective myositis.
4. Myositis due to parasites (Trichinosis).
5. Secondary myositis, including the embolic form.

This classification is no doubt artificial but it probably represents the state of our knowledge of the subject at the present time.

1. The term simple myositis may be applied to such inflammations of muscle as depend upon injury or extension, and may be illustrated by the changes that occur in lacerated muscles (e.g. about fractures) and in such a condition as psoas abscess. This variety can hardly be said to have a clinical being.

2. Myositis from cold is an affection the existence of which is doubted by many. Cold as a factor in the etiology of disease occupies a somewhat uncertain position. It is often used to cover ignorance of the real cause of a disorder, and has probably been credited with an influence it does not possess.

Hayem,† whose monograph on diseases of muscle is the most complete with which I am acquainted, recognises the variety under the title of acute or subacute spontaneous myositis. He states that it usually occurs in young subjects and is met with most often in the calf and pectoral muscles, then in the outer muscles of the forearm, the deltoid, the biceps and the outer muscles of the leg. It commences with rigor and is attended

* Journal Méd. de Corvisart, Feb., 1814.
with moderate fever. The local trouble begins with pain and tenderness. Swelling in the muscles appears from the third to the seventh day, and becomes peculiarly hard. ÒEdema of the subcutaneous tissue is common. Suppuration of the affected muscles is rare, and the average duration of the myositis is twenty-eight days. It ends then in resolution.

The case I have described appears to me to belong to this category, although I am aware, as already stated, that there are some who doubt the existence of such an affection. The chief features of this case coincide markedly with Hayem's description, and the localisation of this disease also very pointedly supports his account. The fact that only a part of the biceps and of the outer forearm-muscles was involved illustrates his assertion that the myositis is more often partial than diffuse.

My patient was certainly exposed to cold in a very definite and severe manner. At the time of the chill he was enjoying the most robust health, and the sequence of events was so precise that there is ample reason for ascribing the myositis to the unusual exposure. It will be observed also in speaking of the other varieties of myositis that there is negative evidence to the effect that the present example cannot well be reckoned with any one of them.

3. Infective myositis. This interesting affection has not received the attention it deserves. It is akin to osteo-myelitis and the infective form of periostitis. It very commonly ends in suppuration, and the more severe aspects of the disease when suppuration occurs cannot well be separated from pyæmia. This form has been described by Dr. P. S. Connor.* The condition is met with in children or in young adults, and especially in the ill-fed and unhealthy. It has been noted, however, in robust individuals. It commences with rigor and is attended with fever. One or many muscles may be involved, and the severer suppurative forms, unless treated by very early and free incisions, are nearly always fatal. Hayem† states that such forms are invariably fatal. There must always be some primary focus of infection. Professor Scriba‡ has recently furnished a valuable account of four examples of the less severe form of the malady. The patients were all healthy males between the ages of seventeen and thirty-three years. The infecting focus was in three instances a small boil, and

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* Ashhurst, Encyclopædia of Surgery, vol. iii.
† Loc. cit.
‡ Deutsche Zeitschrift für Chirurgie, 1885, p. 496.
in the fourth case an abscess of the mouth. While suppuration was proceeding a rigor occurred followed by fever and great pain in certain muscles. A hard swelling in the affected muscles next appeared. In one case one muscle only was involved, in the others three or four muscles. The structures selected were the triceps humeri, the vasti, the pectoralis major, the adductor longus, and the soleus. In two cases suppuration of the muscle occurred, and was treated by free incision. In all instances perfect recovery followed in from three to eight weeks. Dr. Garré* and others have demonstrated the infectious character of both boils and carbuncles, and have proved that they may serve as foci for osteo-myelitis. The resemblance between osteo-myelitis and the present affection is striking. In my own patient there was no reason to suspect this form of myositis. He had had no boil, and no suppurating focus of any kind could be discovered.

4. Trichinosis. The feature of the myositis that forms so conspicuous an element in this disease need not be dwelt upon. With regard to my own patient it is by no means easy to deny the possibility of trichinosis. There was this element in the history. The man was a bachelor and lived alone. He for the most part cooked his own food, and the staple article of his diet was pork. Pork would constitute his sole meat meal on at least four days in the week. There is no evidence to show that he was an inefficient cook. Now, the clinical picture of severe and fatal forms of trichinosis has been elaborately drawn, but more information is required with regard to mild attacks. That "man may tolerate a considerable number of trichina without becoming ill" is allowed by others than Heller,† the author of the statement. There are again mild cases associated with marked muscle symptoms that end in recovery in from six to eight weeks. Heller states that "the most marked initial symptom, present in the mildest as well as in the most severe case, is muscular lameness." The muscles so affected are flabby, weak, and painful if called into action. This symptom may be said to have been present in my patient. Other points in favour of the diagnosis of trichinosis are these. The temperature chart has a curious resemblance to the chart given by Heller as typical of mild trichinosis. Then again in rare cases the disease may commence by a rigor. Trichinae are found most often and in greatest numbers near the tendinous end of muscles. The more tendinous end of

* Fortschrifte der Medicin, 1885, p. 105.
† Ziemssen’s Cyclopaedia of Medicine.
the biceps was conspicuously affected in my case. Against the theory of trichinosis, on the other hand, are these points. The onset of the affection was sudden and violent, the local symptoms were severe. It is difficult to understand that two or three muscles should be rigorously attacked, and yet the disease appear in no other part, and steadily progress towards recovery. If the muscular lameness and rigor belong to trichinosis then the myositis followed at too short an interval. This, I imagine, is a vital objection to this special diagnosis. From Heller's cases it would appear that in trichinosis the muscle phenomena do not become manifest until from the eighteenth to the fortieth day. In my patient there were no preliminary gastric symptoms, although I am aware that they may be absent in the parasitic disease, there was no oedema of the eyelids or face and no sweating. On these grounds, I think, any suspicion aroused by the patient's diet may be put at rest.

5. Secondary myositis. Under this heading may be classed those forms of muscle inflammation that appear during certain acute fevers, typhoid, typhus, smallpox, &c., the myositis of syphilis, that met with in erysipelas, diphtheria, pyæmia and puerperal fever, and the myositis of scurvy, glanders, and ulcerative endocarditis.

The myositis of syphilis is met with in the familiar chronic swelling on the sterno-mastoid of infants, and in the manifestations of the acquired disease. (I may here note that my patient had never had syphilis.) In scurvy the trouble follows upon haemorrhages into the muscle, and in farcy upon bacterial deposits therein.

The myositis that attends ulcerative endocarditis depends upon embolism, and probably the same accident underlies the muscle complications in other of the diseases enumerated. Several cases have been recorded of acute inflammation with suppuration and even necrosis of muscle depending upon embolism in patients who at the time were suffering from no specified disease. In the present patient there was no evidence to suggest that the myositis was secondary in the sense in which the term is here used.
XVI.—On a Case of Gastric Ulcer with Unusual Complications. By H. Montague Murray, M.D. Read December 10, 1886.

A G., an anaemic-looking girl, æt. 23, was admitted into Charing Cross Hospital on February 15, 1886, under the care of Dr. Mitchell Bruce, to whose kindness I am indebted for permission to use the notes from which the following summary is principally taken.

Excepting occasional indefinite attacks of indigestion, her previous health had been uniformly good. There was no family history of disease. Her work, as housemaid, had been, for the last year, exceptionally heavy. The menses had been scanty for nearly all that time, and she had "seen nothing" for two months. Three months before admission she "caught cold," and had been ailing ever since. During this time she had complained of frequent headache, of dyspnœa and palpitation on the slightest exertion, and of nausea and epigastric pain occasionally followed, and still more rarely relieved, by vomiting. There had been at times an aching, inter-scapular pain. She had never vomited any bright blood, "coffee grounds," or anything except partly digested food.

On admission, beyond the general pallor of skin and mucous membranes, there were no physical signs of disease. The tongue was clean and moist. Temperature was 99.2° F. Her diet was restricted to fluids, and for nearly twenty-four hours she was free from pain or discomfort.

During the afternoon of the next day (February 16) the temperature suddenly rose to 103.6° F.; a sharp pain was experienced in the lower part of the left side of the chest, and in the epigastrium. Over the former area a distinct pleuritic rub could be heard. The movements of the left side of the chest were diminished. There was no abdominal pain or distension. The legs were not drawn up. Vomiting occurred during the evening. The vomit was free from blood. Hot fomentations were applied to the seats of pain, and morphia (gr. ½) was given hypodermically.

Next day (February 17) the pain had increased in severity, and, in addition to that just described, was now felt between the shoulders. Vomiting was almost continuous, and seemed
to be provoked by the sucking of ice. The vomit consisted of small quantities of pale, yellowish-green fluid without any solid matter. The abdomen was slightly but uniformly tender and distended. Nutrient enemata were substituted for the fluid diet. Opium pills (gr. j) were given every three hours and morphia (gr. ½) was injected in the evening.

On the 18th the pills were rejected from the stomach and therefore discontinued. Four only had been given. All the symptoms were aggravated. The distension of the abdomen was most marked in the epigastric region. The tongue was dry and red and patient complained of great thirst. Temperature varied from 100° F. to 103° F.

Early in the morning of the 19th patient suddenly vomited without any effort 10 oz. of bright red blood. The pain and thirst were worse than ever, and persistent hiccough increased the general distress. Two grains of ergotine were injected, and to each enema Tr. Opii ⅛ were added. As the pain was in no way relieved, injections of morphia (gr. ½) were continued every four hours. The hiccough ceased toward evening.

On the 20th patient was in every way worse; the pain was quite unrelieved although she was taking three grains of morphia in the twenty-four hours. The vomiting had persisted at frequent intervals since the previous morning, more than 25 oz. of coffee-ground material being vomited during the day. The abdomen was much more tender and much more distended, especially at the upper part. The urine was free from albumin. There was no drowsiness, nor any marked contraction of pupils.

On the 21st patient was much weaker. There was no abatement of any of the symptoms. The character and quantity of the vomit remained about the same. The pulse, feeble and rapid throughout, now became imperceptible. The vomiting ceased about 10 p.m., and the patient died quietly at 2.30 on the morning of the 22nd.

At the post-mortem examination, the acute left pleurisy and acute peritonitis were very distinct. The lower lobe of the left lung was collapsed; the pleural surfaces were roughened, and covered with a flaky inflammatory deposit, which extended from the parietal to the visceral surfaces, and from the meshes of which nearly half a pint of sero-purulent fluid escaped.

The peritonitis was general in distribution and uniform in degree. Nothing suggestive of the escaped contents of the
Dr. Murray's Case of Gastric Ulcer.

stomach could anywhere be found. The appearances of the inflammatory exudation were very similar to those in the pleural cavity already described.

On opening the stomach after its removal from the body it was found to contain 2 oz. of acid, reddish-brown, grumous fluid.

On the posterior wall, about an inch to the right of the cardiac opening, and the same distance from the lesser curvature, there was a deeply excavated ulcer. Its outline was roughly, but not accurately, circular; its diameter an inch; its walls thickened and precipitous; and its floor composed solely of the peritoneal coat. In the centre of the floor was a small circular aperture one eighth of an inch in diameter; the rest of the mucous membrane was healthy. The peritoneal surface of the stomach in the neighbourhood of the ulcer showed no evidence of special local inflammation.

No other organ was found to be diseased.

Three very interesting questions presented themselves while the case was under observation:

1. What was the exact condition of the stomach at the time of admission?
2. When did the perforation occur?
3. Was the ulcer the exciting cause of the pleurisy and peritonitis?

1. My own diagnosis, made on the day of admission, was, "Anaemia with such functional disturbance of the stomach, or such slight impairment of its nutrition, as is common in that condition." It was based on the absence of any definite signs of ulceration. Beyond the general liability incurred by persons of patient's age, occupation, and condition of health, there was no reliable evidence pointing to it. The vomiting was infrequent, bore no definite relation to the ingestion of food, and was not usually followed by any distinct relief of pain. There was no tenderness, and the patient's statements were not sufficiently consistent to justify any great importance being attached to the character of either the epigastric or the interscapular pain. Yet the sequel shows that the ulceration must have been going on at this time—both for anatomical and for clinical reasons. Only six full days elapsed between the patient's admission and the fatal issue. The somewhat irregular outline of the ulcer, the absence of the other coats of the stomach in its floor, and the distinct swelling of its walls, point to a longer duration than this. Nor on a priori grounds was ulceration likely to have arisen after admission; for though
rest in bed and fluid diet may be, and in this case were, powerless to arrest the process, they were not likely themselves to take any active part in bringing it about. On the other hand, the ulcer could hardly have existed as long as the gastric symptoms—three months. It is impossible, with the post-mortem appearances as our only guide, to speak with certainty concerning its duration. It began insidiously, possibly two weeks or more before admission, and did not give rise to any absolutely characteristic symptoms, until the hæmorrhage of the 19th, which occurred after the onset of the pleurisy and peritonitis, and was probably induced by the persistent vomiting of the previous day.

2. When did the perforation of the stomach take place? As a rule, whenever perforation of the stomach or intestine is found in connection with peritonitis, the former is the immediate cause of the latter. But this case seems to be an exception to, rather than an example of, the rule. Both the post-mortem appearances and the clinical history were inconsistent with a perforation of long standing, but compatible with one of only a few hours' duration. When the stomach was first lifted from its place no perforation could be seen, none of its contents escaped, and there was not the faintest local evidence of extravasation. The organ was then carefully removed and opened, and it was not until this was done that the aperture before described was found. There were no adhesions in its neighbourhood, and even slight movements must have caused expulsion of the contents through the opening. If the perforation preceded the peritonitis, it follows that continuous and violent vomiting followed by hæmatemesis can go on for five days from a perforated stomach without any signs of extravasation into the peritoneal cavity or any local irritation of the membrane being present at the termination of that time. Nor were any of the symptoms of perforation present. There was no sudden abdominal pain followed by collapse, and the vomiting continued up to a few hours before death. The missing symptoms were not masked by the anodynes. The patient was never fully under their influence. Mr. Freeman, the house physician, assured me that the patient experienced no marked relief after the opium pills, or even after the morphia injections. Assuming then that the perforation did not occur in time to cause the inflammation of the serous membranes it seems most likely that it took place somewhere about the time the vomiting ceased—four hours before death—and so near it that it had no obvious clinical concern in the case. Whether it took any part in
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... bringing about the final result it is impossible to say. I entertain no doubt but that the action of the gastric juice—of which some was found in the stomach at the *post-mortem*—upon the thin peritoneal floor of the ulcer was the chief factor in the production of the perforation. Had it been solely due to post-mortem digestion, there would have been no adequate reason why the centre of the ulcer should have been the only spot showing any traces of such action, for it was not thinner or more dependent than the rest of the floor, and the ulcer itself did not occupy the lowest portion of the stomach as the body lay after death.

3. The existence of any relationship between the ulcer on the one hand, and the pleurisy and peritonitis on the other is not easy to prove. In the affirmative may be urged: (1) the existence of lymphatics through the diaphragm capable of conveying an irritant from the ulcer to the pleura; (2) the anatomical coincidence afforded by the combination of *left* pleurisy, with an ulcer at the *cardiac* end of the stomach; (3) the rarity of the simultaneous occurrence of pleurisy and peritonitis, and the consequent probability of the existence of a cause common to the two; and (4) the absence of any other discoverable cause. Against this hypothesis, it may be argued that if these complications can come about so easily they should be more frequently associated, and that the peritonitis should have preceded the pleurisy, which apparently was not the case.
XVII.—Right Hemiplegia with Aphasia, occurring during a paroxysm of Whooping-cough, with slight rigidity, subsequently passing into a condition of Athetosis. By Samuel West, M.D. Read January 14, 1887.

Florence F., aged 3, was admitted into the Royal Free Hospital with paralysis of the right arm and leg.

The child had always been healthy and intelligent, and talked fairly well, though she began late. She had measles when an infant but had been perfectly well since, until whooping-cough began fifteen weeks ago; the paroxysms were not very frequent, but were very severe.

Seven weeks ago during one of these fits the child became convulsed; she screamed and moved both sides of the face and body. The fit lasted about an hour, and at its end the right arm and leg were found to be completely paralysed, and the mouth drawn to one side. The child seemed quite unconscious. It took its food well and did not seem in pain; it sometimes laughed in a vacant way. These conditions gradually improved, and in four weeks the child had regained its intelligence, together with partial power in the arm and leg. Three days later it had another fit, and was unconscious for five hours; the movements were slight, hardly more than tremors, but the body was bent to the left side, and there was foaming at the mouth. Twelve days ago the mother found the child again unconscious, and it remained so for an hour. Since then the child has been in the present condition.

June 6, 1884.—Present condition. The child is in good health and well nourished, still troubled with occasional paroxysms of whooping-cough. The lines of the face are rather less marked on the right side, but there is no distinct paralysis. The pupils are equal and react normally; the tongue is protruded straight.

The right forearm and hand are held in a semi-flexed and prone position, and the right thumb is bent into the palm. The muscles are slightly rigid, but this rigidity can be overcome easily when the attention is otherwise engaged. Voluntary power is lost in the hand and arm, but the fingers are in constant slight movement, which increases on every
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attempt to use the limb, there is no wasting of muscle or impairment of sensation. The right thigh and leg are in the state of half-flexion, the foot is extended and the toes curled over the sole. The child cannot bear any weight on this side, and when made to walk with assistance, the hip and knee are still further flexed, the foot and toes remaining in the position described, the quadriceps not acting at all. The patellar and plantar reflexes are good and equal on both sides. Sensation is normal on the affected side; no vaso-motor disturbances are to be observed.

July 7.—Patient has a little more power in both hand and foot; the fingers of the right hand are in constant movement, and occasionally the arm is extended and the hand rotated backwards and outwards as in some cases of athetosis. The foot is in the condition of talipes equinus with a little varus.

July 31.—Gradual improvement has taken place; the hand can grasp a little, but the arm is held in the same position of semi-flexion even when the hand is put out to shake; the arm can be extended passively without difficulty; the thumb is still bent slightly into the palm, but the fingers are straight. When at rest in bed the leg is straight, and the foot in the natural position, but the foot is at once turned in when attention is drawn to it.

August 18.—Patient developed scarlet fever and was sent to the Fever Hospital. It was a mild attack and had no influence upon the condition of the limbs.

October 18.—The child had an attack of convulsions yesterday and was brought back. Great improvement had taken place since she was last seen.

November 3.—The general health has greatly improved; the child has begun to talk again and uses now many new words. For some weeks after the commencement of her illness she did not speak at all, although she clearly understood all that was said to her as well as before. Before she left the hospital the first time she had begun to use a few words, but now she was able to speak rather better than before her illness.

Both the arm and leg are a good deal used now; the hand can grasp fairly, but its movements are rather jerky and irregular. There is no stiffness, but the elbow assumes the semi-flexed position as soon as the arm is used; the leg too is used a good deal, but the movements are peculiar. On walking, the foot is placed flat and straight upon the ground, the heel is then raised but not the toes, so that the forward movement of the leg causes the heel as it were to revolve round
the toes, which remain fixed to the ground. When the foot has reached the right angle it is dragged in that position along the ground and the child often trips over the toes and falls; it would appear as if the extensors and peroneæ were paralysed, but there is no evident defect of these muscles, and they react normally to the faradic current. When still in bed the leg lies naturally, but on movement the foot passes into the position of talipes equino-varus.

When walking the elbow is at once bent and slightly removed from the side, the hand being held on a level with the shoulder with the palm turned forwards.

June, 1885.—The condition is much the same, but the speech is much improved.

June 30, 1886.—Since last seen the child has been in good health and has grown much; the speech is good, so that she can say almost anything, but it is still behind other children of her age. She is very intelligent. She has no pain or headache and has never squinted. There has never been any eye change.

The right hand and arm are in constant slow movement as in athetosis. The fingers and thumb are extended, and the wrist also. The arm is flexed and partly pronated, so that the palm faces nearly forwards. The elbow is kept about 6 inches from the side, the arm being nearly parallel with the body. This is the position at once taken by the limb if the child starts to walk. At other times it may be almost quiescent and in the natural position.

The foot, when the child is at rest lying or sitting, is often in the natural position, but directly the leg is moved it turns at once into the position of varus, and when walking it is often so much rotated inwards as to be nearly at right angles. The leg is then carried round with a sweep, to avoid catching the other foot, the great toe is greatly extended and drawn up over the dorsum of the foot as if to avoid catching the ground. This is probably an acquired position, for the child used often to tumble over the toes, though she does it much less now.

When quiet the movements may be almost absent, but movement or excitement bring them on again at once. They become most marked on attempting to walk. The position of the foot varies greatly from time to time, even when walking, and is sometimes hardly varus at all.

When asleep the movements cease and the limbs are in normal positions.
There is no wasting or obvious paralysis of muscles, and the limbs are equally developed on the two sides. The eyes are and have been normal throughout.

It is now two years and a half since the hemiplegia came on. The child has developed well and normally in brain and body and looks in excellent health. Though the power on the hemiplegic side is good and the child has become better able to manage its affected limbs, the condition of athetosis is certainly not less than it was.

Athetosis is defined by Dr. Gowers to be a post-hemiplegic disorder of movement, characterised by slow mobile spasms of intermittent type and continuous; these this child has, but she also has spasms of a similar kind which are developed on movement, slow cramp-like inco-ordination, a form of spastic contracture. The association of these two forms in the same case is interesting as showing the connection between them.

From the sudden onset of the attack during a paroxysm of whooping-cough it would seem that the lesion must have been due to haemorrhage, although athetosis is usually attributed to softening rather than to haemorrhage. There is nothing further in the case to show the seat of the lesion, but there is nothing to negative the probability that it is in the seat described in the few cases recorded, viz. in some part of the great central ganglia. I should fear that the prognosis as regards ultimate recovery must after the lapse of so long a time be bad.
XVIII.—A Method of Treating Thyroid Cysts; with Cases. By A. W. Mayo Robson. Read January 14, 1887.

HAVING adopted a method of treatment for cysts of the thyroid which I have found to be both simple and efficient and which, I think, may be found generally applicable, I venture to bring forward the notes of two cases to illustrate this method.

Case 1.—Eliza N., æt. 22, a spinner, residing in Leeds, was admitted into the Infirmary on April 1, 1885. She said that twelve months previously she had first noticed a lump at the front of her neck, which had gradually grown up to two months before admission, since which time it had rapidly increased and had, by its pressure, interfered with her breathing.

On admission, there was a smooth, doubtfully fluctuating, somewhat globular swelling, about the size of a Tangerine orange, situated over the front of the trachea and moving on deglutition.

The tumour, which inclined to the left of the middle line, gave rise to dyspncea if it was pressed on, either by her collar or by manipulation.

Operation, April 3.—The skin having previously been rendered aseptic and under the usual antiseptic precautions, an incision of an inch was made over the tumour in the middle line of the neck through skin and fasciae, thus laying bare the cyst; this having been drawn forward with nibbed forceps, was incised for half an inch, giving exit to a glairy fluid mixed with blood; the edges of the cyst were sutured to the skin by means of several No. 2 catgut sutures, and a drainage-tube was inserted to the bottom of the cavity; the remaining part of the wound having been closed an antiseptic dressing was applied.

The wound was dressed on the 5th and on the 7th, when the tube was removed and red lotion applied, so as to secure healing from the bottom. She was made an out-patient on the 14th, i. e. ten days after operation, and presented herself a month afterwards with the wound perfectly healed, with a very slight scar and without any sign of the tumour. The
Mr. Robson's Method of Treating Thyroid Cysts.

Temperature was normal throughout and she had no pain or discomfort.

Case 2.—Mary A., æt. 24, consulted me in June, 1885, on account of a large swelling of the right lobe of the thyroid which inconvenienced her by reason of its size and unsightly appearance. Otherwise she was perfectly well.

As there was distinct fluctuation I aspirated it and drew off about two drachms of thick, gelatinous material mixed with blood; this lessened the swelling, but not very materially, and I advised her to have it incised.

Operation.—With the same precautions and in a similar manner to the last case, except that the incision was over the right lobe, I cut down on and incised the cyst, which bled rather freely from its interior after I had cleared out a large amount of colloid material by means of a Volkmann's spoon. The cavity seemed to occupy the greater part of the right lobe of the gland and readily admitted the whole length of the index finger. Pressure having checked the oozing I stitched the edge of the cyst to the skin and inserted a drainage-tube which was left in for a week and then replaced by a pledget of lint saturated with red lotion. She left the hospital in a fortnight and when seen a month afterwards presented a small scar where the tumour had been.

That the treatment of thyroid cysts is not altogether satisfactory is proved by the various methods in use, both medical and surgical. I think one may safely pass over the medical treatment by simply mentioning it, as I am not aware that it ever materially benefits; and of the surgical treatment by setons and by injections with iodine or perchloride of iron I need only quote from Mr. Erichsen's work on surgery to prove that the former plan is not by any means unattended by danger: "The introduction of a seton is occasionally attended by beneficial results. The operation, however, is not unaccompanied by danger, a patient near London having lost his life by the puncture of a vein and the entrance of air into the circulation." But besides this danger, there is the serious one of setting up inflammation of the whole gland or of lighting up diffuse cellulitis of the neck.

Of the danger of injection by perchloride of iron, Mr. Erichsen again says that rapidly fatal embolism has followed its use; and to the fact that sudden death has on several occasions occurred after the injection of iodine I need only refer to recent articles in the medical journals. The method of
antiseptic incision with suture of the sac to the skin was suggested to my mind by the mode of treating abdominal cysts that cannot be removed but which can be safely drained when they are stitched to the skin.

The advantages which this plan of treatment seems to me to possess are: 1st, its simplicity; 2nd, its absolute safety, for should any vessels be wounded they can be secured at the time and the bringing the sac to the surface effectually shuts off the cellular tissue of the neck; and 3rd, its certainty, for the sac heals from the bottom.

In the articles by Mr. Harrison Cripps in Treves's Manual of Surgery, on the operation for imperforate anus, occurs the following passage:—"If after careful dissection in situ the operator fails to find the bowel he may perform Littre's operation," and again, in the article by Mr. Holmes in his System of Surgery, we read, "And then the surgeon will be justified in proposing to open a higher part of the large intestine and form an artificial anus, in order to save the child's life."

This is, I believe, the recognised rule in cases of imperforate anus where there is no bulging in the site where the anus ought to be and where the large intestine terminates at an uncertain distance from the perinaeum; in fact in the British Medical Journal a short time ago I saw a report by a London Hospital surgeon, of a successful colotomy for imperforate anus, and the operator raised the question as to whether colotomy should not be the first and only operation attempted in such cases.

My present paper has nothing to do with cases of obstruction due to a septum across the rectum at a greater or less distance from the anus, or to a thin membrane obliterating the anal aperture, or to cases where a bulging of the meconium can be seen where the anus ought to be; in such instances the surgeon cannot have a moment's doubt as to the course to be pursued, but it refers to those difficult cases where there is no sign of an anus or of bulging of bowel and where in fact one cannot say whether the rectum is distant half an inch or two inches or more from the perinaeum.

I would propose that such cases should be treated on the uniform plan of incising the perinaeum in the middle line from the central point to the tip of the coccyx, dissecting onwards carefully until either the rectum is reached or until the peritoneum is opened, when if the upper part of the rectum is present it can be brought down, opened and sutured to the skin as in the case related below; or if there is no rectum the
sigmoid flexure of the colon may be sought for, brought down, opened and sutured at the anal site. A catheter in the bladder serves as a guide to the position of that viscus as well as to indicate the line of reflection of the peritoneum from the bladder to the rectum.

A. K., aged 1 day, was admitted into the Leeds General Infirmary, on account of imperforate anus, on March 20, 1886.

On examination the raphé continued uninterruptedly from the scrotum to the coccyx, and when the child cried there was no bulging to indicate the contiguity of the bowel to the perineum.

The child was put in the lithotomy position and an incision made from the central point of the perineum to the coccyx; the dissection was carried somewhat deeply, and several small vessels had to be ligatured; a catheter was introduced into the bladder so as to secure its being empty and to serve as a guide in the dissection. No bowel was seen, nor could any bulging be felt until the peritoneum was reached and opened, when the upper end of the rectum was found terminating in a cul de sac; it was easily brought through the peritoneal wound, opened and sutured to the skin of the perineum, the wound in front and behind being closed by deep catgut sutures.

The child never had a bad symptom and passed its motions through the new anus without any difficulty.

On the 24th, i.e. four days afterwards, the wound was all healed, and on the 28th the friends took the child home, taking with them a bougie equal to a No. 16 English catheter, which they were directed to pass daily. The temperature was normal throughout. Three months afterwards the child was quite well, the bougie was easily passed, and the anus looked perfectly normal.

In considering the advisability of this method, it must be looked at from the several points of view of (1) the danger, (2) the difficulties, and (3) the results.

1. Of the danger.—It seems to me that to make a clean wound in the most dependent peritoneal pouch, which wound is quickly occupied by the bowel covered by the visceral layer of peritoneum, offers very little danger from peritonitis. The shock must be much less than if a double dissection in the perineum and in the loin were made. On the other side the statistics of operation and of colotomy in such cases are frightful, e.g. M. Guersant opened the colon eleven times in the groin and once in the loins without saving one of his patients, and Mr. Holmes stated, in 1883, that since the publication of
M. Rochard's paper in 1859 he had only been able to find a record of two successful cases.

2. Of the difficulties.—There can be none of a serious nature until the peritoneum is opened, when, if there is any portion of a rectum, it will be easily found, but if not, the finger or forceps can be easily passed into the opening so as to bring down a loop of bowel which, if small intestine, can be returned, but if large can be sutured to the skin. A good horizontal light is a great help, and a catheter in the bladder prevents that viscus being wounded. Hæmorrhage is slight, but in any case can be easily controlled.

3. The results.—The anus is formed in the normal position instead of in the loin or groin; and, as proved by dissection, the muscular fibres of the external sphincter are present in the skin of the perinæum, we may expect complete muscular power over the lower bowel; in fact in the case reported above, the child has one motion a day and in every way seems to be formed naturally.
XX.—On a Case of Calculous Disease of both Kidneys, with Remarks on the Surgical Treatment of Calculous Kidneys. By Henry Morris, M.B. Read January 28, 1887.

Priscilla B., aged 40, a housekeeper, was sent to me at the Middlesex Hospital by Mr. Nunn, and admitted on June 15, 1886, with renal calculus and a purulent fistula in the right loin.

History (from notes by Mr. Larkham).—Two and a half years before admission she had typhoid fever, and two months after the fever she observed a swelling in the right loin. This swelling resulted in a large abscess, which eight months later was lanced. The abscess also burst in two or three places and slowly contracted but never quite healed, for a sinus was left in the loin. She has ever since suffered pain in the right renal region, and for the last seven months has felt a scalding or cutting pain in the urethra when passing water, and has micturated much more frequently than natural. Quite recently she has been obliged to pass urine every half or quarter of an hour.

For a long time past the urine has been albuminous, and her feet and legs edematous; and some weeks before admission she passed several minute calculi.

During the day or two prior to admission she had, at times, had a dull aching pain in the left loin, as well as the old pain in the right.

State on admission.—In the right lumbar region are three or four cicatrices, the result of the openings of the abscess, and a sinus, from which there escapes a small amount of thin pus. There is pain and some fulness in the right loin, and some tenderness on pressure over the right side of the abdomen. Pain is felt also in the left loin and in the urethra during micturition, which occurs every quarter to half hour. The urine is acid, sp. gr. 1012, and contains small amounts of blood and pus and one third albumen.

On June 17, two days after admission, she passed with her urine two small calculi, and afterwards a clot of blood. Thirty ounces of urine were voided during the twelve hours next following.
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The fulness, pain, and tenderness, and the sinus in the right side, together with the frequency of micturition, the quality of the urine, and the passage of calculi, pointed to stone affecting the right kidney. The pain which she had suffered for a few days in the left renal region gave rise to the suspicion that there might be stone there also, and the minute size of the calculi passed suggested general nephrolithiasis. But the stress of her symptoms pointed to the right kidney as the one first to explore; moreover, as calculus in one kidney will sometimes give rise to pain in both, it was considered possible that the pain in the left loin was merely sympathetic.

On June 18 an anaesthetic was given and the bladder was digitally explored, but with negative result. An oblique incision 3 inches long was then made in the right lumbar region about an inch below the last rib, and the posterior surface of the kidney was brought into view. This surface of the kidney was carefully examined with finger and sharp probe, and then the anterior surface of the kidney was examined with the finger. Finally, the kidney was freely incised on its posterior aspect, and several grain-like calculi and some fragments of pyogenic membrane were removed from within the organ, but no large calculus was found, and no pus evacuated. All around the kidney the cellular tissue was condensed and firmly adherent, and strong fibrous bands had to be divided here and there during the process of freeing the kidney for digital examination. The pelvis and calyces of the kidney were sacculated but not greatly enlarged, whilst the secreting substance was tough and fibrous and smaller than normal.

A drainage-tube was introduced and fastened in the pelvis of the kidney, the external wound partially closed by sutures, dusted over with iodoform and covered with boracic charpie dressing.

June 19.—It was noted that she had passed a restless night, owing to pain in the right loin and back, and very acute pain in the urethra. There was some stammering or delay in micturating. During the first twenty-four hours after the operation only 26 oz. of urine were passed; it was acid, sp. gr. 1012, contained blood and pus; blood in considerable quantity. A large quantity of watery discharge escaped through the wound.

June 20.—Discharge from wound scanty and without urinous odour. Was very sick in the evening.

June 21.—Very faint and sick. Complains of dragging pain in the left renal region, increased on a deep inspiration.
Wound looks healthy, very little discharge from it. Only takes a little milk and lime water.

June 22.—Very drowsy. Still in great pain in left renal region. Feels sick after everything she takes, but has not vomited through the night. Passes very little urine. Wound covered with a greyish-yellow membrane.

June 23.—At 9 p.m. last evening one tenth of a grain of pilocarpine was given by subcutaneous injection. After this she sweated freely, and passed 2 oz. of urine. Subsequently she passed no more urine, and died comatose at 9.30 this morning.

The quantity of urine passed in each twenty-four hours after the operation was as follows:

<table>
<thead>
<tr>
<th>Time</th>
<th>Quantity</th>
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<tbody>
<tr>
<td>From 4 p.m.</td>
<td>June 18-19, 20 oz.</td>
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<tr>
<td>From 4 p.m.</td>
<td>June 19-20, 20 oz.</td>
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<tr>
<td>From 4 p.m.</td>
<td>June 20-21, 5 oz.</td>
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<tr>
<td>From 4 p.m.</td>
<td>June 21-22, 5 oz.</td>
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<tr>
<td>From 4 p.m.</td>
<td>June 22 till death at 9.30 a.m. on June 23, 2 oz. shortly after the pilocarpine, which was injected at 9 p.m. on the 22nd.</td>
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Post-mortem examination.—The following notes of the post-mortem are abstracted from the pathologist's report:

In the right loin is a wound about 3 inches long, taking the course of the lowest rib, but below it. Emphysema of each lung. Heart fatty and small. A large ante-mortem clot occupied the pulmonary artery.

The right kidney communicated on its posterior surface with the wound in the ilio-costal space. It was small, cystic, hard, and contracted. A wound in this kidney communicated with the renal pelvis; there was no stone in the wound nor in the ureter or renal pelvis, but on section of the kidney one or two millet-seed-sized calculi were found scattered in its tissue. Around the kidney the fatty cellular structures were much thickened and indurated. The colon was adherent to the kidney.

The left kidney was very large and white, with a great increase of its cortical structure. On the anterior surface and in its middle third was found a small cyst containing light yellow pus. On section this kidney presented in its upper third a cavity with ragged ulcerated walls, and large enough to contain a cob-nut; it was filled with about 200 small facetted calculi. Numerous other small stones were scattered in little groups throughout the organ. The ureter was normal in size, and no stone was found in it.
Extending from one kidney to the other behind the peritoneum was a line of inflammatory exudation, and in the small omentum there was some breaking-down tissue.

Remarks.—The cause of the death of this woman was the supernention of acute calculous disease in the large white left kidney, the right kidney having been previously in great part destroyed. The points of surgical importance suggested by the case are two: first, that both kidneys may be too much affected by diffused calculi for any operation to be of service; second, that one kidney may have been long ago and extensively damaged by calculous disease, either without causing symptoms of any kind whatever, or else by a process which results in perinephritic abscess and lumbar fistula, and that subsequently calculous disease in the other kidney may give rise to alarming symptoms, or death.

In such a case, an exploratory operation may be made upon the wrong kidney, a stone may be detected in, and removed from, an organ in which disease has long been quiescent. If the abdominal operation be employed the detection of a calculus on the side first affected and the enlarged and apparently healthy condition of the opposite organ may mislead the surgeon into cutting into the kidney which is not the seat of the symptoms. Thus, for example, it is quite possible for a patient to refer his symptoms to the left side, but on an abdominal exploration this organ may to sight and touch seem quite healthy, and a stone may be felt in the opposite kidney.

Under these circumstances the right kidney will be operated upon, whereas the left kidney may be loaded with calculi and suppurating, and the real seat of the danger to the patient's life.

In my case, no relief having been afforded by the operation on the right side, I contemplated an exploration of the left, but the disinclination of the friends, and more especially the enfeebled and prostrated state of the patient, restrained me.

The post-mortem showed that any operation, short of excision of the large left kidney, would have been unattended with relief, but that to have removed the left organ would have left the patient with an insufficiency of renal substance in the right organ.

Renal calculus cases differ widely one from another, and we must recognise at least three classes for which we may be consulted. (I) Those where one or more calculi of appreciable size are found, and could be removed before the kidney has been
destroyed. In these, nephrolithotomy is followed by complete cure.

(2) Those where one or more calculi of appreciable size have been allowed to remain until they have irritated and destroyed the kidney. In these, nephrotomy should be employed as a means of preserving life and sparing suffering; but the operation is too late to save the kidney intact.

And (3) cases of nephrolithiasis, such as the above, where minute calculi or sabulous particles are diffused throughout the renal tissue. Such cases can be only dealt with surgically, if at all, by nephrectomy, an operation which the very nature of the disease must prohibit, if both organs are known to be similarly affected.

This third class is fortunately the least frequent of calculous cases. For the second class, surgery has repeatedly been of great service.

For the first class there is a great future, if only the aid of the surgeon is sought soon enough.
XXI.—A Case of successful Nephrolithotomy. By Henry Morris, M.B. Read January 28, 1887.

John M., aged 42, a compositor, was sent to me on May 4, 1886, by Dr. George Johnson, who wrote, “The bearer comes with a history which indicates, I think, pretty clearly renal calculus. Perhaps you will take the man into the Middlesex Hospital, and if you decide to operate be so good as to let me know the time in order that I may be present.”

I admitted him into the Middlesex Hospital on May 12, 1886. He gave the following account, which is summarized from notes taken by the dresser, Mr. Couch: His family history was rheumatic, his personal history very good till the onset of the present illness. He has always been sober, active, and healthy, but has suffered from time to time from indigestion. In 1867 during a visit to Paris he drank very freely of red wine, and shortly after noticed a quantity of pink sediment in his urine. This continued the same till eighteen months ago, except on one occasion when there was a well marked fawn-coloured deposit like sugar. During all these years he has suffered pain in his right loin and hip, but no pain on micturation. The character of the urinary sediment altered eighteen months ago, and his symptoms increased in severity. There has frequently been a deposit of coarse gravel from the urine, and severe paroxysms of pain from time to time have attacked the right loin, and extended to the groin along the course of the right ureter. The urine has often been smoky in colour, and has sometimes shown well-marked naked-eye evidence of blood.

At short intervals, for a month or two before his admission, the urine had undergone changes of a definite character. Thus from a light colour it became daily darker until it reached the blackness of porter, then it rapidly became lighter until it resumed a natural pale yellow. This change from porter colour to pale yellow was sometimes completed in a day. When the urine was darkest there was but little voided, but after one of these attacks the quantity varied from 73 to 85 oz. of pale urine. Ordinarily the quantity of urine varied from 45 to 60 oz.
For a long time the pain had been so severe that he has taken large quantities of alcohol to relieve it. In fact he has kept himself constantly under the influence of brandy, but not to an intoxicating degree. On an average he has taken one bottle of brandy a day, and often a bottle and a half, and during the last six months he has added to the alcohol frequent doses of laudanum, taking generally from $\frac{3}{2}$i to $\frac{3}{4}$iss daily. In addition he has taken a great many morphia lozenges.

The effect of these habits on his health and purse were becoming disastrous; moreover, the remedies were only partially successful in keeping him free enough from suffering to enable him to follow his occupation. Therefore he was willing to undergo any operation which afforded him a chance of cure.

State on admission.—He is a spare but well-proportioned man, slightly over medium height. His abdomen is much sunken, so that the viscera can be easily defined. His face is wan, and has a look of suffering, his tongue furred, and his bowels costive. There is a considerable tremor of his hands.

There is no enlargement in either renal region, but the patient said that when lying in bed he has sometimes, during a paroxysm of pain, felt a tense band, extending from opposite the iliac crest to the groin over a line corresponding to the middle part of the ureter. He complains bitterly of pain in the right loin, and along the ureter to the groin.

The urine, as a rule, is acid, sp. gr. 1025 to 1030, and contains occasionally a few pus-corpuscles, a few epithelial cells, and one or two blood-casts.

On May 15 he was anaestheticised, and nephrolithotomy performed, Dr. George Johnson being present. Mr. Pound, my house surgeon, assisted me.

The usual incision in the ilio-costal space only brought into view the lower end of the kidney, owing to an unusually high position of the organ, the lower end not reaching beyond the twelfth rib. This made the operation somewhat more difficult and protracted than usual, and necessitated a second incision $1\frac{1}{2}$ inches long in a direction at right angles to the first.

The calculus was not at once felt, but after due examination of both the anterior and posterior surfaces of the kidney, a spot below the level of the hilum was selected for incision on account of its increased resistance to the finger. The incision was made into the posterior surface of the kidney substance,
and the opening was enlarged by the index finger, which readily passed through it into the renal pelvis, and there detected a calculus. The stone being small and the pelvis of the kidney somewhat enlarged, the stone easily slipped from point to point within the pelvis. An attempt was made to seize the calculus with a pair of slender forceps, but it was not successful, therefore the index finger alone was trusted to, and by a scooping action the calculus was lifted out of the kidney into the wound.

The stone was small, circular, and biconvex, of a rich brown colour, and roughly mammillated on the surface. It weighed 23½ grains, and consisted of oxalate of lime. The wound in the kidney was left as it was, and no drainage-tube was introduced through it. The wound in the parietes was sewn up by five sutures, a large drain inserted at its centre, and a dressing of boracic charpie and gauze was applied over it.

The patient was kept upon his back, with the shoulder and hip of the side operated upon resting upon separate pillows, so as to get full advantage of drainage without pressing on the wound. After the operation he vomited a good deal until the evening of the following day. The urine during the first twenty-four hours was highly loaded with bright red blood. On May 17 (the second day after operation) the urine was of a light amber colour, acid, sp. gr. 1024, and contained only traces of blood. So it continued till May 27, when it was perfectly normal. There was no retention of urine, and no pain or undue frequency of micturition after the operation.

The wound was dressed on the day after the operation, and again on the second day, but the dressings contained no urine, and only a little bloody serum. Subsequently they were not changed oftener than every other day. On May 22 the drainage-tube was removed, and the wound had healed perfectly, except where the tube had passed. On May 31 the patient sat out of bed, and on June 6 he was discharged well—just three weeks and a day after the operation.

The temperature did not rise above 99·6°, the pulse did not exceed 80. Opium and alcohol were entirely discontinued within a day or two after the operation, and on the fourth day all food was taken by the mouth; up till the fourth day he had been fed chiefly by enemata.
Mr. Morris’s Case of Successful Nephrolithotomy.

He has been seen at intervals since the operation several times, at the hospital and by Dr. George Johnson. He remains quite well, and his urine is in every respect normal.

Dr. Johnson informs me that the man called on him on June 4, and said that he was quite well, that he had given up all stimulants and narcotics, and, expressing his gratitude for the cure, he said “it had changed a very gloomy prospect into a very bright one.” Dr. Johnson added that he had tested the urine and found it quite normal.

June, 1887.—The man has frequently been seen since the date of the last note, and was shown at the meeting of the Society when this paper was read. He is perfectly well, and has been ever since the operation.

It may also be added here that the man Edward G., whose case was reported in vol. xviii, p. 185, of the Society’s Transactions, was also shown to the Society at the same meeting, and that he too has remained quite free of disease since his operation.
XXII.—A Case in which very severe symptoms were due to two small Stones in an Atrophied and Movable Kidney; Failure to detect the Kidney by an Anterior Lumbar Incision; Discovery by Laparotomy; Successful removal of the Organ. By Howard Marsh. Read January 28, 1887.

Emma D., aged 25, unmarried, was admitted into St. Bartholomew's Hospital on June 29, 1886, with the following symptoms, which led me to believe that she had stone in the left kidney:—1. Pain in the left lumbar region of a dull, aching character. This was constantly present, and had, she said, existed since she was between thirteen and fourteen. 2. Very sharp exacerbations of this pain, occurring almost daily, sometimes spontaneously, but usually provoked by walking or some ordinary movement. During these attacks, which lasted from an hour to twelve or fifteen hours, or even longer, she sat in a crouching position with her trunk bent over to the affected side, or lay on that side with her limbs flexed on the abdomen. In these attacks the temperature occasionally rose to 99.4°F, but was never above this point. After the attacks she complained of feeling cold and was prostrate and feeble. 3. Frequency of micturition. This had been present for many years, but had lately become much more marked. In the attacks just described the urine was passed six or seven times in the hour. 4. She complained of a feeling of sickness, and sometimes vomited. 5. There was pus in the urine. This was often only microscopical in amount, but sometimes it formed a distinct sediment. 6. There was blood in the urine, but this was now in such small quantities that it could be detected only either by a chemical test or the microscope. The patient, however, stated that on one occasion she passed a considerable amount. The urine, of which as much as five or six pints were generally passed in the twenty-four hours, had a sp. gr. varying from 1022 to 1010. Sometimes crystals of oxalate of lime were present.

On repeated examination, twice when the patient was under ether, I was unable to detect any enlargement of the
kidney, or even to feel the organ. There was a well-marked history of gout in the patient's family. Her mother died of cancer.

As the patient was in no degree better when she had been in the hospital for a fortnight, several of the staff who saw the case agreed that the kidney should be explored. For this purpose I adopted the incision which Mr. Willett has suggested. This runs downwards and slightly forwards, and is placed in the mid-axillary line, between the ribs and the iliac crest.

Having divided the muscles of the abdominal wall and the fascia transversalis I searched for the kidney, but was unable to find it, and the same difficulty occurred to two or three of my colleagues who were present at the time. During this examination the parts in the neighbourhood were exposed as freely as seemed compatible with the safety of the patient, and the cardiac end of the stomach, the spleen, the left lobe of the liver, the vertebral column, and the aorta were all distinctly felt. Under these circumstances I introduced a drainage-tube and closed the wound around it. The patient was violently sick for nearly twelve hours after the operation, but she was never seriously ill, and the wound, though there was at first free suppuration, soon closed. It seemed clear, however, that the kidney had been in some way overlooked, for all the symptoms observed before the operation were still present. I therefore determined to search for the kidney by opening the abdomen in the middle line. On doing this, and passing in my hand, I found the kidney without the least difficulty lying in its normal position. I also found why I could not detect it on the previous occasion. It was atrophied, and so freely movable that I could displace it in all directions for a distance of between 2 and 3 inches. Owing to its free mobility it had, no doubt, been carried up with the peritoneum when that membrane was raised in the opening of the deeper part of the wound. On carefully examining the kidney I could only detect that it was small and withered. I could feel nothing like a stone in it. As it was extensively diseased I determined to remove it, having found, by passing my hand across the middle line, that the right kidney was apparently normal. The wound in the middle line was temporarily closed by a suture, a large sponge having been passed into the abdomen to prevent protrusion of the intestines. The patient being turned on her right side, I made an incision from the angle between the last rib and the erector spinae obliquely down-
wards and forwards to the crest of the ilium. As soon as the abdominal wall was divided the kidney came readily into view, and from its small size and free mobility was removed without difficulty. First the ureter was reached, tied just below the pelvis, and cut beyond the ligature, the distal portion leading to the bladder was syringed out with carbolic lotion, and its end tied; the pedicle of the kidney, consisting of the vessels, was ligatured in two portions, a blunt needle, threaded with strong Chinese silk, having been passed between the artery and vein, or at least through the middle of the pedicle where the finger and thumb came into contact with each other. There was no haemorrhage of any importance. The wound was closed around a small-drainage tube. The abdominal cavity was sponged out, and the anterior wound closed. The patient made a good recovery, indeed she bore the operation better than the former one. Three weeks afterwards, when the wound was reduced to a small sinus, the thermometer registered a temperature of 103° and on some days 104°. Thinking that there must be some matter retained either in the deeper part of the wound or in the ureter I carefully examined the patient but could find nothing. At length it was discovered that she was playing tricks with the thermometer and that her temperature was really normal. She was therefore allowed to be up, and a few days later she was discharged. Shortly afterwards she returned to her work as a household servant, apparently in perfect health.

When the kidney was first examined after its removal no stone could be felt in it, but when it was laid open three small calculi were found embedded in one of the calyces. These were about three times the size of a grain of wheat, and consisted of uric acid.

Remarks.—The surgery of the kidney can attain its complete development only by the publication and discussion of the various difficulties with which operators find themselves confronted. There are from this point of view some circumstances which it seems worth while to record in the present case.

That the kidney could not be found on the first occasion was due in part to its being so freely movable that it readily admitted of displacement when the intestines and peritoneum were raised, but in part to the line of incision which I adopted. Everyone who has attempted to remove a large kidney through any available incision in the loin is well aware of the difficulty that arises from want of space. This difficulty
is to a great extent avoided by the anterior incision of Mr. Willett. By this incision a full exposure of the kidney under ordinary conditions is obtained. As, however, the kidney was in the present case movable it was lifted out of reach as I distended the wound.

Considering the facts before us bearing on the important subject of the choice of an incision, I think that when the kidney can be felt, and is found to be of large size, Mr. Willett's incision should certainly be adopted, but that when the kidney cannot be felt through the abdominal wall, and when it is proposed merely to examine it, or to remove a stone from it, an incision in the loin should be preferred, since there is then less danger of overlooking a small or a movable or floating kidney, and also less disturbance of the surrounding parts.

I have nothing to say upon the method of searching for the kidney through an anterior incision except that in this instance it at once disclosed the existence and situation of the kidney, and put me in a position to venture on its removal, since it enabled me to ascertain that the other kidney was in a fairly healthy state. The abdominal exploration added nothing, so far as I could see, to the danger of the patient. A highly important fact in regard to the surgery of the kidney was illustrated by this case. I mean, that a stone, or a collection of stones too small to be felt when the kidney is freely exposed, may yet cause such severe suffering as entirely to incapacitate the patient for any active occupation. Even if the existence of these small stones could have been ascertained it would have been very difficult indeed to remove them. This might perhaps have been effected by opening the calyces and using a syringe; but even this method would have been very uncertain. It was, I think, fortunate for the patient that the kidney was known to be atrophied and movable so that its immediate removal was determined upon; for an attempt to find the stone would not only have involved prolonged manipulation which might have contained elements of danger, but would, I believe, have ended in failure and the conclusion that after all no stone was present, and so the patient would have been left without relief. Further investigation will no doubt place within our reach more precise knowledge as to what are the conclusive or most reliable evidences of stone in the kidney, and perhaps some new method of searching for small stones may be introduced, so that when strongly suggestive symptoms are observed we may be able to settle the question by
a reliable physical examination, as we are able to do in the case of the urinary bladder. Possibly this will consist of direct sounding with an instrument introduced through a small opening in the pelvis of the ureter, for by this method the greater part of the area of the infundibula might be explored, while, were any calculous material detected on opening in the kidney substance an incision might be made directly over it, so that the stone could either be extracted or washed out by syringing (Bruce Clarke). In the account I have given of this case I have attributed the patient's symptoms to the presence of the small stones found on opening the kidney. But some may be inclined to believe the patient's suffering in part due to the fact that the kidney was movable. For many cases are on record in which severe pain, paroxysmal in character, was due to movable kidney. This point I hope may receive attention in the discussion this evening. Since the paper was written the patient has been readmitted into St. Bartholomew's Hospital, with symptoms pointing to stone in the opposite kidney; but as the case is incomplete I will not enter now into a description of her present illness.

L. F., a married woman æt. 27, was admitted into St. George’s Hospital in 1884, on account of an intra-uterine tumour which was removed, and the patient left the hospital “cured” in the course of a month. The exact nature of the growth was not ascertained. On December 28, 1885, she was again admitted under the care, on this occasion, of my colleague, Dr. Champneys, with the following history.

For some months she had been feeling not altogether well, but had noticed nothing very definite to complain of until about five or six weeks before her admission, when she became conscious of a somewhat vague pain about the lower part of the abdomen, extending upwards to the left kidney, a pain which at times became acute and shot down into the left groin and thigh. This pain increased rather rapidly, and on several occasions there had been attacks of vomiting and shivering.

Micturition had, as a rule, been frequent and at times painful, the urine being thick when passed, but containing no blood.

The catamenia had been regular and natural in all respects. She had noticed no swelling about the abdomen, but had found considerable general tenderness upon any slight pressure.

On admission.—She was delicate looking, pallid, and rather emaciated. There was much tenderness over the whole of the lower part of the abdomen. On the left side of the navel a rounded mass about the size of a kidney could be felt, it was dull on percussion, immovable, and extremely painful on pressure, but did not extend into the flank, which, however, was very tender.

An examination of the uterus was attended by a negative result. The urine was slightly turbid, neutral in reaction, and contained a small quantity of pus.

The pulse was regular and fairly strong. The temperature was 99°F. From the date of her coming into the hospital the tumour steadily increased in size, the pain was at times very acute, and there was occasional vomiting.

By January 8 the mass could be plainly felt in the left
loin, the urine contained rather more pus, was natural in quantity, and was not passed more frequently than normal.

On January 14 there was a severe rigor, the first since admission, the temperature rose to 103°, and an exhausting almost uncontrollable attack of vomiting occurred.

Rapid increase in the tumour followed, and on the 16th, fluctuation was detected. As the patient was becoming exhausted by pain and vomiting, I was asked to see her with Dr. Champneys on February 3 with a view to the adoption of some surgical measure for her relief. Upon examining the patient I found her in a condition sufficiently indicated for my present purpose by the foregoing notes, and as, so far as we could judge after consultation, the tumour appeared to be a renal or perirenal abscess, it was decided to make a free lumbar incision without delay. Accordingly I cut down through the loin as if for the purpose of performing nephrotomy and exposed a tense, fluctuating, circumscribed mass intimately adherent to the surrounding parts. Upon incising this freely a large quantity of foul, urinous-smelling discharge escaped; in parts this was curdy and almost caseating, like that which usually comes from a scrofulous abscess of the kidney. Upon introducing the finger into the cavity it seemed to enter the interior of a greatly disorganised kidney, there being merely a large sac with the irregular imperfect septa formed apparently by the undestroyed calyces crossing it in the usual directions. At the inner aspect of the cavity a hardish adherent portion could be felt, below which passed a pouch, presumably the upper end of the dilated ureter, into which, at the request of Dr. Champneys, a catheter was passed for some distance. The appearance of the discharge, and the characteristic nature of the interior of the sac, left no doubt in my own mind, nor in that of either of my colleagues who were present, that I had opened a disorganised scrofulous kidney.

Great relief followed the operation, the tenderness became less, and the pain much diminished, the vomiting did not entirely cease at once, but was less violent. The condition of the urine was not affected by the operation. The improvement continued, in spite of some discomfort from slight symptoms of carbolic acid poisoning, which showed themselves on the 8th, but which soon subsided upon the substitution of another antiseptic for washing out the abscess.

By February 12 the tongue was clean, the pulse quiet and fairly strong; the patient was sleeping well, the discharge, though still free, had much decreased and was not offensive,
Steady improvement occurred till February 18, when a rapid change for the worse supervened. The breathing was quick, the face congested, but there was no alteration in the condition of the discharge, nor was there any perceptible rise in the temperature, which was at this time normal. By the 22nd the patient was evidently sinking, the temperature was still normal, the pulse quick and feeble. Breathing throughout the left lung was very deficient, there was crepitation throughout the right.

The precise condition of the lungs, however, could not be determined, the patient being too ill to admit of a proper examination.

She died on February 28 from the lung complication.

The discharge from the wound gradually diminished from the time of the operation, and the abscess was manifestly rapidly contracting until two days before death, when it became foul in consequence of the cavity having been imperfectly washed out as the patient was not in a condition to bear any movement.

At the post-mortem examination there was found a small wound in the left loin, resulting from the operation, which led into an abscess of small size, the interior of which was much sacculated.

At the inner and anterior part of this abscess, the kidney lay somewhat enlarged but *quite intact*, and, indeed, had not been involved at all in my operation.

On laying open the kidney itself, which was intimately adherent to the pancreas and intestine, it presented a typical example of scrofulous disease, but had not broken down to any extent. The opposite kidney was apparently healthy.

The base of the left lung was solid; in the upper part on its external aspect were a number of rounded whitish nodules projecting from the surface, which proved to be of a sarcomatous nature. The right lung was emphysematous and greatly congested throughout. The uterus contained a small myoma.

Difficult as may be the differential diagnosis of abscess in and around the kidney under ordinary circumstances, I am unacquainted with any case in which doubt has been felt by the surgeon *after incision* as to the situation of the disease.

In the case which I now venture to bring under the notice of the Society, so exactly in every respect did the cavity into which I cut resemble the interior of the kidney, that I am certain no surgeon would have had any doubt whatever that the organ had been laid open.
Mr. Bennett's Case of Supposed Nephrotyomy.

So perfect in fact was the resemblance that I shall, I am bound to admit, in the future hesitate to say positively at the time of operation in a similar case whether the kidney has been opened or not. Had my patient survived, as I think she would have done if the lung complication had not supervened, the abscess would undoubtedly have healed, and the case have been recorded as an instance of successful nephrotyomy in scrofulous disease.

As it is, perhaps, not absolutely impossible that similar sacculated abscesses may have been opened by other surgeons under the impression that the kidney had been incised, it seems to me that this case is not without some interest in connection with the statistics of the results of the treatment of scrofulous kidney by incision.

History.—S. V., a widow, æt. 40, stated that about fifteen years ago she fell from some steps; the injury was followed by severe pain in the right loin, and for nearly a week her urine was "like port wine." The pain in the right side had continued on and off ever since, but latterly the attacks had become more frequent and more severe. The pain was described as extending from the right lower ribs down to the iliac crest and groin, and round to the back; lately it had been "throbbing" in character, and "like knives," and often accompanied with sickness. With each attack of pain the urine was thick when passed, and deposited a tenacious white sediment, but there had been no blood since the time of the accident. For five to six years she had been conscious of a lump in her side. She had never passed gravel.

When admitted to Middlesex Hospital on November 23, 1886, she was a thin, pale, delicate-looking woman. The right flank was obviously fullier than the left, and in it a tumour was detected reaching up under the floating ribs, downwards to the iliac fossa, backwards to the spine, and inwards almost to the middle line. There was dulness in the flank, and tympanitic resonance over the front of the swelling. The surface of the tumour was smooth; it was tense, and obscure fluctuation could be detected in it. The urine was passed about four or five times during the day and twice during the night. Riding in an omnibus or any similar shaking always excited pains and a desire to micturate. On the day of admission the urine was clear and contained only a trace of albumen, but subsequently it was found to have a sp. gr. of 1015, to be acid in reaction, and to contain about one eighth part of pus and a trace of blood.

Operation.—On November 27, with the patient under an anaesthetic, I passed a grooved trocar into the tumour from the loin; pus flowed through it. An incision was then made in the loin, down to the kidney, and into the pelvis, which was found to be dilated; a considerable quantity of pus
escaped. With the finger a stone was felt in the lower part of the pelvis, and this was extracted; no others could be felt. The kidney and the wound were well irrigated with bichloride of mercury solution 1 in 2000, a drainage-tube introduced, and the wound closed with sutures and dressed with alembroth wool. The tube was removed in forty-eight hours. The patient made an uninterrupted recovery, and left the hospital with the wound firmly healed on December 29. The urine was collected and examined each day. Before the operation the daily amount was 48 oz. For successive twenty-four hours after the operation the total passed per urethram was 17 oz., 18, 28, 47, 44, 52, 50, 32, 62, 62, 62, 32, 56 oz., and so on. This showed that by December 1, all the urine from both kidneys was passing into the bladder. On November 28 the urine contained a faint trace of albumen; after this it was quite normal until December 4, and then for three days a faint trace of albumen was again observed; after that the urine became and continued normal. The calculus is ovoid in shape, weighs, now that it is perfectly dry, 49 grains, and consists of urates. There is no central "nucleus" or cavity.

The latest and fullest statistics of the results of operations upon the kidney are those furnished by Dr. Brodeur in his work entitled *De l'Intervention Chirurgicale dans les Affections du Rein*. He has collected 327 cases of renal operations, and from this large number we may gather some important facts. Of 125 lumbar nephrectomies 78 ended in recovery. Of 110 abdominal nephrectomies 55 were successful. Of 34 lumbar nephrotoomies 23 recovered. Of 9 abdominal nephrotoomies 7 recovered. Of 36 lumbar nephrolithotomies 28 recovered, while all the three abdominal nephrolithotomies were fatal. If we limit ourselves to operations performed for renal calculus or calculus pyelitis we find 36 lumbar nephrectomies with 20 recoveries, 10 abdominal nephrectomies with 5 recoveries, 36 lumbar nephrolithotomies with 28 recoveries, 3 abdominal nephrolithotomies, all fatal, and 6 lumbar nephrotoomies with 2 deaths. The cases hitherto published are therefore in favour of the lumbar as opposed to the abdominal operation.

While recording cases of nephrolithotomy it is important not to lose sight of the fact that many kidneys have been explored without finding a stone, and yet the symptoms complained of before have been relieved. I recently had, under my care a young woman who suffered from extreme pain and
tenderness over the right kidney, shooting into the groin, and accompanied by the passage of blood and pus in the urine. The kidney was explored through the loin, but no stone could be detected. The pain, however, was greatly relieved from the time of the operation and has not returned. There have been many other instances of a similar kind.
XXV.—A Case of Nephrectomy for Hydronephrosis, being the sequel of a Case of Nephrotomy exhibited at the Meeting of the International Medical Congress, held in London, 1881. By W. Morrant Baker. Read February 11, 1887.

The early notes of this case I quote from the Transactions of the International Medical Congress, vol. ii, p. 264.

"A Case of Nephrotomy. (For the details of the notes I am indebted to Mr. Hewer, surgicaldresser.) "Frederick M., a lad aet. 16, was admitted into St. Bartholomew's Hospital February 26, 1881, under my care, on account of a large swelling in the region of the left kidney. He had been for some time previously under the care of Dr. Dyce Duckworth. He was in perfect health until about four years before admission into the hospital, when his illness began with a sudden sharp pain in the left renal region which obliged him to go home and lie in bed. By the next morning, however, the pain had ceased, and he was as well as ever. A second attack, with like symptoms, occurred a fortnight afterwards; and, subsequently, attacks came on at almost uniform intervals of a week, and these have continued to the date of his admission into the hospital; the attacks lasting usually from eight to sixteen hours, and the pain being very acute and always felt in the same region—that of the left kidney. During an attack a large swelling, fluctuating to pressure, has been perceptible on examination in the painful region. The swelling has, however, subsided soon afterwards, and no trace of it can be discovered until the onset of a fresh attack.

"Before an attack it is said the urine is clear; immediately afterwards it becomes thick with pus, the thickness then again disappearing gradually, and the urine remaining clear until the patient has suffered from another onset of pain. During an attack he always lies on his back, with the knees drawn up; and while the pain lasts he is said to take no food, nor does he pass urine, even when the attack is prolonged for many hours.

"In June, 1879, he was admitted into a medical ward, under the care of Dr. Andrew, and remained there for two
months, but without permanent benefit; the attacks of pain and the accompanying symptoms appearing with great regularity.

"The general health was somewhat impaired by the repeated attacks; but the patient was able to attend as an outpatient, under the care of Dr. Duckworth, until the date of his readmission into the hospital.

"March 15, 1881.—At this date, rather more than a fortnight after the patient’s readmission, he had a severe attack of pain, and, the left renal region being occupied by a large fluctuating and tense swelling, I decided on seizing the opportunity and exploring the tumour by operation.

"The incisions made resembled those commonly adopted for lumbar colotomy, the wound being enlarged subsequently by making an incision upwards at right angles to the first which lay parallel to the last rib. After exposure of the tumour, which proved to be a largely dilated kidney, it was punctured, and about a pint and a half of pale slightly turbid fluid escaped. [Sp. gr. 1005, neutral in reaction, and containing pus-cells and a trace of albumen.]

"The opening was then enlarged, and the dilated kidney was explored as thoroughly as possible by the finger and afterwards by a metal sound. The lower boundaries, which seemed to extend into the pelvis, could not be satisfactorily reached. No calculus was discovered.

"The edges of the wound in the kidney-cyst were now stitched, as to part of their extent, to the edges of the wound in the abdominal wall, and an india-rubber tracheotomy tube was afterwards inserted to ensure good drainage. The wound was dressed lightly with carbolized oil.

"Carbolic spray was used during the early stages of the operation, but was discontinued as soon as all danger of wounding the peritoneum was over.

"The subsequent history of this case is only one of almost uninterrupted progress towards recovery—that is, so far as the operation and its immediate results are concerned. The patient has completely regained his health and is free from pain. He still wears a drainage-tube, through which a considerable quantity of urinary fluid is daily discharged. Quite recently Mr. Buckland, one of the surgical dressers, has arranged a simple apparatus, by means of which the fluid is discharged into a bottle which the patient, when walking about, carries slung to his waist.

"Mr. Buckland has kindly furnished me with the follow-
ing account of the urinary fluid passed through the renal fistula, and of the urine passed *per urethram*:

"Fluid discharged by the renal fistula: average amount (daily) 21 ounces, sp. gr. 1005. *Albumen*:—July 22, ½. July 29, ½. August 1, ½. *Urea*:—July 22, 0·2 per cent. July 29, 0·1 per cent. August 1, 0·1 per cent. Acid, slightly; blood, a trace; sugar, none.

"Urine discharged *per urethram*: average amount (daily) 28 ounces, sp. gr. 1015. *Albumen*, none. *Urea*:—July 22, 1·0 per cent. July 29, 1·4 per cent. August 1, 2·0 per cent. Acid, slightly; sugar, none; blood, none."

The patient continued in fair comfort and able to get about and do his work until the end of December, 1881, when he came to the hospital, stating that, three weeks previously, the tube came out in the night, and he had since been unable to introduce it. He had shivered several times, and had suffered from a return of the former pain and discomfort.

He was admitted into the hospital, and the sinus and cyst were explored under an anaesthetic. The boundaries of the cyst were still too extensive to be reached, either with the finger or with the longest metal sound and probe obtainable, the cyst seeming to extend beyond reach both towards the middle of the abdomen and downwards to the pelvis.

The elastic tube was reintroduced and the cyst well syringed out with an antiseptic solution.

The patient left the hospital January 22, 1882, relieved of the symptoms for which he had been readmitted and wearing the apparatus as before.

November 27, 1882.—At this date the patient was readmitted into the hospital with the history that he had remained free from pain and able to work without discomfort until a few days since, when he suddenly began to suffer from a renewal of pain in his left side. The pain was aggravated by pressure, and on examination seemed limited to the left hypochondriac and renal regions. The left side of the abdomen offered a sense of resistance and felt harder than the right; the pain was said, however, to be less severe than before the last exploration. No definite tumour could be discovered.

The patient was weak and had lost much flesh. He passed less urine *per urethram*, and more from the sinus in the loin. The urine passed *per urethram* was normal. The urinous fluid discharged from the sinus was very foul and contained much pus.
February 1, 1882.—The patient suffers great pain. During the last few days the temperature has varied from 99° to 102.8° F.

On December 5, 1882, the sinus was enlarged and the interior of the cyst again thoroughly explored. Some contraction and thickening seemed to have occurred at its upper part; but the lower boundary could not be reached. The kidney-substance apparently could be felt above.

The cyst was thoroughly washed out and a large drainage-tube was reintroduced.

December 28.—The patient has been much relieved by the last operation, his temperature falling to normal and remaining so for about a week. During the past fortnight, however, there has been a renewal of the former symptoms: pain, nausea, and general loss of strength, the temperature varying from 100° F. to 103° F. A profuse discharge of purulent urinous fluid, more or less foul, is discharged from the wound.

It was evident at this period that the patient was steadily getting worse and that the symptoms could not be relieved as before by mere drainage. The advisability, therefore, of the removal of the kidney was discussed, and the further question whether the kidney only should be removed or with it the large suppurating cyst also.

It was decided to remove the kidney alone, for the following reasons: (a) The operation of removing the large cyst (the boundaries of which had not hitherto been reached) would be attended by extreme risk, on account of the adhesions to the peritoneum and other parts which it was thought would probably have been the result of the long-standing inflammation and suppuration. (b) The cyst being probably not a pathological entity per se, but the result of some obstruction in the ureter, might be expected to wither, if the kidney or, in other words, the secreting organ which formed the physiological or pathological raison d'être of the cyst, were removed.

The operation of nephrectomy was accordingly performed December 28, 1882, by an incision in the loin, extending from the old sinus in that region, and by careful dissection through the thickened tissues which lay over the kidney and adjacent parts. The kidney was gradually enucleated by the fingers from within its proper capsule, the latter being left adherent to surrounding parts, and was tied up in portions with strong silk ligatures passed through its substance by
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means of stout aneurism needles, as near the level of the hilus as possible. As each piece was tied it was cut off beyond the ligature with scissors. Four or five portions altogether were thus tied before the kidney was completely removed. Very little haemorrhage occurred, and no damage was done to the peritoneum, which could be seen flapping as a thin membrane at the bottom of the wound with each respiratory movement. One end of the skin-incision was closed with a couple of silver sutures, and the remainder left open with a tube for drainage. Lint, with sanitas oil diluted with olive oil, was applied to the wound and covered by a quantity of Lister's shredded gauze.

The patient did well after the operation, although his convalescence was somewhat prolonged. For many weeks he was in the habit of perspiring profusely, especially at night, a symptom which, on physiological grounds, might be expected, and which I have noticed as a leading symptom during convalescence in another case of nephrectomy since under my care.

Some time elapsed before the silk ligatures came away; the last between two and three months after the operation. This minor trouble may seem too trifling even for reference, but it has happened to others as well as myself, and it may be worth mentioning that the probable cause will be found to be in the fact that the mass of tissue within the ligature does not perish in its whole thickness. The result, therefore, is that a cord of tough tissue remains surrounded by what is now a loose circle of silk, too loose to permit it to release itself by ulceration of the tissue which it surrounds. This, at least, was the cause of the delay in the separation of the ligature in the present case. By steady traction on the ligature a glimpse of the state of affairs could be just obtained at the bottom of the wound, and of course with a snip of the loop the ligature at once came away.

It seems unnecessary to give the detailed notes of the patient's progress after the operation, but it may be mentioned that the temperature was persistently somewhat high and variable for several weeks after the operation, notwithstanding the patient's steady progress towards convalescence. The pulse-rate, too, was high.

The readiness with which, so far as could be ascertained, the one kidney took up and duly performed the work of two, is also worthy of record although not, I believe, exceptional. In the first twenty-four hours after the operation one pint of urine was collected, in the next two pints, and thenceforward
the quantity varied only as it might normally, the average daily quantity being from two to two and a half pints.

The sp. gr. of the urine varied as a rule from 1012 to 1020. At no time was either albumen or sugar detected in it, nor could any pus be detected by the microscope.

The quantity of urea was below the normal average, but probably not below that proper to a patient under similar conditions of convalescence.

The patient left the hospital about the middle of April, 1883, and for several months was able to work. His general health was good, and although a small quantity of pus was discharged daily from the sinus in the loin it gave him but little trouble.

In November, 1884, he was again admitted into the hospital with pain in the left hypochondrium and increased discharge of an offensive character from the sinus in the loin. The pain was increased by pressure, and somewhat relieved by keeping the left leg drawn up.

January 3, 1885.—The various local and general measures adopted having failed to give any permanent relief, the patient was put under the influence of ether, and the sinus in the loin was enlarged. At the bottom of the sinus, what was left of the old hydronephrotic cyst was explored and found now to be much diminished in all its dimensions. It was of about the size of a large orange, and every part of its wall could be now easily felt by the finger. The cyst was found filled by a quantity of horribly offensive material, semi-solid and putty-like, consisting probably of pus, blood, and what seemed to be the degenerated lining membrane of the old cyst wall. At the lower part of this cyst was a narrow channel or diver-ticulum, along which, however, a probe could not be passed for more than about an inch. No calculous matter could be detected.

The cavity was thoroughly emptied of its contents and syringed out with an antiseptic lotion. It was dressed with diluted sanitas oil, a drainage-tube being left in.

From this time onwards nothing occurred worthy of detailed notes. The patient rapidly regained health and strength and the cyst cavity gradually contracted. For some months after leaving off the drainage-tube a narrow sinus remained from which a small quantity of pus was daily discharged; but a piece of cotton-wool, changed once daily, sufficed for a dressing.

At the date of the patient's being exhibited to the Society
Mr. Baker's Case of Nephrectomy for Hydronephrosis. 133

(February 11, 1887) he was perfectly well in every respect. On examining the loin closely it was possible to detect the situation of the old sinus track; but this was obvious only on a close examination, and the opening would barely admit a very fine probe. The patient was unconscious of any discharge from it.

The urine was perfectly normal. The patient was strong and healthy, and able, he said, to perform any amount of heavy work.
A WOMAN, æt. 52, was admitted into the London Hospital under my care December 28, 1885. For the notes of the case I am indebted to Mr. Rawes, who was at the time my house physician. She was an ill-nourished woman, but except that she had been losing flesh for three or four months, she declared that her general health had been good. (It may be stated at once that no visceral disease, except from cancerous changes, was found post mortem.)

On the afternoon of December 7, 1885, she, whilst standing at her work, had suddenly loss of power of the left leg; she turned quickly round, and caught hold of a table for support. She was carried home to bed, and was kept there until she was brought to the hospital December 28, three weeks after the attack.

There was paralysis of the left leg and of that limb only. According to her statement, there had not been paralysis of any other part. The paralysis was nearly complete; she could only just move the toes. The left knee-jerk was greatly exaggerated, the right was perhaps a little more glib than is common. There was well-marked left ankle-clonus. There was no defect of sensation. Except for changes of myopia there is nothing to say of the eyes.

December 30, two days after admission, whilst the patient was being lifted from the bed-pan, the left femur suddenly broke just below the great trochanter. No violence whatever had been used. Great deformity resulted with 2½ inches shortening. After this accident she was closely questioned as to previous pain in the limb. She said she had had pains in the left knee and about the hip, but not severe pains. She had not had pain in any other part, and did not complain of headache. She was transferred to the care of my surgical colleague, Mr. Rivington. I have nothing further to report of the case, except that she gradually sank and died January 10.

Necropsy.—Malignant growths were found in the right ovary, which measured about 3 inches in diameter. In the liver were nodules of the growth, varying in size from that of a
bean to that of a walnut; the viscus was otherwise normal. In the apices of the lungs there were small cavities, with cheesy deposits, in some places calcareous. A nodule of new growth was found in the right lung near the apex. The bronchial glands were affected by the growth. There were posterior hypostatic congestion and oedema of the lungs. There were two or three nodules of the growth in the skin; one was of the scalp over the posterior superior angle of the right parietal bone, the size of a walnut; it was beginning to ulcerate. Other organs were healthy, except for the left femur and the brain.

There was a fracture through the left femur just below the great trochanter; the medulla of this bone was affected by malignant growth, and the compact tissue was greatly thinned. The growth was small and could not have been felt during life. The brain was examined for me by my friend and colleague, Dr. James Anderson.

Brain and cord in spirit for one day. External surface of brain apparently normal. Vertebral and basilar arteries slightly thickened, meninges and branches of the cerebral arteries normal. In the fibres descending from the posterior portion of the right marginal gyrus and neighbouring portion of central convolutions is a nodule of oval shape; long axis $\frac{7}{8}$ inch, lying antero-posteriorly; short axis $\frac{3}{8}$ inch, lying transversely; its posterior extremity on the same vertical plane with the ascending portion of the calloso-marginal fissure, and about $\frac{1}{4}$ inch above the upper plane of the corpus callosum. The nodule is $\frac{1}{2}$ inch from the median surface, separated from it by the white fibres of the gyrus fimbriatus, and the grey matter of the calloso-marginal fissure, which lies as usual in a plane downward and outward. It has the naked-eye appearance of blood-clot, is sharply limited from the surrounding brain tissue, which is normally firm, and it exercises evidently considerable pressure inwards, perhaps less outwards.

In the extreme anterior portion of the middle frontal convolution under the cortex is a nodule the size of a small bean, vascular and solid posteriorly, breaking down anteriorly into a grey grumous fluid with a firm, grey wall, dotted with vascular points. Just below and external to it is a smaller grey nodule. In the left hemisphere, at a point precisely symmetrical with the large nodule above mentioned, is a pea-sized nodule, dark purple, studded with vascular points, lying $\frac{3}{4}$ inch from the median surface, and about $\frac{1}{4}$ inch above the upper plane of the corpus callosum. Half an inch behind and
below this is a second nodule of the same appearance, half the size.

Careful examination of the remainder of the hemispheres, of the central ganglia, pons, medulla and cerebellum fails to detect any further nodules. The left cerebellar lobe is unusually adherent to the restiform columns posteriorly, and there is some matting of the meninges and slight softening of the subjacent tissue at this point, but no naked-eye evidence of tumour. The spinal cord shows to the naked eye no pathological change.

Microscopically, one of the small nodules shows the structure of carcinoma with extremely scanty stroma, large vessels and numerous minute haemorrhages scattered throughout the nodule. The structure of the large nodule in the right hemisphere was not examined microscopically. It was undoubtedly, from naked-eye appearances, almost wholly haemorrhagic.

Remarks.—This case is of interest in showing the value of Ferrier's researches; the lesion answering to paralysis of the leg would cut off fibres coming from his "leg centre." Schäfer and Horsley have shown that the marginal convolution forms part of the leg centre. The diagnosis of monoplegia from cortical disease was enforced by the existence of exaggerated knee-jerk and foot-clonus. The occurrence of fracture on simply lifting the patient off the bed-pan was startling, and might have caused transitory illogical doubts as to the correctness of the diagnosis. Had there been no necropsy it might have been surmised that the disability of the left leg was from first to last fracture, and that there had been no paralysis at all. Especially easy would this hypothesis be to those, who do not believe in the diagnostic value of exaggerated knee-jerk and foot-clonus, and above all to those, who do not accept the results of recent researches on representation of movements in the cerebral cortex. The coincidence of fracture of a paralysed leg from cancerous disease, when that paralysis was owing to secondary cancer in the region of the leg centre, is a very strange one. The absence of headache and of optic neuritis up to December 30, at least eleven days before death, is worth notice.

Cases of monoplegia from limited subcortical lesions are not common. Dr. Hughes Bennett has reported one of the most important cases of this kind (brachioplegia); in that case there was a very limited lesion of the internal capsule (Brain, part xxix).
XXVII.—A Case of Hyperpyrexia in Acute Rheumatism successfully treated by the Cold Bath. By the late R. E. Carrington, M.D. Read February 25, 1887.

ALTHOUGH there is nothing new in the case which I have the honour of bringing before the Society to-night, I am led to do so because I venture to think the more often such cases are reported the more quickly shall we be able to accumulate a solid body of unimpeachable facts, which will enable us to combat the prejudices of the friends of patients, of necessity founded on ignorance, against a remedy which, desperate though it may apparently be, is the only one available under the circumstances,—I mean the use of the cold bath in hyperpyrexia. I have also been led to do so because I believe, at least so far as my experience has gone, that practitioners regard it in many cases as hopeless to suggest this mode of treatment. At all events the difficulty occurred with the father in the case I am about to narrate, even although he saw his son livid and comatose.

The patient was not in the general but in the paying wards of the hospital. In such a department it, of necessity, follows that the decision of friends has more authority than would be permitted in the general wards. The patient was under the supervision of his father, a gentleman of sterling good sense, and yet in spite of the desperate condition in which he saw his son, and which I am about to describe, he urgently wished me to temporise and use every effort short of the only one of any possible use.

The facts, which are of course well known, that cases of rheumatism complicated with hyperpyrexia are by no means proportionately severe as regards the articular affection, and that there may be sudden cessation of the pain, and of other symptoms, with apparent recovery of the patient, which excite our anxiety, come out markedly in the history I am about to detail.

For these reasons I cannot but think that it is our duty to publish and bring forward cases as often as they occur, and, so to speak, educate the public, and show them that this mode
of treatment is by no means so desperate as it seems to be, but on the contrary offers a fair hope of success. The case has also a sad personal interest, for it was the last that the late Dr. Mahomed saw, and who, though far gone in the illness that caused his death, came down late at night to aid me in its treatment.

The patient was a medical student, a strong muscular young man twenty-three years of age, of regular habits. He had been ill for a week with pains in the joints and malaise, and had been treated by a medical practitioner with salicylate of soda for what was seemingly a mild attack of acute articular rheumatism.

On the evening of October 31, 1885, he felt much better, and had risen from bed and lay on his sofa. During this evening he was visited by several friends. Perhaps the consequent excitement had something to do with the subsequent complication. His temperature at this time was 101·4°.

Next morning there had been nothing to excite suspicion, so that his temperature was not taken until between 12 and 1 p.m.—I am not sure of the exact time—but it was found to be 107°. Then I was asked to see him, and arrived at his lodging at 2 p.m. I found him then dressed and lying on the sofa, and on taking his temperature the thermometer marked 106°. He gave me a full account of his illness. He expressed himself as feeling quite well and asked how long he would have to keep indoors. He had neither pain nor swelling in any of his joints, but there was a rough systolic bruit all over the praecordial region which I thought was probably pericardial.

Fearing what was impending I made arrangements for having him removed to Guy's Hospital as quickly as possible. There was some little but unavoidable delay in doing this, and he did not arrive until 4.15 p.m. He had since my visit become rapidly delirious and was very noisy in the cab.

At 4.30 p.m. I was notified of his arrival, and I found him in bed, quite unconscious. His face was livid; he had general convulsions; his eyes were fixed and staring, and there was double external strabismus. His pulse was very feeble, and the fatal event seemed almost a question of minutes. The temperature in the axilla was then 109·8°.

Of course I had taken measures for having everything
PLATE III, ILLUSTRATING THE LATE DR. CARRINGTON'S CASE OF RHEUMATIC HYPERPYREXIA.
Dr. Carrington's Case of Hyperpyrexia.

ready for the cold-bath treatment. He was at once placed in the water at a temperature of 70° F., and then we proceeded to rapidly cool it down with ice. Thirty minims of brandy were administered to him subcutaneously. The quantity of ice used was very great, literally by the bucketful, for it is no exaggeration to state that the patient actually warmed the water. I was assiduously helped by Mr. Targett and Mr. Knaggs, the then house physicians, who diligently rubbed the patient with large pieces of ice. Further, we gave him fifteen grains of antipyrin per rectum.

In the bath the convulsions continued and he constantly made a groaning noise. He vomited once, bringing up, however, only a little frothy mucus. It was found that pouring ice-cold water over his head partially restored consciousness, and he became restless and strongly resisted our taking his rectal temperature. Subsequently he became quiet and his respiration deep and tranquil. He remained in the bath for a full hour, during which time he was unconscious. With a rectal temperature of 102°6, and being somewhat collapsed, he was removed from the bath, placed in bed on a blanket and lightly covered by a sheet. He still remained unconscious and soon became very restless, rolling about and tossing his arms, and then strongly marked tetanic convulsions supervened. Opisthotonus was strongly marked, and the legs and thighs were rigidly extended, to such an extreme degree that he could be lifted by his head and heels. There was distinct risus sardonicus and double external strabismus.

The temperature all this time was falling, and at 7 p.m. it was 97°4°. The spasms were now less frequent. His feet were very cold, and hot-water bottles were applied. I am not sure how far the antipyrin had to do with this somewhat alarming fall.

At 8 p.m. consciousness began to return. He took a pint of milk. The skin became warmer and red. The temperature 97°2° and the pulse 120.

At 9 p.m. the temperature was 99°8°.

At 10 p.m. he was quite conscious and sweating freely, and he had been sleeping comfortably. He was of course carefully watched, and his temperature taken every hour.

I do not propose to incorporate the whole record of temperature in the body of the report but will append it at the end (vide Plate III).

Progress of the case.—November 2, 7 a.m.—He felt very comfortable but was not quite rational at times. He was in
no pain, and his only complaint was want of sleep. He took milk and soda-water freely and twenty grains of salicylate of soda every two hours. His temperature now was 101·8°. During the day he slept at intervals. The bruit was now inaudible, and there were no respiratory complications. The temperature, however, rose slowly until at 12 p.m. it reached 103·8°. He was given fifteen grains of antipyrin by the mouth and quarter of a grain of morphia subcutaneously.

On November 3, at 2 a.m., the temperature had risen to 105·4°. He was now placed in the ice bath, and remained in about fifteen minutes. The temperature fell to 101·4° and he was removed. The quarter of a grain of morphia subcutaneously was repeated with the view of obtaining sleep.

6 a.m.—Temperature again up to 104·4°. The ice bath was repeated for another fifteen minutes and it fell to 100·2°. He then had another quarter of a grain of morphia subcutaneously.

11 a.m.—He had slept well; he was in no pain, but the temperature was again rising, as will be seen by the appended record.

At 2 p.m. I had the advantage of consulting with Dr. Goodhart. The temperature had reached 105·2°. The bath was repeated and we now kept him in forty minutes. The temperature fell to 100·8°. We gave him fifteen grains of antipyrin at one dose, and twenty-five grains of salicin were ordered every two hours. Further, by Dr. Goodhart's advice, he was placed on a water-bed, and ice bags applied to both axillae.

From that time I am glad to be able to record that we had no further trouble as regards temperature. He had a slight return of joint trouble on November 13 and 14, and the temperature rose to 101°, then fell, and he left the hospital in the first week in December quite well, save that he unfortunately had a systolic apex bruit.

I may say that I have seen him several times since, that he rapidly regained his strength, and has had no return of rheumatism. The bruit, however, persisted up to my last examination about a year ago; since then I have not had an opportunity of ascertaining the condition of the heart. It may be convenient to append the treatment as regards the medicines used, viz. salicylate of soda, salicin, and antipyrin.

November 2, 1 a.m.—Salicylate of soda twenty grains
Dr. Carrington’s *Case of Hyperpyrexia.*

every two hours. This was continued to November 3, on the afternoon of which day the following was substituted:

| B. Salicin, gr. xxv. |
| Liq. Ammoniae Acetatis, 3ij. |
| Syrupi Aurantii, 3ij. |
| Aquæ ad 3iss. Misce. |

Every two hours.

November 5.—The above mixture was given every three hours; on November 7 every six hours. On November 12 it was given every four hours; on November 14 every two hours; on November 15 every three hours; on November 19 every four hours; and finally, on November 22, a quinine mixture was substituted.

He took three fifteen-grain doses of antipyrin during the period of the hyperpyrexia, viz. on November 1, per rectum; in the bath on November 2, at 12 p.m.; and on November 3, at 2 p.m. It was not persisted in on account of its tendency to produce collapse.

He was given fish on November 7, chicken on November 12, and returned to meat diet on November 20.

The record of the temperature was as follows:

| Nov. 1.—2 p.m...106° | 6 p.m...102.2° |
| 4.30 p.m...109.8° Bath | 8 p.m...102° |
| 7 p.m... 97.4° | 10 p.m...103.2° |
| 9 p.m... 99.8° | 12 p.m...103.8° |
| 12 p.m...100° | Nov. 3.—2 a.m...105.4° Bath |

| Nov. 2.—1 a.m...100.2° | When removed from bath...101.4° |
| 2 a.m...101° | Soon after bath...100.4° |
| 3 a.m...101.2° | 4 a.m...100.2° |
| 4 a.m...101.4° | 6 a.m...104.4° Bath |
| 5 a.m...101.6° | Just after bath...100.2° |
| 6 a.m...101.8° | 8 a.m... 99.2° |
| 7 a.m...101.8° | 10 a.m...101.6° |
| 8 a.m...101.6° | 12 noon...103.8° |
| 9 a.m...102.2° | 2 p.m...105.2° Bath |
| 10 a.m...102° | Soon after bath...100.8° |
| 11 a.m...102.2° | 4 p.m... 97.2° |
| 12 noon...102.3° | 6 p.m... 99.2° |
| 1 p.m...102.3° | 8 p.m... 98.8° |
| 2 p.m...102.2° | 10 p.m... 98.2° |
| 3 p.m...102° | 12 p.m... 97° |
| 4 p.m...102.2° |

It would be tedious and useless to record further, although, of course, the temperature was frequently and continuously noted, but after this date it never rose above 99° save during the time of the slight return of articular pain, when it reached on November 13 and 14, 100° and 100.2° respectively.
Dr. Carrington's Case of Hyperpyrexia.

There are two points concerning which I should be glad to ascertain the opinion of the Society.

These are, first, as to the tendency of antipyrin to produce collapse; and secondly, whether, as I think, my case shows that a prolonged immersion has more effect than numerous shorter ones, of course watching the effect narrowly.
XXVIII.—A Case of Epilepsy following an Injury to the Leg. By W. B. HADDEN, M.D. Read February 25, 1887.

CHARLES S., st. 32, was admitted into St. Thomas’s Hospital under the care of Mr. Croft on June 23, 1886.

There was no family history of epilepsy. The patient had always been healthy until 1872, when he had an attack of yellow fever in Ashantee.

After this, in the same year, 1872, he was wounded in the left calf during an action with the natives. Several shots were extracted and the wound was quite healed in six or seven weeks. On his return to England, about four months after the injury, he began to have fits. For nine months they were occasional, but at the end of that time they became frequent, and he had as many as twelve in a day.

In July, 1873, the sciatic nerve was stretched by Sir Peter Eade, of Norwich. From that date up to seven weeks before his admission into St. Thomas’s Hospital, the fits entirely ceased. He then had a recurrence of the attacks. He was coming down a ladder one day when he suddenly lost consciousness and fell to the ground. After this he had four or five fits daily, and during the week preceding admission he had as many as eight or ten a day. The fit began with a sharp twitching pain in the scar at the lower part of the left calf; then there was a creeping, heating sensation passing up the leg and left side of the body to the head. Sometimes the patient had time to sit or lie down before he lost consciousness. The left foot, hand, and left side of the face became slightly convulsed. The right side was said never to be affected at this period. The pupils did not react to light during the attack, and the conjunctivae were insensitive. The breathing became slightly stertorous. After this condition had lasted for some seconds he had some general convulsive movements, and with a gasp or two he at once regained consciousness. The tongue was not bitten, nor were the urine and faeces passed during the attack.

The patient has since informed me that he occasionally
used to ramble in his speech after the convulsion, and at other times would remain quite quiet unless disturbed. There was nothing special to note in the appearance of the scar in the left calf, and there was no wasting or anaesthesia of the leg. The scar was somewhat tender and when pressed he would at times have the sensation passing up the left side, and (he has since informed me) occasionally a convulsion would follow.

On June 25 (two days after admission), it was noted that the fits were occurring about every two hours. In some of the seizures he would roll about in bed, and sometimes there was opisthotones. He complained that after some attacks his teeth became clenched and he could not open his mouth or articulate. On the evening of June 25 an attempt was made to stretch the sciatic nerve by means of extreme flexion of the hip, but the result was to throw him into the most violent convulsion he had yet had. Shortly after this I was invited by Mr. Croft to see the case, and give an opinion on the advisability of stretching the great sciatic nerve. I thought the procedure justifiable, especially in view of the relief which followed the operation thirteen years previously. The operation was performed on July 5. The nerve was exposed, freed from its surrounding fascia, and then pulled smartly in each direction. Something was felt to give way; but whether this was some branch of the nerve or the fascia around was not ascertained. Finally, the extremity was flexed on the pelvis and the nerve was stretched in this way also. No fits occurred for nine days after the operation. At the end of that time (July 13) he had a fit; next day he had three, and it is interesting to note that one of these commenced while the plaster splint was being bandaged at the knee. It is stated in the notes of the case that the left leg was violently convulsed.

July 15.—One fit. Thinks he may have had an attack in his sleep, as there was blood in his mouth when he awoke. He has never bitten his tongue during any previous fits.

July 16.—Two slight fits. One occurred after a fresh plaster-of-Paris bandage was applied, and the other when it was taken off to be padded.

July 19.—Three slight fits since the last note was taken.

On July 22 Mr. Makins summed up his condition as follows:—"fits still occurring with some regularity, sometimes two in a day, sometimes one every other day. Wound still granulating. Presented at own request. Sent out with plaster-of-Paris splint, and Lot. Sod. Chlor. dressing."
He took no bromide of potassium or other drug likely to influence the fits during his stay in the hospital. I must add that the urine was examined on admission and found free from albumen.

I wrote to the man five months after he left the hospital and received additional information, some of which is embodied in the account just given. He says, "I have had no fits since I left St. Thomas's Hospital, but I cannot walk far. If I walk a mile my left leg becomes useless, a pain commences at the back of the calf of the leg (not in the thigh, where Mr. Croft operated). This pain becomes so intense that my leg loses its usefulness, and I cannot get the foot off the ground. Then I have the fitty sensations, but do not lose consciousness, but my memory for the time being entirely fails me." He goes on to say that he has severe attacks of cramp in the leg, so that he almost screams with pain. He believes that there are still some shots in his leg, but he seems to have no reason for this opinion, except the pain from which he suffers. In answer to my inquiry, he states that, so far as he knows, no nerve was damaged when he was wounded fourteen years previously.

Remarks.—I do not think there can be a doubt that the fits were genuine. So far as I know, there was no motive for malingering, and it must also be added that he left the hospital at his own request.

The character of the fits, the effect of nerve stretching, and, what is much more important, the immobility of the pupils on exposure to light, and the insensibility of the conjunctivæ negative the idea of imposture. The condition of the pupils and conjunctivæ I tested myself during the solitary attack which I had the opportunity of seeing.

The case, however, is especially interesting with reference to Brown-Séquard's experiments on guinea-pigs. This observer, as is well known, produced epilepsy by injury to the sciatic nerve. Other investigators have since confirmed the accuracy of Brown-Séquard's observation.

Mr. Victor Horsley has recently pointed out that authorities differ as to the length of interval between the injury to the nerve and the onset of the first convolution. The incubation period varies between two and six weeks, and Mr. Horsley is inclined to ascribe this difference in latency to racial peculiarities.

It will be observed that in the case just described a period

* Lancet, December 25, 1886.
of four months intervened between the gunshot wound and the onset of the first convulsions. I must also call attention to the occurrence of opisthotonos in this case; a well-marked condition in the artificial epilepsy of guinea-pigs. It is also of importance to note the effect of nerve stretching. Looking back on the case, I am now inclined to believe that it would have been desirable to explore the site of the old injury with the view of ascertaining the presence or absence of shot. It is possible too that the removal of the scar might have proved more beneficial than nerve stretching.

CASE 1.—J. P., æt. 73, was admitted into Kenton Ward, under the care of Mr. Savory, on October 20, 1885, suffering from retention of urine.

He said that for some time past he had had difficulty in micturition, and that he took a long time to pass his water. Latterly he had used an india-rubber catheter.

On October 13, for the first time, he had complete retention which he could not himself relieve. After a time urine began to dribble away, and he then noticed that it was of a red colour. It had not been so before. Hæmaturia, with dribbling of water from the hyperdistended bladder, continued until admission.

On examination, the bladder was found to reach about midway up to the umbilicus, the prostate, felt per rectum, was greatly enlarged, and blood-stained urine in small quantities dribbled from the meatus. The patient was in great pain and very feeble. A catheter was passed by the house surgeon, Mr. Field, without much difficulty, and a couple of pints of dark red urine were withdrawn with much temporary relief. After this the patient was able to micturate without assistance, and during the next twelve hours he passed three pints of bloody urine.

On the morning of the 21st a catheter was passed and the bladder was gently washed out with a solution of Condy’s fluid. The hæmaturia continued during the 21st, and in the evening the patient died exhausted.

The urine passed, or drawn off, whilst the patient was in the hospital, contained a great quantity of blood, but no clots. The specific gravity varied from 1015 to 1025. The reaction was alkaline.

A post-mortem examination showed the prostate to be generally enlarged, the middle lobe forming a distinct projection into the floor of the bladder. The bladder was slightly dilated, though otherwise normal. It contained about a pint of almost pure blood. Both kidneys were
small, contracted, and granular on the surface. The cortex of each was slightly diminished. The right kidney was infiltrated with blood throughout the cortex, and in the bases of the pyramids, giving it a dark purple colour. The ureters were empty, but the renal pelves contained a little blood.

There were calcareous deposits on the aortic and mitral valves, but otherwise the viscera were normal.

Microscopically examined, the right kidney was found to be in a state of interstitial inflammation. Its connective tissue was increased in quantity and was the seat of a small-cell infiltration. There was also some proliferation of the renal epithelium with granular degeneration of the same. The whole of the kidney was infiltrated with blood, both the renal tubes and the fibrous stroma being alike affected. The haemorrhage was most profuse in the cortex, but was not limited to this part of the gland.

Case 2.—J. L., æt. 49, was admitted into Kenton Ward, under the care of Mr. Savory, on October 21, 1885, having for the previous ten days been under treatment in Mark Ward by Dr. Andrew.

His history was that until three months previously he had been in pretty good health. At that time he had pain in the loins, and his urine became very red. He passed a good quantity of water, and had no rigors or sickness. For the past six years he had suffered from stricture of the urethra and difficulty in micturition. Since his illness began three months ago, he had passed his urine more frequently than previously and said that it had always been red.

The last few days previous to admission he had had much headache and had slept badly. He was not, and had not been, an intemperate man.

Present condition.—Badly nourished. No oedema, arteries rigid, pulse strong and jerky. Heart dulness increased, and impulse heaving. Soft apex murmur and loud second sound. No oedema of legs or face. Slight cough. Temp. 97°, pulse 80.

Urine bright red, sp. gr. 1015, alkaline, contains much blood, and about ⅓ albumen. It smells foully. On standing, it separates into three layers. The upper layer is smoky, the middle layer bright red. The lower layer is gritty and whitish in colour. Microscopically examined, blood-corpuscles and crystals of triple phosphate are found, but no renal casts.

October 23.—Slept badly. Is very thirsty. Passed 46 oz.
of water in past twenty-four hours, bright red, and mixed with much blood.

October 24.—Better night. Passed 68 oz. of water of much the same nature as on admission.

October 31.—Patient seems very ill. Is constantly micturating. Slight cough with râles at bases of lungs. Headache, and pain in the loins and supra-pubic region.

An attempt was made to pass a catheter, but no instrument could be introduced.

November 1 (7 p.m.).—Severe vomiting. Shortly after this he passed into a semi-conscious state.

November 2.—Slept badly and occasionally vomited. Is in a stupid condition and has slight twitchings of the left arm. Passed about 50 oz. of water of much the same nature as on admission.

November 3.—During yesterday afternoon the patient became gradually weaker. At 1 a.m. he became quite unconscious, and at 1.45 a.m. he died. At no time had he any convulsions.

Post-mortem examination.—Heart: left side much hypertrophied. Weight 18 oz. No valvular disease.

Lungs: Broncho-pneumonia with consolidation at left side.

Kidneys: Typical specimens of interstitial nephritis; 3½ oz. each, contracted, granular, and tough, with adherent capsules and shrunken cortex. The pelves and ureters, neither of which were dilated, contained a little blood.

Bladder: Slight hypertrophy of muscle-coat and inflammation with pigmentation of mucous membrane.

Urethra: Tightly strictured in the membranous portion, and a little dilated behind the seat of stricture.

Case 3.—J. D., æt. 64, was admitted into St. Bartholomew's Hospital under the care of Mr. Willett, on February 3, 1886. He was passing considerable quantities of blood in his water, and gave the following history:

His health had always been good. He had never lived out of England. Had scarlet fever when young and acute rheumatism at the age of 17. He had never suffered from any venereal disease. For the last two or three years he had noticed that his urine was not passed in so forcible a stream as formerly, and he was generally obliged to get up once or twice in the night to empty his bladder. The act of micturition was never painful or difficult; he had never had retention.
Sixteen months before admission he had an attack of pain in the loins, with slight shivering, and noticed that his water was of a dark colour. He felt ill for five or six days, and then recovered.

Five weeks before he came under notice he had another attack of pain in the loins, more severe than on the previous occasion, and accompanied by much shivering and nausea. This lasted several days, and he was obliged to keep his bed. A week after the attack commenced he noticed that his water was red. The pain in the back steadily decreased for a fortnight, but had not completely passed away when he was admitted. During the attack micturition became very frequent, but was never painful. He said that he had to pass his water every half hour at first, but as the pain subsided the frequency of micturition also passed away, so that when he came into the hospital he passed his water five or six times in the day, and once or twice at night. He said that the blood was always mixed with the urine, that it never came by itself at the end of micturition, and that he never passed any clots.

Condition on admission.—A man of middle height, with pallid countenance, and looking as though he had recently lost a good deal of flesh. His viscera are apparently healthy. His pulse is 60 and the arteries are hard and tortuous.

The act of micturition is nearly natural. The stream is of full size, and forcibly propelled. There is, however, slight dribbling when the bladder is almost empty.

The urine is bright red, but not turbid. It is acid, contains \( \frac{1}{12} \)th albumen and a considerable quantity of blood. The presence of the latter was determined both by microscopic examination and by testing with guaiacum and ozonic ether. No casts or crystals were found.

The blood is uniformly mixed with the urine, and there is no bright blood passed at the end of micturition.

Examined per rectum the prostate was not found to be definitely enlarged, but there was a slightly greater resistance and induration in its situation than are natural.

An ophthalmoscopic examination showed on the right side a blurred, ill-defined disc, with some retinitis and effusion along the blood-vessels. The media were not clear on the left side, the lens was so opaque that nothing definite could be seen in the retina and optic disc.

For sixteen days after admission he continued to pass urine with very large quantities of blood. Some days it
seemed that at least one fourth of the total quantity of fluid passed consisted of blood. His temperature was persistently subnormal. He passed about three pints of urine in every twenty-four hours.

On February 17 the urine quite suddenly became almost clear. It remained tinged with blood for four days, but from the 22nd day of the month the haematuria entirely ceased.

He remained in the hospital until March 12. From the time the haematuria ceased the quantity of urine was much diminished, averaging from two pints to two pints and a half. It was acid, its specific gravity varied from 1008 to 1013, and it always contained a little albumen.

I have brought these cases before the Society, because I find that the subject of haematuria in connection with granular kidney is not mentioned in most works on medicine, or surgery, and is not, I think, sufficiently recognised as a clinical symptom. Quite recently (Lancet, July, 1885) my friend Dr. West has recorded some instances of renal haemorrhage in patients suffering from interstitial nephritis, but these are the only published cases with which I am acquainted.

The majority of the cases which I bring before you this evening differ from Dr. West’s in the profuseness of the haemorrhage, and I do not know of any parallel instances. In one of these death appeared to be directly due to loss of blood, and in the others the patients were reduced to a very anaemic state. I am not aware of any recorded post-mortem examination of such cases, or of such a condition of the kidneys as I have described in connection with the first case.

From a surgical point of view the amount of blood passed is of much importance, and will in future, in my opinion, merit very careful attention in the diagnosis of the source of a urinary haemorrhage. The condition of the urine in the first of the cases I have narrated certainly deceived most of those who saw it, myself amongst the number, and the brightness of the blood seemed to point clearly to a vesical origin, though with the post-mortem results of this case in my mind I was able to make a more correct diagnosis in the remainder. But even if it is clear that the haemorrhage is of renal origin, the question of diagnosis remains of much importance, and unless it be recognised that the blood may emanate from a kidney which is simply granular, operations may be undertaken under the impression that a renal calculus is present, or that other conditions amenable to operation may be found.
In one of my cases an ophthalmoscopic examination was of much service, and in the others the hypertrophied heart, and the high arterial tension in their early stages, helped the diagnosis. If the blood clears up, the urine will be found to present the characters common to all cases of interstitial nephritis.

It is worthy of remark that in the two fatal cases there was obstruction to the escape of urine from the bladder, in one the result of an enlarged prostate, in the other of a strictured urethra. Judging by post-mortem experience, as well as by clinical practice, I should say that interstitial nephritis is frequently the result of such obstruction, and is found quite independently of those other conditions of dilatation and absorption which go to make up the so-called "surgical kidney."

I do not suppose that cases such as these are really of great rarity; no doubt others have been observed, though not recorded, and the hope of inducing members of this Society to relate their experience is my excuse for this communication.
XXX.—On Cases of Pneumothorax in persons apparently healthy. By F. de Havilland Hall, M.D.
Read March 11, 1887.

Cases of pneumothorax occurring in persons apparently healthy and resulting in complete recovery have attracted a considerable amount of attention recently, so I do not think I need any excuse for bringing before the Society a case observed by myself, and one communicated to me by my friend Mr. B. Rix, of Tunbridge Wells, and I will also give a few notes of three other cases which are not included in Dr. Samuel West’s summary of twenty-four cases in vol. xvii of this Society’s Transactions.

F. R. P., æt. 24, a clerk. The patient was sent to me by Dr. W. L. Penny on June 27, 1885. From Dr. Penny I obtained the following particulars. On May 8, 1885, the patient applied at his house suffering from a sharp pain in the left side of the chest. “Low down in the mid-axillary line there was distinct friction, with slight dulness on percussion, and breathing somewhat tubular. Pulse 110, temp. 100·4°. The dulness covered a very small area; all the rest of the chest on both sides was quite normal. Heart-sounds natural. I sent him home to bed. The next day, to my astonishment, all breath-sounds had disappeared over the whole of the left chest except at one point over the lower part of the scapula where the respiratory murmur was faintly and distantly audible. Percussion note hyper-resonant all over left side and extending half an inch over the sternum to the right. Breathing replaced by amphoteric roaring and loud tinkling. Heart-sounds muffled, apex-beat half an inch inside right nipple. Pulse 140, temp. 99°. He stated that after getting home the previous evening whilst going to bed he suddenly felt faint and had a peculiar sensation about the heart, and I concluded that the rupture into the pleura took place then. He kept perfectly quiet for a month, the temperature being generally 98°. During that time the symptoms slowly subsided, the heart returning to its natural position, and the breath-sounds returning. At the end of a month he went out against my wish, and kept about for nearly a fortnight, but on June 23, he complained of pain and shortness of breath and I found that
the symptoms had returned, though not to so marked an extent as at first. My treatment has consisted chiefly in giving him Syrupus Ferri Iodidi and Oleum Morrhuae, and trying to keep him quiet, which was a difficult task."

When I saw the patient on June 27, I learnt from him that in spite of the return of pain he had on June 23 he had continued at business. He now complained of only slight pain, and there was not much dyspnoea. Pulse 120. Heart-sounds clear. Impulse in epigastrium. The left side of the chest hardly moved at all; there was general hyper-resonance and obliteration of the cardiac dulness. No breathing sounds were audible over the left side. The bell-sound could not be elicited. The right lung was normal. The urine contained a trace of albumen.

The patient's height was 6 feet and weight 9 st. 11 lbs. which is much below the average for his height. There was no family history of phthisis, and his previous health had been good.

On September 30 the patient came again to report himself. He said that after seeing me in June he had followed my advice and kept perfectly quiet in bed for a fortnight; that he had kept at home for another fortnight. He then went to business for a week, and after that he spent some time at Bournemouth. He told me that he felt perfectly well. On examining him I found that the heart was in its normal position. Pulse 92. The lungs were quite free from any signs of disease. There was a doubtful trace of albumen in the urine. His weight was 9 st. 13 lbs.

The particulars of the following case were sent to me by my friend Mr. B. Rix, of Tunbridge Wells.

J. W., æt. 39, mason's labourer. Father and mother alive and well. Has seven brothers and three sisters alive and well. Lost one sister, æt. 16, cause unknown. He has lost one child fifteen months old of inflammation of the lungs, another thirteen months old of broncho-pneumonia, and one, aged seventeen, of phthisis. Has six children alive, and, with the exception of one which had threatening of tubercular meningitis, all healthy.

He had typhoid fever at the age of fifteen, otherwise healthy, and has been in constant work for the last eleven years. He is of fair complexion, florid face, sandy hair. He has always been a very temperate man.

On May 23, 1886, while sweeping, he felt a sudden pain just above the left nipple and came over faint. He continued
at work for five days, but found himself gradually getting breathless, and on the fifth day his breath was so short that he had to give up work, the pain getting daily worse. At the time of the attack he was feeling quite well; he had had no cough in particular. When in bed the breathing was comfortable, except one night, when he had to lie with his head high, and he could not lie on the left side all through the illness. He was first seen on the fifth day, when the left side was found not to move during respiration. Apex-beat to right of sternum. Hyper-resonance all over left side. No respiratory sound to be heard. Slight metallic tinkling at the base of axillary region. Severe pain all over left side, worse at pit of stomach. No cough, no rise of temperature, no vomiting. Appetite good, but he was losing flesh fast and looked very pale. Suffered from palpitation on exertion. He was under treatment seven weeks, during which time the heart gradually resumed its normal situation, hyper-resonance disappeared, and respiration became normal over left side of thorax. The man was able to return to work, only feeling a little weak, but now (October 18, 1886) he has gained flesh and feels as strong as ever he did, only now and then suffering from palpitation on exertion. The left side of the chest is decidedly more resonant than the right, and the respiratory murmur is also louder on the left, but vocal resonance and fremitus are more marked on the right side. Both sides move equally well, and the apex-beat is exactly in a line with the left nipple, one finger's breadth below it.

The second case is recorded by Dr. Delgrange.*

“A youth, aged 18, possessing an excellent constitution, amused himself one evening in exhibiting the power of his chest and the force of his breath. Suddenly he felt a pain in his left side, his face became blanched, and he experienced great oppression. During the night he made attempts at vomiting, and in one of these efforts was seized with a sense of something tearing in his side. Syncope immediately followed. Dr. Delgrange was called in, and found unmistakable evidence of pneumothorax. Under appropriate treatment, the patient recovered after about eight days.”

The third case is recorded by Biermer.†

“Karl D., law student, aged 19, was present at a ball on February 3, 1857, and during a quick dance he was suddenly seized with a peculiar sensation in the neighbourhood of the

* Journ. des Sciences Méd. de Lille; London Medical Record, 1881, p. 196.
heart, of which he said 'that it seemed to him as though the heart had moved from its place and had jumped up.' This unpleasant and strange sensation was accompanied with giddiness, oppression, and sweating, so that the patient was obliged to discontinue dancing immediately. He remained at the ball, but felt a continual inclination to cough, with a stitch in the side, and noticed that his voice had become somewhat hoarse and toneless. Soon after his return home he was seized with shivering followed by heat; he had severe pain in the head during the night and constant cough. Towards morning these symptoms abated somewhat. He therefore got up as usual and went to his employment, and though in the following days he felt shortness of breath and oppression, especially on walking and going upstairs, dull pain in the side on talking and taking a deep breath, hoarseness, pain in the head and recurrent fever in the evening, he, however, only sought medical aid on February 7.

"The physical examination showed the signs of a pneumothorax on the left side, accompanied by a moderate amount of liquid effusion.

"In seven weeks all objective signs of the pleural affection had disappeared, and the patient was discharged from treatment, and some months later he appeared better than before his illness."

The case recorded by Ricker* is that of a badly-developed young man eighteen years of age, of pale countenance and defective nutrition. He had suffered from scrofulous glands from childhood. Both parents were healthy, but there was the history of glandular affections and lupus in the father's family. The patient had been prevented from working all the winter by his affection, but when he came under Dr. Ricker's observation he had an occasional cough; the examination of the chest showed no other signs except a slight catarrh.

Ten days later he began to cough more and complained at the same time of a stabbing pain in the left side. When the patient was seen again about a week later he complained of considerable shortness of breath. The features were pale, the skin cool and sweating, the respirations very frequent and laboured, the pulse small and accelerated. The physical signs were those of a pneumothorax. Two days later the patient's distress had much increased; he was unable to lie down for an instant without causing the greatest danger of suffocation. The countenance was cyanotic and bloated. The respirations

were extremely laboured, about 60 in the minute. There was no trace of a pleuritic effusion. To relieve this threatening condition a puncture was made with a trocar in the sixth interspace in the axillary line and the air rushed out with a hissing noise. The patient immediately perceived a considerable alleviation, the cyanosis disappeared, and he could now lie on his back. During the evacuation of the air the heart, which had been displaced downwards and to the right, moved upwards. The patient gradually improved, and in the course of a week was able to leave his bed for some hours. His nutrition quickly improved and he presented a more healthy appearance than formerly. When examined some two months later the heart had regained its former position, there was a difference in the tone of the percussion note on the two sides, and the respiratory murmur anteriorly was weaker on the left than on the right side.

The sudden occurrence of pneumothorax in a person of apparently good health and followed by complete recovery in the course of a few weeks is a circumstance calculated to rivet attention, and one is astonished to find that it is only comparatively recently that an attempt has been made to collect the cases and to endeavour to discover the pathology of this condition. That these cases are not very common may be inferred from some remarks of Biermer,* who says that the recovery of a non-traumatic pneumothorax is so rare that only a few well-authenticated examples of it are to be found in literature, and he quotes Lebert to the effect that though his 'Handbook of Practical Medicine' is founded on the basis of 6000 histories of illness observed by himself, he has never been so lucky as to see a single case of this kind end in recovery.

To-night I invite your attention to thirty-one instances of this condition. Of these twenty-one were communicated to the Society in 1883 by Dr. Samuel West,† five were brought before the notice of the Medical Society in March, 1886, by Dr. Whipham, and the remaining five are the cases on which my paper is based.

In analysing these thirty-one cases one is at once struck with the enormous preponderance of males, the ten cases which I propose to add to Dr. West's list being all males. In looking up Vogel's case (No. 4 in Dr. West's list) I find

† I exclude three of Dr. West's cases as being of traumatic origin. Traumatic pneumothorax is so common, and the explanation of the mode in which it occurs is so obvious, that I shall say nothing further on the subject.
that the patient was a female at 29. That will make therefore eleven cases in which the rupture occurred in males, and only two in females, or taking the two lists together, twenty-one males to two females. The explanation of this great preponderance of males over females is to be found, I believe, in the greater muscular exertion of males compared with females.

Whatever may be the predisposing cause there is little doubt in my own mind that the immediate cause of the pneumothorax is some strain. This came out very markedly from an examination of the ten cases of Dr. Whipham and myself. In only three of the cases there was no evidence of over-exertion, in two the pneumothorax occurred three days and one day respectively after unusual exertion, in the remaining five the rupture occurred while the exertion was taking place, and, as if to clinch the argument, one patient had a recurrence a month later while doing exactly the same thing as had produced the original attack.

The next striking point is the age of the patients. It is, as Dr. Whipham writes, not a little remarkable that, on looking through Dr. West’s table, with two exceptions (in one of these the age is stated at fifty, and in the other the patient was said to be elderly), the patients are under thirty-eight years of age, and most of them under thirty. The table of ten cases which I now bring forward is in agreement with this statement, the age of the eldest patient being thirty-nine, and the youngest eighteen, the average age being just over twenty-four and a half years. As regards the time occupied in recovery from this affection, in the ten cases one recovered in about eight days. With this exception they varied from one to two months, so we may regard five or six weeks as about the average period for complete recovery from non-traumatic pneumothorax occurring in apparently healthy persons.

The above are all the facts which the cases before us offer, and when we attempt to give an explanation of the pathology of this condition we at once leave the sure foundation of fact and embark on the uncertain sea of conjecture, and I have no explanations to put forward other than those advanced by Dr. Whipham, viz. that the causes may be divided into two groups: “1. Those which are presumably external to the lungs; and, 2. Those which are probably the result of change in the pulmonary tissue.” The first explanation will account for only a very small number of cases, and is only applicable where there is a history of old pleurisy. It is quite conceiv-
able that, given pleurisy with adhesions, an unusually severe exertion may throw so much strain upon the adhesions as to cause them to tear away part of the visceral layer of the pleura, and thus allow air to enter the pleural cavity. In the majority of the cases, however, pneumothorax is due to changes taking place in the lung tissue itself, and from a study of the cases on record I have no doubt in my own mind that the rapid breaking down of a small tuberculous mass before a secondary pleurisy has had time to set up adhesions, and thus prevent the escape of air into the pleural cavity, is by far the most frequent cause. As is well known, it is impossible to recognise these small infiltrations, by physical signs, and it is only by the after-history of these cases that the cause of the pneumothorax can, with any degree of certainty, be determined.

As an objection to this theory it may be said that though phthisis is so common, how comes it to pass that we meet with so few cases of this kind of pneumothorax? An observation of Vogel's* will furnish the reply. He has pointed out that cavities at the apex of the lung more rarely perforate than those in other parts of the lungs. The explanation is that on the one hand the interval between the pulmonary and costal pleura is here very slight, and on the other hand that the expansible tension of the parenchyma of the lung during inspiration is less considerable in the upper than the lower part of the lung. Now, as the majority of the cases of pulmonary phthisis begin in the apex it will at once be seen that this fact affords an answer to the objection. The other cause of pneumothorax occurring in the apparently healthy is the rupture of an emphysematous vesicle. The case of a pedlar, described by Flint, who developed this condition while carrying a heavy weight, is probably an instance of this kind.

The physical signs of pneumothorax are usually sufficiently well marked to render the diagnosis very easy. The distention of the affected side, the obliteration of the intercostal spaces, the displacement of the heart, and the abolition of cardiac dulness, if the effusion be on the left side, the tympanic percussion note, the loss of vocal fremitus and resonance, the absence of all breathing sounds, or the presence of amphoric breathing, and the bell-sound, constitute an aggregate of signs which is very striking. The only cases of difficulty are those in which we have to deal with a limited pneumothorax. Here the diagnosis has to be made from cavities in the lungs, dilated bronchi, lungs compressed by pleuritic

effusion, and from certain rare cases of pneumonia. Biermer has entered very fully into this question in an able article in the Würzburger Medicinische Zeitschrift for 1860. But there is one sign as to the value of which in a diagnostic point of view I am uncertain, and this is metallic tinkling. That it occurs in cases of pneumothorax, where there is no other evidence of fluid in the pleural cavity, seems highly probable, but I am unable to give any explanation as to its mode of origin in these cases.

Walshe* mentions that some observers have heard metallic tinkling in the absence of a liquid effusion. He remarks on this, "Low-pitched echo may intelligibly be produced under the circumstances; but the absence of fluid, especially where vocal sound is the alleged cause of the phenomena, makes it desirable that, in respect of tinkling, the observations should be repeated." In a footnote he adds, "I must admit, however, in respect of the necessity of the presence of fluid as a condition of metallic tinkling, that I have met with one or two cases where I was not perfectly sure, though true metallic tinkling existed, that liquid was present."

The subject of treatment can be speedily dismissed. If there be symptoms of shock at the time of the entrance of air into the pleural cavity the exhibition of diffusible stimulants may be required. The injection of twenty to sixty minims of ether subcutaneously would fulfil this indication admirably. Many of the cases seem to have been accompanied with severe pain, and the subcutaneous use of morphia has generally given relief. As Dr. Douglas Powell* pointed out some years ago, the engorgement of the venous system, and more particularly of the portal system usually consequent upon pneumothorax, may be relieved by the cautious administration of such purgatives as produce a watery flow from the intestines. Moreover, it is certainly desirable that there should be no occasion for the patient to strain at stool, as this exertion might cause a fresh rupture in those cases in which healing had occurred.

The only other point is the question of paracentesis. This operation is required when there is excessive dyspnoea or failing action of the displaced heart. Paracentesis was had recourse to with great benefit in four out of the twenty-one non-traumatic cases of Dr. West. In the ten cases to which I am now directing your attention it was only required in one

† Med. Times and Gazette, 1869, p. 194.
patient. A single paracentesis seems sufficient to give relief. In all cases the main element in treatment is absolute rest in bed.

### Table of Cases of Pneumothorax.

<table>
<thead>
<tr>
<th>No.</th>
<th>Author and reference</th>
<th>Sex</th>
<th>Age</th>
<th>Side affected</th>
<th>Duration</th>
<th>Cause and observations</th>
</tr>
</thead>
<tbody>
<tr>
<td>3</td>
<td>Ditto</td>
<td>M.</td>
<td>37</td>
<td>Right</td>
<td>2 mos.</td>
<td>Came on after unusual exercise in garden.</td>
</tr>
<tr>
<td>4</td>
<td>Flint, Principles and Practice of Medicine, 1883, p. 156</td>
<td>M.</td>
<td>29</td>
<td>Left</td>
<td>7 wks.</td>
<td>Came on while carrying weight of about 70 lbs. A month later another attack from similar cause. Emphysema.</td>
</tr>
<tr>
<td>5</td>
<td>Fraentzel, Ziemssen’s Cyclopaedia, vol. iv, p. 746</td>
<td>M.</td>
<td>19</td>
<td>—</td>
<td>6 wks.</td>
<td>Came on while rolling a heavy barrel. Nothing abnormal to be detected in the lungs.</td>
</tr>
<tr>
<td>6</td>
<td>Delgrange, Journ. des Sciences Méd. de Lille</td>
<td>M.</td>
<td>18</td>
<td>Left</td>
<td>About 8 days</td>
<td>Came on while exhibiting the power of his chest and the force of his breath.</td>
</tr>
<tr>
<td>9</td>
<td>B. Rix</td>
<td>M.</td>
<td>39</td>
<td>Left</td>
<td>7 wks.</td>
<td>Came on while sweeping.</td>
</tr>
<tr>
<td>10</td>
<td>F. de Havilland Hall</td>
<td>M.</td>
<td>24</td>
<td>Left</td>
<td>1 month</td>
<td>Fortnight later he had a similar attack.</td>
</tr>
</tbody>
</table>
ELIZA P., æt. 34, was admitted to the London Hospital with cancer of the rectum. The symptoms began nine months before, when she complained of severe pain in the rectum and frequency of defaecation with diarrhoea. She had passed blood for three months. During the last few weeks she had only passed one motion daily.

**Condition of rectum.**—The anus was unaffected. Above it the rectum was involved throughout its whole circumference by an ulcerated cancerous growth. While the patient was under an anaesthetic the finger could apparently be passed above the growth, and Mr. Treves, who had previously examined the case, was of the same opinion as myself. The rectum was not adherent to the vagina nor at any other point.

**Operation on August 3.**—The operation was performed in the usual manner, an incision having first been made down to the coccyx. After separating the rectum from its surroundings as high as was deemed advisable it was found that the upper limit of the growth had not been reached. The operation was continued and the peritoneal cavity opened anteriorly. The entire circumference of the reflection of peritoneum upon the rectum was severed partly by tearing and by touching resisting points with the thermo-cautery. After its complete division the rectum, which had previously resisted traction, could readily be drawn down. The diseased portion was removed with a wire écraseur. Hæmorrhage was slight. The portion of bowel removed measured six inches in length and the line of section was at least half an inch above the upper limit of the disease. After thoroughly cleansing the cavity of the wound the bowel was secured to the anal margin by two or three sutures, and a large drainage-tube was passed into the cavity of the sacrum. Not the slightest traction was necessary in bringing down the bowel. Intestine was seen, but there was no tendency to protrusion. Iodoform with wood-wool pads were used as dressing. The constitutional disturbance after the operation was exceedingly slight and there was at no time any evidence of peritonitis. The temperature did
not reach 100° until the seventh day, when slight secondary fever occurred, which rapidly subsided, the temperature being quite normal on the fourteenth day.

Drainage of the wound was facilitated by keeping the patient as much as possible supported by pillows in a semi-recumbent position.

Portions of the lower edge of the bowel sloughed and slight retraction took place, but after healing was well advanced the bowel was only an inch to an inch and a half above the anus.

August 17. — Wound covered with healthy granulations.

October 5. — She left the hospital quite well, and having gained flesh considerably.

The happy issue of the case was probably in a considerable measure due to the assiduity of Mr. Perry, the house surgeon, who washed out the wound with carbolic acid or perchloride of mercury lotion after each passage of feces. The bowels were at first confined with opium.

The portion of bowel removed, after contraction from immersion in strong spirit, measures five inches in length, and the line of section is three inches above the recto-vaginal reflection of peritoneum. The disease consists of an annular band of firm cancer, three inches broad. It is chiefly limited to the mucous membrane and mucosa, but the muscular coat shows considerable fibrous thickening. The lower margin of the growth is an inch to an inch and a half above the anus and the upper margin is half an inch below the line of section. The cancerous nature of the disease was confirmed by microscopic examination.

Remarks. — In relation with this case some points connected with the pathology of peritonitis and with the operation of excision of the rectum may appropriately be discussed. The experiments of Wegner, recently confirmed and more exactly followed out by Grawitz (Charité Annalen, xi, Jahrgang, p. 770, and Cblt. f. Chirurgie, November 6, 1886), show the exceeding tolerance of this membrane to septic and pathogenic micro-organisms. But to avoid inflammation certain conditions must be complied with. A stream of air may be for some hours passed through the peritoneal cavity without ill effect. Even organisms which induce suppuration, such as the Staphylococcus aureus, may be injected in solution in water without exciting suppuration, provided that the fluid is not in too great amount to be absorbed in a few hours. Grawitz sums up the conditions under which the
introduction of pyogenic organisms produces suppurative peritonitis as follows:—

(a) If the peritoneal cavity contain a stagnant nutrient fluid, in which bacteria can increase in numbers and penetrate the serosa, which reacts with cell growth and out-wandering of leucocytes.

(b) If by caustics the tissue of the peritoneum is destroyed so that the bacteria can become disseminated in it; simple irritant substances, as turpentine, are harmless.

(c) If the dissemination of the organisms is favoured by phlegmonous inflammation in a wound.

Applying these conditions to an opening into the peritoneal cavity in excision of the rectum we find the conditions not unusually favorable for the occurrence of peritonitis, certainly not so much so as in many operations for strangulated hernia.

In the first place the opening is in the lowest part of the cavity and the external wound is dependent. These circumstances, with attention to position, should render the accumulation in the peritoneum of the discharges from the wound impossible.

2ndly. Only a limited disturbance of the peritoneum takes place.

3rdly. The danger of phlegmonous inflammation should not be great with the use of modern antiseptics and due cleanliness.

In offering these remarks I am far from advocating reckless and unnecessary opening of the peritoneum in excision of the lower bowel. But I submit that the pathological considerations mentioned, and the successful result in this case and in others in which the peritoneum has been wounded in this operation, may encourage us to operate in otherwise favorable cases in which it is doubtful if the disease outsteps the limits laid down by those who have made this operation a special study. In one respect I venture to think the complete intra-peritoneal section of the bowel has an advantage over the ordinary operation; and this consists in the readiness with which the rectum can be brought down. The reflection of peritoneum resists strongly any attempt to draw it down beyond a limited extent; but I found that directly the last shred was divided the upper limit of the disease could be exposed to view without using any traction.

Six months after the operation, and since this account of her case was written, the patient wrote to me, in answer to my inquiries, stating that she continued in perfect health.

HAVING now for many years been familiar with a painful deformity of the foot which appears to have escaped the notice of our surgical writers, I venture to bring before the Society a short account of five cases which have been under my care. The disease consists simply of the flexion of the first phalanx of the great toe through an angle of from $30^\circ$ to $60^\circ$ upon the first metatarsal bone. There is no ankylosis, but the phalanx cannot be extended, and the attempt to execute this movement by external force gives rise to considerable pain. The metatarso-phalangeal joint is usually somewhat enlarged and thickened. There is no deviation of the first phalanx outwards, as in the common affection called hallux valgus. Walking is very painful and difficult, and the patient is obliged to bear his weight as much as possible upon the outer border of the foot. In four of my cases the disease was limited to one foot. The phalangeal joint is healthy, and the ungual phalanx is maintained in a straight line with the first.

There are two causes which I have been able to assign to this condition, viz.: (1) A blow, setting up some inflammation in the metatarso-phalangeal point; and (2) ill-fitting and unyielding boots, especially in individuals in whom the first toe is much longer than, and therefore in advance of, the other toes. In the former class of cases, I suppose that during the rest following an injury the toe becomes flexed, in order to relieve the painful tension of the swollen joint, much in the same way as the knee-joint is affected by a similar condition. By the time that the more acute symptoms have subsided the short fleshy muscles of the sole have contracted, and adhesions have formed, which prevent the restoration of the normal position of the joint. In the latter class it is easy to understand that the constant backward pressure of a short strong boot upon the tip of the great toe must lead to some deviation from its ordinary form. If this deviation be outwards hallux valgus is produced, but if the toe be forced directly backwards, flexion takes place at the metatarso-
phalangeal joint; a projecting angle is formed on the dorsal aspect of the joint, and the pressure of the rigid upper leather upon this projection causes inflammation, which fixes the bones in their new position. When once a pronounced deviation of this kind has been produced the flexor muscles act with greater advantage, and tend to increase the deformity. All my five cases have been in young men. I suppose it affects the male sex because men have more walking to do, are more liable to injury, and wear stiffer boots. I am in some doubt as to why I have met no well-marked cases in older men, but I am inclined to think that in later life the great toe may leave its position of direct flexion and the condition may change to that of hallux valgus. Of this, however, the only evidence which I can adduce is the alteration which had taken place in the third of my cases, when he came to me after two years' interval for further treatment. It is possible, however, though not probable, that it is merely accidental that these five cases have been in young men between the ages of fourteen and twenty-three.

I have seen no reason to attribute the condition to gout, although I have certainly in one instance noticed a much slighter but very painful flexion of the great toe, in which there was fair ground for ascribing this origin.

I have also examined carefully to find whether there was any paralysis or spasmodic contraction of muscles which would account for the deformity. In no case could I detect any feebleness of the extensor proprius hallucis, which would lead to dropping of the first phalanx, nor on the other hand could I ascertain that the abductor, flexor, and adductor hallucis, the function of which muscles is to flex the first phalanx, were hypertrophied, or firmer than usual. Again I have been unable to make out any contraction of the inner-most band of the plantar fascia.

As to treatment, I suppose that in the earlier stages it would be easy to cure the condition by rest and some form of splint, which should maintain the joint in the straight position. In all five of the cases which I have recorded in this paper the deformity was too great for this method of treatment.

In my first three cases I adopted the plan of dividing subcutaneously the tendons and muscular fibres of the short muscles which go to the sesamoid bones and the under surface of the first phalanx of the great toe. In all there was considerable improvement.
After a few months two ceased attending, so I cannot say whether they were permanently cured. A third returned in two years with increased pain and deformity. The phalanx had been flexed 45° and now it was flexed through 60°; and in addition considerable adduction had also come on, so that the great toe was displaced beneath the second toe, in the condition, in fact, of slight hallux valgus. I excised the joint with the sesamoid bones and he made a good recovery. It seemed, however, to me when another severe case came under my care, that a smaller operation and one which would leave intact the important ball of the great toe, would be equally effective. Accordingly in this, my fourth, case I made an incision along the upper aspect of the inner border of the foot, and after exposing the proximal half of the first phalanx with as little injury as possible to the tendons, I nipped it through with a strong bone forceps, and removed it, leaving the head of the metatarsal bone and the sesamoid bones undisturbed. The wound healed by primary union, and twenty-two months later the toe was in good position, and the patient had walked without pain twenty miles in the day. There was but little movement left in the joint, but in other respects it would have been difficult to detect that there had ever been any deformity, or that the joint had been operated on.

Since that time I have had another case of this deformity under my care, in which both great toes were affected, and I performed the same operation upon both joints. Primary union followed, and in three weeks the patient was walking about with the toes quite straight. It is too soon, however, to say in this case whether the relief has been permanent.

Case 1. Hallux flexus; tenotomy; cured.—Frederick R., æt. 23, steward, was admitted on July 23, 1878, under my care, into Luke Ward, Guy's Hospital, suffering from flexion of the metatarso-phalangeal joint of the right great toe, with pain extending thence to the ankle, and considerable difficulty in walking. On February 6 of the same year, while he was at sea, a spar had fallen on the foot and jammed it against a water tank, laying open the inner side of the ankle. He was in a hospital at New York for six weeks, and the wound healed up. After that his right great toe gradually became contracted. On admission, it was flexed through an angle of 45°. On July 29, I divided subcutaneously a portion of the plantar fascia, the flexor brevis hallucis and abductor hallucis muscles just behind the ball of the great toe.
The carbolic spray was used, with carbolic gauze dressings, and a splint was applied to the sole of the foot.

On August 18 the splint was taken off and the patient was discharged cured. He could walk well, with only a slight limp.

**Case 2. Hallux flexus; tenotomy, cure; recurrence, resection of metatarsophalangeal joint.**—James W., æt. 14, shoemaker, was admitted under my care into Luke Ward, Guy’s Hospital, on July 3, 1878. Nine months before a fourteen pound weight had fallen upon his right great toe. It became inflamed but not displaced. He walked about as usual and attended to his work. A week after the accident, however, he noticed that the metatarsophalangeal joint began to project on the dorsal aspect. The deformity has gone on increasing up to the present time, and the pain became so great that he had to give up his work a fortnight ago. On admission, the first phalanx was directly flexed through 45°, and the metatarsophalangeal joint was much thickened.* Passive movement of the joint caused much pain and very distinct grating.

On August 9 he was put under chloroform and I divided subcutaneously the inner border of the plantar fascia, the abductor flexor brevis, and adductor hallucis, about three quarters of an inch behind their insertions. The proper position of the great toe could then be restored.

* It is added also in the report that the first phalanx was partially dislocated backwards. I suspect, however, that this statement is inaccurate.
The operation was performed under the spray, and recovery took place rapidly without any rise of temperature. On the 15th the wounds were healed and the position of the toe normal. On the 24th he went out well.

On June 26, 1880, two years later, he applied for readmission, with a worse condition of deformity (vide Fig. 1). He attributed the return of his trouble to having let a heavy weight fall on the toe about a month after he went out.

The great toe was flexed through 65°; very little movement was possible. There was not much pain except when he had been walking a long time. Besides the flexion there was adduction through 55°, which had placed the great toe slightly under the second toe.

On the 29th I excised the joint and the sesamoid bones under chloroform. By July 16 he was up in a wheelchair, and by the 20th the wound was healed. On the 26th he was walking about, and he left the hospital on August 5 practically well.

Case 3. Hallux flexus; tenotomy; cure.—Thomas II., æt. 16, a carman, came to me as an out-patient at Guy's Hospital on January 7, 1880. Nine months ago he hurt his left great toe, through kicking it against a wall in his efforts to put on a short boot. The toe has been bent ever since. It is now flexed through an ankle of about 40°, and I cannot extend it by using moderate force. He thinks that it is out, but I can find no evidence of dislocation. It interferes with his walking.

He would not come into the hospital, so upon January 13 I operated upon him as an out-patient. I divided the soft parts subcutaneously under ether through two openings, external and internal to the ball of the great toe. As soon as this was done I could easily hyperextend the joint. Carbolic gauze dressings were used, and a short plantar splint applied. No pain followed the operation and on the 23rd the wounds were quite healed. On February 13, I note that the splint is still on but that he is able to walk about and do a little work. On March 5, I saw him for the last time. The toe was then in fair position, and he walked comfortably.

Case 4. Hallux flexus; resection of proximal half of first phalanx; cure.—William J., æt. 21, painter, was admitted under my care into Luke Ward, Guy's Hospital, on February 11, 1885, suffering from flexion of first phalanx of the left great toe through 45° (vide Fig. 2). He had had it for three years,
and could assign no cause. It began with pain in the joint and occasional shooting pain up the leg. At the same time he noticed that the skin over the joint was red. He was not aware that he had worn a tight boot. Walking was difficult and painful. He could use the extensor proprius hallucis well, and no band could be felt on the inner side of the sole. Abductors and adductors were not affected, and he could slightly flex and extend the toe. There was a little prominence of the metatarso-phalangeal joint of the right great toe on the dorsum, but no marked flexion of the phalanx.

On a subsequent occasion I found that the right great toe was so abnormally long that it projected three eighths to half an inch in front of the tip of the second toe.

On February 13 I excised the proximal half of the first phalanx under ether, by an incision one and a half inches long at the junction of the upper and inner surface of the joint. The articular cartilage was a little worn and fibrous, but there was no other evidence of disease in the joint.

By March 5 the wound had healed by primary union. By the 30th he could walk well on the flat foot.

Twenty-two months after the operation he came to show himself, and my late dresser, Mr. Fisher, made a drawing of his foot (vide Fig. 3). The first phalanx was in a line with the metatarsal bone. The joint was stiff but not ankylosed, the range of passive movement being $5^\circ$ to $10^\circ$. The scar of the operation was smooth, and difficult to recognise, and there was nothing abnormal about the appearance of the foot.

The only pain he had in the joint was when he put on his boots. The day before I saw him he had walked twenty miles without any discomfort.
Case 5. Double hallux flexus; resection of proximal half of first phalanx on each side; cure.—Henry C., æt. 20, engineer, was admitted under my care into Luke Ward, Guy's Hospital, on October 25, 1886, suffering from flexion of the first phalanges of both great toes. It came two years ago with no cause, except that he thinks he may have hurt himself by tight boots, but he does not remember any special inconvenience or pain from this cause. He has to walk upon the outer borders of his feet, and suffers a good deal of pain.

The right great toe is flexed through about 30°, and the left through 40°. I can feel no band in the plantar fascia, and the short flexors are not thickened. The extensor proprius hallucis acts well. There is no grating in the slight movements which remain, but some can be felt in the left phalangeal joint. In the left foot there is also abduction through about 10° in the metatarsophalangeal joint, which causes the great toe to be separated by a considerable interval from the other toes. It projects also a quarter of an inch in advance of the second toe. The toenails are remarkably small, of an oval shape, and, so to speak, embedded in the soft parts on the dorsum of the first phalanx. Walking is difficult and painful.

October 26.—I removed under ether the proximal portion of the first phalanx of each great toe, in all rather more than half an inch of the bone. There was very little hæmorrhage, and in less than three weeks the wounds had healed by primary union. He went out soon afterwards, and was then able to walk upon the flat of his feet. During the operation I observed that the cartilage of the joint was thin, and had lost some of its pearly lustre. There was a well-marked transverse furrow upon the head of the metatarsal bone, which corresponded to the usual position of the upper border of the base of the first phalanx; and the articular cartilage of the base of this phalanx had been somewhat encroached upon by fibrous tissue near to its plantar border.

Note (July 9, 1887).—In these five cases, and in three others which I have seen since writing this paper, the deformity has not been accompanied by flat foot. I do not think therefore that it can be attributed to yielding of the arch of the instep.
XXXIII.—Cases of Internal Suppuration, Acute and Chronic, without Fever. By Samuel West, M.D. Read March 25, 1887.

Although the rule is generally true that internal suppuration is associated with fever, the exceptions are sufficiently common to deserve more notice than is often given them. It is of course well known that in the case of serous membranes the temperature is not of itself alone sufficient to determine the diagnosis between serous and purulent effusions; in other words, that the temperature may be as high with serous as with purulent exudation. It is not, however, so generally accepted that the converse is also true, viz. that fever may be absent in both cases alike. The knowledge of this fact is perhaps of the greatest clinical importance. In the following cases suppuration ran its course without the occurrence of any of the symptoms which we are in the habit of associating with the presence of pus.

Case 1.—Martha G., æt. 21, servant, caught cold during menstruation three months before admission; she suffered pain in the lower part of the abdomen, and remained in bed for a week. Although she still suffered pain, she did her work until about three weeks ago. An indefinite history of chills, though not of distinct rigors, was given about one month before admission; she had not lost flesh much, nor did she feel or look ill.

On admission, a large swelling rising out of the pelvis and extending some inches upwards into the abdomen was found. It occupied chiefly the left side. It was irregular in shape on the surface, and along its edge, which was well defined. It was painful on pressure. Per vaginam a hard mass was felt, chiefly on the left side, to which the uterus was firmly fixed.

The patient neither sweated at night, shivered at any time, nor had any rise of temperature, though she was under observation for many weeks; the swelling, however, slowly increased in size, although the tenderness became much less, so that it could be easily handled and examined. The temperature was taken at frequent intervals and at all times of the day and night, and never rose above normal.

As the swelling continued to increase the patient was
Dr. West’s Cases of Internal Suppuration.

examined by Mr. Rose under chloroform, but nothing fresh was discovered, and the treatment was continued as before. Even after this necessarily somewhat rough handling the temperature still never rose.

A slight discharge of pus from the vagina had been noticed soon after admission and continued throughout, but it was never more than sufficient to stain the linen.

By January 9, i.e. two months after admission, the swelling had so far increased as to reach nearly to the umbilicus and to extend nearly to the right iliac spine. While on the left side it occupied the whole iliac fossa.

The patient seemed to be losing ground, but the temperature did not even now rise above 99°.

Shortly afterwards she was transferred to the care of Dr. Hayes, and an exploratory puncture was made. Pus was removed, which was extremely foetid, and a few days later Mr. Rose made an incision in the loin, reflected the peritoneum, without laying its cavity open, reached the pus, evacuated it, and drained the cavity.

Recovery was rapid and complete without any bad symptoms or elevation of temperature.

Case 2.—Alfred N., æt. 16, was admitted into the Chest Hospital, Victoria Park, with purulent pericarditis. He was taken suddenly ill three weeks before, and seemed almost moribund on admission. The pericardium was tapped thrice and finally laid freely open and drained. The patient recovered completely, the temperature throughout only on one occasion reached 100° F., and that shortly before the operation. The case is fully described in the Med.-Chir. Trans. for 1883.

Case 3.—Julia S., æt. 10 (Clin. Soc. Trans., 1885), was seized with sudden abdominal pain on June 8, and admitted on June 12 into the Royal Free Hospital. The diagnosis was made of purulent peritonitis, and on the 15th the abdomen was opened, a great deal of very foetid pus was evacuated, but the child never rallied after the operation and died the same evening. The autopsy proved the case to be one of idiopathic primary purulent peritonitis. In this case again the temperature only once reached 99·8°.

Case 4.—Hannah A., æt. 48, was seized with sudden pain in the epigastrium on May 2, and from that time the pain continued, and she lost flesh and strength, perspired greatly during sleep, and was confined to bed.
On admission she was thought to be suffering from suppuration in the hepatic region, but during the four days that she was under observation the temperature only once rose to \(99^\circ\). On the post-mortem examination an abscess was found between the liver and diaphragm and abdominal walls, due to the rupture of an old duodenal ulcer. \(\text{Clin. Soc. Trans. for 1886.}\)

**Case 5.**—Arthur L., æt. 13, a boy messenger, was admitted into the Chest Hospital, Victoria Park, for shortness of breath and cough. Two months and a half ago he was attacked one night with vomiting and after a few days with pain in the right side. This had continued since. The dyspnœa increased, and finally he was so ill that he came to the hospital.

The whole right side was dull, though not absolutely at the apex. Pus was found, and the side was twice tapped; on the first occasion twenty-four ounces were removed, and ten days later ten ounces more. The pus was thick and sweet; ten days later the side was laid freely open, and about half a pint of pus evacuated.

From this time recovery was rapid, and on the twenty-second day the tube fell out and could not be replaced. The wound closed, and the patient was well. He gained sixteen pounds in thirty-two days, his total weight being only about four stones.

On the day of admission the temperature reached \(100^\circ\) F., but afterwards only once reached \(99.2^\circ\), being at other times normal or a little below.

Different as these cases are from one another, they all present one feature in common, viz. the absence of fever in spite of suppuration. In Cases 1 and 3 this is the more remarkable as the pus was very foetid.

In the first case the accumulation of pus was gradual, and it might be inferred that the absence of febrile temperature was due to its chronicity. This, however, is not the explanation, for Cases 2 and 3 were extremely acute, the duration in both being not many days; nor is the explanation to be found in the rate of formation of pus, the temperature being high where it is formed rapidly and in a large amount, and absent where it is formed slowly; for in Cases 3 and 5 there was a very active and rapid development of pus, and during the whole time fever was absent. In Case 5 it is true fever may have been present before the patient came under
observation, but the analogy of Case 3 makes this possible explanation improbable, and we have in Case 5 to deal with what from other symptoms appeared to be a case of active suppuration, yet with no febrile temperature.

Lastly, the explanation might be sought in the possibility of the patient being in a state of collapse, but this condition was not present in any one of the cases.

I see therefore no explanation of the clinical fact. It must be noted as a fact and borne in mind, for it greatly increases the difficulties of diagnosis in many cases, and may, if not remembered, lead to error.
THE pathology of lymphatic gland tumours is peculiarly ill-defined, and, so far as it has yet been written, is distinguished by a certain confusion. The clinical conception of these growths has been marred by an indefinite morbid anatomy, and the whole subject has been obscured by an exuberant nomenclature. The following terms have been applied to such chronic glandular swellings as are assumed to be non-inflammatory—hypertrophy of glands, lymphoma, lymphadenoma, Hodgkin's disease, lymphosarcoma, and some others. The definitions assigned to these terms by various authors differ considerably. Certain of them may be synonymous on the one hand or they may all express different affections on the other. I do not propose to enter upon this subject in the present paper. I have alluded to it in order to explain why no specific term is applied to the gland swellings under consideration. The terms themselves in the present state of our knowledge can be little other than misleading. With regard to the affections included under the above names there are two facts that may here be noticed. In their clinical features these growths show a sufficient likeness to one another to suggest that they may differ in degree rather than in kind. In the second place, it is especially to be observed that in their histological characters they are closely allied. Indeed those authors who enter into the abstruse question of differential diagnosis make little or no use of histological peculiarities.

The fact to which I wish to draw attention in this paper is the following: certain of these glandular swellings are influenced by arsenic in a remarkable manner, and can indeed be cured by the persistent use of that drug. I am not anxious to assign any name to these tumours, but while the pathological nebula is clearing away, it may be well to bring out as distinctly as possible the clinical features of these growths.

I have selected the following case—out of many—as a typical one.

The patient was a woman, æt. 68, a widow in comfortable circumstances. She was of medium height, was moderately...
Mr. Treves's Case of Glandular Swelling.

stout, and had the aspect of health. She was fair complexioned, and her hair, which was plentiful, was almost white. Her eyes were grey, and there was no arcus senilis. She had lost about half of her teeth; such as remained were in good condition, and she had no need of false teeth.

Her family history was good. She knew of no case of phthisis among her nearer relatives, no instance of cancer or tumour, and no example of glandular swelling. She had always enjoyed excellent health. She had never had any serious illness, and was not the subject of rheumatism in any form. When a child she had never suffered from enlarged glands, or from any affection that could be considered scrofulous.

She had had two children (sons). They were both in the enjoyment of good health, and had never exhibited any strumenous manifestation nor any glandular affection.

In January, 1886, some small glandular swellings were discovered by accident under the patient's right lower jaw. In a little while like swellings appeared in the inframaxillary region on the opposite side of the neck. The patient could not account for these tumours. She was in her usual health at the time, was not exposed to cold, had had no injury, and had experienced no trouble in either the nose, the mouth, or the throat. The tumours were perfectly painless, soft, rounded, and very movable. They gradually increased in size and number, and, spreading downwards, occupied—as early as March, 1886—both sides of the neck from the jaw to the clavicle. They still caused no inconvenience.

The patient now consulted Dr. Wiles, of Mile-end, who subsequently sent the patient to me at the London Hospital. During the months of March and April the woman continued under Dr. Wiles's care. He treated her with iodide of potasium in increasing doses—from five grains to fifteen, and ultimately twenty grains three times a day. To the neck the Unguentum Plumbi Iodidi was applied. The patient's health suffered somewhat under this treatment. She lost flesh, became weaker, and a little anæmic. The tumours, on the other hand, steadily increased. I first saw the patient on May 6th, 1886. She said she was much thinner than she had been six months ago; she was weak, and there was some anæmia. Apart from the swellings in the neck she complained of no trouble. The cervical tumours were considerable and produced great deformity. Both sides of the neck from the jaw to the clavicle were occupied. The outline of the lower
maxilla was obliterated. The swelling consisted of roundish, soft, and elastic tumours, varying in size from a hazel nut to a duck’s egg. They were homogeneous to the touch, of even surface, and very movable. They were of distinct outline, and entirely free from tenderness and adhesion to the skin. Of late there had been some pain in the neck. A number of the gland-tumours were under the sterno-mastoid muscle. A few quite small and movable glands were found high up in the axilla. There were no enlarged glands to be felt elsewhere. The temperature was normal. The regions of the periphery from which the lymphatics of the neck and axilla converge were carefully examined, but no primary disturbance discovered. The patient had not had sore-throat. I imagine that the condition would be met by the terms lymphadenoma or Hodgkin’s disease.

I advised that the iodide of potassium be discontinued, and that arsenic be given. The patient commenced with five minims of liquor arsenicalis three times a day.

She continued the drug for five months, i.e. until the commencement of October, 1886. The dose was gradually increased to fifteen minims three times a day. It often caused distress, and was then discontinued for a while and recommenced in smaller doses. No local application was employed.

I made a careful examination of the patient in December, 1886, two months after all treatment had been discontinued. She was much improved in health, had gained flesh, and was no longer anaemic. To the eye nothing remained of the cervical swellings. On the right side one large gland had suppurated, and on the left side there had been two suppurative foci. These collections of pus had appeared within a month of taking the arsenic. They had discharged spontaneously, and the sinuses that resulted had long since undergone spontaneous cure. Nothing remained but three small, round, red and depressed cicatrices.

On both sides of the neck a few small hard and fixed masses could be felt. In number they did not correspond to more than one third of the original collection of separate gland-swellings. The neck had resumed its normal outline, and on a superficial examination appeared free of any traces of the disease. Nothing could be felt in the axilla.

I think it would be quite safe to infer that the cure was entirely due to the arsenic.

Many monographs have been written upon the use of arsenic in certain malignant or quasi-malignant gland-tumours.
I will allude only to one of the most recent, since it presents a fair account of the present position of this question. The paper is by Dr. Köbel, of Tübingen (‘Über die Arsenbehandlung maligner Tumoren,’ 1886). It has this fault, that it does not clearly depict the clinical aspects of the kind of glandular swelling that is benefited by arsenic. The following is a brief abstract of the contribution: The treatment was applied to all varieties of malignant disease. The arsenic was taken internally for months in increasing doses, and was also employed locally in the form of a subcutaneous injection. The drug was found to be quite useless in cancer and in round-and spindle-celled sarcomata of lymphatic glands. A cure followed the use of arsenic in one case of multiple and rapidly-growing sarcoma, and in many cases of so-called malignant lymphoma. The first-named case is remarkable. The patient was a man aged thirty-six. During a few months no less than four tumours appeared at various parts; one developed in the right axilla, another under the left clavicle (with the result that the bone underwent spontaneous fracture), a third appeared on the acromion, and a fourth about the spine of the seventh cervical vertebra. Arsenic was administered by the mouth and subcutaneous injections were used. The axillary tumour and that under the clavicle suppurated, and with the evacuation of the abscess the growths disappeared. The other two shrunk away without suppuration. The treatment was kept up for two months, and at the end of this time a new swelling was observed over the back. This was excised and submitted to a microscopic examination. It proved to be a spindle-celled sarcoma. In time recurrence took place in the cicatrix. It was now treated with injections of arsenic. It suppurated and necrosed and entirely disappeared. At the time of the writing of the paper three years had elapsed. The patient was well and there had been no sign of a relapse.

In seven cases of malignant lymphoma the use of arsenic was attended with excellent results. The term "malignant lymphoma" would correspond to the more familiar "non-leukaemic lymphadenoma."

Dr. Köbel has collected in addition fifty-nine recorded cases of this affection in which arsenic was employed. Of these fifty-nine cases the arsenic was administered by mouth and by injection in thirty instances. In twenty-four the administration was by mouth only, and in five by injection only. In seventeen cases a cure followed after the treatment had been kept up from one to six months.
In at least five of these patients some recurrence took place at the termination of several months. The growth, however, appears to have again yielded to arsenic. A diminution of the tumour was noted in fourteen cases out of the fifty-nine. In the remaining twenty-eight cases the use of the drug was attended with no benefit. Dr. Köbel's experience is in favour of supplementing the general use of arsenic by local injections.

The illustrative case here given may be considered as a type of the gland-tumour that is benefited by arsenic. The patients are all about or beyond middle age.

I have never found arsenic of value in the treatment of the glandular enlargements of childhood.
DESCRIPTION OF PLATE IV, ILLUSTRATING MESSRS. PARKER AND ROBINSON'S CASE OF CONGENITAL DEFORMITY.

Fig. 1.—The condition of the hands.

Fig. 2.—The condition of the feet. Before operation.

Fig. 3.—The foot. After operation.

ELIZABETH W., aet. 3½ years, was admitted into the East London Hospital for Children on June 29, 1886, with the congenital malformations depicted in Plate IV. She was brought by her mother with a view to some plastic operation on the feet.

The mother, and several brothers and sisters, had inherited a similar deformity, and experienced considerable inconvenience from the feet. The feet supported the body less firmly than normal; when standing, the cleft between the bones tended to open. The toes were powerless and uncontrolled, they were apt to become sore, and suffered from corns. There was difficulty also in getting fitted with comfortable boots.

The following operation was therefore undertaken on the left foot. The toes were removed; the skin in the cleft between the metatarsal bones was dissected away. The parts were then approximated and the contiguous skin-edges carefully adjusted as shown in Plate IV, fig. 3.

After the foot had been well irrigated with corrosive sublimate solution (1-2000) a soft bandage was applied so as to keep the parts in good apposition, and over this a large dressing of corrosive wool. On October 27 an identical operation was performed on the right foot, and a similar dressing applied.

Fifteen days after the first operation the child developed scarlet fever. A few days after the second operation an attack of measles set in. In neither case, however, did the exanthem interfere with primary union.

Remarks.—It was the experience of older members of this family, who had themselves personally felt the inconvenience of deformed feet, which induced the mother to seek surgical help for this child. The peculiar susceptibility which the child manifested to contract the exanthemata, may be briefly referred to. The occurrence of exanthemata after surgical operation is a possible sequela, and must always be kept in mind when oper-
ating on young children who have not yet had them. Scarlet fever is a more frequent sequela than measles, possibly for the reason that fewer children escape measles, that is to say, more children have had measles when they come into a surgical ward and are, therefore, in a measure protected; many children escape scarlet fever, even though exposed to its infection, unless the circumstances be especially favorable to its development, as after surgical operation.

Appended is a family tree, with a carefully worked out history of the deformity through three generations.

The case just detailed of congenital deformity affecting the feet and hands adds a not unimportant item to the cases previously reported by Mr. Fotherby in the British Medical Journal, May 22, 1886, and by Mr. Anderson in the same journal, June 12, 1886.

The deformity first made its appearance in Sarah F.; her parents were healthy and in no way deformed, nor, as far as can be ascertained, had any previous member of the family ever presented any similar malformation. This woman is one of seven children; the rest in no way are deformed, and all have had large families, not one member of which has so far shown any deformity. Sarah F. has deformed hands and feet; her feet she would not permit to be examined, but they are said to have the same appearance as the feet of the child Elizabeth W. The right hand (Fig. 2) shows the following:

**Fig. 1.**

**Fig. 2.**

abnormalities: there are only four metacarpal bones, the one that is absent being apparently the first; the second metacarpal is perfect and ends in a rounded extremity beneath a pad of skin on the radial border of the hand; the third metacarpal
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Roman = Perfect. Italic = Malformed

Compiled by H. B. R.
bone has a dwarfed first phalanx which ends in a pointed extremity; the fourth and fifth fingers are perfect except they are united by a web of skin in their whole length. The left hand (Fig. 1) has also only four metacarpal bones, the first appearing to be the one absent; the second metacarpal bone is perfect and ends in a free extremity beneath a pad of skin; the third and fourth fingers have phalanges common to them, the first phalanx, however, is bifid at its proximal end and articulates with each metacarpal bone; the fifth finger is perfect. On inquiring for any reputed cause, it is said that when Sarah F's mother was pregnant a beggar came to the door and held up his deformed hands. The defect in these hands, if known, is not handed down, nor is it known whether the same man had deformed feet. At what period of the gestation this impression was experienced is not known.

Sarah F. had thirteen children, of which the following were deformed:

(1) Mary Ann W. Hands perfect. The right foot (Fig. 3) presents a deep cleft passing between the second and third metatarsal bones almost to their junction with the tarsus; the first metatarsal with its phalanges, which are tilted outwards, is normal; the second metatarsal is rounded off and projects beneath the skin, reaching down three fourths the length of the first metatarsal bone; the third metatarsal is short and rounded off, and, like the second, has no phalanges attached, projecting beneath the skin on the inner side of the external claw; the fourth and fifth metatarsals with their phalanges, which are tilted inwards, are normal except, that these toes are united by
a web. The left foot (Fig. 4) has the cleft between the first and second metatarsals. The inner claw is formed by the first metatarsal bone and its phalanges, which are not tilted outwards. The second and third metatarsals project on the inner side of the outer claw, the second having no phalanx attached, whilst the third has a small one. The fourth and fifth toes are perfect but they are united by a web.

(2) Emma H. Hands perfect. Feet said to be like Eliz. W.'s.

(3) Susan F. Hands and feet said to have been deformed like Elizabeth W.'s. This girl was twin with James F., who showed no malformation at all. (Both dead.)

(5) Annie F. Hands and feet said to be deformed in the same way. (Dead.)

(6) Thomas F. (i). Hands and feet deformed. (Dead.)

(9) James F. Feet deformed. Not married.

(10) George F. Both hands and feet deformed. The right hand shows the following abnormalities:—the first metacarpal bone is about half the ordinary length, rounded off at the end and having no phalanges attached; the second metacarpal is present and has a dwarfed first phalanx; the third and fourth fingers have the bones perfect, but they are closely united by a web and have a nail in common, which, however, shows sign of its dual formation; the fifth finger is normal. The left hand (Fig. 5) is deformed as follows:—the first metacarpal bone is perfect but its first phalanx is tilted directly outwards and the terminal phalanx is placed at right angles to this; the second finger is like the same finger of the right hand; the third and fourth fingers are united by a web and tilted a little to the ulnar side; in the third finger, however, only the first phalanx is
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present; the fifth finger is normal. Both feet (Fig. 6) are alike. The great and little toes have their metatarsal bones and phalanges normal except that they are displaced outwards and inwards respectively. The second, third, and fourth metatarsal bones are not present. This man is unmarried.

(12) Samuel F. Hands and feet said to be deformed like the child Elizabeth W.

(13) Henry F. Hands and feet deformed. (Dead.) The third generation consists of the families of Mary Ann W., Ellen H., and William F.

Mary Ann W. has had ten children, those mentioned below being deformed.

(2) Alice W. Right hand much smaller than the left; muscular development very defective, which is explained by the girl being left handed. The tubercle on the outer side of the first metacarpal bone is very prominent; nail on its terminal phalanx very imperfect. The second phalanx of left index finger is displaced to the ulnar side. The right foot (Fig. 7) shows these defects: The first metatarsal and its phalanges are normal except for the displacement of the terminal phalanx outwards. No second and third metatarsals to be felt. The fourth and fifth metatarsals are normal, but their first phalanges, although they have separate articulations for the metatarsal bones, are fused and bear only single second and third phalanges which are tilted inwards to touch the great toe. The left foot only differs from the right in that here the third metatarsal bone is present and its rounded end may be felt beneath the skin on the outer claw.

(4) Harry W. The hands are perfect. The bones of both feet are normal. In the right foot the great toe and second toe are united by a lax web; the bones of the third and fourth toes, although distinct, are kept in close union by another web. In the left foot the third and fourth toes are united.

(6) Boy (dead). Feet deformed like Elizabeth W.'s.

(7) Elizabeth W. Both hands alike (Fig. 8). The metacarpal bones are perfect and also the phalanges of the thumb and little fingers. The index fingers have only the first phalanx. The middle and ring fingers have first phalanges distinct at their metatarsal end, but fused beyond; the second and third phalanges are common to the two fingers. The right foot is deformed as follows:—The great toe has its metatarsal bone and its phalanges normal, the latter being displaced inwards. The cleft is situated in the position of the second metatarsal bone, which is not to be felt. The third metatarsal bone
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may be felt with a rounded end beneath the skin on the inner side of the outer claw. The first phalanges of the fourth and little toes are separate at their metatarsal ends but fused below and have only second and third phalanges common to the two, toes, which are bent inwards, forming a hook meeting the outwardly pointing great toe. The left foot (Fig. 9) is the same as the right except that the third metatarsal bone bears a small first phalanx.

(9) Boy (dead). Hands perfect. Feet like Elizabeth W.’s. Emma H. has had ten children, two of which have been deformed.

(4) George H. Feet perfect except that both feet have a web between the great and second toes.

(7) Arthur H. One hand perfect and the other and both feet deformed like Elizabeth W.’s.

William F., himself perfect, has had four children, two boys and two girls, all perfect.

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The following points seem to us worthy of notice:

1. Out of 37 descendants of Sarah F. 16 are malformed.

2. Unlike the two cases previously reported a female was the first to show the imperfection, and the defect is, thus far, being transmitted through females.

3. In these cases it is the hands that first regain the perfect form. Out of 16 descendants malformed 10 have both hands and feet defective, 6 the feet alone, but not a single instance occurs in which the hands are defective whilst the feet are perfect.

4. Although a limb has regained its proper form in one
Granting potentialities person. certain the tendency to feasible occurrence the mission "to transmission environment ance asserted stances for happen fright opinion embryo, of children. there are imperfect, whilst in the third generation 7 only out of 23 children.

(6) As in Mr. Anderson's, and unlike Mr. Fotherby's case, there are no associated mental or physical defects.

(7) The deformities now recorded show far greater symmetry than in the two other collections of cases referred to.

(8) As in Mr. Anderson's case, the defect is attributed to a fright, but it is not known at what period of gestation this fright was experienced. Whether, supposing such an event happen during the early months of pregnancy, mental shock has any influence over the subsequent development of the embryo, is a much-disputed and doubtful point. Beyond the asserted fright there is complete absence of cause to account for the first deformity.

From the symmetry and extent of the lesion intrauterine causes, such as amniotic bands, would not in our opinion suffice to explain them. Under these circumstances we cannot attempt an explanation of the first appearance of this deformity. What is the cause, however, of the transmission of the deformity? Granting even that uterine environment might account for its first appearance, it seems to us that something further is needed to explain its transmission to 16 out of 37 descendants. We do not think that "mental impression" suffices, although in such a case as this, the constant presence of the deformity in the house and its occurrence in successive members of the family are calculated to exercise a powerful impression on the pregnant mother and that too from the very earliest period of pregnancy. A more feasible explanation, in our opinion, is "hereditary influence." When the deformity has once appeared, there is an inherited tendency for it to reappear in the descendants of the malformed person. We venture to think that the individual cells of which the complete organism is formed are invested with certain potentialities just as is the complete organism itself. As certain intellectual or bodily characteristics are transmissible
only to some members of a family, so is this deformity; and just as certain peculiarities, after having for a time become lost, tend to crop up again among the descendants, so, in the transmission of this deformity, we notice reversions back to types present in a former generation after the deformity has apparently been lost in the generations actually under observation.
XXXVI.—A Case of Psoriasis, with associated Rheumatism, which passed into general Pityriasis rubra (Dermatitis exfoliativa). By Sir Dyce Duckworth, M.D. Read April 1st, 1887.

F. A., set. 24, married eighteen months, was admitted into St. Bartholomew's Hospital under my care on November 14, 1885. She has a healthy child aged eight months, suckled up to four months ago.

She has lived chiefly in London, and latterly at Stratford. She occupies the ground floor of a damp house. When a child she had rheumatic fever. Ten years ago, and five years ago, she had attacks of psoriasis, each time accompanied by painful joints. There is no family history of rheumatism to be obtained. She had been ailing for five months before admission, having first noticed small spots on the arms, around the elbows, with pain in the joints. The eruption gradually increased. She had advice at the London and afterwards at St. Mary's Hospital. Mr. Malcolm Morris commended her to my care as an in-patient. The whole of the body was covered irregularly with patches of unequivocal psoriasis, erythematous areas extending beyond the limits of scaliness; on the face, neck, and mammae there were only small patches. Over the abdomen the erythema was almost continuous. In the groins were large scales. The umbilicus was hard like a nut. Dense scaly patches over the patellae and olecrana with tendency to crack. The flexor aspects of the fingers were thick, and showed similar tendency. Palms of hands thick and horny, nails rough and indented like a thimble both on hands and feet. The whole surface of the body was dry and tender, burning and pricking sensations being experienced. The ears were free, except on the concha of left ear, where there were branny scales. The complexion was pallid; hair light brown. Tongue somewhat dry and thickly coated; buccal membrane unaffected. Some enlarged glands at the angle of the right jaw, and behind the right sterno-mastoid muscle. All the large joints, except the shoulders, were tender and slightly swollen.

The heart's impulse was quiet, apex displaced to left nipple line in fifth space. Booming first sound with systolic
murmur at apex, fading towards base; second sound clear, never had any palpitation. Pulse 120, full, rather incompressible; lungs natural; temperature elevated, varying from 100° to 102·6°, with nightly rises, for two weeks after admission, becoming higher subsequently, as will be described presently. The catamenia had been regular since the birth of her child. There was occasionally found a trace of albumen in the urine, which was acid, often containing abundant urates, but no glucose, and averaging in specific gravity 1015 to 1025.

The treatment ordered was as follows: An alkaline bath every second night, followed by inunction with olive oil all over the body. Milk diet, no beef-tea. Internally, four minims of Fowler's solution with liquor potassii, tincture of cardamoms and peppermint water three times daily.

In a few days there was slight general improvement. The temperature, however, kept up. The tenderness in the skin and joint-pains prevented good rest at nights. She felt hungry and asked for meat; fish and potatoes were then allowed. The scales began to fall off freely into the bed. Erythema very red and spreading up the neck. She was kept in blankets because of the oily inunction.

On November 19 right wrist and knee painful; effusion into the latter.

On November 20 she was put on sodium salicylate gr. xv, 6tis horis. The head was covered with a lotion consisting of one ounce of soft soap dissolved in two ounces of rectified spirit. Linimentum calcis was applied to the limbs. Twenty minims of paraldehyde were given at night to secure rest. Confection of senna ordered as the bowels were rather confined. During the next week the temperature kept up, being unaffected by the salicylate, and the joints remained painful, the ankles being oedematous. The salicylate was increased to eighty grains in the day, and milk diet again enjoined. Size baths were substituted for the alkaline ones.

On November 29 the temperature rose to 103·6° at night. It had not been below 100° for a fortnight. No improvement in the state of the joints, but, on the contrary, the left wrist and knee were worse, also the hands and wrists were affected. Vaseline was found of service in relieving the dry and scaly state of the face. Notwithstanding her condition the patient complained of being hungry, and begged for meat. There was also thirst. The tongue was rather dry and furred in patches.

November 30.—An ointment of oleate of zinc, one drachm
Sir Dyce Duckworth’s Case of Psoriasis.

to the ounce of benzoated lard, was now ordered instead of the former applications, and the quino-alkaline draught, containing two grains of quinine and twenty of bicarbonate of potassium, 6tis horis.

December 3.—Some improvement generally in joints; can straighten right knee better. Skin softer and less red, but desquamating freely. Allowed some mashed potatoes and butter, also custard pudding. Prefers blankets to sheets.

On December 5 temperature fell to 99°, having been 103° on previous evening; pulse 100. Urine still contains a cloud of albumen. Joints certainly better. Soft systolic murmurs heard at apex and base of the heart.

December 11.—Joints improving; temperature falling; no albumen in urine; linimentum calcis employed for skin, and a change made in the treatment. Five minims of liq. sodii arseniatis out of the soda and calumba draught thrice daily, and full diet, with a mutton chop and greens, and two bottles of soda water. In three days’ time was more comfortable. Temperature still 100° to 101°. Large epidermic scales separating, leaving healthy-looking skin beneath; joints less tender. Fifteen grains of antipyrin were now given, and repeated in two hours, with a view of reducing the temperature. Forty-five grains in all were given, but without effect on the body heat. Tongue clean and moist, appetite good. Four ounces of port wine ordered, and a varied meat diet.

December 18.—Arseniate of sodium increased to seven minims at each dose, and in three days’ time to eight minims.

December 23.—Gets out of bed, and sits up each evening.

December 28.—Improving gradually; temperature over 99° each night. Pulse still frequent, 112, and of increased tension. Only slight desquamation, but much irritability of skin. A paste of starch, oxide of zinc, glycerine and lime-water ordered.

December 31.—Joints stiff and thickened. Sterno-clavicular joints loose, the clavicle being unduly prominent at the sternal end on right side.

January 7, 1886.—Temperature slowly becoming normal. Wrists strapped with soap plaster. Still very weak, but generally making progress. Skin assuming soft and natural condition.

January 29.—Skin quite recovered; conjunctivæ irritable owing to arsenic; rapidly gaining flesh; pulse 80.

February 12.—But little improvement in joints of fingers and wrists; the former are becoming everted to ulnar side;
heart-murmur at apex remains; urine natural. Returned home.

This case illustrated a transformation of a well-defined and well-recognised form of skin affection into another equally well-differentiated. This is not the first case in which psoriasis has been observed to pass into general exfoliative dermatitis. Pye-Smith and other authorities have recognised this occurrence, as also the origin of the latter from eczema, impetigo, and traumatic dermatitis. It is not, however, common to meet with such transformation, and the disease, as a rule, begins without any other previous cutaneous lesion. The association with rheumatism was very distinct and noteworthy in this instance. I use the term rheumatism in a somewhat wide sense, however. I know of no better term to use for the disorder met with in this patient. There was plain history of a previous attack when a child, and there was a mitral lesion present on admission. The history of two previous attacks of psoriasis, each accompanied with rheumatism, is very worthy of note in the case. From this point of view it was, however, very remarkable to find that the rheumatic symptoms and the pyrexia accompanying the whole illness were both quite unrelieved and unaffected by treatment with salicylate of sodium, pushed freely. Neither did appropriate dietetic treatment prove of the usual value in promoting recovery from the rheumatism or the fever. Hence, some would affirm that true rheumatism was not really a factor in the case. At any rate, there were fever, severe articular pains, swelling, subsequent stiffness and thickening, and ultimately enlargements and deformities of the affected joints; also the before-mentioned cardiac affection. It would be hard to find a more complete grouping of symptoms wherewith to form a diagnosis of rheumatism. Again, the skin affections in this case are such as attach to rheumatic proclivity. It was noteworthy that the patient was often hungry, and instinctively craved for meat and more sustaining diet than I felt justified for a long time in permitting her to have. When, yielding to her instincts, and recasting the whole treatment of the case, prescribing arsenic in gradually increasing doses, and giving good diet and wine, rapid and decided improvement took place, ending in perfect recovery of all but the joint-affections, new light was thrown upon the case. What did good to the integumentary trouble certainly did good to the rheumatic element which was present. But for the rheumatic affection, I should have sooner resorted to the plan of treatment which

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ultimately succeeded. I have not found difficulty before in the treatment of simple pityriasis rubra. Baths, inunction, and alkaline tonic treatment have commonly sufficed, and I have never yet met with the intractable cases recorded by German authorities. I believe supporting treatment is generally called for, and arsenic has virtues in obstinate cases little, if at all, inferior to those it possesses against ordinary psoriasis and pemphigus. The rheumatic, or rheumatoid, element in such cases has to be studied along with the same symptoms which are met with, and which I and others have already described, in cases of diffuse scleroderma, and to which I hope some day to again direct attention.
DESCRIPTION OF PLATE V.

Figs. 1, 2, 3 and 4 illustrate Mr. William Anderson's paper on Hammer-toe. (From drawings by Mr. Anderson.)

**Fig. 1.**—Longitudinal section of hammer-toe.
   a. Angular prominence over head of first phalanx. A subcutaneous bursa is usually found at this point.
   b. Head of first phalanx. The articular cartilage, above the point of contact with the middle phalanx, is usually more or less atrophied.
   c. Glenoid ligament.

**Fig. 2.**—Dissection of first interphalangeal articulation in hammer-toe.
   a. Extensor tendon.
   b. Lateral ligament.
   c. The contracted portion of the lateral ligament blending with the glenoid plate.

**Fig. 3.**—The same preparation. The glenoid plate and inferior fibres of the lateral ligaments divided to allow the extension of the joint.

**Fig. 4.**—The first interphalangeal articulation of a normal toe, exposed from the dorsal surface by the removal of the extensor tendon and head of the first phalanx.
   a. Shaft of first phalanx.
   b. Lateral ligament.
   c. Base of second phalanx.
   d. Scattered capsular fibres above glenoid plate loosely attached to periosteum of first phalanx.
   e. Glenoid plate strongly attached to plantar margin of base of middle phalanx.

**Fig. 5.**—Illustrates Mr. Bellamy's case of Symmetrical Gangrene following Varicella. (From a drawing by Mr. John Morgan.)
XXXVII.—A Case of Symmetrical Gangrene following Varicella in a child four years old, causing death on the fifth day. By Edward Bellamy. Read April 1, 1887.

Elizabeth P., æt. 4, was admitted into the Charing Cross Hospital under my care, on January 19, 1887, with the following history. On Sunday, January 16, the parents of the patient noticed spots over her body and face (red spots and very painful). The patient was allowed to run about, and was not attended by any medical man. The only treatment consisted of a powder administered by her mother. On Wednesday, January 19, at 7 p.m., the patient complained of her right leg being sore, and a small, circular, black-looking patch about the size of half a crown was observed below the inner side of the knee. By ten o'clock this patch had extended down the leg to the foot and the child was brought at once to the hospital. On arrival a similar patch had made its appearance just above the left ankle, which rapidly extended up to the knee. The patient was unable to stand, and complained of great pain in both legs, not increased on pressure. The spots still remaining on the patient's trunk were identified by my house surgeon as those of varicella.

Present condition.—The patient has a bluish-black lividity of both legs extending from below knee to the foot.

Right leg.—Anterior aspect: The lividity extends to within a quarter of an inch of the lower border of the patella. Internal: Lividity extends above knee for 3 inches. Posterior: To within 2 inches of flexure of knee-joint. External: As high up as on the posterior aspect.

On the outer aspect of the right thigh, about 4 inches above highest point of the patella, there is an oblong patch (similar in colour to legs), 3 inches long by half an inch wide. The second, third, and fourth toes are quite exempt from discolouration, and the fifth toe is but slightly discoloured.

There is a bleb on dorsum of foot, three quarters of an inch from web of third and fourth toes. Also an excoriation of dorsum of foot 3 inches in length, to which cotton wool has adhered.

Left leg.—Anterior aspect: Lividity extends up to lower
border of patella. Internal: Beyond knee for 3 inches above upper border of patella, and then winds round to back of thigh. Posterior: To flexure of knee-joint. External: To level of flexure of knee-joint.

On outside of left thigh, 2 inches from the upper border of the patella, there is an oblong patch 1½ inches long and 2 inches wide. There is a bleb about the size of a shilling on dorsum of foot half an inch from web of second and third toes. An excoriation on dorsum of foot an inch in length.

Right arm.—Two inches below olecranon on outer aspect of forearm is a small patch, first observed on Friday morning. Slight discolouration on either side of vertebral column on a level with crest of the ilium. In the middle of left cheek discoloration began to appear at 12 o’clock on Friday morning, and at 4 o’clock Friday afternoon a similar patch appeared on right cheek, and also in concha of left ear.

Complexion very white, face sallow and expression dull and heavy. Tongue dry and brown, but reddish at tip. Pulse 150, weak. Heart-sounds normal, breathing normal. Temp. 100°. Both legs are cold to touch and irressistive. Pulsation in left femoral artery can be indistinctly felt, but not at all in right. Urine 1023, alkaline, clear, pale, no trace of albumen, nor could hemoglobin be detected. Patient complained frequently of thirst, and was given warm milk.

On January 20, patient was very depressed and continually dozing, but on the 21st was livelier and brighter, especially after a visit from her mother, whom she recognised and spoke to. Patient when left alone will undress her legs and attempt to pull off cotton wool in places where it has stuck.

Before death patches on both cheeks increased with a slight bleeding over bridge of nose. Also discolouration appeared on buttocks and increased on back; another patch also appeared on right forearm and one on left chest, an inch external to nipple.

At 8 p.m., January 21, patient became suddenly delirious, breathing was stertorous at 12 o’clock, and at 1.45 a.m. she died.

Family history good. Father a potman, in good health, has lately suffered considerably from epistaxis. Mother healthy, no miscarriages or stillborn children, one brother older and one sister younger, both healthy, who had just recovered from varicella when patient was attacked.

Post-mortem examination made January 22, by Mr. Stanley Boyd. Body: Well-nourished, showing patches of gangrene
and petechiae (noted during life). Rigor mortis strongly marked, surface still warm. Superficial view of abdomen normal. No fluid in peritoneum. Thorax: Superficial view normal; three or four oz. of blood-stained fluid in either pleura. Pericardium: One or two drachms straw-coloured fluid. On outer side of right auricle one or two large patches bounded by sanious haemorrhagic margins. Heart: The right heart is flaccid and contains only a little fluid blood. Left ventricle strongly contracted. Valve on right side normal. There is an enormous opening in the foramen ovale, admitting freely the tip of the thumb; it is bounded below by a valve a quarter of an inch deep, having a free crescentic margin. The aortic valves are normal. On the anterior cusp of the mitral valve, just above the attachment of the chordae tendineae of the largest papillary muscle, there is an oval, reddish-brown, slightly raised patch, about the size of a split pea, due to interstitial thickening in the valve. There are no vegetations or any other signs of endocarditis. The cavities of the heart appear to be normal, and the heart-substance healthy.

Right lung: Surface normal; the upper part of the posterior border of the lower lobe is much congested, probably from post-mortem gravitation only; the rest of the lung appears quite normal. Some thick mucopustus occupies the larger bronchi; there is but slight injection of mucosa. Left lung: Apex only in part congested, and there seems to have been some haemorrhage beneath the pleura. Decomposition is beginning here as shown by small air-vesicles beneath the pleura. In a great fissure close to root of lung there is small subpleural haemorrhage; there are no petechiae; the apex of lung seems much inflated, being rounder and larger than it should be in proportion to rest of viscus. It is crepitant throughout, and presents upon section only the appearance of moderate hyperaemia, limited somewhat sharply by the outlines of lobules about 1·5 inch below the surface; otherwise the appearance is natural and the consistence is not diminished; the lower lobe is hyperaemic posteriorly.

Liver: Surface normal on section. Spleen: Size normal, firm; upon outer surface, near lower end posteriorly, is a dark patch about the size of the tip of the finger, looking as if a little extravasation beneath the capsule had taken place, and upon section it would appear as if this were the case. The haemorrhage was very slight.

Right kidney: Surface and size normal; capsule strips
normally, and showing one or two patches of superficial congestion. Left kidney normal. Suprarenals normal. Dissection of right leg: The fat and skin and gangrenous parts are dark purple, from hæmorrhagic infiltration, and this appearance dies away about apex of Scarpa's space. The obvious gangrene ceases just above the inner condyle of the femur, the skin above this being apparently normal; the leg, which appears quite gangrenous, being uniformly purple. There are only some petechiae or small hæmorrhages in the intermuscular areolar planes, the muscles themselves being very strongly contracted in rigor mortis, and appearing almost healthy; just here and there they show small hæmorrhages. This is especially the case in the deeper fibres of the soleus. The femoral glands are a good deal enlarged, and either hæmorrhagic or containing a quantity of blood-pigment. The saphena vein contains only post-mortem clot. The femoral vessels examined throughout are found perfectly healthy and empty.

Brain surface normal.
XXXVIII.—Intussusception produced by the presence of
a Tumour growing from the Mucous Membrane of the
Great Gut. By W. Arbuthnot Lane, M.S. Read
April 1, 1887.

A. B., æt. 8, was a feeble, wasted lad who had been under
my care for some time suffering from disease of the
right hip-joint.

One morning after the child had passed a very fluid motion
the nurse noticed a red mass about as big as a walnut bulg-
ing from the anus. She, supposing it to be a prolapse of the
mucous membrane of the rectum, made several attempts to
return it within the anal aperture but without success.

I saw the child some hours after the occurrence and found
a very congested mass, about an inch or more in diameter,
projecting from the anus. Blood oozed rapidly from the con-
gested surface of the tumour.

On introducing the finger into the rectum, I felt a very
narrow pedicle about an inch and a half long, extending
upwards from the tumour. This narrow pedicle gradually
increased in thickness till it became apparent that it was
intussuscepted gut.

Though I could reach beyond the brim of the true pelvis
with the tip of my finger I was unable to feel the point of
intussusception. On pulling gently on the tumour it was
possible to expose some of the tense pedicle, which was then
seen to be very pale.

As the child had no symptoms of strangulation, and also
because I felt doubtful as to how much was pedicle and how
much intussuscepted gut, and being curious to determine the
seat of the tumour, I replaced it within the anus. It was at
once drawn up to the level of the brim by the traction exerted
upon it by the intussuscepted gut.

On again examining the child next day, Dr. Lees and I
were able to feel the tumour through the wall of the rectum,
and it was readily fixed between the finger in the rectum
and the hand placed on the surface of the abdomen. It was
situated in the middle line about 2 inches above the sym-
physis. It could be moved readily in a vertical plane but less
readily from side to side. It appeared as if it were in connection with the transverse colon about its centre.

Five days afterwards, after an attack of diarrhoea, the tumour reappeared at the anus. This time it was even more congested than on the previous occasion, and bloody mucus oozed freely from its surface when it was squeezed. The intussuscepted gut was also much more congested than it was on the previous occasion, and instead of being tense its mucous surface bulged into the anal aperture. It was now seen that the tumour was attached to the mucous membrane by a very constricted neck and that it had no true pedicle as it appeared to have on its first appearance.

I removed the tumour and returned the gut up to the level of the pelvic brim.

On the following day there was no sign of the presence of any intussuscepted gut, and since that time there has been no recurrence of the intussusception. No tumour could be felt in the abdomen on either occasion on which the growth protruded from the anus.

The tumour resembled in appearance and in microscopical structure the ordinary rectal polypus of young life.
XXXIX.—Three Cases illustrating the Difficulties in establishing Natural Respiration after Tracheotomy and their Treatment by Tracheal Catheterism. By Bilton Pollard. Read April 22, 1887.

Two of the patients who form the subject of this communication were for a long time under the care of Mr. Godlee and passed into my hands when I succeeded him at the North Eastern Hospital for Children. Mr. Godlee suggested to me that the cases should be published and has looked over the notes of the earlier part of the cases which had been under his care.

The difficulty experienced in establishing natural respiration after tracheotomy is by no means an uncommon one, and in two of the cases now recorded the difficulty still remained after most determined and persevering attempts had been made for more than a year and a half by the usual methods.

The plan which at last proved successful was a very simple one. It is hoped that in similar cases a like result may follow its employment.

My first case was a boy, aged 2½, on whom tracheotomy had been performed by the house surgeon at the North Eastern Hospital, for laryngitis, on November 17, 1884. A fortnight later the child was convalescent, but the tube could not be removed on account of the great dyspnœa with retraction of the chest which at once came on. During the following eight months frequent attempts were made to dispense with the tube, but it was impossible to leave it out for more than a few minutes. Mr. Godlee then catheterised the larynx, but no improvement followed. About four months later the tracheal portion of a vulcanite tracheotomy tube broke off and slipped down the trachea. It was removed from the right bronchus two days later by Mr. Godlee by means of a pair of curved forceps.

On January 2, 1886 (thirteen and a half months after the tracheotomy), Mr. Godlee passed a piece of india-rubber drainage-tube, about the size of No. 8 English catheter, through the larynx from the tracheotomy wound and brought it out at the mouth. The two ends of the tube were tied together and a metal tube was inserted in the trachea. The
piece of drainage-tube was removed from the larynx after forty-eight hours, but the patient's condition remained unchanged. This operation was repeated four months later but without success. The frequent application of nitrate of silver to the trachea above and below the wound was next tried, but still the removal of the tube at once induced urgent dyspnœa and caused the child to plunge about its bed in great distress. Repeated attempts to get rid of the tube still failed, but it was noticed that the inhalation of chloroform relieved the dyspnœa. On July 3, 1886 (over a year and seven months after the tracheotomy was done), I passed a Macewen's tracheal catheter from the mouth through the glottis and into the trachea beyond the tracheotomy opening. The tube was tied in position and the child breathed comfortably through it for thirty-one hours. The tube then became blocked and had to be suddenly removed. The tracheal opening, which had nearly closed, was covered up and the patient continued to breathe comfortably through his mouth. A fortnight later the tracheotomy wound was soundly healed and the child began to play about the ward. He never suffered from dyspnœa after the removal of the Macewen's tube; but though he soon spoke plainly in a loud whisper, it was not till three months after the last operation that he recovered the power of phonation. The boy is now, after an interval of six months, perfectly well.

My second case was a boy, æt. 6, who was admitted to University College Hospital in a state of semi-asphyxia owing to laryngitis. The house surgeon operated at once without an anaesthetic, and in his hurry to open the windpipe he divided the cricoid cartilage. The patient soon recovered from the laryngitis but it was found impossible to get rid of the tube, so the patient was transferred to the surgical wards under the care of Mr. Godlee, who allowed me to treat the case. In addition to laryngeal spasm there appeared to be some obstruction in the trachea, for dyspnœa came on when the tube was removed even though the tracheal opening was kept dilated. Three months after the tracheotomy I attempted to pass the Macewen's tube, but it was only after many attempts and after dilating the rima glottidis by the passage of a series of steel bougies that I succeeded in doing so. As the tube passed down the trachea it detached a piece of granulation tissue which was coughed up through the tube. After the tube had been in position for thirty hours it was removed owing to an attack of pneumonia in the left lung, induced, I believe, by the Macewen's tube having slipped down into the right bronchus.
A pad was placed over the tracheotomy wound and the boy breathed through his mouth for fifty-four hours, but by that time urgent dyspnœa had come on and it was necessary to replace the tracheotomy tube. This was worn for ten days, and then, the boy being convalescent from his attack of pneumonia, a very short india-rubber tube, which was just long enough to enter the trachea, was substituted for it. Two days later it was found that the boy was breathing almost entirely through his mouth, so the tracheotomy tube was removed. Three days afterwards the tracheotomy wound was closed, respiration was natural and the power of phonation was good. The patient was then discharged. The boy is now, after an interval of three months, perfectly well.

My third case was a boy, 3\textdegree 4, who was brought to the North Eastern Hospital on July 1, 1884. He was nearly asphyxiated owing to the impaction of a foreign body, which the house surgeon dislodged whilst exploring the pharynx. On the following day the child had paroxysmal dyspnœa and signs of blocking of the right bronchus. Tracheotomy was performed by the house surgeon, but in this case also the cricoid cartilage was divided. Nineteen days later a large cherry-stone was coughed out of the tracheotomy wound, but it was found impossible to dispense with the tube as the child became rapidly asphyxiated on its removal. The usual methods were had recourse to during the following seven months but without success. Mr. Godlee then enlarged the tracheal wound downwards and removed some granulation tissue from the trachea. He also passed a series of French bougies upwards through the larynx. Seven weeks later Mr. Godlee removed more granulation tissue from the trachea, and the larynx was frequently catheterised after this date, but the passage still remained very small. The patient was discharged on May 14, 1885 (ten and a half months after admission). He was then taken to the Golden Square Throat Hospital, where he remained for ten months, and wore a variety of tubes, but was never without one. The patient was readmitted to the North Eastern Hospital on September 29, 1886 (two years and three months after the tracheotomy). He was wearing a large-sized silver tube with a laryngeal opening, but his voice was only just audible. On October 19 I found that the upper end of the wound was at the level of the vocal cords and that the latter were adherent to one another in front. I separated them by an incision with a curved bistoury and passed a silver catheter through the larynx. The
catheterism was repeated two days later, and two days after
that I dilated the larynx up to No. 13 of the English scale
with Lister's steel bougies and then passed the Macewen's
tracheal catheter. The catheter was kept in position for
thirty-eight hours, but after its removal the boy only breathed
comfortably through his mouth for an hour. A very short
india-rubber tracheotomy tube was inserted. This was
repeatedly removed for longer and longer periods, and seven
days after the operation it was removed at 10 A.M. and left
out till 10 A.M. on the following day. A fortnight later I
again dilated the larynx up to No. 15 Eng. with steel bougies.
The tracheotomy tube was not replaced. Five days later the
tracheotomy wound was closed, and in five days more the boy
could phonate well with an effort, but his control over his
laryngeal muscles was not perfect and sometimes the last word of
a sentence was uttered in a very shrill tone. So far the result
in this case was very pleasing, but after the tracheal wound
had been healed for about three weeks some retraction of the
chest was noticed, and during sleep this symptom became
exaggerated. Dr. Semon kindly examined the larynx for me
and reported that there was a growth of granulation tissue on
the right vocal cord and that the cords were adherent in front.
A week later the boy's condition was worse and the tracheal
opening was re-established.

The difference between the plan I followed and that adopted
by Mr. Godlee was, that by keeping the tube in the trachea
whilst the tracheotomy wound was allowed to close, the
patients were left, on its removal, without the opportunity of
making use of the passage which, from constant employment,
had become for them the natural one. In the first case I
thought the obstruction was due chiefly to spasm, because the
child could breathe through its mouth under the influence of
chloroform. I also thought that the anxiety which the child
manifested when the tracheotomy tube was removed might
be a powerful factor in inducing the spasm, and that if the boy
could be inspired with confidence in his powers of breathing
whilst the tracheal opening was closed the chief impediment
to cure would be removed. In the second case there was
a good deal of laryngeal spasm but there was also a growth of
granulation tissue in the trachea, and it seems probable that
the steady pressure of the tube caused absorption of this. In
the third case there was a genuine stricture of the larynx
owing to adhesion of the vocal cords. After their separation
by the knife the Macewen's tube served as a means of continu-
ous dilatation, but though it rendered laryngeal respiration and the closure of the tracheotomy wound possible, it failed to effect a cure owing to the vocal cords growing together again. The dilatation should in this case have been continued for a longer period before the tracheotomy wound was allowed to close.

The rapid cure in the first case was, I think, due largely to the previous mechanical treatment to which the larynx had been subjected. It was more callous and so the Macewen's tube caused less irritation and swelling. In the other two cases it probably induced some laryngitis, and it was only after the subsidence of this that the full effect of the treatment was obtained. I believe the adhesion of the vocal cords in the third case resulted either from some original injury of the glottis owing to the high position of the tracheotomy wound or from the irritation of the tracheotomy tube, the convex surface of which rested against their lower surface.

The insertion of the tracheal catheter was attended with much difficulty. In two of the cases I succeeded by first passing a long silver probe into the mouth from the tracheotomy wound and then passing the catheter downwards over the probe. In the other case this method failed, but I succeeded by passing the catheter upwards from the tracheotomy wound until the lower end could be placed in the trachea and then pushing it down again until the end was well beyond the tracheotomy wound.

I found small brushes such as are employed for cleaning pipes very useful for keeping the catheter free from mucus.
XL.—A Case of large Omental Lipoma successfully removed by Abdominal Section. By W. A. Meredith. Read April 22, 1887.

Intra-abdominal fatty growths of sufficient dimensions to call for surgical interference are of very uncommon occurrence to judge from the few cases which appear on record. Of those reported, the majority are described as lipomatous or myxo-lipomatous retro-peritoneal tumours, either originating in the peri-renal tissues or else developed between the layers of the mesentery.

The only instance which I have been able to find where the growth is described as omental is one recorded in the nineteenth vol. of the Pathological Society’s Transactions (1868), by the late Mr. Cooper Foster. The tumour, an enormous fibro-lipoma fifty-five pounds in weight, was removed post mortem from a woman sixty-three years of age, and was considered to have developed between the layers of the omentum.

In view of these facts the following case seems worth recording.

The patient first came under my notice in October, 1884, in the New Hospital for Women. She was then suffering from a large abdominal tumour, supposed to be ovarian, and with this view of its nature an operation for its removal was undertaken. On exposure, however, the tumour was found to be solid, consisting apparently of fat, and under these circumstances the operator, declining to interfere further with it, closed the abdominal incision. Convalescence from this exploratory operation was extremely tedious owing to the feebleness of the patient, who, however, ultimately recovered and left the hospital.

After her return home, the tumour continued slowly but steadily increasing in bulk, causing so much discomfort that she became more than ever anxious to be rid of her burden. Under these circumstances I was asked by her medical attendant, Dr. Slimon, of Hackney, to admit her under my care at the Samaritan Free Hospital; and I accordingly did so with a view to operation in January of last year (1886).
The following history was then obtained. The patient, a widow sixty-two years of age, was the mother of seven children, the youngest twenty-four years old. She had never been strong, but until latterly had not been seriously ill. There was no family history of tumour, cancer, or phthisis. The menopause had occurred at the age of fifty-two.

About four and a half years previously she first noticed some abdominal swelling, and this had steadily increased from that time. Ever since the operation in October, 1884, she had been more or less constantly bed-ridden owing to increasing difficulty in locomotion from the size and weight of the tumour.

She was a feeble old woman of spare habit, but fairly well nourished. The thoracic organs were healthy; pulse 96 in the minute, quite regular, small and somewhat incompressible. There was no oedema of face or lower limbs. Urine was normal in appearance and quantity, of acid reaction and free from albumen; specific gravity 1020. The tongue was clean, appetite indifferent. The bowels acted regularly, but there was considerable trouble from flatulence.

The abdomen measured 46 inches in girth at the level of the umbilicus, and 23½ inches vertically from the ensiform cartilage to the symphysis pubis. The tumour, bordered above and in either flank by intestinal resonance, was uniformly dull on percussion and quite elastic to the touch, almost conveying a sense of fluctuation. It was most prominent below the umbilicus, whence it extended to the pelvic brim and overhung the pubes and groins. The entire mass could be readily swayed from side to side as the patient lay on her back, but could not be pushed upwards to any extent. On vaginal examination the uterus was found atrophied and quite free from the growth, which could not be felt in the pelvis.

A diagnosis of omental lipoma was based upon the apparent mobility of the tumour, and further upon the absence of any intestinal resonance in front of the mass.

The following are the details of the operation performed on February 6, 1886.

The cicatrix of the former incision in the linea alba having first been excised, the subjacent peritoneum was opened and the tumour was found to be closely adherent to the parietes anteriorly. No attempt was made to separate the adhesions; the capsule of the growth was freely divided longitudinally, and, introducing my hand, I succeeded without any great diffi-
culty in enucleating it *en masse.* During this process, however, the size of the tumour necessitated a prolongation of the original incision to the extent in all of fully 12 inches, in order to permit of its extraction. Some smart haemorrhage occurred during the enucleation, and eight or ten fine silk ligatures were subsequently required for bleeding vessels on the inner surface of the capsule.

On examining its connections, the base of the emptied sac was found to be continuous with the covering of an elongated mass of fat seated higher up in the omentum immediately below the transverse colon. This was not interfered with as the patient's condition rendered any further enucleation unadvisable. No other growths were detected, but the liver was felt to be considerably enlarged. The emptied capsule remained adherent to the parietes on either side of the abdominal incision, but was freely open to the peritoneal cavity above and below. The intestines lay altogether behind it, and neither the ascending nor the descending colon came into view.

After careful sponging out, the abdomen was closed without the insertion of a drainage-tube.

The tumour, after removal, weighed fifteen and a half pounds. It was solid throughout, consisting of dense lobules of fat with a scanty amount of fibro-cellular tissue.

The patient was much exhausted after the operation, but rallied in the course of the evening. On the following day the temperature rose to 101·2°, with a very feeble and intermittent pulse of 120, and there was constant vomiting of light-brown watery fluid devoid of odour. In the course of the next thirty-six hours the temperature fell to 99·2° and the pulse to 104, but sickness continued very troublesome, and the urine was very scanty in amount and dark coloured, although free from albumen.

From this time, however, matters gradually improved as the sickness abated, and the patient began to take nourishment by the mouth in place of the rectal injections of beef-tea with port wine and quinine which had been administered at three hours' intervals since the operation.

On February 11 (sixth day) the sutures were taken out from the upper two thirds of the abdominal incision, the remainder being removed on the following day after the bowels had been relieved with the help of an enema. The wound was found well united throughout.

Convalescence subsequently progressed steadily, and the
patient was able to leave her bed on February 24 (the nineteenth day after operation). She quitted the hospital four days later.

When seen in June last the woman was in good health. She had gained flesh and strength; abdominal examination revealed no evidence of any further growth.
XLI.—**Abdominal exploration for Chronic Intestinal Obstruction**: Relief of Symptoms by formation of artificial Anus: Subsequent removal of growth involving Splenic Flexure of Colon: Recovery. **By Bernard Pitts, M.C.** Read April 22, 1887.

Mrs. D., æt. 37, dates her troubles as commencing in October, 1885. She was carrying in from the yard of her house a wash-tub, and as she was going through the door, owing to her not steering straight, she hit the wash-tub against the portal, and this made it hit her in the abdomen on the left side. She immediately felt faint, and had to sit down; shortly after the faintness passed off, and she was able to go for a walk with her husband; but after walking about a mile, pain in the belly and gurgling came on; she had to lean on her husband for support, and to return home and go to bed. She was in great colicky pain for some hours. Previous to this accident Mrs. D. had enjoyed perfect health; she had been married for ten years, and had three children; she had always been troubled with a certain amount of constipation.

One month after the accident she had an attack of sick headache, vomiting, constipation, and pain in the abdomen; this passed off in three or four days.

On January 1, 1886, she had another attack which lasted rather more than a month. The constipation was so severe that she had only one action of the bowels in twenty-one days: the vomiting was continuous, and very offensive, smelling like faeces. She was treated with enemata and morphia, and gradually got better. From this time, up to admission into St. Thomas's Home, she could only obtain an action of the bowels by use of enemata. She had lost flesh and strength, and had frequent attacks of distension of the abdomen, with vomiting; for the last six weeks she had had no satisfactory evacuation of the bowels, even after repeated enemas.

State on admission. August 21, 1886.—Thin, pale woman, weak pulse, abdomen distended, coils to be seen and felt; much peristaltic movement; occasional vomiting, the vomit has a brownish tinge, but not offensive. One large coil can be
felt in particular stretched across the abdomen, running downwards, and to the left groin; this coil is very hard, but varies in tenseness.

The case was seen by Mr. Edmunds with me, and we concluded that there was mechanical obstruction, probably due to a malignant stricture of large bowel. Enemata were ordered, to be given with long tube.

On August 24, when we saw the case together again, we found the patient somewhat better, small lumps of faeces had passed, and the sickness had ceased. The large coil stretching obliquely across the abdomen still very prominent, but chiefly distended with air. There was no visible distension of the caecum, and no distension to be made out in left loin.

August 27.—No vomiting since 22nd, seems much better, but still quantity of faeces passed with enemata is insufficient even for the food taken. She passes wind freely.

August 30.—By the use of oil enemata every night, and soap enemata every morning, sufficient stools are now obtained, and patient is relieved of obstruction, and is able to get up today for first time.

On September 7 an examination was made of the abdomen and rectum under anaesthetic. No tumour was felt, and the result was negative. From this time her bowels did not respond to the enemata so well, and it was thought right to try 3ss Ol. Ricini; there was no action after this, and patient was sick for two days.

September 21.—Distension of abdomen had quietly been taking place in spite of enemata, and there was this day much faecal vomiting, worse in the evening and at night. She rapidly became exhausted, with feeble pulse, and her general condition was so bad that, after consultation, we decided to give morphia freely, hoping the acute symptoms would pass off (as they had before), and agreed that then an exploratory operation should be performed. Fortunately the vomiting completely ceased with morphia, and on September 24 she was so far recovered as to allow an abdominal exploration. There had been no relief obtained by rectum for some days. After making a small median incision, we found that the transverse colon was greatly distended, and displaced downwards, and that the descending colon was completely empty. The transverse colon was lying at a lower level than the umbilicus, and then the distended bowel passed upwards on the left side under the ribs, and so greatly was it distended that it would have been dangerous to manipulate sufficiently to trace the
exact connection of the distended with the collapsed bowel. No tumour could be felt.

By pressing up the abdominal wall a small incision was made (cutting on the finger) in the upper part of the left linea semilunaris, and the left end of the distended transverse colon was fastened by superficial sutures to the margins of the opening. A packing of iodoform gauze was employed, and a small piece of drainage-tube placed opposite the centre of the exposed part of bowel. The stitches were passed with extreme care through the superficial coverings of the bowel, for the distension was extreme.

The median incision was now closed, and the whole wound dressed antiseptically. No spray was, however, employed.

Under morphia patient remained very comfortable, and free from sickness; she passed some flatus per anum.

On September 26 I dressed the side wound, and punctured what I believed to be the colon at the bottom of the opening, with a narrow tenotomy knife. No gas escaped, and there was no smell on the knife. No further attempt was made then to establish an opening, and the place was again dressed with iodoform.

September 28.—Temperature still normal, no sickness; central wound dressed, and sutures removed; good union.

September 29.—The side wound was again examined, and the iodoform plugs taken out; but as one wished to gain firm adhesion, and patient was fairly comfortable, the dressings were replaced.

October 2.—As the abdomen was now considerably distended, an exploration for the supposed attached colon was again made, a director was introduced for some distance, but with no result, and it was quite clear that the bowel had broken loose, and that the opening had been made through the mesocolon. This was very disappointing; it was necessary to wait still one more day, to give time for the puncture to close.

On October 3, under ether, the whole of the dressings were taken off, and the coil of distended colon was seen through the abdominal wall; it passed below the level of the lateral wound, dipping down below the level of the umbilicus in the middle of abdomen, but was high up in its hepatic and splenic flexures. The lateral wound was enlarged downward so as to come over the colon, and by cutting through successive layers the omentum was reached and the colon identified. A longitudinal band was found, and at a time when the colon was relaxed it was seized with pile forceps and
held in the wound. The amount of movement the colon made in its peristaltic contraction was now well seen, for the pile forceps were drawn inwards considerably when a spasm occurred. The colon was now secured to the margins of the abdominal opening, a small opening was then made in it, and a second lot of sutures were used to attach the mucous mem-
brane of the bowel to the skin. Flatus and faeces escaped freely.

October 4.—A very large amount of faeces have escaped
during the night, and patient much exhausted. Temp. 100·6°,
abdomen greatly distended with flatus. Poultices were
ordered, and brandy given in small quantities by mouth; no
sickness. In the evening abdomen became less distended,
and temperature fell to normal.

October 8.—A large amount of faeces have been con-
stantly passing (since the last note) by the colotomy wound.
Yesterday and to-day a small action of the bowel occurred the
natural way.

During the last few days the patient has had curious
mental symptoms, with faecal odour of breath. She has been
lethargic, and was semi-comatose for some time (all the 5th),
not recognising her husband, sleeping a great deal, fractious,
imagines everyone is against her. She has a difficulty in
finding the right names for things, makes a great difficulty
about taking her food. These symptoms seem to be chiefly
due to toxæmia from feculent reabsorption. There is now no
abdominal distension and no tenderness.

November 5.—Greatly improved since last note; gets up
every day; has gained weight, now 7 st. 4 lbs., and weight
before operation 6 st. 10½ lbs. The major part of her faeces come
through the colotomy wound, but a little is passed per anum.
By introducing the finger through the colotomy opening, one
can feel towards the left flank a mass surrounding and growing
in the colon, and the contracted opening of the bowel through
this mass can be felt. The mass appears freely movable, of
about the size of a small orange, and well under cover of the
ribs. It was therefore decided to attempt the removal of the
affected bowel.

After careful preliminary treatment, by washing out both
upper and lower bowel, and by restricting the patient for a
week to milk diet, on November 22 the third abdominal opera-
tion was performed.

An oblique incision was made in the left side below the
ribs, about 2 inches above the colotomy opening. Great care
was taken whilst opening the abdomen to keep this colotomy opening shut off.  

On introducing the finger into the abdominal cavity, the collapsed descending colon was drawn out, and by hauling on this it was comparatively easy to pull the tumour into view from under the ribs, and with a little patience it was made to extrude from the wound. Clamps were placed on the healthy bowel above and below the strictured portion and the intervening part, with a portion of adherent omentum, and a V-shaped piece of mesentery (in which several small glands could be detected) was then cut away. Numerous vessels had to be tied, but by careful use of sponges no hæmorrhage took place into the abdominal cavity.

The mesenteric gap was now sutured with catgut, and the two ends of bowel were carefully united by silk sutures throughout the posterior and lateral aspects. The muscular coat of this united part of the bowel was then attached by numerous sutures to the abdominal opening, and the everted mucous membrane was carefully stitched to the skin. The patient was thus left in the condition of possessing a second colotomy opening, the first opening having contracted to the size of the little finger.

One may dismiss the after-treatment very briefly; everything went on as well as one could wish. The temperature remained normal throughout, and on the tenth day the patient was able to get up. She was then taking ordinary diet; she had no shock after the operation. She left for home on January 2, a perfectly different woman; she had gained a stone in weight, and had very little inconvenience from the artificial openings. The original opening had contracted to a sinus.

An apparatus was constructed to try and press back the projecting posterior wall and thus to restore the natural passage.

I saw Mrs. D.’s husband on April 19, 1887, or five months after the removal of the growth, and he informs me that his wife remains perfectly well, and is able to do her household work; her weight is now nine stones, or an increase of over two stones since the first operation. Except on two or three occasions all motion has been passed by the artificial anus; she wears the vulcanite shield, and attends to the bowels at a fixed time every morning. Syringing the opening, she obtains by this means a good action of the bowel, and has no further trouble or inconvenience during the remainder of the twenty-four hours. She does not suffer from any prolapse of bowel.
I obtained a small voluntary addition to the previous history, viz. that for a couple of years prior to her first symptoms of obstruction, she had been troubled with a slight cough and that, with the cough, she always had a little pain on the left side under the ribs, but both cough and pain had vanished since the colectomy. I mention this circumstance as told to me, for it may be that the pain felt with the cough was due to the commencing mischief in the bowel.

_Description of specimen._—The portion of colon removed measured in the fresh state about $4 \frac{1}{2}$ inches in length, and practically represents the splenic flexure; the new growth is singularly hard, scirrhus-like in density and appearance. The lumen of the intestine is greatly narrowed for about $1 \frac{1}{2}$ inches, and admits only a catheter of No. 1 size. The bowel above the constriction is much hypertrophied and dilated. On the mesenteric border several small glands were found and removed. A portion of omentum which was adherent to the bowel is to be seen in the specimen. A very marked contraction is to be seen on the outside of the bowel, much as if a cord had been tied round it; and it would seem as if the obstruction had commenced as an annular stricture.

The growth was examined microscopically by Mr. Shattock and proved to be a columnar epithelioma.

_General remarks._—It was from the first highly probable that the obstruction in this case was in the large bowel, but the position in the large bowel was doubtful. In making an abdominal exploration, I followed a practice that I have adhered to now for some years, when no stricture can be discovered by rectal examination, for I think a chance colotomy is an unsurgical procedure, and is very likely to fail even in relieving the patient of the obstruction.

One is also enabled by the exploration to gain definite information as to the cause and extent of the mischief, and this is of immense advantage when any further radical measure is contemplated. I may be allowed here very briefly to refer to two other cases of chronic obstruction in which I have made use of an exploratory incision and then performed colotomy.

Sarah C., æt. 42, came under my care in St. Thomas's Hospital in February, 1881, with complete obstruction of the bowels of three weeks' standing; she had suffered from constipation and some epigastric pain for several months. No stricture of rectum was discovered, and no relief could be obtained by enemata. By central abdominal incision an annular stricture
of sigmoid flexure was discovered, and the distended colon was attached to a separate incision in the left groin, made by cutting on to one’s finger; the bowel was opened two days later, and relief afforded; the central wound healed by direct intention, and the patient made a rapid recovery. One was much impressed during the operation by the ease with which this annular stricture might be removed, and so soon as the woman had recovered from the obstruction, this further operative measure was proposed to her, but she declined, and I could not press such a departure from orthodox surgery. During the next year this woman was able to do her household work in comfort, and all or almost all motion passed by the rectum, the opening in the groin merely acting as a safety-valve.

In 1882 Mr. Bryant published a successful case of removal of such a stricture secondary to a lumbar colotomy. Feeling that one was now justified in pressing more strongly the operation, I sent for the woman and again pressed it on her, but she was so satisfied with her present condition that she would not consent, and I was also afraid that the stricture might then have got adherent to surrounding structures.

This woman was again admitted into hospital in 1883, two and a half years after the relief of her obstruction, and died in a few days. During the last year of her life all the faeces had passed through the artificial opening.

On post-mortem examination there was found a very large growth pressing on the left ureter and causing hydronephrosis. Secondary growths were found in the liver. This case proved how slow to advance a malignant annular stricture may be, and made me determine in any future case to remove such a growth whenever possible.

The next case was one of obstruction in a man, &t. 48, who was under the care of Dr. Sharkey in St. Thomas’s Hospital, and I operated at Dr. Sharkey’s request.

A malignant stricture of cæcum was found, which was already adherent to surrounding structures, and the artificial anus had to be made at the end of the small bowel. The opening was made in a similar manner to the other cases, viz. by means of a small separate incision in the right groin.

This patient died three days after operation, and at the post-mortem secondary deposits were found in the liver. By the exploratory operation one learned: first, that no ordinary colotomy in either loin would have relieved the obstruction; and, secondly, that if he had recovered from the obstruction to the bowel it was not a case suitable for any attempt at
colectomy, for the growth was much too adherent to the structures round to permit of its removal.

In each of these cases, the selected portion of bowel was attached to a separate opening in the abdominal wall, mainly for three reasons: first, it allows the exploratory incision to heal by first intention, and as the second opening is exactly the size wanted, and the bowel is stitched all round, there is no wound near the opening to heal; secondly, the bowel is not displaced from its natural position by being drawn up to the central wound; and lastly and most important, as is evident from the case I have brought forward this evening, it is of great advantage for after-examination to have the opening near the seat of mischief; in the case of Mrs. D., if I had made an opening in the dilated transverse colon, opposite the upper part of the central incision, I should have been unable afterwards, by exploration with the finger, to examine the stricture and its relations.

With regard to the colectomy in Mrs. D.'s case it would have been clearly inviting disaster to have performed it before the obstruction had been relieved; the operation when the bowel was empty was comparatively easy, although the situation of the stricture under the ribs was most unfavorable, but owing to the great strain that had been going on for a year above the stricture, the mesenteric attachment had no doubt been stretched, and I was thus enabled more easily to draw the strictured bowel out of the wound.

It was very tempting, after removal of the affected bowel, to proceed to the complete suturing of the two ends, and thus restore the natural channel, but the great dilatation of the transverse colon made me determined to be content with the union of the hinder part of the gut, and give complete rest to the dilated bowel for a few months. I was in hopes also that by use of the vulcanite tube the feces might find their way into the descending colon, and a natural cure of the opening be possibly effected. The angle, however, at which the two parts of bowel are joined is too great to expect such a result.

The patient is now so little inconvenienced by the artificial opening that I hesitate to advise further operation, for suture of large bowel in this position must be attended with considerable risk and chance of failure. I trust I may have the advantage of the advice and experience of any members present on this question.

With regard to prognosis, it can only be said that it must be more favorable than if the disease had been left. It is
now one and a half years since her first symptoms of obstruction, and she is now in perfect health. It must be remembered, however, that some enlarged glands were found and removed at the operation.

The case of Sarah C., who lived two and a half years after colotomy, was a most favorable one for colectomy, for the stricture was favorably situated on the sigmoid flexure; at the time of exploration it was a mere hard ring in the bowel. Unfortunately it must be rare for obstruction to call attention to the disease at so early a period. It is surely not presuming to believe that if she had submitted to colectomy and had survived the operation, she might have had a much longer lease of life, and would certainly have been spared much suffering from the local affection.

In conclusion, I would draw attention to the very peculiar state Mrs. D. was in for the first few days after opening the colon, and whilst the enormous collection of faeces was being got rid of. A state of great and even fatal depression is not uncommon at this period in cases of colotomy for chronic obstructions, but I never remember to have seen a patient so evidently poisoned by the gases generated during the evacuation of a faecal collection. The breath had a strong odour of faeces, and the mental condition was most peculiar.
XLII.—Some Cases of Abdominal Cysts following injury.

By Rickman John Godlee, M.S. Read May 13, 1887.

I propose in the following paper to call the attention of the Society to three cases in each of which a large cystic tumour was developed as the result of the passage of a cartwheel over the abdomen. As that which occurred last in point of time is the only one in which the diagnosis is quite clear I will proceed at once to describe it, and will begin by saying that it is a case of ruptured ureter.

A little girl (M. A. E., No. in hospital register 1422), four years of age, was admitted into University College Hospital on July 21, 1886, having been run over by a cab on the previous day. She then complained of pain and tenderness in the abdomen, principally in the left inguinal and lumbar regions; here there was considerable bruising, and there was also bruising about the left elbow. The temperature was about 100° F. and the pulse 122.

She had been examined by a medical man at another hospital before admission, and nothing except the symptoms mentioned above had been detected and nothing further appears to have been noted for the first few days. In fact for the first fortnight, at the end of which time I first saw her, she remained much in the same condition. The bruising of the abdomen, however, disappeared, but increasing tension of the region previously bruised declared itself, and a little dulness was detected at the lower part of the chest on the left side. There was occasional but very slight vomiting and a dry cough, apparently the remains of an attack of whooping-cough; the temperature, which occasionally rose to 101° and over, sometimes sank to normal, and on the whole had a downward tendency. The urine, I regret to say, was not then examined, but nothing was ever noted amiss with it, and when it was examined on August 14, 15, and 16, it was acid, sp. gr. 1026—1028, and to the naked eye and under the microscope normal.

The indefinite swelling gradually gave place to a large well-defined elastic and fluctuating tumour which by August 12 extended inwards to within half an inch of the middle line, downwards to the umbilicus and the anterior superior spine of
the ilium, backwards to the erector spinae and upwards under the ribs. The stomach and spleen appeared to be pushed up by it and there was dulness in the left back to the level of the eighth rib. The tumour was quite dull; the rest of the abdomen was resonant as were the upper part of the left side and the whole of the right side of the chest.

On this day (August 12) I drew off with the aspirator, through a puncture between the iliac crest and the ribs, three inches from the spine, 550 c.c. of turbid, slightly alkaline urine containing 0·3 per cent. urea (total 1·53 grammes), half albumen, a large quantity of mucus, a small quantity of pus, and some phosphate crystals.

The child was not in any way upset by this proceeding, but the cyst rapidly refilled and in two days was about as large as before. The urine passed amounted to 250 c.c. on the 13th, 290 c.c. on the 15th, and 150 c.c. on the 16th, and was normal in character. The temperature remained slightly raised.

On the 19th (that is just a month after the accident) I opened the swelling, making the incision across the puncture previously made. The cyst wall was carefully exposed before opening it and was dense and fibrous, the lining perfectly smooth. The kidney was felt projecting into the upper and anterior part and seemed rough at its lower end; a drainage-tube was inserted and the wound was dressed with salicylic wool, the deeper parts of which were soaked in 1—2000 corrosive sublimate solution.

The amount of fluid drawn off was 1214 c.c.; it contained 0·15 per cent. urea, some pus, and 1/10 albumen.

For three weeks the patient was quite comfortable except for the copious leakage of urine from the wound, which showed no sign of diminution; the tube had been removed and the cavity and the sinus had both very much contracted. Then began the first of a series of pyrexial attacks, no doubt due to some accumulation of pus and urine in the deeper parts of the wound, which continued off and on till the middle of October. In these, the temperature often rose to 103° or 104° and the child was becoming pale, thin, and pasty looking. The reinsertion of a tube had no effect in stopping these attacks.

I could not doubt that the ureter was completely ruptured, because no blood had appeared at any time in the urine, and I argued that if the laceration had been in the kidney it would probably by this time have closed. I accordingly thought it best to remove the kidney, and this was done on October 28,
through a crucial incision in the loin. The operation was very difficult; it was very difficult to find the organ at all, as it was embedded in dense fibrous tissue, very high up and, of course, small. However, the nephrectomy was accomplished, but I fear that the upper end of the ureter was left behind and is the cause of the non-closure of the sinus, which still discharges a little pus. The temperature remained high, 101°—103°, for nearly a week and then fell to normal, the ligatures coming away en masse on November 25, a month after the operation. The child has made an excellent recovery and is now fat and strong, but, as was said, a minute sinus still remains.

It is difficult to collect all the urine of a child of this age. It may be said, however, that there was no marked diminution in amount after the operation. It varied from 10 or 12 up to 20 or 30 oz., the sp. gr. and the percentage of urea varying in proportion from 1030 to 1008 and from 2 to 1.2 respectively.

Other cases of this sort have been recorded. I submit that it is the best treatment to adopt when the patient is distinctly loosing-ground and there is evidence that the other kidney is healthy. In fact in this instance, after the first opening, no other course seemed open to me as it appeared likely that if the pyrexial attacks continued the child would ultimately fall a victim to them. Undoubtedly cases may arise where a permanent fistula communicating with the ureter does no harm beyond the inconvenience it causes, and then the patient must weight this inconvenience against the dangers of a nephrectomy.

Whether it might have been better to have delayed interference longer on the chance of the kidney becoming atrophied is another question. Possibly an answer may be suggested by the two cases which are to follow.

The first of these almost certainly received some injury to the kidney, but I am quite unable to say what the natures of that injury was and how far it accounted for the symptoms.

J. R., aged 23, a valet, was admitted under the care of my colleague Mr. Beck, on July 13, 1885 (No. in hospital register 1420), having been knocked down on that day and run over by a heavy waggon as he lay on his back, the wheel passing over his abdomen from the right hip to the left hypochondrium. He was suffering from shock on admission and vomited the contents of the stomach, flecked over with streaks of bright arterial blood. There was considerable pain all over the abdomen. A drop of blood was found at the end of the catheter when the water was drawn off; on this and the follow-
ing two days, as he was unable to pass his water, it was drawn off and was on each occasion found to be smoky, but after this it was passed naturally and was normal. Vomiting of bilious matter, not continued nor excessive, lasted till the 18th. The temperature rose to 100° on the second day and gradually mounted to 101.8° on the sixth day. After the eighth day it did not rise to 100°, but it remained over 99° till the day before his discharge, July 29, sixteen days after the accident. He became slightly jaundiced during his stay in the hospital, but left it feeling fairly comfortable and apparently without any tumour or dulness in either flank.

On August 12, a fortnight after his discharge, he came back, telling us that since that time he had been occasionally sick after taking solid food and had suffered slightly from diarrhœa. We found him pyrexial, with a temperature of 100° to 101.8°, with the abdomen slightly but uniformly distended and giving no signs of free fluid nor of a localized cyst; there was pain, however, in the hypogastric region and the left flank. He was put upon spoon diet and the vomiting did not recur. Hot fomentations were applied to the belly and the symptoms referred to this region subsided. There was no trouble of any kind with the water. In about four days the temperature came down almost to normal.

Between the date of admission and September 10, nearly a month, he was kept mostly in bed, and for a considerable part of the time on spoon diet, as it seemed clear that vomiting was on more than one occasion started by giving him solid food and allowing him to get about. Ultimately, however, this restriction was relaxed and the vomiting did not reappear. In the meanwhile a tumour developed itself, projecting slightly below the margin of the ribs on the left side, dull on percussion (the dulness extending in the axilla to the level of the nipples) and apparently pushing the heart up, for marked pulsation was apparent in the second intercostal space.

On September 10 an aspirator needle was passed through the seventh intercostal space a little in front of the line of the posterior axillary fold and so no doubt through the attachment of the diaphragm. It entered a cyst, and 7 oz. of turbid fluid were drawn off, containing a mere trace of urea (0.01 per cent.), a certain amount of albumen, and showing, under the microscope, blood-corpuscles, some indefinite granular cells, and some flat epithelium.

On the following day it is noted that the heart's apex was
beating in the third space; that dulness in the back began at the eighth rib and extended forwards as far as the sixth rib in the posterior axillary line; that in front of this there was stomach resonance as high as the fifth rib on the mid-axillary line; and that no tumour was to be felt below the ribs. The intercostal spaces were collapsed.

Four days after the aspiration (September 14) the physical signs were the same as before the fluid was drawn off. About this patient there is little more to be said; he gradually improved in general condition, though suffering occasional attacks of sharp pain in the abdomen, modified apparently by position, but he had no more actual sickness. The temperature, which rose to 100° the day after the aspiration, came down on the following day to normal and remained so until his discharge on October 9. There were still signs of unnatural distension of the stomach, the tympanitic note varying in extent but sometimes reaching the third interspace. The dulness in the back and axilla did not alter, but breath-sounds could be heard over the dull area. The intercostal spaces were again collapsed and no tumour was to be felt in the abdomen.

He has kept well since, though he appeared the other day at the hospital complaining of some pain in the abdomen, again of a dragging character, due, I believe, to a left inguinal hernia, for which he was supplied with a truss.

It seems almost useless to speculate upon the exact nature of the injury in this case. I do not think that the fluid was in the pleura, though it is not possible to prove the contrary. I think it most probable that it was contained in a cyst above the kidney and resulted from a laceration of the organ, and that by pressure on the stomach it gave rise to the troublesome gastric symptoms, and also as it extended backwards it pressed the diaphragm against the ribs and so caused the dulness in the back and axilla. The possibility of the existence of a diaphragmatic hernia was discussed, but the idea was discarded.

The third case is not like either of the others, and is perhaps more remarkable than the last.

W. W., a boy at. 7 (No. in hospital register 2450), was knocked down by a van-horse about September 23, 1885, and the front wheel of the van pressed against him on the right side between the iliac crest and the ribs, but did not pass over him. He remembered being pulled out and thinks that he could have walked home. He was seen by Mr. Adams Clarke,
of Bushey, who found him suffering from pain in the abdomen, and administered some medicine, after which vomiting took place and the patient was relieved. He was kept in bed and fed with spoon diet for four days, after which he was able to go out for a walk and in the following week he went to school. At the end of this week he vomited after eating some fresh pork, and from that time to November 28, about two months, he had occasional attacks of indefinite illness and gradually lost flesh.

I saw him on November 28, and found him with a temperature of 103° and felt, when he was under chloroform, a distinct tumour in the left side of the upper part of the abdomen; but when he was admitted into the ward this tumour could not be made out. The next day the temperature was normal.

He was kept in the hospital for nine weeks, during the whole of which time the temperature was slightly raised, being generally a little below, sometimes a little above normal. The tumour, which was sometimes only detected with difficulty, gradually because more distinct, occupying the epigastric and the left hypochondriac regions; it extended to the middle line in front and downwards to an inch above the umbilicus. There was a resonant percussion note over it, and a curious splashing sound was heard on sharply tapping it above. This was no doubt caused by the movements of the contents of the stomach, and as I was convinced that I could feel the transverse colon crossing the lower part of the cyst I did not think it safe to explore with the aspirator needle, and so the boy was sent home for a while.

He never had any urinary symptoms and all his other functions were natural. There were no signs of tubercle. The weight remained stationary: about 3 st. 2 lbs.

On August 24, 1886, he came back (eleven months after the accident). The tumour had much increased in size; it occupied the whole of the epigastric region and was mesial in position reaching, on each side, to the cartilage of the eighth rib and below to the umbilicus. It was movable from side to side and descended during inspiration. The stomach was clearly above it and the transverse colon apparently crossed its lower part as before. The boy's general health was good. His temperature was normal.

On August 25 I made an incision through the abdominal wall 8 inches long, the lower end of which was 1½ inches from the umbilicus. There were no peritoneal adhesions. Omentum (probably great omentum) appeared in the wound and also a
piece of a viscus, presumably flattened great intestine, but I am not prepared to assert that it was not the pyloric end of the stomach. I tried to pull up the omentum but failed. So I incised it above the exposed viscus and so laid bare the surface of the tumour. In doing so the small sac of the peritoneum was distinctly opened; we had thus not to do with a dropsy of the small sac.

After protecting the abdominal cavity with sponges, an aspirator needle was inserted into the cyst and 43 oz. of turbid, whitish-yellow fluid were drawn off, neutral, sp. gr. 1015, containing 5 per cent. urea and albumen in large quantity, but no sugar. As the cyst became lax, it was seized with forceps and incised and the margins of the opening in its wall were attached by stitches to the margins of the incision in the abdominal wall, the upper and lower parts of this latter incision being approximated above and below the opening into the cyst; a drainage-tube was inserted.

The wall of the cyst was dense fibrous tissue one sixteenths of an inch or more in thickness. The lining of it was perfectly smooth and soft and fell into folds when the fluid had escaped. The cavity extended back to the spine, up to the loin, and laterally as far as the kidney on each side.

The wound was dressed with salicylic wool, the deeper parts of which were soaked in 1—2000 corrosive sublimate solution. It behaved perfectly well and the boy did not have a bad symptom. The sutures came away in good time, and after occasional fluctuations in the amount of discharge (it was at first very slight, then very copious and watery, then gradually diminished), closure of the wound was complete by October 13, seven weeks after the operation.

The amount of urea in the fluid obtained from this case, 5 per cent. was larger than that found in the cyst resulting from the ruptured ureter in the first case. Yet there is no evidence that any injury of the kidney had occurred at any time. I thought on examining with the finger that it might possibly be an enormous hydronephrosis; but the mesial position and the fact that closure took place so comparatively quickly would seem to negative this hypothesis.

In view of the length to which this paper has already attained, and the obscurity of the last two cases, I will not add any further comment, but will simply leave these notes on record for comparison should similar cases occur to other members of the Society.
XLIII.—A Case of Spasmodic Torticollis probably caused by Cerebral Lesion. By G. V. Poore, M.D. Read May 13, 1887.

J. C., æt. 36, a bank clerk, consulted me in August, 1886, by the advice of Dr. Shingleton Smith, of Bristol.

The patient was suffering from spasmodic torticollis of unusual severity. At frequent intervals the head was turned violently towards the right shoulder. The spasm was so strong that the whole body rotated with the head, and not unfrequently the patient was obliged to sink upon the floor or cling to a chair or couch to prevent himself from falling. It was by far the most severe case of torticollis which I had ever seen, and, following my advice, the patient came into University College Hospital on August 5, where he occupied a private ward.

A few weeks previously the sternal attachment of the left sterno-mastoid muscle had been divided at Bristol, but this operation had not checked the spasm appreciably. When first I saw him there was no marked excess in the contraction of the left sterno-mastoid, although, previous to the tenotomy, Dr. Shingleton Smith assured me that the contraction of this muscle was very excessive. The rotation of the head when first I saw the patient was effected mainly by the right splenius capitis (assisted probably by the right obliquus capitis inferior), which could be felt to be in an extreme state of spasmodic contraction. After the patient had been a few weeks under observation (the re-union of the divided tendon having been perfected) the action of the left sterno-mastoid became far more violent than at first.

This shifting of the spasmodic action from one rotator muscle to another and from one side of the body to the other (though no new fact), seemed to preclude us from localising the lesion which caused the rotation either in the muscles or the nerves supplying them, for it is very improbable that a lesion should spread from the spinal-accessory nerve and sterno-mastoid muscle of one side to the splenius and obliquus and cervical nerves of the opposite side; and it is inconceivable that the latter muscles and nerves should become
diseased as the immediate consequence of the former being placed *hors de combat* by tenotomy.

These facts become comprehensible by referring the lesion to some centre specially associated with the function of rotating the head and having command (to this end) of muscles on both sides of the body.

The question, "Is the lesion central"? naturally arose in connection with this case; and on going in search of evidence of central mischief a scar was discovered (shown in the accompanying Woodcut from a photograph) close to the middle line and very near the coronal suture on the left side. The scar was rather more than an inch in length.

![Illustration of a patient's head]

Questioned as to this scar the patient stated that it was produced more than ten years ago by striking his head against a stone while diving into shallow water. He wounded his head severely by the blow, and was "half stunned" thereby.

In 1873 he contracted syphilis, which was followed by sore-throat, skin eruption, and loss of hair.

He says that he has "always" been liable to headache, and that in November, 1885, these headaches became more
severe and were limited to that part of the head in the neighbourhood of the scar. These headaches were accompanied by giddiness, and, occasionally, when walking, he felt as if he were going to fall. The headaches continuing he sought advice for them in March, 1886, and he was treated by mercurial inunction. About March 28 the headache diminished but the torticollis commenced, and has continued ever since.

Owing to the violence of the torticollis he was obliged to leave off work and made a voyage to Gibraltar, being away about three weeks. He returned to Bristol and was treated at the Infirmary, mainly with galvanism, for about three months, but without benefit. An attempt was then made to control the spasm by means of a mechanical apparatus, but he states that when he wore it he felt "like a restive horse in harness," and that it made him worse. Tenotomy was then tried, but all modes of treatment had been of no use up to the time of his admission to University College Hospital.

His habits were moderate, but he appeared to drink and smoke more than was good for him under the circumstances. The tongue was coated. The optic discs were normal. There were no other facts in the family or personal history which seemed to be of any importance.

The spasm never occurred during sleep and never woke him up. It seemed to disappear when he was quite quiet and composed himself to sleep with the head and neck in absolute repose, and without a thought of voluntary effort. When he awoke the spasm began again gradually, and became most intense when he attempted to walk about or became mentally excited.

Although the spasm was terribly severe, and made his life a positive burden, it was curious to note how small a thing would control it. When, for example, the head was twisted with utmost violence to the right, a very slight amount of traction exercised upon the nose, either by himself or another, was sufficient to bring the chin to the middle line.

The faulty position of the head was easily rectified by the artificial stimulation of the antagonising muscles. Thus, if during the height of the spasm the rheophores of an induction battery were placed, the one upon the left splenius and the other upon the right sterno-mastoid, the position of the head was immediately rectified.

The application of the constant current, the positive pole being placed over the anterior superior angle of the right
parietal bone and the negative over to left splenius, also had the effect of counteracting the spasm and keeping the head quiet. This effect of the constant current, even when very weak, was most marked. The patient very distinctly stated that he "derived great support" from a small chain battery worn with its poles placed over sponges on the spots indicated. The effect of electricity was transient and produced no permanent good. Both forms of current were, however, employed during the greater part of his stay in hospital.

On August 16, eleven days after admission, and after a most methodical electrical treatment, Mr. H. H. Brown, the house physician, makes the following note, which I can fully endorse: "No improvement in symptoms; the rotation of the head to the right is quite as forcible as when patient was first seen, or rather more so because the left sterno-mastoid has now quite recovered power." At this time his tongue had cleaned.

All things considered, the probability as to the lesion being central seemed very great.

1st. There was the absence of any evidence of disease in the nerves or muscles of either side, and the entire failure to relieve the patient by local measures, such as electricity and tenotomy.

2ndly. It was evident that the source of the trouble was to be looked for at some spot which, so to say, had command of muscles on both sides of the body; and such a spot was only to be found in the so-called motor area of the brain on the left side.

3rdly. We have the scar and the history of the injury which caused it, combined with a record of syphilis, and troublesome headache referred to the neighbourhood of the scar.

4thly. The scar is situated over that portion of the cortex cerebri which is said to be concerned with the function of rotating the head to the opposite side, and it seemed probable that some meningeal thickening was interfering with the working of this area of the brain.

The first step in treatment was to admit the patient to hospital, and thus to insure (a) rest and quiet; (b) a bland, nourishing diet, without tobacco or stimulants; (c) the discontinuance of narcotics to which he was getting habituated. During his stay in University College Hospital he took neither morphia, nor chloral, nor bromide, nor any other narcotic. Counter-irritation was employed over the scar,
first, by means of a blister, and subsequently by tincture of iodine. The main indication in treatment seemed to be the administration of mercury. This was commenced on August 10 by inunction, and on August 23, no constitutional effect having been produced, one grain of Hydrargyrum cum Cretà was given three times daily. This was increased to three grains three times a day on September 3, and as adjuvants a hot bath was ordered daily, and one third of a grain of calcium sulphide with each dose of mercury. There being still no soreness of the gums, on September 9 the dose of mercury was further increased to four grains of Hydrargyrum cum Cretà three times daily. The gums then became slightly sore, and on September 14 he suffered from a sharp dysenteric attack which lasted a few days. This reduced his strength somewhat, but the mercury being discontinued he soon recovered, and on September 23 it was noted that the spasm of the head was less violent, and from this date the torticollis lessened day by day. He was discharged on September 30 almost well, and a month later I had an opportunity of seeing that his improvement was maintained.

Another method of treatment had been mooted, viz. trephining, and the exploration of the parts of the meninges and brain beneath the scar. Soon after his admission my colleague Mr. Victor Horsley saw the patient with me, and he quite agreed with me in my views as to the seat of the lesion. As the patient’s life was not only useless but a burden to him we agreed, seeing the very unfavorable prognosis of such cases, that if the constitutional treatment produced no effect trephining would be warrantable, and it was, indeed, ultimately arranged that the operation should be performed on September 28. In the face of the improvement which set in on September 23 it was, however, decided by us that any operative procedure ought certainly to be postponed. Mr. Horsley’s dresser, unaware of the change which had taken place in our opinion, had, indeed, shaved the head preparatory to the operation, and we took advantage of this fact to blacken the scar and have the patient photographed.

The photograph, it will be observed, shows the exact position of the scar. The scar lies over the posterior ends of the superior and middle frontal convolutions, and certainly within the area (No. 12) which, according to Professor Ferrier, is associated with lateral movements of the head and eyes and dilatation of the pupil.
In this case there was no deviation of the eyes nor dilatation of the pupil, and this might be taken as an argument against the hypothesis which has been put forward. Area No. 12, as figured by Ferrier, is, however, a big one, and it is quite reasonable to suppose that the movements with which it is concerned are more specialised in man than in the monkey.

Some of Mr. Horsley's recent experiments point, I believe, to the fact that stimulation of a spot in the cerebral cortex in close proximity to that with which we have been dealing, produces a rotation of the head to the opposite side without any deviation of the eyes.

This case, I am well aware, proves nothing; but I think the hypothesis which I have put forward with regard to it is at least warrantable. It will serve to direct the attention of others to the point which I have mainly insisted upon, and may lead to the elucidation of an obscure subject.

The extreme severity of the spasm and the fact that it yielded to treatment constitute in themselves a sufficient reason for bringing the case before the Society.

JAMES W., æt. 49, but looking much older. He has had gout for seventeen years. It began in the great toe, and then settled in both hands. A year after the first attack he had a second, and two years later he had another. Five years later he had what was called erysipelas of the right leg.

For the last four years he has been unable to do any work, being crippled in all his limbs. His knees have been for many years weak and stiff and painful, and both toe-joints are stiff and deformed. He has chronic ulcers of both legs. His hands are fixed and distorted, with the knuckles swollen, and the fingers drawn over to the ulnar side. There is a soft gouty deposit over one of his knuckles, and over both elbows are soft masses as big as walnuts, which discharge a friable, half-fluid substance through the thin ulcerated skin.

As regards his history, his grandfather suffered severely from gout. He himself was strong and healthy all his early life. He has never had rheumatic fever, nor syphilis, nor any form of scrofulous disease. He has been unable to work for the last four years, but has managed just to get about.

On October 17 he was in his usual health. He went out on an errand that evening, and went to bed without any pain or discomfort. He woke with intense pain in the right knee, which was flexed, red, and swollen. There was also swelling of the lower fourth of the thigh, with a few faint dusky lines about the knee. He was treated with cold lotions, quinine, and opium.

I saw him first with Dr. Stocker on November 12. He had then a big abscess reaching two thirds of the way up the front and outer side of the thigh, and communicating freely with the joint, so that a wave could be felt and seen to pass from one to the other. The joint grated and felt disorganised. I aspirated the abscess, and drew off seven ounces of perfectly sweet, healthy pus.

November 14.—Aspirated again. Six ounces of perfectly healthy pus.

November 18.—Incision high up in the thigh, near the
Mr. Paget's Case of Suppuration in a Gouty Joint. 233
trochanter; let out nearly half a pint of healthy pus. Next
day the dressings were saturated, and the skin of the thigh
was acutely inflamed, as if irritated by the discharge.

November 22.—Sleeps and takes food well; temperature
normal. Knee much reduced in size; discharge profuse and
slightly offensive. Slight sore-throat, with dusky redness of
palate and fauces.

November 24.—Less inflammation of the skin of the thigh
and of the gluteal region. The sore-throat has disappeared.
Two counter openings made to-day in the thigh.

November 26.—An incision was made in the calf of the
leg, and nearly half a pint of offensive, blood-stained pus was
let out.

November 28.—He sleeps and takes food fairly well; tem-
perature normal. There is a vesicular rash on his forehead and
the back of his neck. The vesicles are small, flat, irregular,
and close set, as though he had been scalded.

December 3.—The rash has disappeared. He complains
much of general itching of the skin. The discharge is very
profuse.

December 8.—He has lost ground; he is delirious at night,
and does not take food well. Temperature remains about
normal, with slight rises. The tongue remains clean and
moist, as it was at first. There is distressing itching of the
whole skin, with desquamation. Counter openings were made
in the calf and at the back of the knee.

For many days after this the case seemed more hopeful.
All suppuration above the knee had ceased, and the incisions
were nearly healed; the outline of the knee was good, and the
big abscess in the calf was closing. The temperature was
never much above normal, and the urine, which was loaded
with greyish urates, was free from albumen. But he slept
and took food badly, and was often delirious. Bedsores
formed, and he died exhausted on December 30.

He was twice seen by Sir James Paget, who advised
against amputation as too dangerous for a man so pulled
down by chronic disease. I wish to thank Mr. Edwards and
Mr. Agnew, of the Western General Dispensary, for their
careful and skilful treatment of the patient.

I could not make a proper post-mortem examination. The
joint shows the bones bare of cartilage, rarefied, and super-
ifically ulcerated, without any marked eburnation or lipping.
The surface of the patella was slightly blood stained. The
crucial ligaments were gone, the synovial membrane was
thick, soft, and in part destroyed by suppuration. There was a small quantity of thick pus still in the cavity of the joint.

Remarks.—Dr. Norman Moore published a case of gout in a plumber, aged forty-six, who died after five weeks' profuse diarrhoea, with cough and dyspnoea. At the post-mortem, among many other gouty lesions, the right knee was gouty and contained a large purulent effusion.

Mr. Rivington published the case of a man of sixty-two, a sufferer from severe gout for twenty years, admitted with a severely-inflamed hand, suppurating over the dorsum. Sloughing extended rapidly, the extensor tendons were involved, the wrist-joint opened, and the adjacent tissues destroyed. On amputation the carpal bones were found extensively necrosed. The great toe-joint was also disorganised, but under treatment it became ankylosed.

Mr. George Critchett had a case of a man of fifty-eight, who had suffered from gout for twenty-three years. At last he had inflammation of the right eye, which recurred more than once; then came acute destructive inflammation, with suppuration of the globe, the sclerotic gave way, the lens escaped, and the globe collapsed.

These cases may perhaps show that, in some cases of gout, suppuration may occur, without any obvious common cause except the gout itself to account for it. In one case there was said to be "marked cachexia," in another there was the appearance of premature old age. But the only element of disease common to all was chronic gout, of many years' duration and of great severity.
XLV.—_Acute Dilatation of the Heart produced by Alcoholism._ By Robert Maguire, M.D. Read May 27, 1887.

The acute effects of alcoholism are shown more particularly in the nervous and circulatory systems. In the nervous system they are now well known, and are always serious. In the circulatory system, on the other hand, they are seldom important, and I therefore venture to bring before the Society two cases which illustrate an acute change in the heart, accompanied by pronounced symptoms, and caused by the abuse of alcohol.

Case 1.—A. B., æt. 23, was admitted under my care as an out-patient at St. Mary's Hospital, on February 18, 1887. His face was very pale and pasty-looking, but was not oedematous. The lips were bluish. There was marked oedema of the legs, extending upwards as far as the knees, but no oedema was seen in the arms. The abdomen was distended and contained a small quantity of fluid. The pulse was small, of low tension and short, and at the time of observation was beating 140 times in the minute. The pulse was also somewhat irregular in force. The heart beat was diffused, but the apex beat was found in the fifth interspace, half an inch outside the nipple line. Epigastric pulsation was excessive. There was no venous pulsation in the neck. The deep cardiac dulness in the parasternal line commenced at the second interspace, while the transverse dulness extended from a little more than an inch beyond the right sternal line to half an inch to the left of the apex beat. The first sound of the heart was short and clicky, and the intervals between the various sounds were almost equal. The second sound was, as is normal, more marked at the pulmonary than at the aortic cartilage, but yet not accentuated. At the junction of the fourth right costal cartilage with the sternum there was heard a soft blowing systolic murmur, not obscuring the first sound, heard best at this point, conducted downwards as far as the ensiform cartilage, upwards only a very short distance, and also heard to the right of the sternal line for about an inch. There was considerable dyspnœa, but no abnormal auscultatory signs over the lungs.
The temperature was normal. The tongue was dry and coated with a slight white fur; there had been morning vomiting. There was some tenderness over the hepatic region, but no enlargement of either liver or spleen could be detected. The urine, it was stated, was passed freely, but no estimation of its quantity could be made. It was of pale yellow colour, sp. gr. 1003, and contained no albumen or sugar. The patient complained of weakness of the legs, nervousness and sleeplessness, and great tremor of the hands was observed.

A diagnosis was made of dilatation of the heart affecting both the right and the left ventricles. The murmur described I believed to be functional, and probably produced at the aortic orifice. It was rather low in situation for an aortic murmur, but, in the absence of venous pulsation in the neck, it seemed improbable that there was any regurgitation through the tricuspid orifice. As there was no history of previous cardiac symptoms, or of any disease likely to cause organic valvular affection, it seemed essential to regard the dilatation as acute in its onset, and its presence accounted for the oedema of the legs and slight ascites. Such a dilatation of the heart is of common occurrence in cases of granular kidney, then supervening acutely upon an amount of hypertrophy sufficient to balance the increased arterial tension. This explanation was thought of in the present case, and was supported by the facts that the arterial tension was defective and the urine was of low specific gravity. But the pulse, in cases of acute dilatation, occurring as a complication of granular kidney, while of diminished tension, is widely different from the pulse found in the present case. In such circumstances the pulse is easily compressible, but the beat is large, soon over, and "flopping" in character, while the artery is still full between the beats. This condition has been styled by Dr. Broadbent "virtual tension." Again, in the case described, even when, as will hereafter appear, the patient quite recovered from the cardiac condition, no increased tension in the pulse, but rather the reverse, was observed. Albumen is frequently absent from the urine of granular kidney, but never, so far as I have seen, when the heart has become acutely dilated. It was therefore impossible that the dilatation of the heart in the case under discussion should be the result of Bright's disease. Muscular exertion, as is well known, will cause such an affection of the heart; but my patient had in no way exerted himself. In fine, no cause whatever for the dilatation was discoverable other than alcoholism, the history of which was well marked. The patient
had been a chronic dram drinker, and an excessive drinking bout had been terminated by the symptoms which led to his seeking relief at the hospital.

There were prescribed cardiac tonics in the shape of iron, nux vomica, and digitalis, and total abstinence from alcohol was ordered. The latter command he did not absolutely obey until after the first week or ten days.

On February 25 the oedema of the legs had greatly diminished and the ascites had disappeared. The pulse was larger in volume, but showed no signs of high arterial tension. It was now regular in force. The apex of the heart was beating in the nipple line, the deep cardiac dulness did not extend more than half an inch beyond the right edge of the sternum, and the systolic murmur previously described had disappeared. The urine had still a specific gravity of 1003, and contained no albumen. The digitalis was now suspended.

On March 1 all oedema had disappeared; the cardiac apex was situated in the fifth space about an inch to the sternal side of the nipple, and the deep cardiac dulness did not begin until the right border of the sternum was reached. The first sound at the apex was somewhat short, but the intervals between the sounds were of normal duration, while the pulse was of normal size and power. The urine was now of specific gravity, 1015, and contained no albumen. A week later the patient was discharged.

Case 2.—G. M., æt. 43, came as an out-patient to St. Mary's Hospital on March 11, 1887. He complained of swelling of the legs. His face was pale and anxious looking, and the conjunctivæ were watery and congested. There was great oedema of the lower extremities, reaching as high as the knees, and equally marked on both sides. There was no sign of fluid in the peritoneum and no oedema of the upper extremities. The hands were tremulous, the tongue also tremulous and coated with a whitish fur, and the patient complained of sleeplessness and nervousness. The pulse at the wrist gave 90 beats in the minute, was small, feeble, easily compressible, and showed no trace of virtual tension. The apex beat was situated outside the nipple line about half an inch, and in the fifth space. The beat was somewhat diffused. The epigastric pulsation was well marked. The deep cardiac dulness in the parasternal line commenced at the third rib. In the transverse direction it was found to correspond with the right edge of the sternum, and to reach a very short
distance beyond the apex beat. The first sound of the heart at the apex was somewhat short and sharp, and the intervals between the sounds were equal; at the base the second sound was more marked at the pulmonary cartilage. No murmurs were to be heard. There was no cough or dyspnœa. The liver and spleen appeared to be of normal size. The urine was scanty and high coloured; its specific gravity could not be taken, but it contained no albumen or sugar, and deposited urates copiously on standing. There was no history of gout or rheumatism, and no account of previous cardiac symptoms or dropsy. The patient had not been specially exposed to cold, and had not undergone exertion in any form likely to cause the condition of the heart. I obtained from him, however, an account of great alcoholic excess, which ceased only a week before he consulted me. Indeed, his debauch was terminated by the onset of his symptoms. He had been under the care of a medical man for some days, but he had not entirely knocked off the alcohol. I ordered entire abstinence from alcohol, and gave him iron, nitro-muriatic acid, and nux vomica.

On March 18 the patient reported himself much better. The œdema of his legs had much diminished. The apex beat of the heart was in the fifth space and one inch within the nipple. The dimensions of the heart were otherwise normal. The first sound at the apex was still somewhat short, but the intervals were normal.

In this second case, by the arguments I have already made use of, I was enabled to exclude valvular disease of the heart and Bright's disease. The cardiac signs were evidently those of dilatation of the left ventricle. Whether there was hypertrophy in addition was perhaps uncertain at the first examination, although the character of the apex impulse, and the fact that the apex although pushed outwards was not lowered, would tend to show that no hypertrophy was present. This is confirmed by the further progress of the case, since on the last examination the heart had only normal dimensions. The absence of dulness to the right of the sternum would indicate that the right ventricle was not affected, and it may be well to mark that in this second case there was no such dyspnœa as was found in the first case described, where, too, there were evident signs of dilatation of the right ventricle.

In both the above cases it would appear that dilatation of the heart was acute in onset; further, there appears little doubt that it was produced by the alcoholism. It may be
permitted to speculate as to the method of production. The absence of any virtual tension in the arteries during the dilatation, and of excessive tension when the heart had resumed its normal size, would show that the organ could not have given way on account of increased peripheric resistance. Moreover, in acute alcoholism the arterial tension is lowered. There cannot have been any acute degeneration of the heart since recovery was so rapid. I should therefore conclude that in both these cases we have to do with an acute poisoning of the heart muscle by the alcohol, causing it to give way before a normal, or even a reduced arterial tension. According to this view the left ventricle would be the first to suffer, as shown in the second case, since it encounters the greater resistance, while a greater degree of alcoholic excess, as in the first case, would cause even the right ventricle to dilate.
XLVI.—A Case of Endemic Hæmaturia due to the presence of the Distoma Hæmatobium (Bilharzia hæmatobia). By H. Handford, M.D. Read May 27, 1887.

In November, 1885, a healthy-looking, fresh-complexioned, well-built young man, æt. 24, a divinity student, came to me complaining of pain in the loins, and of passing blood in the urine, especially after walking.* He had left England for Natal three and a half years previously, and while there had been in robust health, except for two attacks of dengue. He returned four months before I saw him, and first passed blood in his urine while on the voyage home. The surgeon of the ship, who was familiar with the endemic hæmaturia of the Cape, at once diagnosed the disease.

When I saw him he experienced no ill effects from the disease except occasional pain in the back, and some slight discomfort, hardly amounting to pain, in the urethra at the end of micturition, when he usually passed a little blood, never exceeding a teaspoonful in amount. There is no appreciable quantity of blood mixed with the urine first passed. No blood is passed on first rising in the morning after a night’s rest in bed. More blood is passed after exercise.

He has no symptoms of cystitis, and usually only passes water three or four times during the day, and not during the night. The urine is acid, of sp. gr. 1020, and contains albumen, though little, if any, more than the blood would account for. There is a copious deposit on standing, consisting of urates, mucus, blood-corpuscles, and fragments of tissue. Embedded in the latter, and also occurring free, are numerous ova of the Distoma hæmatobium. Under a three-quarter inch cover glass from ten to twenty ova can be discovered. Free-swimming ciliated embryos were also found in the urine immediately after it was passed, but whether they had escaped from the ovum before the urine left the bladder, or whether they were set free by the pressure of the cover glass during examination, I cannot say. Certainly many were

* He had been under the care of Mr. Burnie, who had diagnosed the disease, and had been kind enough to send the case to me on account of its clinical interest.
DESCRIPTION OF PLATE VII, ILLUSTRATING DR. HANDFORD'S CASE OF BILHARZIA HÆMATOBIIUM.

Fig. 1.—Embryo of Bilharzia magnified about 600 diameters (½ oil immersion objective).

At a are shown the four rapidly-pulsating tubes, each surrounded by a sheath. From the upper left one a vessel leads for some distance and is gradually lost.

b. Gullet.
c. Primitive digestive area (stomach?).
d. The embryo was dying during the observations, and the protoplasm of the lower part of the body, from deficient moisture, shrunk away from the ectosarc at this point.

Fig. 2.—Embryo escaped from the ovum shown in Fig. 3. × 250.

Fig. 3.—Embryo of Bilharzia, within ovum, head towards spike. × 250.
hatched in the latter way, and the process of the escape from the ovum could be readily watched. He has not been subject to diarrhœa; on the contrary, the bowels are usually constipated. There is, therefore, no reason to suspect any implication of the mucous membrane of the intestine, as sometimes happens.

He is a total abstainer, and while in Natal the water he drank was usually filtered, but occasionally he may have drunk unfiltered water.

He has remained under observation until the present time, (May, 1887) and has continued in robust health, though working hard as a student. He but seldom experiences any pain in the back now, and is more comfortable when he drinks freely of water. He passes less blood, frequently none for several days together, and can usually tell, by a feeling of fulness in the perinæum, when he is about to pass blood. The ova are still present in the urine but in diminished numbers, and require more searching for, many slides not containing any. All the ova contain fully-developed embryos. None are found in the early stage of development. Several of them were found surrounded by a cluster of leucocytes, indicating the existence of an inflammatory process, leading to suppuration which probably set them free.

Walking does not now cause pain in the back or bring on the hæmorrhage. When he is passing blood the frequency of micturition is increased to five or six times a day instead of three or four.

In regard to treatment, he took small doses of turpentine regularly from November, 1885, to March, 1886; and from January to March, 1887, he took ten-grain doses of iodide of potassium, with an equal quantity of the ammonio-citrate of iron, thrice daily. No attempt has been made to apply parasiticide remedies to the mucous membrane of the bladder by means of injections. He was somewhat anxious on first hearing the nature of his malady, but is now in hopes of outliving his guests.

The points of chief clinical interest are—

1. Can the parasite multiply within the human body, or is an "intermediate host" necessary?

2. In the latter case, and in the absence of re-infection, (and the conditions for re-infection are not known to exist in this country), what is the "expectation of life" of the parasites, and may the host reasonably hope to outlive his guests?

3. Is any parasiticide treatment useful?
4. What is the method of infection and what precautions should be recommended with a view to prophylaxis?

The life-history of the Distoma hæmatobium, like that of many of the other Distomidae, has not yet been worked out, but I believe I am correct in saying that in all those in which the cycle has been completely observed an intermediate host has been found to be necessary.* All analogy, therefore, teaches that the parasite cannot multiply within the human body, or in fact within the body of any vertebrate. What, then, is likely to be the duration of life of the parasite? Leuckart† says, "Concerning some we know that they continue to live in exposed situations, not only for several years, but even through a whole decade (Bothriocephalus latus, Tænia saginata); but others again scarcely live longer than a few weeks. On the whole, the period of existence of entozoa may be assumed to be longer than that of free-living animals of a similar size. In the somewhat allied fluke disease in sheep it is known that the parasites may live at least a year and probably longer; but when the animal is infected with only a limited number of flukes (twenty to thirty) it may eventually completely recover by the death of the parasites. In my patient the adult bilharzia must have lived at least one and three quarter years. How much longer will they continue to live?

With regard to parasiticide treatment, the parasites are said to inhabit the branches of the portal system of veins, and to migrate in pairs to the veins of the pelvis for the purpose of depositing eggs.‡ Remedies must reach them therefore through the blood. Vesical or rectal injections may destroy those that are situated superficially in the mucous membrane, but cannot affect those more deeply placed in the veins. Neither can injections influence the mucous membrane of the ureters from which much of the hæmorrhage is supposed to come. So far as I am aware, no remedy taken by the mouth has yet been found to exert any evident unfavorable influence on the life of the parasites.

Little is known of the mode of infection, and therefore nothing certain can be said as to precautionary measures. We must be content with generalities till the life-history of

* The only exception is Aspidogaster, which inhabits the pericardial cavity of the fresh-water mussel, and attains sexual maturity without any further change of habitation. Among the internal parasites of the Vertebrae we do not know of a single analogous example (Leuckart, p. 70).
† Ibid., p. 87.
‡ Ibid., p. 44. The ova, however, have been discovered in the tissues of the viscera, and even in the skin.
the *Distoma haematobium* has been worked out, as was done so beautifully in the case of the liver-fluke (*Fasciola hepatica*) at Oxford in 1881 and 1883 by Professor Thomas, now of Auckland, New Zealand, who showed that *Limnaeus truncatulus* was the intermediate host.

"The sporosacs of Distomidae have only been found in molluscs and fish, but the encysted larvae are more widely distributed, and have been found not only in molluscs, but also in aquatic larvae of insects, in crustacea, in hydrachna, rarely in worms, in small fish, tadpoles, frogs, and newts. Some cercariae encyst themselves on plants, or on the sides of the vessel which contains them."*

Cobbold "has tried to induce the ciliated embryos of the *Distoma haematobium* to enter the bodies of a variety of animals, such as gammari, dipterous larvae, entomostraca, lymnae, paludinae, different species of planorbis, and other molluscs; but neither in these nor in sticklebacks, roach, gudgeon, or carp, did they seem inclined to take up their abode."†

The failure of these experiments, and the fact that the disease has now frequently been brought to this country, but has not become indigenous (and I believe no case has been recorded which cannot be traced to infection abroad), point to the conclusion that British forms do not afford a suitable intermediate host in which the embryos may develop. We must look for the intermediate host in some form of life common to Egypt and the Cape, probably an invertebrate, and possibly a mollusc.

Analogy with the liver-fluke, however, would suggest that the organism gains admittance to the body, either as a free cercaria in unfiltered water, or in the encysted condition on fruit or uncooked vegetables. Boiling or filtration in the one case, and cooking in the other, is the remedy. For fruit or vegetables that are eaten uncooked, soaking in salt and water is to be recommended.

N.B.—The following observations on the structure of the ovum and embryo, and on the points of contrast with those of the liver-fluke, are worthy of note:

"The development of the ovum of the liver-fluke does not proceed beyond the stage of yolk cleavage within the bile-ducts

† Parasites, 1879, p. 52.
of the sheep. The complete development of the embryo can only take place out of the body of the bearer, and at a lower temperature."

All the ova of the bilharzia that I examined on more than a dozen occasions during the space of eighteen months contained fully-developed embryos which could be set free by rupturing the ovum, by pressure on the cover-glass, and after the space of half to one minute the liberated embryo began to swim actively. No ova were seen showing simply the yolk cleavage of the early stage of development. It is stated that in the bilharzia, embryos may be set free within the urinary passages, and may even penetrate the tissues. Though in this case embryos were seen in the urine within a few minutes of its being passed, I am of opinion that they were set free by the pressure of the cover-glass during the preparation of the specimen for examination.

The cilia covering the embryo were in several cases seen in motion before the rupture of the ovum. The embryo sometimes has its head towards the spiked end of the shell, sometimes towards the other end. The eye-spot observed in the embryo of the liver-fluke, which very closely resembles the embryo of the bilharzia, is absent in the latter.

"In the embryo of the liver-fluke, just behind the head papilla, there is a globular portion of the body which has a somewhat different appearance from the rest of the parenchyma, and reacts differently with staining agents." This, says Thomas, may possibly be the rudiment of a digestive tract. In the bilharzia embryo this differentiation of the protoplasm is very marked, and there is in addition a distinct gullet. In the embryo of the fluke, "narrow passages may be indistinctly seen on either side of the embryo; these are the excretory vessels, which have been found in several other trematode embryos. Midway between the extremities of the body, and on the right and left of the middle line, are two funnel-shaped spaces provided with cilia in constant motion." In the embryo of the bilharzia, four at least of these have been observed instead of two. But I look upon them as (? contractile) tubes forming part of a water vascular system, suspended in a perivascular sheath, and having a rapid, waving, to and fro movement of from 200 to 400 per minute. The two at the upper part move much less quickly than those at the lower part. Vessels can be distinctly traced from them.

With regard to the duration of life of the embryo, in the

case of the fluke "the average in water is about eight hours; occasionally one may live over night. In an alkaline solution of peptone they have been kept alive three days, but though they increased somewhat in size, no advance in organization took place." In the bilharzia five hours is the longest time I have kept an embryo alive under a cover-glass. In urine passed on October 14, 1886, active free-swimming embryos were found on October 17, and again on October 19. On the latter date, all the ova found contained living active embryos; but within ten minutes of the time they were placed on a slide under a cover-glass they had all ruptured and set free their embryos. A few days later, the embryos in all the remaining ova in the urine were dead. They were granular in appearance, and seemed to be penetrated by innumerable bacteria.
XLVII.—Case of a Child in whom a Patch of Lupus had been removed partly by scraping, partly by Salicylic Acid and Creosote. By F. Dawtrey Drewitt, M.D. Read May 27, 1887.

The case is brought before the Society only because it affords an unusual opportunity of seeing the result of lupus treated on the same subject by two different methods. The patient, who has come here this evening, is a strumous, sallow-complexioned girl, eleven years of age. She was brought to the Victoria Hospital for Children in December last. On the left side of the face was a more or less circular patch of lupus vulgaris four inches by five in diameter. It started four years ago from an old suppurating gland at the angle of the jaw, and it had gone on spreading steadily. It extended downwards underneath the chin beyond the middle line, forwards and upwards to within an inch of the lower eyelid, and to within half an inch of the angle of the mouth, and it included the whole ear, the lobe of which had disappeared. The centre of the patch contained irregular bands of whitish cicatricial tissue, over which minute dilated veins were spread. The advancing edge formed on the cheek a ridge of piled-up granulation nodules. This ridge was about half an inch in breadth, and from it there was a scanty discharge of thin yellowish secretion. The lower edge on the neck was red, scarcely raised above the skin, and less actively advancing.

Wishing to compare the salicylic acid treatment as practised by Unna of Hamburg with the treatment by scraping, I asked one of my surgical colleagues at the hospital to undertake the treatment of one portion of the patch while I undertook the other. This he was good enough to do; and so all the diseased tissue above a line drawn from the angle of the mouth to the lower border of the ear was thoroughly removed by scraping, and three days afterwards I applied a plaster of creosote and salicylic acid (fifteen parts of the former to one of the latter) to all the remaining portion, carefully cutting the plaster to slightly overlap the patch of lupus, and then covering it with oiled silk and strapping.

Three days afterwards, on removing the plaster, the unhealthy growth was suppurating, little pits being left corre-
sponding to the tubercles, while the healthy skin remained unaffected. The application of the plaster had been painful, so after bathing the part I rubbed in with cotton wool a 20 per cent. solution of cocaine until sensibility had gone before applying the second plaster. This was repeated in subsequent dressings, and in four weeks' time after eight plasters had been applied the diseased part had become smooth and healthy, and the plasters were discontinued. There has been no return of the growth.

Unna claimed for the salicylic acid treatment that it removed old bands of cicatricial tissue. This it has altogether failed to do, and not only that but some new bands of thin cicatricial tissue have been formed, but still, on comparing the scar left by the salicylic acid treatment with that left by completely successful scraping, it will, I think, be seen that the salicylic acid scar is softer, smaller, and has less tendency to contract, and judging from this case alone, there is distinct evidence in favour of the salicylic acid treatment of lupus.

THE patient in attendance, a youth æt. 19, was admitted into St. Thomas’s Hospital on March 7, 1887, with “hammer-toe” affecting the second digit of the right foot.

The deformity had the usual characters. The first phalanx was retracted at the metatarso-phalangeal joint but could be straightened without difficulty, while the second phalanx was flexed at right angles upon the first and strongly resisted extension; in all other respects the foot was perfectly well formed.

The left foot was normal in aspect, but it was found that the range of extension at the first phalangeal joint of the second toe was much more limited than that which existed at the same articulation in the third, fourth, and fifth toes. This was regarded as the result of an affection of the same nature as that in the corresponding part of the opposite foot but in a less advanced stage.

The patient states that the condition was first observed at the age of five years. Two cousins on the paternal side have suffered from the same complaint.

The deformity was treated by excision of the head of the first phalanx. The soft parts over the dorsal aspect of the articulation were divided by a longitudinal incision down to the bone, the head of the phalanx was then made to protrude through the wound and was removed by bone forceps in front of the attachment of the lateral ligaments. The joint was straightened and fixed in position by means of plaster of paris. Dressings antiseptic.

The wound healed by first intention, and the patient was discharged at the end of three weeks without apparatus of any kind. At the present time the toe remains stiff and straight at the affected joint, and locomotion is perfect.

The subjoined remarks, based chiefly upon an analysis of twenty-two cases, may serve to draw attention to certain features in connection with the disease that appear to have hitherto escaped comment.

1. The complaint is in its onset peculiarly one of childhood
and youth. In the whole of the cases referred to the deformity was first noticed in the period between infancy and early adult life, in one instance shortly after birth, in the rest, between the ages of 5 and 21, with a slight preference for the third quinquennial period. In many of these, however, it is probable that the actual pathological process commenced long before the resulting changes in the joint had become sufficiently marked to attract attention.

2. The lesion appears to be confined to the first phalangeal joint of the lesser toes, and the metatarso-phalangeal joint of the great toe (an articulation which may perhaps be regarded from the developmental point of view as belonging to the interphalangeal series). The two examples of the affection of the great toe which have come under my own observation resembled exactly the condition described by Mr. Davies-Colley a few weeks since; and I may be permitted to state that a preparation of the affected structures in "hallux flexus," which, through the courtesy of that gentleman, I have been able to examine, confirms my view of the pathological identity of the disease with "hammer-toe."*

The affection is comparatively rare in the third, fourth, and fifth digits. The second toe was the seat of the deformity in twenty of the cases in my list, the fourth and fifth together in one case, and the fifth alone in one case.

3. Hammer-toe is often bilateral. This obtained in ten out of the twenty-two cases. In the remaining twelve the left and right foot suffered in equal proportions. Where both feet were affected the two attacks were separated by an interval of from three to seven years.

4. The tendency to the disease often appears to be inherited. In four cases in my list the complaint had appeared in other members of the patient's family. In one of these (male) the mother and maternal grandfather had been affected in the same manner; in two others (females) the deformity had been present in the mother, and in the case before the Society two female cousins on the father's side were suffering from a similar condition.

5. The disease appears to be equally common in the two sexes, and proportionately as frequent in the middle as in the

* "Hallux flexus," like hammer-toe, appears always to arise in the young, but, unlike the latter, has not yet been seen in the female subject. The name proposed by Mr. Davies-Colley is not quite appropriate, as in the less extreme examples of the disease the limitation of the range of extension at the metatarso-phalangeal joint does not become sufficient to cause the toe to assume a permanent flexed position.
lower classes. No special constitutional diathesis has been observed in any of my own cases.

6. The deformity is almost always attributed to the use of ill-made boots. There is, however, no doubt that "hammer-toe" may arise quite independently of defect in the shape of the foot covering, and it is open to question whether the connection between the complaint and the evil ways of the shoemaker is ever more than a coincidence. In one example in my own series the retraction of the toe was noticed in the first year of life before any shoes had been worn, and as a rule the shape of the foot was perfectly normal, except for the hammer-toe, but an associated hallux valgus was present in three cases, and pes cavus in two. It must be remembered that the deformities of the toes for which the shoemaker is responsible are very frequent and often very complex, while "hammer-toe" is comparatively rare.

7. The most important feature in the morbid anatomy of the disease was first shown by Mr. Shatlock in a card specimen exhibited at the Pathological Society on December 21. I may, however, be allowed to state that my first dissection of the condition, which revealed the same lesion, was made over two years ago and that I have since that time taught to my class the facts now demonstrated.

The essential elements in every case is a contraction of those fibres of the lateral ligaments which pass to the under surface of the attached (distal) portion of the glenoid plate. In addition to this the articular extremities of the bone may undergo secondary changes of shape in long-standing cases, the head of the proximal phalanx becoming flattened, in consequence of atrophic lesions in that portion of the articular cartilage which in the normal condition plays upon the base of the middle phalanx during extension of the joint but in "hammer-toe" is permanently separated from it; and a similar process may take place also in the base of the middle phalanx. The peculiarity is scarcely visible in the preparations now shown, but is admirably exemplified in a specimen which Mr. Walter Edmunds has been kind enough to place at my disposal.

When the joint has been dissected it is sometimes possible to extend the middle phalanx by the exercise of a moderate degree of force; and it is noticed that this movement, as well as that of restitution, is accompanied by a spring-like action very similar to that which takes place in the condition known as the "trigger finger." The phenomenon is undoubtedly a result of the alteration in the shape of the articular ends of
the bones, and is explained by the sudden relaxation of tension which occurs when the shorter radius corresponding to the atrophied portion of the head of the first phalanx is brought into opposition to the corresponding surface of the middle phalanx (Plate V).

The nature of the changes leading to the shortening of the ligaments is unknown. It is probable, however, that the starting-point is a peculiar form of chronic inflammation of the glenoid plate and associated portion of the two lateral ligaments. The muscles, tendons, and fasciae take no part in the causation of the deformity but may possibly become implicated secondarily in long-standing cases.

8. Our knowledge of the true seat of the disease places the treatment upon a more satisfactory basis. In the past the recognised methods have been: (1) Section of tendons—flexors, extensors, or both; (2) division of the ligamentous (and other) tissues on the plantar aspect of the affected joint; (3) amputation, partial or complete, of the member; and (4) excision of the articulation.

The first of these measures is unserviceable. In the second it would be difficult to limit the section to the ligamentous structures, and moreover it is not easy to prevent a relapse; amputation is objectionable because it entails an unnecessary mutilation and may lead to a secondary hallux valgus; but excision is free from the drawbacks attending the other plans. The operation described in connection with the case brought forward this evening is extremely easy of performance, the healing process is rapid and the result all that could be desired with regard to appearance and utility.

In conclusion, I have to thank Mr. Shattock and Mr. Adams for two of the preparations shown to the Society, and my colleagues at St. Thomas's, for permission to make use of the notes of their cases.
LIVING SPECIMENS

DESCRIBED BY CARD.*

I.—Further Notes of a Case of Obliterative Arteritis.
By A. Pearce Gould, M.S. Exhibited October 8, 1886.

This patient was shown to the Society two years ago. Since then he remained at work as a labourer until six months ago, when he was attacked with numbness in the right arm and forearm, accompanied by severe headache; this headache has continued ever since. Four months ago he suddenly lost power in the left arm, and the same evening he became unconscious. Next day he spoke and moved his arm. He noticed the pulsation of his right subclavian artery until four months ago, not since.

The right forearm is distinctly colder, and measures one inch less in circumference than the left. The skin on the back of the right hand is of a deep red hue, and when the blood is displaced by pressure it only returns very slowly. Neither the radial nor the ulnar artery can be felt on the right side, and there is no pulsation in the brachial artery. The axillary and the third part of the subclavian artery can be plainly felt as a hard pulseless cord. The right superficial temporal artery cannot be felt pulsating in front of the tragus, the left is very readily felt. The right common carotid artery appears to be normal. The heart's apex-beat is in the fifth left interspace, vertically below nipple; the sounds are normal; there is no bruit. Pupils equal, react to light; the optic discs are similar in appearance on the two sides.

* 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."

S., H., æt. 4½. Female. At three months of age she was let fall downstairs, and was picked up unconscious. She remained in this condition two days, but was seen by no medical man.

Her mother states that during the two days she lay very quiet, had no convulsions, and did not vomit. On coming round it was noticed that she did not move the left arm and cried when it was touched; at the time this was ascribed to local injury. A large swelling in the position of that now existing ("as large as her mother’s fist") was noted on the second day; this gradually decreased until it reached its present size. Pulsation was noticed two to three months later. The child has never seemed to suffer in any way as a result of the injury, she has never complained of her head, and was as forward in speech and walking as any of her brothers and sisters.

On examination the patient is healthy looking, bright, and active. A prominent swelling of oval outline (3½ by 1½ inches, the long diameter coursing transversely) is found over the most prominent part of the right parietal bone, rather behind its centre, and extending slightly beyond the sagittal suture over the left parietal bone. It is elastic and pulsates visibly; moderate pressure causes no pain or slowing of the pulse. On palpation a large oval defect in the skull is readily made out, rounded at its left extremity, at its right continued into a fissure which passes downwards 1½ inches, reaching the squamous portion of the temporal bone. Its edges are strongly everted, and from the posterior margin a triangular spicule of bone projects forwards.

On examining the trunk and limbs the left upper and lower extremities are evidently smaller than the right (a difference in circumference in the leg of half an inch, in the forearm of a quarter of an inch). The patient slightly drags the left leg in walking; there is no rigidity, and the reflexes are normal.

The large opening, existing in this case, strongly supports the statement of Mr. T. Smith that in these cases considerable absorption of bone occurs, while the want of development on the left side points to probably considerable injury to the brain, although not enough to warrant one in supposing the swelling to communicate with the lateral ventricles.

Note.—The patient was seen again in February, 1887.
The opening appears to be rapidly increasing in size, the skull defect being now 2 inches by $3\frac{1}{2}$.

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Ellen W., æt. 13½, was admitted into the East London Hospital for Children on May 26th, 1886, with a tumour, irregularly hemispherical, and as large as an orange, growing from the inner and lower part of the right patella; it was somewhat movable, firm, almost cartilaginous to the touch, and not painful. It had followed an injury sustained about nine months previously. There were some enlarged glands in Scarpa's triangle; they were movable, painless, and not very obviously dependent on, or in relation to, the tumour.

The tumour was removed through a linear incision, parallel with the inner border of the patella; it was found to be growing from the patella, and being unmistakably sarcomatous, the patella was also removed. The skin and neighbouring parts were unimplicated. The girl made a rapid recovery, with a stiff joint.

She has been seen several times since the operation, and there is at present no sign of recurrence.

Addendum, June, 1886.—Still no sign of recurrence.

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The patient, who is also the subject of pulmonary phthisis, presents the following appearance on laryngoscopic examination:

Slight reddening of the laryngeal mucous membrane. Both vocal cords slightly eroded. Right ventricular band much swollen, of a pale yellowish pink colour; surface irregu-
larly nodulated and probably ulcerated. At the posterior extremity of the right ventricular band there is an excavated ulcer. Inter-arytenoid fold swollen, and forms a rounded projection at its right edge. In the subglottic region on the right side, just below the vocal cord, there is a small tumour of the size and shape of a small bean, of greyish pink colour. The surface of the tumour is fairly smooth, and its edges shade off into the surrounding mucous membrane.

*History.*—The patient first came under observation in June, 1886, as an out-patient under Dr. Biss at the Brompton Hospital. Dr. Biss was absent at the time, and the man was seen by Mr. H. H. Taylor, the Assistant Resident Medical Officer, who drew my attention to the case.

At that time there were no physical signs of disease in the chest, but there were ulceration of both vocal cords, and a tumour in the subglottic region. The tumour was then more conspicuous than it now is, and did not shade off into the adjacent tissues. The diagnosis lay between tuberculosis and syphilis. The history given by the man was as follows:

Winter cough for three or four years. Continued cough and hoarseness for one year.

A few weeks after I first saw the patient his sputum was examined, and found to contain tubercle bacilli; slight signs of phthisis were also detected. He was then admitted into the hospital under my care through the kindness of Dr. Roberts, and subsequently became an out-patient again under Dr. Biss, to whom I am greatly indebted for allowing me to show the case to the Society.

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V.—*A Case of Congenital Deficiency of Right Fibula, and Fusion of Left fourth and fifth Metacarpal Bones.*

By A. Pearce Gould, M.S. *Exhibited November 12, 1886.*

The right leg is shorter than the left. The right tibia is bent forwards, with a vertical groove in the skin over the convexity of bone. There is no fibula. The three inner toes only are present. The foot is turned outwards.

*Left hand.*—Only four metacarpal bones are present; the little and ring fingers articulate with the inner of the four, which is larger than either of the others.

Patient is recovering from her third attack of optic neuritis and other evidences of intracranial growth, retaining, practically, perfect vision. Her first attack occurred in 1884, with paralysis of both sixth nerves. She hears a grating or clanging or singing sound in her head, and this sound, varying at different times, can be heard by auscultation, at maximum behind and in front of left ear.

VII.—A Case of Paralysis of Supinator Longus, Biceps, and Deltoid Muscles from Pressure on fifth and sixth Cervical Nerves. By C. E. Beevor, M.D. Exhibited November 26, 1886.

Five weeks ago the patient, a woman aet. 36, went to sleep, leaning the left side of the neck on the edge of a table for three hours. On waking up she found that she was paralysed. The muscles affected were the biceps, deltoid and supinator longus, supra- and infra-spinatus, and slightly the trapezius; also she had anaesthesia over the shoulder and in the thumb. The deltoid, biceps and supinator longus muscles re-acted very slightly to a strong faradic current; with constant currents, $CCC = ACC$, in the affected muscles.

The case is important as it shows that pressure on the fifth and sixth cervical roots of the brachial plexus, above the plexus, produces paralysis in the supinator longus, biceps, and deltoid muscles, though all these muscles are supplied by different peripheral nerves in the arm. Attention was called to this class of cases by Professor Erb in 1874, and other cases have been described in Germany by other authors since that time. It is believed that this is the first case published in this country, and also that the point of pressure has in no other case been more exactly localised than in this one. In most of the other cases recorded, the injury has been caused by falling on to the head and shoulder, but here there was distinct tenderness over the course of the fifth and sixth cervical nerves in the posterior triangle of the neck, the
part which came in contact with the edge of the table. The combination of the muscles supplied by the fifth and sixth cervical roots, viz. the supinator longus, biceps, and deltoid, is considered to be of a physiological nature, i.e. to produce a definite movement, and not to be a mere anatomical arrangement.

The prognosis of these cases is favorable.


T. M., a schoolboy, æt. 11, has had this swelling in the calf of his right leg as long as he can recollect anything. He is an orphan, and nothing is known of his antecedents. He states that he was run over by a cab some years ago and that the swelling followed this accident, but no other information of such accident can be obtained.

He has been under observation for some months, and the swelling has been somewhat larger than it is this evening, the diminution being due to rest in bed. By means of the application of an elastic bandage the swelling can be reduced.
upwards of 1 1/2 inches in circumference. No pulsation can be detected in it. The skin moves freely over it in all parts. It has occasionally been temporarily enlarged, owing to prolonged standing or walking, and at such times the boy has complained of pain and tenderness.

Scattered over the surface of the head, trunk, and limbs are upwards of twenty small naevi, varying in size from that of a pin's head to a pea. Many of these appear to be degenerating, and are probably followed by the growth of fresh ones.


J. Q., æt. 17, single, a shoeblack by occupation, no relations living. Patient had good health till he was seven years old, when he caught cold, and has since been in his present condition. The upper extremities, chest, and back are covered with a dry scaly rash, which is present also over the general surface of the skin. On the outer side of each elbow-joint the skin is much thickened, and presents a dark, muddy appearance; the scales are easily picked off. The same condition is found below the knees. The hands and feet are perfectly normal. Patient seldom perspires, except on his face.


J. N., a female, by occupation a cook, æt. 40, single, was first admitted to the Marylebone Infirmary in 1884 with shooting pains in both legs. Family history good, catamenia regular. Patient says she was obliged to give up work a year before. She was admitted here, as she could not stand, though she states positively that at that time her feet had not altered in shape and that she was able to wear ordinary boots as usual; she never wore high heels. The patient presents all the
PLATE VIII, TO ILLUSTRATE MR. LUNN'S CASE OF DEFORMITY OF THE FEET IN A PATIENT SUFFERING FROM LOCOMOTOR ATAXY.
A Case of Cyanosis of Feet (Raynaud's Disease).

By John R. Lunn. Exhibited January 28, 1887.

C., æt. 56, a mason by trade, admitted to the Marylebone Infirmary, December, 1886. History good. Has had several healthy children. Never had syphilis nor any serious illness. Laid up with rheumatic pains in 1855 and 1866, after catching cold. He noticed his present condition of feet in 1884, twitchings and numbness coming on in paroxysms, and lasting a few days; then he would have a short relief. Since 1884 he has not been able to do a day's work. His feet are always cold to the touch, and frequently turn black, resembling gangrene. After they are wrapped in cotton wool they become the natural colour and feel very hot to the patient. He often gets patches of anaesthesia just above both ankles, also hyperaesthesia. The soles of the feet were ironed in 1885, in the hopes of improving the flat condition, and setting up some new reaction of circulation, but with very little success. Patella reflex normal. No other nerve-symptoms. Muscles act well to the continuous current. Ankle-joints and toe-joints somewhat rigid.

During the last three months the patient has developed a large, firm, round, non-pulsatile swelling in the left groin below Poupart's ligament, about the size of an orange, which is gradually getting larger.

H., æt. 51, single, a stableman by occupation, was admitted to the Marylebone Infirmary September 23, 1886. His father died in the infirmary four years ago from erysipelas of the face (he was a well-developed man). His mother died young. Had no brothers nor sisters.

Present condition.—Patient very anaemic, and general pallor of mucous surfaces. Puffiness of eyelids, dryness of skin. Never perspires, nails good, skin generally glistening, legs scaly, extremities cold, thyroid glands not to be felt; special senses good. Tactile sensation delayed and impaired, tongue thick and enlarged. Tonsils not well developed; slowness of thought and action, gradually getting worse. Feels the cold very much, and is very drowsy; has no whiskers; no hair on pubes or axilla; eyelashes very scanty. Knee and plantar reflexes normal; no anaesthesia or paralysis; urine normal.

XIII.—A Case of Removal of both Upper Jaws. By Rickman J. Godlee, M.S. Exhibited January 28, 1887.

W. W., æt. 55, was admitted into University College Hospital April 14th, 1886. He had a gumboil nine years previously, which continued to collect and break till July, 1885. Mischief then spread along the roof of mouth and pieces of bone came away.

On admission.—He had a large squamous epithelioma involving most of the hard, and part of the soft palate, perforating the left antrum and involving most of the alveoli.

First operation, April 18th. The upper lip was split, and the nose turned up after making incisions round the alæ. The parts removed were the lower parts of the right and left maxillæ, the lower part of the septum, and the left inferior
Living Specimens.

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turbinate bone, but leaving the floors of the orbits and the
tuberosity of the left maxilla.

Second operation, August 15. Recurrence having taken
place the parts removed were the floors of the orbits, the upper
part of the septum nasi, the remains of the left superior
maxilla, and parts of the turbinate bones.

There is at present no apparent reappearance of the disease.

XIV.—A Case of Sporadic Cretinism, with Myxoedema.
By A. H. Robinson, M.D. Exhibited February
11, 1887.

E. B., æt. 25, was born in a low-lying portion of the parish
of Stepney, and in the neighbourhood of a canal, gas-
works, and other unsavoury surroundings. He is the fourth
child of his parents, and was born at full time, labour being
in every respect natural. At his birth the practitioner in
attendance expressed the opinion that the child “would not
live a month.” He was so weakly as to be unable to take the
breast, and was fed by spoon for some time, his food consist-
ing of sugar, butter, gruel, &c. The mother states that the
house they inhabited was to all appearance sanitary, and they
lived in it after the birth of this child uninterrupted for
eighteen years, when they removed into Essex. Up to twelve
years of age the child refused to assume the vertical posture,
all attempts to make him do so being followed by screaming.
He was unable to walk until twelve years of age. He made
no attempt to speak until thirteen years of age, and he only
reached his present very imperfect attempt at speech a few
years ago. The first dentition took place about the usual
time. The mother is unable to say when he ceased to grow
in stature. He has always been of costive habit, the bowels,
as a rule, not acting for three weeks, and even a month has
elapsed without any action. Defaecation is accompanied with
great pain, and he has occasionally fainted owing to its
severity. He has always been capricious in matters of diet,
being more particularly fond of lean mutton and custards, but
always refusing fat meat. He has complained of his throat
from time to time, but the cause of this has never been ascer-
tained. He has never had fits or any other illness. The supra-clavicular swellings have been present from birth.

*Family history.*—E. B.'s father was a whitesmith, and suffered severely from lead-poisoning. Owing to this he was incapacitated from all employment for some years before his death, which occurred at about fifty years of age. He had several enlarged joints, and there was some deformity of his lower limbs. He also was born in Stepney. So far as his wife knows, there is no history of neurosis in his family.

E. B.'s mother is a native of Northamptonshire, and is now fifty-three years of age. She is a tall spare woman of decidedly nervous temperament. Some time ago she was under my care with aphonia and general debility. The aphonia passed off without any special treatment. She has endured much anxiety and fatigue in the nursing of her husband and children. She had epilepsy prior to her marriage, but has had no fits since. Her youngest sister was also subject to fits. Several other brothers and sisters died of consumption in early life, and one brother died, aged fifty-three, of "gout." She remembers seeing cases of enlarged thyroid gland in her native place in Northamptonshire, but no case occurred among her relatives. She has been seven times pregnant, the pregnancies ending as follows:—1, a male, born at full time, still living, subject to fits, and once under treatment for supposed hydrophobia following the bite of a dog; 2, a female, stillborn, full period. 3, a male, died under two years of age, of wasting; 4, the Cretin, to whom this paper refers; 5, a male, living, healthy; 6, a male, stillborn, full period; 7, a male, living, and with him what was called a "false conception."

*Present condition.*—E. B. is 3 feet 6 inches in height, but broadly built (Plate IX), his chest measurement being 24 inches in circumference. His limbs are well and strongly formed. The belly markedly pendulous. The head is deformed, the face being excessively developed, the cranium deficient, the imperfection being most noticeable at the occiput. There is a marked transverse depression, the width of two fingers, crossing the vertex at the fronto-parietal suture. The nose is broad and stunted, the nose approaching very nearly the vertical. As a rule the chin droops upon the sternum. The eyes are somewhat obliquely placed. The lower jaw is projected forwards to an extreme degree; the forehead is low and somewhat receding. The circumference of the head is barely 20 inches. The lips are thick, more especially the lower, which hangs everted, and they are never in apposition,
PLATE IX, TO ILLUSTRATE DR. ROBINSON'S CASE OF SPORADIC CRETINISM.
the tongue being visible between them, though not actually protruding. There are no fissures at the angles of the mouth, but there are several linear scars on the upper lip. The hair is light brown, scanty towards the occiput, short, dry, and coarse. The scalp is with difficulty kept free from pityriasis. The skin everywhere is of a tawny hue, readily picked up between the fingers owing to its extreme looseness; over the hands and feet it is thrown into wrinkles with every movement. It is, moreover, extremely dry and harsh, no approach to perspiration ever having been noticed. There is marked puffiness of both upper and lower eyelids, but no real oedema anywhere. The teeth are deeply discoloured; the molars are partly decayed and irregular, the upper incisors notched; there is a double row of lower incisors, the cutting edge of several of them being removed. The palate is unusually high and vaulted. The ears are large and flat, but well formed. The hands and feet are large, the former being remarkably spade-like, the latter flat. Over each clavicle, and quite filling and projecting from the supra-clavicular fossæ are soft, flabby masses. The gait is waddling, from side to side, the hands being carried in a flexed position over the front of the abdomen, the body held stiffly, the eyes downcast, and the head oscillating finely as he walks. The feet are raised from the ground in walking so as to resemble equine progression. He cannot run. His power of grasp is almost nil. There does not appear to be any affection of the organs of special sense. Speech, however, is very rudimentary, and he is unable to do more than partially pronounce a few words,—for instance, "doctor," for doctor, and so on. He can imitate the mewing of a cat very perfectly, and makes use of that species of vocalisation for any other animal he is requested to imitate. The knee-jerk is difficult to elicit on both sides. There is no ankle-clonus. No response follows tickling the soles of the feet, but the slightest prick of a pin is readily detected and recognised in every part of the body. He cannot readily stand with his feet together, but there is no absolute loss of power of co-ordination. Respiration is of normal frequency, abdominal in character, expansion of chest being deficient, percussion note everywhere flat, breath-sounds feeble. Pulse beats at eighty-four per minute, is full and regular. Heart's apex-beat cannot be felt in any position, cardiac sounds clear, and conducted to right apex in front, as well as to back of chest, but no bruit is detected. The superficial veins of the front of the thorax and abdomen are very prominent. His temperature is normal,
and probably occasionally subnormal. He is particular in his choice of diet, preferring puddings, eggs, and the like to meat, and he refuses anything of a fatty nature. He frequently complains of pain in the abdomen, no doubt due to his costive habit. The bowels act at long intervals, never less than three weeks elapsing. A thyroid gland has been carefully sought for, and no trace of one can be found. Micturition is infrequent, two days often passing without the bladder being emptied. As a rule he signifies his desire, but occasionally he passes it involuntarily. The urine has been examined from time to time, and has always been markedly acid, very pale, 1005 sp. gr., and free from albumen. Urea was estimated at 2 per cent. The sexual function is apparently quite in abeyance; there is no evidence of masturbation. He is of cheerful disposition, though his expression is somewhat melancholic. He takes interest in what is going on around him, but there appears to be no inclination to intellectual development, though various means are afforded him.


The case is that of a boy, æt. 15, who has had lupus vulgaris of nose for four years. He first came under my care in February, 1885. The nose was then scraped with a sharp spoon, and the surface almost entirely healed.

Early in 1886 he came to me again with recurrent lupus of nose, and ulceration of pharynx and epiglottis. The latter had a granular, pitted and "worm-eaten" appearance, such as is seen in tubercular cases.

In the autumn of the same year the palate first became affected, and at the same time it was seen that the septum nasi was perforated, and that some destructive inflammation was going on inside the nose.

The ulceration of palate began immediately behind the
DESCRIPTION OF PLATE X, ILLUSTRATING MR. GLUT-TON'S CASE OF OLD LUPUS OF NOSE, WITH RECENT TUBERCULAR ULCERATION OF LIP, PALATE, PHARYNX, AND LARYNX.

It shows the recent ulceration of upper lip, imperfect cicatricial tissue just behind the incisor teeth, tubercular nodules over soft palat and cicatrized mucous membrane on the posterior wall of pharynx.
incisor teeth around the opening of the anterior palatine canals, a fact which would lend some support to the theory that the disease spread by direct extension from the nose. It was at first in a condition similar to that now seen over the soft palate. The upper lip also became affected, apparently from friction against teeth which were covered with the secretion of the parts behind.

Present condition.—The nose has a few scattered patches of advancing lupus. The inner surface of the upper lip, and the mucous membrane over the anterior part of hard palate opposite the incisor teeth is occupied by a granular ulcerating surface showing no tendency to heal. The mucous membrane over posterior part of hard palate, and nearly all the soft palate, is swollen and red. Scattered over this surface are a few minute yellow points, which are thought to be tubercular ulcers. The parts now deeply ulcerated were previously in a similar condition (Plate X).

The pillars of the fauces, the pharynx and the remains of the epiglottis are swollen, red, and granular. The posterior wall of pharynx has at times in part cicatrized, and the epiglottis has been partially destroyed.

The lungs: Dr. Sharkey tells me that there are slight abnormal signs in apex of left lung which probably indicate early tubercular disease.

Eyes: Mr. Nettleship is of opinion that the eyes show undoubted signs of inherited syphilis.

Teeth normal.


L. L., æt. 6½, family history and previous history good. Eighteen months ago the patient slipped into a hole, and was said to have put out his hip; there was inability to walk, and (?) displacement; this was easily reduced, and the patient was able to run about next day. Is said to “have put it out” twice since then, and wriggled it in himself on both occasions.
Living Specimens.

When he first applied at the hospital on November 5 the leg was slightly flexed and rotated outwards, but the deformity quickly disappeared under manipulation, but how it did so was not very evident. Since then he has continued lame.

The left lower extremity is shortened to the extent of an inch, and everted slightly; it is somewhat wasted, and on rotation there is at times a distinct grating. There is also slight wasting of the muscles about the hip, and the great trochanter appears prominent. In front of this is a well-marked, rounded boss, immovable excepting with the femur. The front of the joint is full, and on external rotation there is increase of this fulness, apparently from pressure of the enlarged head. There is no pain on manipulation. The great trochanter is an inch above Nélaton's line. No other joint is affected. The patient about with no more limp than would be caused by the runs shortening.

XVII.—A Case of Saturnine Cachexia, illustrating Paralysis of Arms and Legs, Subcutaneous Nodules, and Gout. By Sir Dyce Duckworth, M.D. Exhibited February 11, 1887.

C. J. D., a single woman, æt. 32, engaged in trimming upholstery. Very sallow and cachectic; of small build. Had employed a cosmetic powder for her face for five years. This was analysed and found to be carbonate of lead. Marked Burtonian line on gums, arms much wasted, double wrist-drop, supinators unimpaired, Gubler's dorsal tumours of the hands well-marked (caused by over-flexion of carpus, deficient support of extensor tendons, and prominence of bones). Muscles of upper arms and scapulas affected, deltoids especially. Legs: muscles much wasted and flabby, tremors, no rigidity. Pains in muscles and bones, and much tenderness on pressure; walks feebly. Faradic contractility completely lost in extensors of thumbs of both hands; much impaired, but not lost, in other extensors; supinators react readily. Galvanic irritability lost in muscles of thumb, the others reacting fairly well except extensor communis digitorum of right arm. Electro-sensibility unimpaired. Muscles of both
legs react readily to both currents (Dr. Steavenson's report). Knee-jerks much increased, also sole-reflexes.

History of colic. Urine of rather low sp. gr. with occasional traces of albumen and blood. Urea much diminished; pulse frequent and of high tension. Attacks of acute gout in left great toe-joint, and left wrist. Appearance of small subcutaneous nodules over the tibiae.

Great improvement in six months under treatment by good diet, warm baths, galvanism, and large doses of iodide of potassium.


GEORGE W., æt. 38. Was born in London, where he has always lived. Is married and the father of three children, of whom one is alive and healthy, one died in infancy, and one was stillborn. Always been temperate; drinks ale. Had scarlet fever at three years of age, water on the brain at nine years of age, and St. Vitus’s dance at ten years of age. Eight years ago fell from the top of a tree to the bottom on his face. Was formerly very active. About five years ago he noticed, or rather his friends told him, that his face was beginning to swell. The swelling appeared first in the eyelids, the sides of the nose, and cheeks. He noticed at the same time that his gait was unsteady, so that he was at times thought to be drunk; he had a tendency to fall on his right side. Formerly he could walk very quickly. He has always felt chilly. During the last four years he has been slower in action and thought, and for the last two years has had difficulty in articulating his words, owing to his tongue being too large for his mouth. He says that his teeth began to fall out and his hair to be loose before the swelling of face began. Patient has a heavy stolid appearance; the hair on the vertex is thin and scanty, the integument of forehead is thickened, of a yellowish pigmentation, and has a few wrinkles. The eyebrows are very arched, and the eyelashes thin. The eyebrows are puffy, transversely wrinkled, and pearly in aspect; the root of the nose is broad and thick, and the alæ nasi thick, fixed, and broad; the cheeks are puffy and full, and present a defined
Living Specimens.

delicate pink flush, whilst the lips are thick, especially the lower, which is often of a purplish hue; the tongue is broad and thick, and there is very marked thickening of the mucous membrane of the cheeks and palate. The gums are lobulated and thickened, and the teeth loose. The voice is monotonous and articulation slow. The thyroid is not perceptible. The hands are clumsy and expressionless, and the skin over the extensor surface of arms and hands is dry and scaly; has occasional numbness in fingers. Pulse is 72, regular, small volume. The chest and abdomen are normal. There is some slight pitting along the course of left tibia. Reflexes (patellar) fairly marked; no ankle-clonus. Some solid œdema over the backs of feet. Urine 1016, no albumen or sugar, urea 1.1 per cent., phosphates. Taste perverted. Temp. subnormal. Hearing fair. Gait slow and heavy. Father died of apoplexy. Has one sister, who is dropsical.


Ada Elizabeth B., æt. 41, single, was born at Brighton, but has lived in London always. Five or six years ago she noticed "suffocation feelings and risings" in stomach. About three years ago she noticed that she was swelling all over, especially in the legs and feet, so that she was unable to bend them. Very shortly afterwards her hands began to swell, especially across the knuckles; one year ago her friends told her that her face was getting stout, and she noticed swelling, especially about the eyelids, nose, and lips, so that she appeared "to look cross and to pout at people." At the same time her throat ached whilst talking, and she was obliged to give up singing, and to talk slowly on account of the thickness of her lips. She was formerly very active and quick, but during the last year or two she has been obliged to give in on account of the difficulty she felt in exerting herself. She has complained of dryness of the skin for two years, and also partial loss of power in her arms. Her teeth have been loose for three or four years, and her hair has come out a good deal of late. During the last two or three years has had a feeling at times as if everything "was going on her left
side," and she has had occasional giddiness in walking. For three years she has been slower in thought and action, so that she used to get lost in her business, and her memory has been impaired for two or three years, and her taste and smell have been lost at times, also hearing. She had "gastric fever" eighteen years ago, and although never laid up, except for this illness, has never been quite well, especially during the last six or eight years. Her father is alive and well; her mother also alive, but delicate. Five sisters and brother died in infancy. Her maternal grandfather died of angina pectoris, and a maternal uncle of "galloping consumption." One paternal aunt had epilepsy. Patient is heavy looking in appearance; hair dark brown; irides grey; the eyelids are pearly in aspect and thickened. Skin of forehead has a yellowish tinge, hair still abundant on vertex; there is thickening of the alæ nasi and also of the lips, especially the lower. Cheeks are puffy, and present a delicate pink flush; root of nose broad; tongue is big and flabby, and the teeth are loose; no swelling of gums. There is some thickening of interior of cheeks and soft palate. The voice is slow; no special fulness above clavicles. Thyroid not felt. Pulse 60, regular, and of fair volume. Chest normal. No marked thickening of hands; skin on the extensor surfaces dry and scaly. No marked œdema of legs or feet. Temperature subnormal. Urine sp. gr. 1020, trace of albumen; no sugar. Patella reflexes absent.


This patient fell from a tree upon his head last March. He was picked up and carried to bed, where he lay quietly for six weeks. All this time there were no nerve symptoms noticed by the patient, who had complete control over all his limbs. At the end of eight weeks he presented himself at University College Hospital, where he was examined by Mr. Barker. The position of the head, and mass of inflammatory material around the upper vertebrae, left no doubt that there had been a fracture of the latter. At this time he was partly paralysed. He could walk, but dragged his right leg heavily
after him. His right arm was also very weak, but he could execute all its movements. There was slight loss of power over the bladder. The knee-jerk was very much exaggerated. The optic discs were normal; sensation appeared normal everywhere.

The patient remained in the same state for some time, and then began to improve gradually under the use of small doses of perchloride of mercury and tonics. There is now very little weakness left, but the deformity of the neck and the stiffness of the latter remain as before.

XXI.—A Case of Rhythmical Tremors affecting one Limb (right arm) only. By Arthur Davies, M.B. Exhibited May 27, 1887.

HENRY B., æt. 46, dog collar maker. He had gonorrhoea at eighteen years of age, but denies syphilis; has always been healthy, and has never met with any injury. His family history is good.

The patient attributes his present illness to a severe mental shock sustained five years ago, when he lost his wife and four children in nine months. He states that about four years ago he first noticed that his right hand began to shake, and that this unsteadiness gradually affected his whole arm, and that it was increased by exertion; he says that whilst awake the arm is in constant movement. During the last eighteen months he has also noted gradual loss of power in the arm; he has never had any pain.

The patient is a well-nourished man. There are constant rhythmical tremors of the arm; the fingers are partially flexed, but the interossei are unaffected, the hand is alternately flexed and extended on the forearm, and the forearm on the arm. The movements are increased on exertion; they continue when the patient is at rest. There is loss of power in all the muscles of the shoulder and arm; the grasp of the right hand is less than that of the left. There is no wasting of the muscles.

Electricial report by Dr. Howard Tooth.—There is much less electro-sensibility in the right arm, but to faradism there is no difference in the irritability as compared with the left, except perhaps in the right triceps, which is a little less irritable than that of the left side. To galvanism, the reactions are absolutely normal.
REPORT

OF A

COMMITTEE OF THE SOCIETY NOMINATED NOVEMBER, 1884.

TO INVESTIGATE

JOINT DISEASE IN CONNECTION WITH LOCOMOTOR
ATAXY.

THE following Report has been drawn up from a study of living specimens, anatomical preparations, and cases recorded in English and foreign literature.

The living specimens examined by the Committee were 27 in number, and in the appendix to this report the history, present state, and progress of such of these cases as have not hitherto been recorded are fully detailed. Full abstracts of 42 English cases and an analytical table of 66 cases, collected from English and foreign sources, are also appended to the report. The number of foreign cases might have been largely augmented, but in many of them the data supplied were insufficient for the purposes of this investigation. The Committee have also examined a numerous series of cases of osteo-arthritis in ten infirmaries in London.

Having accumulated all this material the Committee came to the conclusion that the knowledge gained with regard to Charcot's disease would be best set forth by an endeavour to answer the following three questions, which comprise the chief points raised in the debate two years ago.

1. Is the joint affection in Charcot's disease the same or a different disease from that which was formerly known as chronic rheumatic arthritis?
2. Is Charcot's joint disease, as described by him, ever found without the nervous symptoms usually regarded as characteristic of tabes dorsalis?

3. Is characteristic osteo-arthritis ever found associated with the symptoms of tabes dorsalis?

It was also the intention of the Committee to investigate the relation of syphilis to Charcot's joint disease, but they found it impossible, in many cases, to determine whether the patients had or had not suffered from syphilis.

In attempting to decide the question whether Charcot's joint disease is, or is not, an instance of what has been long known as chronic rheumatic arthritis, the Committee have formed a strong opinion that no investigation of the subject would be adequate which did not embrace a study of the clinical as well as the anatomical features of the two diseases, and that to take only the final products of a pathological process and to ignore the mode of their development, would be unreasonable and unscientific.

The very essence of the difficulty of answering this question consists in the circumstance that we cannot trust to a definition of chronic rheumatic arthritis founded upon pathological specimens and clinical records of cases made before the recognition of locomotor ataxy, or at least before any suggestion was made that an affection of joints might be related to locomotor ataxy. In the ordinary descriptions of cases of chronic rheumatic arthritis no reference is made to the state of the nervous system. The Committee, therefore, visited some of the workhouse infirmaries in London and made a study of numerous cases of osteo-arthritis.

Robert Adams, in his work on rheumatic gout, says, "The wrist-joint and the joints of the carpus, metacarpus, and the phalanges are more frequently affected with chronic rheumatic arthritis than any other of the articulations." Our own observations at the London Workhouse Infirmaries bear out this statement very fully; for amongst the numerous cases of undoubted osteo-arthritis which we saw in those institutions, there were very few in which these joints escaped, and in the majority of the cases they had been affected since the early stages of the disease. We saw very few cases in which the disease was limited to one or even to two or three joints. On the other hand, we have collected from English and foreign sources the records of 66 cases of joint disease associated either with well-marked symptoms of tabes dorsalis or with some of the symptoms of that disease, and we find that,
although joints as small as the phalangeal were diseased in one case, joints of smaller size than the knee and elbow were only diseased in 10 cases. In 41 cases only a single joint was affected. In 19 cases two joints were affected, and in only 6 cases were more than two joints affected. The total number of joints diseased in the 66 cases was 100, and of these the knees were diseased 45 times, the hips 24 times, the shoulders 13 times, the elbows 7 times, the wrist once, the tarsus 5 times, the metatarso-phalangeal joints 4 times, and the phalangeal once. From these figures it is evident that tabic arthropathy usually affects single joints, and rarely more than two in the same patient, and that it selects the larger joints in preference to the smaller. In distribution, therefore, we may conclude that there is a striking difference between osteo-arthritis and the joint-affection met with in association with locomotor ataxy.

In all the joints, which have been for a long time diseased in association with locomotor ataxy, we have found relaxation of ligaments and unnatural and free mobility. These two conditions contrast strongly with the opposite conditions, which are almost constantly met with in osteo-arthritis.

Reference to standard works on such a point as this cannot be conclusive, for it is only natural that diseases, so closely allied anatomically as are tabic arthropathy and osteo-arthritis, should have been confidently included under a single title at a time when locomotor ataxy was only beginning to be recognised. Adams, although he emphasises the fact that chronic rheumatic arthritis usually leads to restriction of movement, mentions cases in which there was unnatural mobility and relaxation of ligaments, and notably the case of J. Stafford, in whom first the left knee and subsequently both shoulder-joints became dislocated. He does not state symptoms from which it is possible to identify the cases as tabic, but it is impossible to read his description without being struck with the resemblance of the joints to those with which tabes is now found to be associated. That he refers to the painless character of the joint disease in these cases, and states that one patient died from a chronic disease of the bladder is suggestive.

In the discussion at the Clinical Society on Mr. Morrant Baker's paper, reference was made to a case of Mr. Stanley's,* in which so many of the clinical symptoms were given as to warrant the conclusion that the patient from whom the joint

was taken was suffering from locomotor ataxy. Mr. Shattock brought before the Society a hip-joint, contained in the University College Museum, which Mr. Richard Quain has described in his Clinical Lectures* as an example of rheumatic gout. The patient suffered from well-marked gastric crises, loss of sexual power, and progressive weakness in his legs. The clinical and pathological condition of the hip-joints correspond well with those met with in association with locomotor ataxy. If we are right in our opinion that Mr. Stanley’s case and Mr. Quain’s case were both instances of tabic arthropathy, it is highly probable that many of the cases classed as hydrops articuli would, if submitted to investigation to-day, be recognised as of tabic origin, or at least as occurring in association with tabes. This position is supported by our own observations at ten workhouse infirmaries in London. We could only find three cases, in addition to those which we classed as tabic, in which the joints presented relaxation of ligaments, and unnatural mobility. In one case the disease was limited to the knee-joint, and had come on quite gradually. The appearances of the joint were characteristic of Charcot’s disease. The patient said that eighteen months ago she suffered from aching pains in her left knee, and that six months later the joint began to swell, and that about the same time she began to suffer from shooting pains in both her legs. The knee-jerks could not be obtained, but no other symptoms of locomotor ataxy were present. The second case was an instance of poly-articular disease. The knee-joints were rigid, but the metacarpo-phalyngeal joints were loose and partially dislocated. In this case the only tabic symptom was absence of knee-jerks, but the rigidity of the joints appeared to afford sufficient explanation of their absence. The joints in the third case were similar to those just described, but there were no symptoms of tabes. A fourth case was brought under the notice of the committee by Mr. Thomas Smith. There was an affection of the hip-joint, which closely resembled Charcot’s disease, but no symptoms of locomotor ataxy were present. We attach some importance to the fact that during our study of these joint diseases, at least four of the cases came under our notice without our knowing or suspecting anything of the history or symptoms of the patients, and from our examinations of the joints alone we diagnosed Charcot’s disease, and found on investigation that many of the symptoms of tabes were present. We must also allow that our examination of the knee-

* P. 104.
Joint Disease in connection with Locomotor Ataxy. 275

joints above mentioned, in which there was distension of the joint, relaxation of the ligaments, and unnatural mobility, suggested the presence of nerve-symptoms. But although we have not classed this case as tabic, there are grounds, in the existence of shooting pains in the limbs and the absence of the knee-jerks, for suspecting that more definite symptoms of locomotor ataxy may occur later on.

With regard to the local pathological product in osteo-arthritis and in Charcot’s disease, we do not find much essential difference. The minute changes which have been noticed in the cartilage are similar in the two diseases. In the records of French cases the changes described are similar to those which characterise osteo-arthritis. This was the case in the joints which Mr. Baker has figured in the Transactions of this Society for 1885, and in a hip- and knee-joint, which we have since had an opportunity of examining. In many of the cases of Charcot’s joint-disease there is an abundant formation of osteophytic growth in connection with the bones and soft tissues of the joints, and in some there is eburnation of the exposed bony surfaces. In one of Mr. Baker’s cases, recorded in the Transactions for 1885, a comparison of the knee-joints strongly suggests the identity of the two diseases, so far as the local lesions of the cartilage and bone are concerned; for in one the signs of Charcot’s disease are well-marked, and in the other the appearances are typically those of an early stage of osteo-arthritis. But whilst we are prepared to allow that the local pathological processes which are at work in the joints in both diseases may be indistinguishable, we consider it unreasonable to thereupon conclude that the two diseases are identical. It would, in our opinion, be quite as reasonable to infer, because a final pathological product—a slough of soft tissues—may result either from an irritative lesion of a nerve or from the local action of a cautery, that the pathological process by which the sloughing is brought about in the two cases is identical. It is not sufficient to study the results of a disease; we must study its mode of eruption, its progress, and its associated conditions.

In osteo-arthritis the joint lesion most commonly comes on with painful swelling, and, after many exacerbations of the disease, it gradually in the course of years leads to destructive changes, which may permit of some unnatural mobility, but the movements in both natural and unnatural directions are limited and painful.

The joint affections which occur in connection with loco-
motor ataxy present a very different picture. In the 66 cases which we have collected 100 joints were diseased. In 79 of these the mode of onset is stated. In 56 it was sudden and in 23 it was gradual. In 26 instances of sudden onset the patients were under fairly continuous observation; in one of these, which we observed ourselves, the right shoulder-joint was disorganised and dislocated in four days after the onset of the swelling: in more than half the cases the joints were either dislocated or disorganised within periods varying from one to six weeks after the onset; of the remainder all but 3 were disorganised within four months, and those 3 were disorganised within nine months. Where the onset was not absolutely sudden, in the cases classed by us as gradual, the patients were not so continuously under observation, but in 2 cases disorganisation is recorded after periods of two months and eight months, and in 3 other cases—the only others in which dates are given—the joints were found disorganised within eighteen months.

The condition as regards pain at the time of onset is referred to in 54 cases. In 34 of these the onset was painless; in 20 it was painful, but in all but two of the latter series the pain rapidly subsided and the joints remained painless in all but two cases, up to the time of observation.

The last case which has come under our observation is most striking in its onset and its rapidity of the destructive changes. On April 19, 1887, this patient,* who had been the subject of locomotor ataxy for eighteen or twenty years, and had suffered a rapidly destructive lesion of his left shoulder-joint, was unaware that anything was wrong with his right shoulder, although we had noticed creaking in the joint three months previously. On the following day he had a most severe attack of lightning pains in his limbs and was very sick. On the 21st the right shoulder felt stiff, and on the 22nd he was unable to raise his right hand to his head. On the 23rd he examined his shoulder and found that it and the neighbouring parts of his chest were immensely swollen and discoloured, and that there was a great rattling in the joint when he moved his arm. On the 25th, four days after the stiffness of the joint was noticed, we found the right shoulder, the whole of the upper arm, and the adjacent parts of the chest enormously swollen, and in some places discoloured. The skin did not pit on pressure, the head of the humerus was dislocated beneath the coracoid process, and the limb could be

* Ziba Bickett, Case 6, p. 295.
vigorously moved and freely manipulated without causing any pain. Rattling noises and rough grating were produced by movement. The patient had had no injury of any kind before the onset of this affection of his right shoulder.

We believe that nowhere in the whole range of the pathology of joints can a lesion similar to this be found apart from locomotor ataxy.

The most characteristic condition in Charcot's joint disease appears to be the rapid wearing away of the opposed bony surfaces without the sclerosis of bone met with in typical cases of osteo-arthritis. Volkman has suggested that the cause of the rapidly destructive changes may be found in the oft-repeated strains to which the joints are subjected by the ataxic movements of the limbs. This may have been the case in some of the patients, in whom the joint lesion was of gradual production, but we do not think that such an explanation would be applicable to a case similar to that just referred to, in which disorganization of a shoulder-joint occurred in four days. In that patient there was no ataxy of the upper limbs, so, as regards it, Volkman's suggestion fails to the ground.

A more extended study of the relation of ataxy to disorganization of the joints leads to a similar conclusion. The date of onset of the ataxy is mentioned in 47 of the 66 cases which we have collected. The joint lesions occurred in the upper limbs in 10 cases, and in 9 of them there was no ataxy at the time of onset of the joint disease. In one case it had been present for two months before the onset of nerve-symptoms. The joints of the lower limbs were affected in 37 cases, and in 20 of these there was no ataxy at the time when the joint lesion occurred. In many of the cases the affected limbs had remained free from ataxy up to the date when the observation of the patient was recorded.

There is another and a more probable explanation of the rapidity with which the destructive changes in the joints occur in some cases, to be found in the atrophy of bone, which obtains in cases of locomotor ataxy. Charcot and others have laid stress on this fact, and the readiness with which fractures occur in ataxic patients is well known. This is illustrated by two of the joints, which have been exhibited at the Clinical Society, viz. by Professor Humphry's case of disease of the knee-joint, in which the joint lesion had occurred within three months of the patient's death, and by one of Mr. Baker's cases of affection of the knee. In both of these, portions of the tuberosities of the tibia had been broken from the rest of the
bone and displaced to a lower level, where they were supported by buttresses of new bone. It appears probable, therefore, that the rarefaction of bone which occurs in cases of Charcot's disease may be partly concerned in the causation of the destructive changes which often progress so rapidly in those cases.

It is difficult to form a positive opinion with regard to the relation which Charcot's disease bears to osteo-arthritis, but we think that we have shown that, although there are resemblances between the local results of the two diseases, there are differences in the distribution, mode of onset, condition as regards pain, and the rapidity of the disorganisation, and that these differences amply warrant the separation of tabic arthropathy from the group of joint diseases commonly known as chronic rheumatic arthritis.

The second question which we have proposed for consideration—whether Charcot's disease, as described by him, is ever found without the nervous symptoms usually regarded as characteristic of tabes dorsalis—involves a definition of the last-mentioned disease. The term "tabes dorsalis" or "locomotor ataxy" is applied to a chronic disease of the cerebral-spinal axis, which may vary considerably in its aspect according as other nervous structures besides the posterior columns of the spinal cord are or are not involved in degenerative changes; symptoms which are present in one undoubted case may be absent in another; the initial symptoms vary in different cases, as does also the rate of progress, which may be very slow or very rapid. We are of opinion, therefore, that the absence of many of the symptoms of locomotor ataxy in cases manifestly in their early stages, is not sufficient to warrant us in excluding that disease when some symptoms of it are present. It is possible that sometimes the occurrence of the joint lesions may bring the subjects of them under medical observation at an earlier date than happens with cases of locomotor ataxy, which are not complicated by so startling an accident.

We shall allude to some of the cases in which the symptoms are most irregular. F. Stokes, æt. 42, came under our observation six and a half months after the gradual onset of a destructive lesion of his right elbow-joint. The joint was a good instance of Charcot's disease, but the symptoms of locomotor ataxy were irregular. Gastric crises had been present for two years, sudden and uncontrollable desire to micturate for one year, and shooting pains in both upper and
lower limbs for six months. With the exception that the right hand felt as though it was "asleep," there were no disturbances of sensation; the pupils reacted to light, and in association with accommodation for near vision; the left knee-jerk was slight, but the right was excessive; there was no ataxic gait. Five months later an attack of neuritis affected the musculo-spiral nerve of the right side. Four months after that the pupils reacted better during accommodation for near vision than they did to light. The right knee-jerk was still brisk. Sixteen months later the left elbow, forearm, and hand swelled up, and in the course of a week became enormously enlarged. The skin was red and brawny, and pitted under firm pressure. Well-marked grating in the joint was caused by movement. The patient complained of a feeling of tightness about his elbow-joint, but movement and manipulation did not cause pain. In the course of a few days the swelling began to subside, and then a good deal of unnatural mobility could be detected. With the exception that the right pupil was quite inactive to light and the left contracted very feebly, the symptoms of locomotor ataxy were unchanged. Frederick Waters,* who was shown at a meeting of this Society by Mr. Barker, is an instance of the absence of the pupillary phenomenon, of anaesthesia, and of ataxic gait, and of the presence of one knee-jerk and exaggeration of the other occurring in association with a most typical instance of Charcot's joint affection. Gastric crises, atony of the bladder and incontinence of urine marked the onset of this patient's disease four and a half years previously. Attacks of lightning pains had occurred in his legs and abdomen during three years. Perforating ulcers had formed on his right foot from time to time during the previous two and a half years. All sexual desire and ability were lost. Other cases in which the symptoms of locomotor ataxy are irregular, and notably that of J. Taphouse,† whose case is fully detailed in the Appendix to this report, might be instanced as examples of Charcot's disease occurring in association with only some of the symptoms of locomotor ataxy; but, unless Mr. Smith's case, referred to earlier in this report, be an instance of it, we have failed to meet with a case in which the mode of onset, and the rapid disorganisation insisted upon by Charcot, has occurred without the presence of some tabic symptoms.

The third question—Is typical osteo-arthritis ever found

* Case 9, p. 302.
† Case 4, p. 292.
associated with any or all the symptoms usually regarded as characteristic of tabes dorsalis?—has been investigated by a study of the numerous instances of typical osteo-arthritis contained in ten of the large workhouse infirmaries in London. In only two cases have we met with any symptom that might suggest an affirmative answer. One of the cases was brought under our notice by Mr. Morrant Baker. The patient was the subject of well-marked osteo-arthritis of the knee; the knee-jerks could not be obtained, and the pupils were inactive to light. No other symptoms of tabes were present. In the second case, many joints were diseased, and one tibia was partially dislocated, but the joint was rigid, the pupils contracted well during accommodation, but not much to light; the knee-jerks could not be obtained, but the fixation of the joints and atrophy of muscles appeared to be a sufficient explanation of their absence. In all the other cases—a very numerous series—the pupils reacted to light, and either the knee-jerks were obtained or the contraction of the quadriceps extensor was felt when the patellar tendon was tapped. In Mr. Baker's patient, just referred to, there was no local condition to explain the absence of the knee-jerks; but if even we admit that both the cases were instances of commencing locomotor ataxy, they prove nothing when we consider the much larger number of cases in which no sign of that disease was found. Charcot admits it, and in the nature of things we can see no reason why true osteo-arthritis should not sometimes affect patients suffering from locomotor ataxy. If tabic symptoms were frequently found associated with typical osteo-arthritis, it would go far to prove that there was a generic relationship between Charcot's disease and osteo-arthritis; but such an association does not occur.

We propose, in conclusion, to review shortly, by the light of our own observations, and our study of recorded cases, the account of the disease as it is summarised by Professor Charcot. We have but little to add to, and but little to take away from, that accurate and guarded description.

Charcot states that the joint lesion in locomotor ataxy is always an early phenomenon, that it occurs between the prodromic period, when lightning pains are the only symptom, and the onset of incoordination, and that when the joint disease appears late in the disease it always affects the joints of the upper limbs. In general terms our statistics bear out this statement, but it is not a rule without exceptions.

In 47 cases, in which both the date of the onset of the
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joint lesion and that of the onset of the incoordination of movements of the affected limbs is mentioned, or in which incoordination is stated to be absent, we find that in 29 cases there was no incoordination at the time the joint disease began, and that in 18 cases there was incoordination.

An analysis of the presumed duration of the locomotor ataxy at the onset of the joint disease shows that in the lower limbs as many onsets occurred over five years as under five years, whilst in the upper limbs twice as many occurred over five years as under five years. In all the cases in which the joints of the upper limbs were affected early lightning pains had been present in them for periods varying from one and a half to three years. When the upper limb joints were affected late in the disease the attacks of lightning pains in those limbs had only occurred for a short time. In 6 cases joints of both the upper and lower limbs were diseased. In one of these the right elbow was affected two years before the right tarsus; but in this case the lightning pains had attacked the upper limbs as early as the lower. In the remaining 5 cases the lesion of the joints of the upper limb occurred after those of the lower. In 2 of these cases the interval was short (four months and one year), but in both of them the lightning pains had been present in the upper limbs for about the same time as in the lower.

It appears to be a fact, therefore, that the joint disease in tabes occurs most commonly in the limbs which have manifested the symptoms of locomotor ataxy for a short period only. Our statistics, however, show that the disease frequently occurs in the knees and hips between ten and fifteen years after the symptoms of locomotor ataxy commenced. In one case the knee became completely disorganised twenty-two years after the onset of lightning pains and ataxic gait.

We agree with Charcot's statement that the early prodromic symptoms in the joints are "crackling sounds." We observed these in a joint which three months later swelled up rapidly and became disorganised in four days. This patient also presented an unusual symptom, which is alluded to by Charcot as of rare occurrence, viz. fever (temperature 100°—102.5° F.) during the time the joint was greatly swollen.

Charcot's statement, that the onset is always sudden and in no way dependent on previous injury, is not completely borne out by our statistics. In 79 cases the mode of onset is stated; in 56 it was absolutely sudden; in 23 it was gradual. We have investigated the question of previous injury in 26 cases, and
in 8 of these the joint disease was attributed by the patients to an injury.

Charcot lays stress on the sudden swelling being due to hydrarthrosis and engorgement of the soft tissues without oedema. We have only observed three joints within the first few days of the onset, and in them the swelling of the joint and the neighbouring soft tissues was enormous. In two of these cases there was no oedema, but in the third case there was marked pitting under pressure. In all the cases which we have observed at later stages there was no oedema, although in many of them the limbs were much swollen beyond the region of the joints.

Charcot makes a guarded statement with regard to the absence of pain. He looks upon the painless course of the disease as characteristic, but allows that pain sometimes occurs. The figures which we have given in the early part of this report are in accordance with his assertion. In rather more than a third of the cases the onset was painful, but the pain rapidly subsided in nearly all of them, and at the time of observation they were painless even on rough manipulation.

By those who hold that there is nothing special in the joint lesion of tabes the painless character of the affection is explained by postulating an anaesthetic state of the joints dependent upon the lesion of the nervous system. We cannot accept this view until it be shown that there is in locomotor ataxy a special anaesthetic state of the joints quite out of proportion to the rest of the limbs, because in a number of the cases examined by us the affected joints were quite painless although a pin-prick of the skin was felt acutely.

We fully agree with Charcot's statement that the disease affects the large joints rather than the small, but we differ from his opinion that, in contrast with osteo-arthritis, the hip-joints enjoy a certain degree of immunity in tabes. He classes the joints as being affected in the following order of frequency: knee, shoulder, elbow, hip. Our statistics show the knees to be affected 45 times, the hips 24 times, the shoulders 13 times, and the elbows 7 times, in a total of 100 joints.

When speaking of the characteristic features of the joint lesions in locomotor ataxy Professor Charcot insists on the predominance of wearing away of bone over the production of bony burrs in recent cases, and mentions the frequency with which true dislocations occur in tabic arthropathy. The occurrence of true dislocations, that is without wearing away of bone, is
proved by some of the cases we have appended to this report, and perhaps best by that of B. W.,* which was recorded by Dr. Dreschfeld. The joint affected was the hip. The head of the femur was found dislocated on the dorsum ili, in one month after the patient took to his bed on account of a severe attack of pains in the affected limb. The head of the bone was freely movable and could easily be returned into its socket. It was not atrophied, and there was no deposit of new bone about the joint. Ziba Bickett† and M. A. Dowsett,‡ whose cases are reported in detail in the Appendix, are also instances of sudden dislocation. The shoulder was the joint affected in both, and in the first the dislocation was found in four days after the onset of the joint disease and in the second in fourteen days after. It seems likely that the rapid dislocations, which occurred in these patients, were caused by the great intra-articular pressure which resulted from effused fluid—that they were instances of dislocation by distension. Many cases might be cited to prove the predominance of wearing away of bone over the production of osteophytes, but it will suffice to refer to those of M. A. Dowsett‡ and J. Fare,§ in both of whom the head and tuberosities of the humerus had disappeared. The upper end of the shafts could be distinctly felt to have their natural size and shape. Both the hip-joints of the first-named patient presented analogous features, but the left elbow, wrist, and knee of the second patient were all much deformed by osteophytic growths.

Irregular attempts at repair are frequently made, but the extent to which these occur appears to depend on the amount of irritation to which the joint is exposed. Some repair may be found quite soon after the onset of the joint disease, and in Professor Humphry's specimen, to which we have already alluded, there is an example of it. The patient from whom it was taken died three months after the onset of the joint disease, but still there were evidences of repair. It should be borne in mind, however, that in this case one of the tuberosities of the tibia had given way, and that the new bone which formed as a support to the displaced fragment may be an instance of the abundant formation of callus which occurs around fractures in tabic patients. Joints which have been diseased for a long time are often greatly deformed by osteophytic growths. We cannot express our views on this subject better than by quoting Charcot's own words. He writes, "Un-

* See Table I, p. 336.
† Case 6, 295.
‡ Case 15, p. 310.
§ Case 2, p. 283.
doubtedly, in cases of old standing, when the articular surfaces, worn and deprived of cartilage, have continued to move on each other, the limbs being still made use of more or less imperfectly, the signs observed are those of dry arthritis, to wit, eburnation and deformation of the articular surfaces, deformation of the osseous extremities, bony burrs, and stalactites, foreign bodies, &c.  The cause of the relationship between locomotor ataxy and the joint disease which may complicate it is finally discussed by Charcot. He considers the disease to be of spinal origin and possibly dependent upon a lesion of the cells in the anterior cornua, but, after referring to facts in support of his view and facts in opposition to it, he says: "On the whole, the question relative to the precise seat of the spinal lesion remains yet to be decided." We believe that the cells of the anterior cornua cannot be justly accused of causing the joint lesion. Atrophy of the spinal cells was found in some of the earlier cases recorded, and it was noted by Dr. Hadden in a case described by him at the Pathological Society, but in other cases no lesion of these cells has been found. Again, destruction of these cells frequently occurs in infantile spinal paralysis without the occurrence of joint disease. It is true that muscular wasting often complicates the joint affections, and in a case recorded in the _Lancet_, July 12, 1884, wasting of the muscles of the leg and thigh soon followed the onset of the disease, first in one hip, and then in the other; but in many cases no such wasting occurs. The great muscular development of the upper limbs of Christina Metcalf was referred to by Dr. Buzzard at the Pathological Society in 1880, and in 1885 it was still very marked. The negative facts above referred to are sufficient to destroy the hypothesis that the joint disease is dependent on a lesion of the anterior cornua. Another theory, that of Dr. Buzzard, places the cause centrally, viz. in a hypothetical joint-centre, in the medulla oblongata. Dr. Buzzard has pointed out, in support of his view, the frequency with which gastric, intestinal, or laryngeal cases are met with in cases of locomotor ataxy complicated by joint lesions. He states that out of 100 cases of locomotor ataxy uncomplicated by joint disease, there were typical gastric sym-

* Diseases of the Nervous System, New Sydenham Society's translation, p. 56.
† Loc. cit., p. 60.
‡ Transactions of the Pathological Society, 1886.
§ Table I, p. 318.
|| Transactions of the Pathological Society, 1880, p. 207.
¶ Clinical Lectures on Diseases of the Nervous System, pp. 265, 266.
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ptoms in 10. Out of 48 cases of recorded tabetic arthropathy gastric crises were present in 24. We have investigated the frequency with which gastric crises occurred in 45 English cases. In 3 of them the symptom is not mentioned, and in 3 more it is doubtful whether the attacks of sickness were true crises. If these 6 cases be added to those in which there were no gastric symptoms, there still remain 50 per cent. of the total number, in which well-marked crises occurred. This proportion corresponds exactly with that previously announced by Dr. Buzzard.

Dr. Hadden* has reported a case in which there was some sclerosis in the neighbourhood of the pneumogastric nuclei, and in one of our own cases—that of John Griffiths†—Dr. Gowers believes there is some sclerosis about the pneumogastric nuclei, though the latter are themselves quite normal. We are not aware of any observation which is opposed to Dr. Buzzard's view, but until the medulla has been examined in a larger number of cases it can only be looked upon as a suggestive hypothesis.

Another explanation of the connection of Charcot's disease with locomotor ataxy may possibly be found in the presence of peripheral neuritis. In a specimen of disease of the ankle-joint shown at the Clinical Society (Transactions, 1885) by Mr. Henry Morris, the posterior tibial nerve presented marked signs of neuritis, but the terminal branches were not examined. In one of the cases recorded below (F. Stokes), an attack of neuritis in the musculo-spiral nerve supervened nearly twelve months after the onset of Charcot's disease of the elbow-joint. The musculo-spiral nerve, however, does not supply the elbow-joint.

In another of our cases (John Griffiths†) there were signs of disseminated myelitis in the lumbar region of the cord, but the lesion of tabes was not present, although the symptoms of locomotor ataxy were well marked. Dr. Gowers, who examined our sections of the spinal cord and medulla oblongata, suggested that the case might be one of peripheral tabes. Both sciatic nerves were probably normal. No examination of the peripheral nerves was made.

Pitres and Vaillard (Archives de Neurologie, vol. vi, p. 180), in a case of arthropathy of the metacarlo-phalangeal joint of the right index finger, found peripheral neuritis in the nerves corresponding to the joint. In their case there were also perforating ulcers of the foot.

* Loc. cit.  † Case 8, p. 299.
The frequency with which perforating ulcers of the feet occur in association with tabic arthropathy, lends some support to the view that the joint disease may be sometimes dependent on peripheral neuritis.

Mr. Baker has advanced the view that we have to do with a widespread disease which may either specially attack the nervous system and give rise to locomotor ataxy or attack a joint or joints and give rise to osteo-arthritis or attack both and cause Charcot's disease. This hypothesis would explain the occurrence of the joint lesion in some cases at a time when the symptoms of locomotor ataxy are absent or but slightly marked. The material at our disposal is not of a nature to warrant a definite conclusion on this point.

James Paget, Chairman.
W. Morrant Baker.
Thomas Barlow.
Thomas Buzzard.
Dyce Duckworth.
Warrington Haward.
Thomas Smith.
Bilton Pollard, Hon. Sec.

APPENDIX.

Full reports of seventeen cases not hitherto recorded.*

Case 1.—J. Saeger, female, æt. 57, shown at the Clinical Society by Messrs. J. R. Lunn and H. Larder, November 28, 1884. Report taken in March, 1885.

This patient was a native of Rotterdam; she married in 1853. She had lived in London since 1867. She was quite well until 1870, when she began to suffer from attacks of severe pain in both her knees. The pains lasted about five minutes at a time but they recurred after an interval. The complete attacks lasted two or three days. The pain was limited to the knees for one year, but at the end of that time it acquired a shooting character. The pain was limited for a time to a single spot in each leg and then it shot down both her legs and rapidly disappeared.

* Two other unrecorded cases (Gill and Jermy) appear in Table I, but as fuller details than there given could not be obtained, they are not included in this list.
In 1875 the patient noticed that she was unsteady in her gait and had to use a stick to walk with. In 1876 she fell down three steps and strained her left knee; the knee and thigh swelled up without pain. The limb was fixed on a splint; the swelling of the thigh soon disappeared, but patient had been unable to walk, even with crutches, since the accident on account of weakness in her legs. In 1880 the limb was put up in plaster of Paris and kept at rest for six weeks, but no improvement followed. In January, 1883, the patient was admitted to the St. Marylebone Infirmary. She was the subject of locomotor ataxy and her symptoms had been slowly progressive since then. During the last nineteen months she had had three attacks of severe pains in her arms. In October, 1884, she complained of a sense of constriction around her waist and of deep-seated boring pains at her epigastrium, and suffered from retching and vomiting. The attack lasted for a few days. She had been free from these symptoms during the last three months.

In March, 1885, the patient's condition was as follows:—She was well nourished and looked healthy. Her sight was good and her other special senses were normal. Her optic discs were healthy. She had slight difficulty in swallowing. Her pupils were equal and measured 2½ mm. in diameter; they did not react to light, but they contracted well during accommodation.

In the upper limbs sensibility was normal but coordination of movements were somewhat impaired. Tactile sensibility was much impaired on the abdomen and legs. Slight touches were not felt at all. The prick of a pin was felt, but only after a few seconds. Differences in temperature were distinguished. There was no wasting of muscles and no loss of power in them, but there was marked incoordination of movements, and the gait was very ataxic. When lying in bed the patient has no knowledge of the relative position of her legs. She said that she was obliged to respond quickly to the desire to pass water, but there was no actual incontinence of urine. The skin reflexes were diminished. Both knee-jerks were absent. There was no ankle-clonus. The left knee was much enlarged and the whole of the limb was swollen to a less extent, but there was no pitting on pressure. The ends of the bones entering into the knee-joint were enlarged, but there were no osteophytic growths. The left patella was broader than the right and was dislocated upwards and outwards. When at rest the left leg lay in a position of abduc-
tion and partial external dislocation. The movements of the left leg on the thigh were much freer and over a larger area than natural. Movements were accompanied by coarse grating but there was no pain.

There was some puffiness about the right knee, and some creaking was caused by movement. There was no laxity of the ligaments and no unnatural mobility.

Case 2.—J. Fare, male, æt. 45, under the care of Dr. A. H. Robinson at the Mile-end Infirmary. Report taken February 22, 1887.

The first symptom noticed by this patient was shooting pains in both his legs fifteen years ago. The pain sometimes occurred in his arms, but they were not so severe in them as in his legs. About ten and a half years ago he began to notice some difficulty in walking in the dark, and ten years ago he decidedly staggered. About ten years ago he had a sensation of pins and needles in his feet.

Just ten years ago (February, 1877) the patient was following his occupation as a sailor, and was at sea, when his left leg from the foot to the knee suddenly swelled up. He could not assign any cause for the affection, and he did not suffer any pain in connection with it. The swelling only lasted for about a week, and as it subsided a sudden painless swelling of his left arm commenced. At this time the patient experienced much more difficulty in walking than formerly. He went to the London Hospital in March, 1877. Blisters were applied to his left knee and elbow. After a stay of three weeks at the hospital he was discharged, and the patient states that at that time (less than three months after the onset of the affection) his knee and elbow were in just the same state as at present. In August, 1878, he had a severe bilious attack, and since that time he had frequently had similar attacks, for which he could not assign any cause. Latterly the attacks had recurred about every three months. For the last five or six years the patient had lost the power of telling the position of his legs without looking at them. For the last ten years he had been unable to write owing to disease of his finger-joints, but it was only during the last three years that he had noticed any difficulty in coordination of the movements of his hands. Sensibility in his hands had been impaired, or, as the patient says, lost for the last five years. He noticed no increase of sexual desire at the onset of his illness, but he had had neither sexual power
nor feeling during the last four or five years. During the last three or four years his bowels had been much constipated, and this symptom had been getting worse, so that now his bowels were never opened without an aperient. During the last three years the patient had had difficulty in micturition, and during the last two years the calls to pass water had been very sudden and urgent. His sight and colour perception had always been good. He stated that the ptosis of his left upper eyelid, and the left external strabismus, which he now has, came on when he was a boy. Since the onset of the disease in his left knee and elbow, his right shoulder, left wrist and the joints of both his hands had been getting affected quite gradually and painlessly. His fingers and left wrist began to get affected as long as nearly ten years ago (a month or six weeks after he left the London Hospital). The disease of his right shoulder commenced a little later. Nineteen years ago the patient had a venereal sore, but no definite history of secondary syphilis could be obtained.

The patient’s condition on February 22, 1887, was as follows:—His pupils were of medium size and did not react either to light or in association with accommodation. With the exception of ptosis of the left eyelid and left external paralytic strabismus, which dated from boyhood, there were no signs of affection of the cerebral nerves. Tactile sensibility in the middle and ring fingers was much impaired. It was impaired, though to a less extent, in the other fingers, the hands and forearms. Painful stimuli were not readily felt, but if of sufficient intensity to be felt there was no delay in the perception of them. Heat and cold were both distinguished, but more readily on the anterior than the posterior surfaces of the upper limbs. There was considerable loss of the power of coordination of the movements of the upper limbs. Tactile sensibility was lost in the feet and legs below the knees. The effect of painful stimuli was not tried, but the patient stated that a few weeks ago his sensibility was tested with a pin, and that, although he did not feel the prick at the time, he felt it severely about a quarter of an hour later. His perception of heat and cold was delayed for four seconds on the right leg, and for three seconds on the left. Heat and cold were both described as heat, but the hot spoon caused a more acute sensation than the cold one. The patient had great loss of the power of coordination of the movements of the lower limbs, and he was quite unable to tell the position in which his legs were lying without looking at them. The muscular power in
his legs was good. Both knee-jerks were absent. There was no ankle-clonus. The plantar reflexes were absent.

The patient suffered from severe gastric crises about every three months. His bowels were obstinately confined. The desire to micturate came on suddenly, and patient was then unable to hold his water. He had lost all sexual power. When the finger was lightly drawn over the skin large wheals gradually rose up, so that the patient's name, lightly traced out on his back, soon stood out in distinct wheals.

The right shoulder was much enlarged, owing to the presence of fluid in the joint. The upper end of the humerus was dislocated. It occupied most readily the position of the head of the bone in a subspinous dislocation, but the patient could voluntarily displace it beneath the coracoid process. There was a movable osteophyte in the capsule just beneath the acromion process, and there was a ledge of new bone projecting from the infraspinous fossa beneath the head of the numerus, when dislocated to that spot. It appeared to act as a support to the dislocated bone. The head of the humerus appeared to be a good deal absorbed, and coarse grating could be felt in the joints on movement. No loose bodies could be felt in the joint.

The left elbow was much enlarged and greatly deformed owing to the presence of two massive osteophytic growths occupying the position of the condyles of the humerus. The olecranon process lay between the two masses, and appeared of normal shape. Free unnatural mobility in all directions, and accompanied by coarse grating, could be obtained. The patient had considerable power of voluntary movement at the joint. There was no fluid in the joint. No loose bodies could be felt, and no bony growths could be detected in the capsule.

The left wrist was slightly enlarged, owing to fluid in the joint and to osteophytic growths attached to the dorsal surface of the carpal bones, and to the ulna. Coarse grating could be felt in the wrist, and the lower radio-ulnar joint. Some bony growths could be felt in the capsule.

The left knee was enlarged, chiefly owing to hypertrophy of the bones. There was but little fluid in the joint. The patella was not much enlarged. There was well-marked lipping of the borders of the condyles of the femur just as in the most typical cases of osteo-arthritis. The tuberosities of the tibia were much enlarged, and osteophytic growths projected from the bone. Coarse grating was produced by movements.
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of the tibia and patella against the femur. There was free lateral mobility, and antero-posterior sliding movements. The bones did not appear to be much eroded.

The fingers of both hands were distorted at all the joints, which allowed of much unnatural mobility. The appearances resembled those of poly-articular osteo-arthritis, with the exception of the increased mobility. The terminal phalanx of the right middle finger could be dislocated and reduced with the greatest ease. Grating could be felt in all the joints. Manipulation of all the affected joints was painless.

Case 3.—J. Smith, female, æt. 56, under the care of Mr. Marcus Beck at University College Hospital. Report taken in December, 1886.

The first symptom of locomotor ataxy noticed by the patient was a sudden attack of vomiting, for which she could assign no cause. The first attack occurred three years ago. At first the attacks recurred about every six months, but during the last year the interval had been reduced to four months. About the time of the onset of the gastric crises the patient noticed a feeling of "deadness," and of pins and needles in her right foot. About the same time, and since, she has always felt to be treading on soft things when walking. Lightning pains first occurred in her lower limbs two years ago. The pains lasted on and off for a week, and then passed away, but returned again after three or four weeks. The interval between the attacks had been getting shorter, and now they recur every week or fortnight. About fourteen months ago the patient fell on the inner side of her right knee. There was much immediate pain in the knee and the joint became gradually swollen. She attended as an out-patient at the Middlesex Hospital, where her knee was bandaged. She still continued her work, and after a fortnight the pain subsided, but the swelling continued and gradually increased up to the date of her admission to University College Hospital in October, 1886. She continued her work until seven months ago, but she was then obliged to give it up, as her knee was getting worse. About this time she noticed that she staggered in walking, but she is quite certain that she was not unsteady, and that she had no difficulty in walking, even in the dark, before the onset of the joint affection. Patient had never had diplopia, or perforating ulcers of her feet.

On December 18, 1886, the patient's condition was as follows:—Her pupils were small and equal. They did not
react to light, but contracted in association with accommodation for near vision. The patient complained of a feeling of numbness, and of pins and needles in her right foot, but tactile sensibility in her feet and legs was fairly good. Painful stimuli were at once appreciated. Cold was rapidly appreciated in legs. Heat was felt rapidly as cold, and after a delay of one and a half seconds it was felt acutely as heat.

Sensibility in upper limb was normal. The patient walked with a hesitating and unsteady gait, but there was no marked ataxy. She could not stand steadily with her eyes shut. Coordination of the movements of her upper limb was good. Both knee-jerks were absent. Plantar reflexes were brisk. Patient had sudden calls to pass water, and sometimes her urine flowed involuntarily. The right knee was swollen, chiefly due to the presence of fluid in the joint. The lower end of the femur, the upper end of the tibia, and the patella, were much enlarged. There was a rounded mass of new bone attached to the posterior part of the upper end of the tibia. There was no marked wearing away of the articular ends of the bones. Coarse grating could be felt during movements at the joint. The ligaments of the joint were much relaxed and there was free lateral mobility at the joint. The movements at the joint were unattended with pain.

During the three months the patient was in the hospital, she had two well-marked gastric crises, and repeated attacks of lightning pains in her lower limbs.

Case 4.—J. Taphouse, male, æt. 39, under the care of Dr. T. D. Savill at the Paddington Infirmary. Report taken December 14, 1886.

The first symptom noticed by the patient was the formation, three years ago, of corns on the balls of both his little toes. That on the right toe broke, and a wound formed. Other wounds formed beneath the first phalanx of both his big toes. They healed up in the course of a few weeks, but often broke open again. For the last three months they had been healed. Two years and nine months ago, without any injury or obvious cause, patient’s left knee swelled up, and in three days reached a large size. There was some redness of the skin, and pain in the joint at first. There was no swelling of either the thigh or the leg. After resting the knee for a few weeks the swelling subsided, but the joint was left disabled. The swelling had returned from time to time since, and the joint had been getting weaker. The patient had suffered from
aching "rheumatic" pains in his legs, but he had not had any typical lightning pains. He had not had any marked gastric crises, but had suffered from "attacks of the bile." These attacks had been unattended with pain, and had not impressed the patient much. Both knee-jerks were found absent by Dr. Savill in July and August, 1886. They were present, though impaired, in September, 1886. The patient had not had syphilis.

On December 14, 1886, the patient's condition was as follows:—The pupils were unequal, the right being larger than the left. Both pupils reacted very badly, if at all, both to light and in association with accommodation for near vision. Tactile sensibility appeared to be fairly normal in both limbs, and in the face, but on all these parts the prick of a pin did not cause pain, although it was stuck deeply into the skin of the legs and forearms. The patient's gait was not ataxic, and he could move both his upper and lower limbs with precision. He could stand steadily with his eyes shut and his feet close together for one minute, but after that time he swayed a little. Plantar reflexes were brisk. Both knee-jerks could be readily obtained. The patient experienced sudden calls to micturate and occasionally he had an involuntary action of his bladder. His sexual power, which had never been excessive, was not impaired. He suffered from constipation of his bowels.

His left knee-joint was enlarged. There was no fluid in the joint, but the internal tuberosity of the tibia and the condyles of the femur were enlarged, and the borders of the latter were distinctly lipped. The patella was not enlarged. Coarse grating was felt on moving the joint, and there appeared to be a number of vegetations within its cavity. No bony growth could be detected in the capsule. There was no displacement of the bones, but there was well-marked lateral mobility at the joint.

Case 5.—M. Winder, female, æt. 58, under the care of Dr. S. H. Moore at the Chelsea Infirmary. Report taken December 16, 1886.

About eight years ago this patient's right knee began to swell gradually without any injury or other known cause. A little later her left knee began to swell. There was no pain in either of the joints, either at the onset of the affection or during its further progress. Both joints had been getting gradually weaker, and their ligaments more and more relaxed up to the present time. About six years ago they got so dis-
abled that the patient was obliged to use crutches, and she has continued to use them ever since. About two years after the onset of the joint affection she began to suffer from pains which darted across her knees, but especially the left one. About the same time she began to suffer from darting pains in her legs. These pains were and have continued to be much worse in her left leg. They came on especially with the onset of wet weather, and usually lasted for two or three days at a time. The above symptoms were the only ones of which she was conscious until about a month ago, when she noticed numbness and a feeling of pins and needles in the sole of her left foot. She had had no gastric crises, and no trouble with her bladder or rectum and no perforating ulcers of her feet. She had not had syphilis.

On December 16, 1886, the patient's condition was as follows:—Her pupils were of medium size and equal. They contracted readily in association with accommodation for near vision, but reacted very slightly if at all to light. She complained of a feeling of numbness in the sole of her left foot. Tactile sensibility was impaired in the left foot and leg. The prick of a pin was only indistinctly felt in the sole of her left foot and left leg. The sensibility of the right lower limb was perfect. The contact of a cold spoon with the left leg was not perceived, and that of a hot spoon was described as a faint prick of a pin. It was not painful, but when it was applied to the right leg it was at once felt as heat, and described as painful. The perception of stimuli which were felt, was not appreciably delayed. The sensibility of the upper limbs was normal. The patient was much crippled, but still it was plain, when she walked with the aid of crutches, that her gait was ataxic. Her legs were thrown a little outwards, and her heels were brought down sharply to the ground. When standing with the aid of crutches she at once became unsteady when she closed her eyes. Her upper limbs appeared normal in all respects. The plantar reflexes were present. The knee-jerks were absent. The functions of the bladder and rectum were normal. The skin of both legs, but particularly of the left, was dry and scaly.

The left knee was much enlarged owing to a great increase in the size of the opposed ends of the femur and tibia, and to the presence of fluid in the joint. Massive osteophytes projected from the bones, particularly from the back of the tibia, and others could be felt in the soft tissues of the joint. The internal condyle of the femur appeared to be much worn.
about. There was no dislocation, but there was very free lateral mobility at the joints, and when the patient stood up, the left leg bent outwards and formed an angle of $135^\circ$ with the thigh. Coarse creaking could be felt in the joint during movement.

The right knee was affected in a similar manner to the left, but the disorganisation of the joint was much less marked.

Case 6.—Ziba Bickett, male, æt. 53, a dock-labourer, under the care of Mr. Rickman J. Godlee at University College Hospital. Report taken on June 8, 1886.

He states that he had been a very healthy man. He contracted gonorrhoea when he was twenty years of age, but he had not suffered from syphilis. His younger brother suffers from locomotor ataxy, but has not any joint disease. The patient attributes the disease to the great exposure to wet to which he and his brother had been subjected. His other brothers had not been so exposed, and did not suffer from locomotor ataxy.

For the last twenty years this patient had suffered from attacks of morning vomiting, which came on directly he got out of bed. The attacks were unattended with pain or even with discomfort. Sometimes instead of the vomiting he had a sudden attack of diarrhœa. During the last eighteen months the attacks of vomiting had been much less frequent. They had only occurred five times. During the last eight years the patient had been troubled with frequent and loose action of his bowels. He has regularly three actions of his bowels during the day, and sometimes as many as five. The desire to go to stool comes on very suddenly, but beyond the discomfort arising from this urgency he has no pain in connection with defecation. During the last eight or nine years this patient had been troubled with sudden calls to pass water, and had experienced much difficulty in holding it after the onset of the desire to pass it. About seventeen years ago he began to suffer from "rheumatic" pains in his legs. It is not possible to separate these pains from the true lightning pains, which were most severe eleven years ago. The pains came on suddenly, e. g. after the slamming of a door or the rattling of a passing cab, and if severe they lasted about twenty-four hours, but if less severe they often lasted several days. About two years ago lightning pains began very severely in his arms. They had been confined to his elbows, wrists, and hand till
four or five months ago, when he first felt them in his left shoulder. He had not suffered from pains in his right upper limb higher than his elbow. About ten or twelve years ago he noticed difficulty in walking in the dark or twilight. At about the same date an internal squint came on in his right eye.

About four or five months ago patient fell through a distance of four feet over a ship's side and struck his left shoulder, but no damage to the joint resulted from the fall. About two months later the whole of the left upper limb turned black and blue without any known cause. Four days ago he fell whilst going downstairs and again struck the left shoulder. He did not suffer from pain in the joint at the time, but during the night his left shoulder began suddenly to swell, and for three hours was the seat of severe pain. After that time the pain subsided, and the patient had been quite free from pain in the joint since.

On June 8, 1886, the patient's condition was as follows:— Both pupils were very small and of equal size. They did not react to light, but a distinct contraction occurred during accommodation for near vision. The right eye was turned strongly inwards, and the patient was unable to turn it outwards. Colour perception was normal. The sensibility of the feet and legs was impaired both to tactile and painful impressions. Heat and cold were felt and readily distinguished. The perception of the sensation produced by pricking the feet was delayed for one and a half seconds. There was neither anaesthesia nor analgesia of the upper limbs, but the perception of stimuli originating in the hands was delayed three quarters of a second. The patient had a well-marked ataxic gait, and was quite unable to stand with his eyes shut. The movements of the upper limbs could be executed with precision. Both knee-jerks were absent. Sexual power was totally lost. The left shoulder was greatly swollen, and the swelling extended down the upper arm as far as the elbow. The swollen parts could not be indented with the finger. There was neither redness of the skin over the shoulder or tenderness on manipulation of the joint. The movements of the joints were impaired and attended by creaking.

On December 14, 1886, the patient's general condition was unaltered, but the head of the left humerus was found dislocated beneath the coracoid process. It could be readily reduced, but could not be maintained in position. Coarse grating was felt during movements at the joint. There
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appeared to be little or no effusion in the joint, and there was no swelling of the soft tissues. No osteophytic growth about the joint could be detected. Manipulation of the joint was perfectly painless.

The right shoulder was the seat of coarse grating during movement, but it was otherwise normal.

On April 19, 1887, the patient was not aware of any alteration in the condition of his right shoulder. On the following day he had a most severe attack of lightning pains in all his limbs, and had an attack of sickness. The attacks of sickness and the lightning pains had been very severe for some weeks previously. On the 21st the patient noticed that his right shoulder was stiff, and on the 22nd he was unable to raise his right hand to his head. On the 23rd he examined his shoulder and found that it, his upper arm, and the neighbouring parts of his chest were greatly swollen, and that there was a great rattling in the joint when he moved his arm.

On the 25th the right shoulder and the whole of the upper arm were greatly swollen. The front of the chest and the axilla were much swollen, and in parts the skin was discoloured, apparently from extravasated blood. The skin did not pit on pressure. The head of the humerus was dislocated beneath the coracoid process. The limb could be vigorously moved and freely manipulated without causing any pain. Rattling noises and rough grating were produced by movement. The patient had had no injury of any kind before the onset of this affection of his right shoulder.

The condition of the left shoulder was the same as on December 14, 1886. There was some loss of coordinating power in the upper limbs.

Case 7.—E. Bewry, female, æt. 48, under the care of Mr. Berkeley Hill at University College Hospital. Report taken in February, 1886.

About fourteen or fifteen years ago this patient began to suffer from neuralgic pains, which shot down her arms and legs and around her body. She was not conscious of any other symptoms of disease, and is quite sure that she had no difficulty in walking until eight years ago, when on returning home after a long day's washing she noticed that her right knee was swollen, and that it felt as if it would give way. Her leg as low as the middle of the calf was also much swollen. Her private doctor stated it as his opinion that a blood-vessel had burst into the joint. The joint had not sustained any
injury, and no cause could be assigned for the sudden swelling of her knee. After a few days' rest the swelling subsided, but the patient noticed that her knee creaked when she moved it. The joint was rather painful whilst it was swollen, but the pain soon disappeared. Since the first onset of the joint affection the knee had often swelled up, and a few days before her admission to University College Hospital it became more swollen than it had ever been before. A few years ago the patient suffered from diplopia, but this symptom disappeared under tonic treatment. Twelve months ago she had a violent attack of sickness, which lasted for about a week. During the last year she had often fallen down in the dark. There was no history of syphilis.

On February 3rd, 1886, the patient's condition was as follows:—Her sight was good with the aid of glasses. Her colour perception was normal. She had no diplopia. Her pupils were small and of equal size. They contracted during accommodation for near vision, but did not react to light. Tactile sensibility in both feet and legs was normal. The prick of a pin could be felt in both legs, but not so acutely as it ought to be, and the perception of the sensation was delayed two seconds after the stimulation. The patient was unable to distinguish with certainty between a hot and a cold spoon applied to her legs. She could move her legs with precision, but when standing up she began to sway to and fro as soon as she closed her eyes. Her gait was ataxic. The plantar reflexes were present, but both knee-jerks were absent. There was no affection of either the motor or sensory apparatus of the upper limbs.

The right knee and leg were a good deal swollen. The swelling of the leg was due largely to oedema of the superficial tissues, and that of the knee to great distension of the joint-cavity with fluid. Over the inner and posterior aspect of the internal tuberosity of the tibia there was an enlarged bursa which communicated with the cavity of the joint. There were free lateral and antero-posterior sliding movements at the knee. The lower end of the femur was partially dislocated backwards as the patient lay in bed, but it could be readily moved forwards into its proper place. The right patella measured three quarters of an inch more across than the left. The circumferential measurement of the limbs at the level of the upper end of the tibia showed an increase of four and a half inches on the right side as compared with the left. This increase was partly accounted for by the swelling of the soft
tissues and by the bursa, but the bone was distinctly enlarged. The lower end of the femur was enlarged, but of normal shape. The extent of the enlargement could not be made out owing to the fluid that surrounded it. There were no large osteophytes, but a little irregularity along the border of the upper end of the tibia could be made out. A soft creaking could be felt in the joint during movement. No loose or pedunculated bodies could be detected within the joint.

Case 8.—J. Griffiths, male, æt. 43, formerly under the care of Mr. Marcus Beck at University College Hospital. Report taken in April, 1885.

Towards the end of the year 1877 this patient began to experience a feeling of pins and needles in his feet. A year later he began to suffer from shooting pains in his legs. The pains were not very severe, but the patient was never free from them for more than a day or two at a time. During the last two or three years he had had no pains in his legs, but about two years ago he suffered from shooting pains in his shoulders. Towards the end of 1879 his right ankle began to swell quite suddenly, and a month later his right knee and the whole of his right lower limb swelled up. The swelling lasted six weeks and then disappeared, except about his knee, which had remained large ever since, and had steadily got more deformed and weaker. About two years ago the patient noticed creaking in his left hip-joint, and about a fortnight later his left leg and thigh began to swell. The general enlargement soon disappeared, but a small swelling was noticed in connection with the horizontal ramus of the right pubic bone, and a second in connection with the right ilium just below the anterior part of the crest. The swellings increased in size gradually until July, 1884, when for a time they enlarged more rapidly. In the early part of his illness the patient had an increase of sexual desire, and in July, 1884, erection of the penis was induced by slight irritation, such as the sudden exposure to cold air, as by the removal of bed-clothes. His urine was frequently passed involuntarily at this time.

In April, 1885, the patient's condition was as follows:—His pupils were small and of equal size. They did not react to light, but they contracted when the patient accommodated for near vision. He could not read small type without a strong light. He had no diplopia.
There was no affection of either the sensory or motor apparatus of the upper limbs.

Tactile sensibility in the lower limbs appeared to be perfect, but the prick of a pin did not evoke pain in the lower half of the right leg or in the lower third of the left one. He could usually distinguish heat from cold, but he sometimes called the touch of a hot spoon the prick of a pin. When touched with a cold spoon he was aware that the surface touched was of some size. Perception of sensation was delayed about one second. The patient could stand and walk a little without support. His gait was not ataxic. He walked unsteadily, but the joint affection amply accounted for that. The plantar and abdominal reflexes were exaggerated. Both knee-jerks were absent. There was no ankle-clonus. The patient occasionally had a sudden call to pass water, and sometimes his urine flowed involuntarily.

His right knee was enormously enlarged. The skin over it was normal, but the superficial veins were enlarged. There was very little fluid in the joint. The tibia was dislocated outwards, and slightly backwards. The ligaments of the joint were relaxed, and when the patient stood up his tibia was still further displaced backwards. There was slight lateral mobility at the joint. The opposed ends of the femur and tibia and the patella were much enlarged. The upper end of the tibia appeared to be dilated into a cup-shaped extremity. The border of it was smooth and free from osteophytic growths, except at the upper part of the external tuberosity, from which there was a bony outgrowth. The circumferential measurement round the middle of the right knee showed an increase of 6½ inches as compared with the left knee. At a distance of 4 inches above the patella the increase was 2½ inches. The right patella was an inch higher than the left.

The left hip-joint appears to be quite disorganised. The trochanter is not enlarged, but it is placed at a higher level than natural. On the outer surface of the anterior part of the ilium there is a large, hard, and apparently bony growth, which extends from the iliac crest to level of the great trochanter. The skin over it is normal, but the subcutaneous veins are dilated. On the inner surface of the horizontal ramus of the pubes there is another bony growth which extends outwards to near the anterior superior iliac spine, and inwards to near the symphysis pubis.

This patient killed himself five months after the date of this report by drinking strong nitric acid. The central nervous
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system, the sciatic nerves, and the two diseased joints were obtained. Numerous sections of the medulla oblongata, the spinal cord, and the nerves were made. The lesion of tabes dorsalis was not present in the spinal cord, but there appeared to be a little overgrowth of connective tissue in the posterior columns of the cord in the lumbar region. The fibrous tissue of the neurilemma and the endoneurium of the sciatic nerves appeared to be excessive, but there was no increase of fibrous tissue in the funiculi of the nerve. Some of the sections were submitted to Dr. Gowers, who reported upon them in the following words:—"I think there is a little sclerosis in the medulla, at the level of the pneumogastric nuclei, around the fibres of origin of the pneumogastric nerve, and between the ascending root of the fifth and the slender column (ascending root of the lateral mixed system), but the fibres of the pneumogastric and all its nuclei, and the slender column itself, are all normal. The cord does not present the lesion of tabes. In some parts it is quite normal. The changes in the lumbar region are dubious. They suggest disseminated myelitis rather than tabes. I doubt whether the sciatics are abnormal." Dr. Gowers suggested that possibly the case was one of peripheral tabes.

The right knee: The capsule of the joint was much enlarged, and the cavity of the joint contained about ten ounces of slightly turbid yellow liquid. Very free movement of the bones was possible in all directions. There were numerous villous projections from the inner surface of the synovial membrane. Some of the processes were very large, and contained nodules of bone. There were three pedunculated bony masses about the size of chestnuts. The patella was much enlarged, and its articular surface was very irregular. In the situation of the ligamentum patellae there was an irregular bony mass. The interarticular ligaments and cartilages had disappeared. The lower end of the femur was enlarged, but it retained its natural shape. The articular cartilages had disappeared, but the peripheral parts of the articular surface were covered in some places by a coarse velvet-like tissue, and in others by a smooth bossy material, having the naked-eye appearances of dense fibrous tissue. The microscopical appearances of this tissue were those of fibrous tissue containing a few cells. No cartilage could be seen. The exposed surface of the condyles was uniformly smooth, but it did not present any patches of eburnation. The upper surface of the tibia was divided into two unequal parts by a ridge which corresponded to the inter-
val between the condyles of the femur. The outer tuberosity was much enlarged by an outgrowth of bone, which projected upwards from it, and embraced the outer surface of the outer condyle of the femur. There was no articular cartilage on the joint surface of the tibia, and the latter was uniformly smooth though not eburnated. The osteophytic growths were everywhere covered on the joint surface by dense fibrous tissue. There was slight lipping of the borders of the articular surface of the femur, but not of that of the tibia.

The left hip: The joint-cavity was much enlarged, and the posterior part of it was supported by a large plate of bone which appeared to have been formed in the tensor vaginae femoris, and the two lesser glutæi muscles. The capsule was greatly thickened. Numerous pendulous vegetations projected from the synovial membrane. The head and much of the neck of the femur was worn away, and the exposed surface was covered by shreds of fibrous tissue. The acetabulum could not be recognised, for it and the adjacent parts of the dorsum iliæ formed a large surface, within the attachments of the capsule, against which the eroded head of the femur had played. There were no osteophytes in connection with the upper part of the femur, which, with the exception of the great wearing away of the head of the bone, appeared normal. There was a large mass of rather dense bone situated apparently in the substance of the iliæ muscle, where it lay over the brim of the pelvis.

Case 9.—F. Waters, male, æt. 39, shown at the Clinical Society November 28, 1884, by Mr. A. E. Barker. Report taken in April, 1885.

This patient's illness commenced four and a half years ago with sudden attacks of sickness, accompanied by abdominal pain, situated especially about the pit of his stomach. The attacks of sickness came on most frequently in the morning. He frequently vomited his breakfast, but rarely his dinner. The attacks had been getting less frequent. About the same time he began to be troubled with difficulty in passing water, and to suffer pain above his pubes and along his urethra. He was sometimes troubled with incontinence of urine. He had suffered from lightning pains in his abdomen and both his legs during the last three years. He still suffered from shooting pains in both legs, but especially in the left one. The attacks came on at intervals of a few weeks, and lasted for twelve or twenty-four hours at a time. They were worse in damp and
cold weather. About two and a half years ago the patient had what he terms an "ague fit," and he then first noticed that his right foot, which had already been rather sore, was swollen and red. On the following day he was unable to get his boot on on account of the swelling, and he noticed that an ulcer had formed beneath the ball of his right little toe. This healed in five weeks, but during the next two years he had four other ulcers, and from one of them on his right big toe some pieces of bone were discharged. In July, 1884, his left knee, thigh, and leg gradually swelled up, and in the course of three weeks the knee-joint became disorganised. He had slight pain in the joint at the onset of the disease, and he suffered from attacks of sickness at the same time. He had lost all sexual power during the last year. About twelve years ago he had a chancre on his penis, but it had not been followed by any secondary symptoms of syphilis.

In April, 1885, the patient's condition was as follows:—There were no signs of disease of his cerebral nerves. His sight was tried, and he could distinguish colours readily. He had no diplopia. His pupils were of medium size. They reacted well to light, and in association with accommodation. Both the sensory and motor apparatus of his upper limbs were normal. Tactile and painful stimuli were readily appreciated in his legs and the soles of his feet. There was no delay in perception of sensation. Heat and cold were much confounded, especially on the inner side of the left leg, and on the left foot. The muscular power of his lower limbs was good. His gait was not ataxic, and he had no loss of muscular sense. The plantar reflexes were exaggerated. His left knee-jerk was normal, and his right one appeared to be excessive. There was no ankle-clonus. The patient had to strain a great deal in order to pass his urine. The urine occasionally dribbled away. All sexual desire and ability were lost. The skin of both legs and the soles of both feet was rough and dry. The epithelium on the soles of both his feet was very thick and hard. There was a deep cleft on the ball of his right little toe at the place where the ulcer had been.

His left knee was swollen, mainly owing to fluid in the joint, but also due to enlargement of the upper end of the tibia, and also, though to a less extent, of the lower end of the femur. There were no osteophytic growths. The ligaments were relaxed, and allowed of the bones being separated from one another, and being knocked together so as to cause a distinct sound. There was free lateral mobility at the
joint. There was no pain in the joint. There was well-marked creaking in the joint. The circumference of the knee at the middle of the joint was $4\frac{1}{2}$ inches more than that of its fellow. The circumference at the level of the tuberole of the tibia was $2\frac{1}{2}$ inches more than on the opposite side. The left patella was $\frac{1}{2}$ inch broader than the right one.

On June 15, 1885, the general symptoms were unchanged. The gastric crises had again become more frequent and more severe. Two perforating ulcers had reformed on the sole of his right foot. The condition of his knee was as above described, with the addition of an osteophytic growth from the posterior part of the external tuberosity of the tibia, and some thickening of the tibia and fibula at the points of insertion of the hamstring tendons.

This patient died in February, 1886. No post-mortem examination was obtained.

Case 10.—J. Pickford, male, æt. 60, shown at the Clinical Society by Mr. Rickman J. Godlee, November 29, 1884. Report taken April 15, 1885.

This patient's illness commenced sixteen years ago, with shooting pains in his thighs and legs, and soon afterwards in his arms as well. The attacks at first recurred about every month, but they gradually became more frequent, and now a day rarely passes without an attack of pain. The patient had never suffered from gastric crises. In 1870 he noticed that he saw double, and three years later it was noticed that his right eye turned inwards. In December, 1872, the fingers of his right hand became contracted, and on this account he attended as an out-patient at the Hospital for the Paralysed and Epileptic in Queen's Square for three months, and at the end of that time his hand was well. Soon afterwards the ring and middle fingers of his right hand became contracted, and a little later the corresponding fingers of his left hand got similarly affected. Three and a half years ago the patient's left thigh suddenly swelled up enormously. He did not suffer from pain in the joint, and movement of the limb was unattended with pain. He was suffering from severe shooting pains in his limbs at the time. After three weeks' rest he again began, though with great difficulty, to get about again. The difficulty in walking had remained ever since. In 1881 he noticed that his urine occasionally dribbled away, and that he sometimes experienced a sudden desire to micturate soon
after he had passed water, and when he was unable to pass any more. The urinary symptoms had persisted. He had noticed no alteration in his sexual functions throughout his illness.

On April 15, 1885, the patient's condition was as follows:—He could not see to read except in a strong light. There was a well-marked internal strabismus of his right eye. He could hear the ticking of a watch at a distance of four inches from his head on the right side, but on the left he could not hear it at contact. Both pupils were moderately contracted. They were sluggish, but they contracted to light, and in association with accommodation for near vision. His feet often felt cold, but never numb. Tactile sensibility in his lower limbs was normal. The prick of a pin did not cause pain in his left foot or in the lower third of his left leg, though it did so in the right lower limb. The plantar reflexes were present, but the knee-jerks were both absent. Patient's gait was not ataxic. There was no affection of either the sensory or motor apparatus of his upper limbs.

His left hip was much enlarged, and the superficial veins over it were enlarged. The swelling was due to enlargement of the great trochanter, which extended two inches above Nélaton's line. The horizontal ramus of the pubes was enlarged, and there was a long spicule of bone which extended from the ilium just below the anterior superior iliac spine for four inches down the front of the thigh in the substance of the muscles. No loose bony fragments could be made out, and no osteophytic growths from the bones entering into the joint could be detected. The movements of rotation at the joint were limited, and all the movements were attended with coarse grating.

The ring and little fingers of both hands were affected with Dupuytren's contraction.

Case 11.—Elizabeth Neale, æt. 53, shown at the Clinical Society, November 14, 1884, by Mr. A. A. Bowlby. Report taken in April, 1885.

In 1860 this patient began to notice difficulty in walking in the dark. In 1861 she had an attack of diplopia which lasted for several months. In the same year her gait became ataxic and she began to suffer from lightning pains in her legs. The attacks of pain lasted for two or three days and the interval between the attacks was usually about three weeks. In 1879 she began to suffer from attacks of sickness accom-
panied by epigastric pain, and about this time the lightning pains were more severe. In September, 1882, she had a fit on two occasions in which she lost consciousness. She also lost the use of both her legs for some weeks. Power gradually returned and she was again able to walk. In August, 1883, her right knee became suddenly greatly swollen. It quickly became very loose, and in a few months she was quite unable to walk on it. It had caused her no pain at any time. The patient was in St. Bartholomew's Hospital for six weeks of July and August, 1884, and during that time the swelling of her knee subsided but the joint became deformed. The deformity had been increasing up to the date of this report. The lightning pains and the attacks of vomiting had continued, but latterly they had not recurred so frequently and had not been so severe.

In April, 1885, the patient's condition was as follows:—Her pupils were of medium size. They did not react to light, but they contracted during accommodation for near vision. She sometimes suffered from shooting pains in her face of a kind quite similar to those she had in her legs. She had slight involuntary twitchings of the muscles of her face and neck. Her speech was slow and measured.

Sensibility in her upper limbs was normal. There was some impairment of coordination of movements in her upper limbs. She had occasionally shooting pains round her abdomen. Ordinary tactile sensibility in her feet and the lower half of her legs was lost and in the upper half of her legs it was diminished. The prick of a pin was only indistinctly felt in the upper half of her legs and in the lower half it was only felt as a touch. Neither tactile nor painful sensations were produced by pricking the soles of her feet. She could distinguish between heat and cold, but sensation was delayed, and for a longer time after stimulation of the right leg than the left. The soles of both feet were sensitive to heat-stimuli, but perception of the sensation was delayed for six seconds. The patient had considerable power in her legs, but she had lost the power of coordination. The plantar reflexes and the knee-jerks were both absent. Micturition was difficult and painful. The patient had frequent desire to defecate.

The right knee-joint was completely disorganised. The tibia and fibula were dislocated backwards into the popliteal space. The upper and anterior part of the tibia was worn away and the lower and posterior part of the femur was worn in a corresponding manner. The lower end of the femur formed a prominence at the front of the knee. The patella
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was displaced so as to lie above and on the outer side of the condyles of the femur. There was free lateral mobility at the joint. Well-marked creaking was produced by moving the joint, but no bony grating could be detected. There was no fluid in the joint. The joint could be moved freely and the bones could be knocked against one another without causing pain. No osteophytic growths could be felt about the ends of the bones. The right patella was a quarter of an inch broader than the left, and the right femur was a little larger than its fellow.

Case 12.—M. Hollis, female, æt. 47, shown at the Clinical Society by Messrs. J. R. Lunn and H. Larder, November 28, 1884. Report taken in March, 1885.

The patient married at sixteen years of age. Her first child was born one year later. One child is living aged 32, and two died young—one from phthisis and one from convulsions. Two were stillborn.

The patient was quite well until nine years ago, when she began to suffer from shooting pains in her epigastrium, and shortly afterwards in her legs. About the same time she experienced great weakness in her legs, and they often gave way under her. She had difficulty in walking in the dark. The shooting pains had gradually increased in severity. The duration of the attacks and the frequency of their recurrence had also increased. They now last for two or three days, and they recur sometimes weekly and sometimes after an interval of two or three weeks.

Rather more than two years ago she was told at the Welbeck Street Dispensary that she was suffering from locomotor ataxy. In March, 1883, she was admitted to the St. Marylebone Infirmary, and since that time her symptoms had become more and more marked. She had never suffered from gastric symptoms.

About two years ago her left knee and the adjacent parts of her thigh and leg swelled. About six months later she noticed a crackling sound in her knee—"like cracking nuts." Since the onset of the joint affection the size of the knee had varied, owing to alteration in the amount of puffiness of the superficial tissues.

In March, 1885, the patient's condition was as follows:—She was a well-nourished and healthy-looking woman. Her sight and colour perceptions were good. There was no diplopia. Her pupils were small and unequal (R. 3 mm., L.
2½ mm.). They did not react to light, but they contracted during accommodation.

Her upper limbs were unaffected.

She complained of numbness in her feet, and of sensations as though she walked upon wool. Tactile sensibility was impaired in the soles of her feet but was normal in her legs. Painful impressions were felt naturally. Differences of temperature were distinguished. There was no loss of muscular power. Her gait was very ataxic. There was no loss of muscular sense.

Patient's face, hands, and feet perspired freely. There was no rectal or vesical trouble. The plantar reflexes were normal. The knee-jerks were absent. There was no ankle-clonus.

The left knee was much enlarged, and the superficial tissues were puffy, but they did not pit under pressure. The bones entering into the joint were enlarged, and the patella was broadened. There were no osteophytic growths. The tibia was abducted, rotated outwards and partially dislocated outwards. Grating was caused by moving the patella on the condyles of the femur, and by flexing and extending the leg on the thigh. There was slight lateral mobility at the joint. The joint was free from pain. The right knee creaked on movement, but there were no other signs of disease in it.

Case 13.—Frewing, female, æt. 37, shown at the Clinical Society, November 28, 1884, by Mr. G. H. Makins. Report taken in March, 1885.

This patient began to suffer from pain in both sides of the small of her back about six years ago. About two months later vomiting accompanied the pain. The vomiting had been worst in the morning. The attacks of vomiting recurred about every five weeks since their commencement. She had been in St. Thomas's Hospital on five occasions on account of these attacks. For the last four years she had had some difficulty in walking, and for the same period she had suffered from aching pains all down her left leg, which also had a feeling "as if it were dead." She ceased to menstruate five and a half years ago, after the birth of her last child, but menstruation returned again a year ago. Two years and three months ago a perforating ulcer formed under the ball of her right big toe; a piece of bone was discharged from the wound eighteen months ago, and three months ago the wound healed. The left big toe began to enlarge and suppurate
twelve months ago. The left foot was the seat of shooting pains at the same time. Three months ago the left metatarso-phalangeal joint was somewhat enlarged, and two sinuses on the inner side of the foot lead down to the joint. Movement of the joint did not cause pain.

There was no history of secondary syphilis, but patient had had one miscarriage, and one stillbirth; two children had died at the ages of sixteen and eighteen months respectively.

In March, 1885, the patient's condition was as follows:—Her pupils were small; the left one was a little larger than the right, they were both inactive to light and during accommodation for near vision the contraction was slight. Her sight was not good, but this might be accounted for by the myopia present. With the exception of some impairment in the sensibility of the left ring and little fingers the sensory and motor apparatus of the upper limbs were normal. The prick of a pin was only dimly felt in the sole and dorsum of the left foot, but it produced a reflex action. The same condition was present in the leg as high as the knee; the analgesia, though less marked, was present in the right foot and legs. The touch of the finger was felt by both lower limbs. Patient's gait was not ataxic, but she swayed a little when she stood with her feet together and her eyes closed. There were no signs of disease of the special lumbar centres.

The left big toe was shortened, apparently due to wasting of the head of the metatarsal bone. There was some lateral mobility and grating in the metatarso-phalangeal joint.

The skin over the ball of the right big toe was rather thicker than that over the left; there was no sinus. There was free mobility in all directions and some grating in the metatarso-phalangeal joint. The toe did not appear to be shortened.

Case 14.—J. Marshall, male, æt. 39, under the care of Dr. Radcliffe at the Hospital for the Paralysed and Epileptic, Queen Square. Report taken March 14, 1887.

This patient began to suffer from shooting pains in his legs eight years ago. They were thought to be rheumatic. No further symptoms occurred till five years ago, when he noticed difficulty in walking in the evening, and in standing steadily whilst washing himself. About this period his left foot and leg swelled up suddenly. In about sixteen months the swelling subsided, and his foot was left in the state in which it was at the date of this report. About five years ago the shooting pains in his legs were more severe than at first, and
they also affected his arms, and, to a less extent, other parts of his body. They were particularly induced by sexual intercourse. He also became affected with the girdle sensation, and that had continued ever since. Sexual power had never been in excess. About three years ago it became greatly diminished, and at the time of this report it was quite lost. Two years ago he had very little power over his bladder, and could not hold his water for more than three hours. On March 14, 1887, the patient's condition was as follows:—

The left pupil was slightly larger than the right in all positions. Both reacted well to light and during accommodation, though the left did not contract so well as the right. The movements of the eyeballs were good. Vision was good. His sensibility to both tactile and painful impressions was markedly impaired in both his legs and the whole of his trunk as high as the third rib, but he could always distinguish between a touch and the prick of a pin. He usually distinguished between heat and cold. The perception of sensations was considerably delayed over the whole of the affected area. The patient could stand with difficulty whilst he looked at his feet, but when he closed his eyes he began to sway about and would have fallen if not supported. His gait was very ataxic. He was unable to tell whether his knees and hips were flexed or extended, and he did not know the position in which his legs were lying. He had no loss of muscular sense in his upper limbs, and he could perform all movements normally. The superficial reflexes were obtainable, but the abdominal and epigastric reflexes were delayed for about one and a half seconds after the stimulation. Both knee-jerks were absent. The functions of the bladder and rectum were normally performed. He had not suffered from gastric crises. The tarsal bones of the left foot were enlarged. There appeared to be some bony thickening of all the cuneiform bones, but especially of the internal one. The scaphoid was much thickened, and there was a large rounded prominence on its inner side. Between the malleoli there was an irregular bony mass, more prominent on each side, which was apparently connected with the astragalus. The sole of the foot was much flattened. There was no pain in the foot. The right foot was normal.

Case 15.—Mary Ann Dowsett, æt. 50, under the care of Dr. C. A. J. Wright at the Islington Infirmary. Report taken March 21, 1887.

The first symptom of locomotor ataxy noticed by the
Joint Disease in connection with Locomotor Ataxy.

Patient was difficulty in walking in the dark. This was ten years ago. This symptom got worse gradually, and eight or nine years ago she felt very giddy in the dark, and had a very staggering gait. The patient gave a history of attacks of epigastric pain and sickness coming on at intervals of a few weeks. The attacks were accompanied by much flatulence. It was questionable whether these were true gastric crises. The attention of the medical officer at the Infirmary had not been drawn to them. The patient had had incontinence of urine for many years, and she attributed it to a confinement. About six and a half years ago (November, 1880) patient was lifting a heavy weight and "ricked" her right thigh. She fell down and suffered much pain in her thigh at the time. A good deal of swelling of the thigh followed. She was taken to St. Bartholomew's Hospital, where she was told that her right thigh was broken. She was treated with a long splint. She left the hospital after three or four weeks, and went about on crutches for about a week, but after that time she was confined either to bed or to a chair. When she left the hospital her right leg was two inches shorter than its fellow. Within six months after leaving the hospital she began to suffer from shooting pains in her legs. The attacks of pain had been increasing in frequency and severity since their onset, and at the date of this report she had them about every fortnight or three weeks. Her arms were first attacked with shooting pains about three years ago. About two and a half years ago, after being bedridden for nearly four years, her right shoulder swelled up rather suddenly and painlessly. There was, however, some pain on movement. The swelling was strictly limited to the region of the joint. It was accidentally noticed by the medical attendant. When first seen there was no dislocation, but in about a week or a fortnight the head of the humerus was found dislocated below the coracoid process.

The patient could give no further account of her illness, and was not aware that anything was wrong with her left hip or her right knee, although both these joints were extensively diseased at the time this history was taken. There was no history of syphilis.

The patient's condition on March 21, 1877, was as follows:—
Both pupils were small, equal in size, and did not react to light, or in association with accommodation. With the exception of slight dimness of sight there were no signs of affection of the cerebral nerves.

Tactile sensation in upper limbs was good. Painful stimuli
were rapidly appreciated, and heat and cold could be readily distinguished. There was some loss of the power of coordination in the upper limbs.

Tactile sensibility in the feet and legs was almost lost. Painful stimuli were acutely felt, but their perception was delayed for about two and a half seconds. Heat and cold were usually distinguished. There was great loss of the power of coordination of movement in the lower limbs, and the patient was quite unable to tell the position of her legs without looking at them. The plantar reflexes were absent. The knee-jerk could not be obtained on either side. There was no ankle-clonus.

The desire to defaecate was quickly followed by an action of the bowels. There were no perforating ulcers of the feet.

The right shoulder-joint was much swollen, owing to distension of the capsule of the joint with fluid. The upper end of the humerus naturally rested beneath the coracoid process, but it could be displaced in all directions. When carried behind the posterior border of the deltoid it could be felt as a rounded end about the size of the bone below the tuberosities. There were no osteophytic growths attached to it. The acromion process had a natural shape and size. No bony growth could be detected in the capsule, but loose bodies or hypertrophied and pedunculated synovial fringes could be felt within the joint. The right humerus was an inch shorter than the left.

The left shoulder-joint was not swollen, and the head of the humerus occupied its natural position, but coarse grating was produced by moving it against the glenoid cavity.

Both hip-joints were similarly diseased. They were both much disorganised. The head and neck of each femur appears to be absent. The trochanters were of about the natural size. They occupied higher positions than natural, and could be moved freely in all directions. The mobility at the joints was so much increased that the anterior surface of the femur could be made to face almost directly backwards. There was very little if any fluid in the joints. No osteophytic growths could be detected. Coarse grating in the joint was produced on movement.

The right knee-joint was not swollen, but the ligaments were lax, and allowed a partial backward dislocation, as the limb lay on the bed. The head of the tibia could be partially dislocated either backwards or forwards, and the movements were attended by coarse grating within the joint. No osteo-
Joint Disease in connection with Locomotor Ataxy. 313

phytes and no lipping of the ends of the bones could be detected.

The ankle-joints were rather stiff, but they did not present any signs of disease.

There were no signs of an old fracture of the shaft of the right femur for which the patient says she was treated six and a half years ago.

Case 16.—Jane White, aet. 40, under the care of Mr. J. R. Lunn, at the St. Marylebone Infirmary. Report taken April 21, 1887.

This patient stated that about eight or nine years ago she had an attack of shooting pains in her legs. The attack lasted for two or three days and then quickly ceased. For the next twelve months she was free from pain, but at the end of that time an attack of pain quite similar to the first occurred. The pains were not very severe at first but they gradually got worse, and the attacks recurred more frequently until at the date of this report they occurred about every week. Three or four years ago the patient began to have attacks of sickness for which she could not assign any cause. The attacks of sickness were associated with the shooting pains. She woke up in the early morning with a feeling of heaviness about her eyes, and soon after the pains commenced quite suddenly, and along with them she had a feeling of nausea and epigastric pain. Retching and vomiting sometimes, though not always, occurred. During an attack she always perspired freely and sometimes she perspired without known cause independently of an attack. About six years ago her feet began to swell and to be painful. She had several attacks of this kind, accompanied sometimes by pain and at others not. During the last three years her feet had been getting deformed, and during the last twelve months they had got much worse. During the last four years she had been unable to walk in the dark. Two years ago, two perforating ulcers formed on her left foot, one on the ball of the big toe, and another on the ball of the little toe. Three ulcers formed on the right foot, two being over the balls of the little and big toes respectively, and the third between the other two. They all healed, but a year later they formed again.

On April 21, 1887, the patient’s condition was as follows:— Her pupils were small and of equal size. They did not react to light, but contracted well during accommodation. There was no impairment of either the sensory or motor functions of
the upper limbs. There was no impairment of tactile sensibility in the lower limbs. Painful impressions were felt, but in the feet and legs below the knees they were not felt so acutely as in other parts. Heat and cold were readily distinguished in the lower limbs, but perception of the sensations was delayed two seconds. The patient was unable to stand when her eyes were closed, but there was no obvious ataxy. She was unable to tell the position of her legs when in bed. The plantar reflexes were present, but were not so brisk as natural. Both knee-jerks were absent. There was no ankle-clonus.

The soles of both feet were much arched and the bones of the tarsi in front of the astragalo-scaphoid joints stood out prominently on the dorsum of the feet. The proximal phalanges were over-extended and the patient stood on her heels and the heads of the metatarsal bones. The tarsal joints were rigid. The feet were free from pain. No other joints were affected. There were no ulcers on the soles of the feet, but the epithelium was much thickened at the places where they had been.

Case 17.—Frederick Stokes, æt. 42, under the care of Mr. Rickman J. Godlee, at University College Hospital. Report taken in April, 1885.

This patient contracted syphilis twenty-one years ago. He had been married fifteen years, but had no children.

He had suffered from sudden attacks of sickness without apparent cause for two years. He had been troubled with aching pains in the lower part of both forearms and in both hands during the last fifteen months, and during the last six months he had had shooting pains in both his upper and lower limbs. During the last year he had had sudden and uncontrollable calls to micturate. One year ago a corn formed on the ball of patient's right great toe; suppuration took place beneath it, and an ulcer, the size of a sixpenny piece, resulted. No necrosis occurred, and the ulcer healed in a little over a month. Six and a half months ago the right elbow began to be affected and gradually swelled up; six weeks later the joint was larger than when the patient came under observation. There had been no pain in the joint except the shooting pain, which affected other parts as well.

In April, 1885, the patient's condition was as follows:—Both pupils reacted to light, and in association with accommodation. The right hand felt as though it was asleep, but
no affection of sensibility of either the upper or the lower limbs could be detected. The gait was not ataxic and there was no incoordination of the movements of the upper limbs. The left knee-jerk was slight, but the right was exaggerated. The right elbow was much swollen, owing largely to distension of the capsule of the joint with fluid. The lower part of the humerus was much enlarged. The forearm bones were partially dislocated inwards. The olecranon fossa of the humerus was empty, and the olecranon could be felt at the inner side of the joint. Neither the head of the radius nor the olecranon process were enlarged. There was a bony nodule in the upper and inner part of the capsule. There was free lateral mobility at the joint, and the bones could be moved freely without causing pain.

In September, 1885, the patient suddenly lost the power of extending the fingers of his right hand, and within a day or two of the onset of this symptom his right musculo-spiral nerve was found enlarged and tender. The loss of power lasted three or four days.

On February 3, 1886, the pupils contracted much better during accommodation for near vision than they did to light. The whole of the right hand, and the left thumb and forefinger felt numb. The power of coordinating the movements of the upper limbs was fair. The attacks of sickness recurred less frequently and were less severe than they were ten months previously. The knee-jerk was brisk on the right side, but on the left it was very slight.

On May 21, 1887, the patient was admitted into the hospital, under the care of Mr. Marcus Beck, on account of a sudden enlargement of the left elbow-joint of about a week's duration.

The patient's condition was as follows:—His pupils were of medium size, and contracted well during accommodation for near vision, but the right pupil did not contract at all to light, and the left one very feebly. His colour vision was normal. There was no diplopia or ocular paralysis. His hearing was good. He complained of shooting pains in both forearms and both legs. The attacks of pain recurred almost every week; the pain began in either forearm or leg, was at first cramp-like, and then shot into one of his fingers or to the dorsum of his foot and disappeared. The pains shifted from place to place, and recurred every few minutes. A complete attack usually lasted two or three days. The patient complained of numbness in both his hands, but there did not
appear to be any loss of sensibility in the upper limbs to
tactile or painful impressions. There was no incoordination
of the movements of the upper limbs. Tactile sensibility in
lower limbs was perfect; painful, hot, and cold impressions
were readily felt. There was no ataxic gait, and the patient
could stand steadily with his eyes closed. He suffered from
attacks of sickness, which came on without any evident cause.
He sometimes had difficulty in passing his urine, and at others,
usually at night, his urine escaped involuntarily. Sexual
functions normal. Bowels regular. The right knee-jerk was
brisk and left slight.

The right elbow was much deformed owing to dislocation
of the radius and ulna, and to osteophytic growths. The outer
side of the lower end of the humerus was much enlarged, and
formed a prominent mass about the size of an orange. The
olecranon was displaced backwards and inwards, was much
enlarged, and had lost its natural shape. At the inner side
of the joint there was an osteophyte, which was connected
with the upper end of the ulna. There was a partially movable
mass of new bone connected with the anterior and inner part
of the capsule of the joint. Pronation and supination were
attended by much creaking and coarse grating sounds. The
joint could be flexed about 15° beyond a right angle, and ex-
tended to about 30° short of complete extension. There was
no lateral mobility.

The left elbow:* On admission the lower fourth of the left
upper arm, the elbow, and the forearm were enormously
swollen; the dorsum of the hand and the fingers were edema-
tous. The skin around the elbow-joint and over the swollen
regions was red and brawny; it pitted under firm pressure of
the finger. The patient complained of a feeling of tightness
in the skin, but he did not suffer from pain in the joint, and
he was able to move the joint freely without causing pain.
Movements, which were a good deal restricted by the swell-
ing, were attended by a coarse grating sensation. No un-
natural mobility could be detected.

On May 27 the swelling, which had gradually been sub-
siding since admission, had almost left the lower part of the
forearm; the tissues around the elbow-joint did not pit on
pressure. There was still much swelling of the joint, but this
was due chiefly to fluid within the capsule. The bones entering

* The affection of the left elbow is not included in Table II, or in the statistics
referred to in the Report, as both had been prepared before the onset of the
disease in that joint.
Joint Disease in connection with Locomotor Ataxy.

into the joint could be made to slide laterally over one another, and some lateral angular movement could be made at the joint. The bones were loosely attached to one another, but there was no dislocation. There was no pain on flexing or extending the elbow-joint.

On May 29 the front of the forearm just below the elbow was the seat of a hot boring and aching sensation, and the upper part of the forearm was again swollen and red. The temperature was 98.8° F.

On June 2 the joint was aspirated on each side of the olecranon and about an ounce of fluid was drawn off; the fluid was tinged with blood; it was free from pus. The temperature was 100.4° F.

On June 3 the temperature was normal, and the swelling was again subsiding.
### Table I.—Full Abstracts of 42 cases of Charcot’s Joint Disease, collected from

<table>
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<tr>
<th>Name and reference</th>
<th>Sex and age</th>
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</tr>
</thead>
<tbody>
<tr>
<td>Christina Metcalf.</td>
<td>F. 30</td>
<td>16 years</td>
<td>Disease commenced with severe epigastric pains and sickness; lightning pains in both legs came on 2 or 3 years later, and still later pains came on in upper limbs. Two or three years after onset of disease patient had a marked ataxic gait. Diplopia 6 years ago. Pupils unequal and dilated; they do not contract either to light or in association with accommodation. Sensation in upper limbs normal, except that perception is delayed. Patient does not feel the prick of a pin in the soles of her feet; sensation is impaired in the lower part of her legs, and perception is delayed 3 seconds on both sides. Patient has difficulty in distinguishing moderate degrees of heat and cold, but a very hot spoon is usually recognised. Plantar reflexes absent; knee-jerks absent. The muscles of the upper limbs are markedly developed; those of legs and thighs are wasted, but there is more power in them than would be expected from their appearance and that of the joints.</td>
<td>Six years ago both legs swelled a good deal but the swelling subsided with rest; soon afterwards she noticed one morning that the right leg was much swollen from the knee to the ankle and on getting out of bed she found that she was unable to stand on her right leg; she had a very severe prickling sensation in the joint and oppression at that time. The knee was shortly afterwards found to be dislocated. The left knee began to be weak and numb before the right, but has not got worse so rapidly. In 1880 the right shoulder began to be affected with severe prickling pains, and the joint suddenly swelled up and dislocation occurred One year and ten months ago right knee began to swell gradually; there has been no pain in the knee from the first swelling of knee subsequent 9 months ago and the joints then became deformed</td>
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<td>Elizabeth Neale.</td>
<td>F. 53</td>
<td>25 years</td>
<td>Difficulty in walking in the dark began 25 years ago. Diplopia 24 years ago, but not now present. Lightning pains in lower extremities, and ataxic gait for 24 years. Gastric crisis 6 years ago and since. Argyll-Robertson phenomenon; pupils of medium size. Tactile sensation absent in both feet and lower half of both legs, and diminished in upper half of legs; the prick of a pin is only indistinctly felt in the upper half of legs, and in the lower half it is only felt as a touch; analgesia and complete anaesthesia of soles of both feet; sensation of heat is felt in soles of both feet, but perception is delayed six seconds; incoordination of movements of legs; plantar reflexes absent; micturition difficult and painful; defaecation difficult and painful, and patient has frequent desire to defecate</td>
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of which 24 have been examined by the Committee, and the rest English sources.

<table>
<thead>
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<th>Description of Joints</th>
<th>Remarks</th>
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<td><strong>Right Knee</strong>.—The tibia is dislocated backwards into the popliteal space; the internal condyle of the femur is much deformed, and the external appears to be nearly worn away; the bones can be separated from one another for 1 inch, and made to knock against one another without causing pain; there is great mobility in all directions. A few bony outgrowths can be detected on the outer side of the internal condyle; some loose or pedunculated bodies can be felt on the inner side of the knee; there is no swelling of the soft tissues of the joints, and no fluid in the cavity. The bones grate when they are moved against one another.</td>
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<tr>
<td><strong>Left Knee</strong>.—The tibia is partially dislocated backwards, but it can be drawn into position; bony prominences can be felt along the borders of the condyles, and loose bodies of firm consistence or vegetations, which are partially movable, can be made out within the joints; there is free lateral mobility of one bone on the other; there is no grating produced on movement.</td>
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<tr>
<td><strong>Right Shoulder</strong> is swollen, due to fluid in the joint; the head of the humerus is dislocated under the coracoid process; it can be easily reduced, but it slips out again when the arm is moved; there is grating in the joint; and a number of loose bodies or vegetations from the capsule can be felt moving beneath the fingers. The acromial end of the clavicle is dislocated upwards on to the acromion process.</td>
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<tr>
<td><strong>Left Shoulder</strong> appears to be normal.</td>
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Patient has twice had "fits" in which she was unconscious. She has slight twitching movements of the face, neck, and fingers; speech is slow and measured.
Janet Sneger.  
Shown at the Clinical Society, Nov. 28, 1884, by Mr. J. R. Lunn. Report taken in March, 1885.  
F.  57 years  
Lightning pains in legs since onset of disease. Three severe attacks of pains in arms during last 18 months; gastric crises 5 months ago, and during the following 3 months; Argyll-Robertson phenomenon; pupils contracted; sensation in lower extremities impaired, and perception of impression delayed; incoordination of movements of legs, and loss of muscular sense in them. Knee-jerk absent in both legs.

Mary Hollis.  
Shown at the Clinical Society, Nov. 28, 1884, by Mr. J. R. Lunn. Report taken in March, 1885.  
F.  47 years  
Lightning pains in epigastrium at onset of disease, and in both legs soon afterwards; pupils unequal and small; Argyll-Robertson phenomenon; sensation impaired in soles of feet; marked ataxic gait; knee-jerks absent.

Frewing.  
Shown at the Clinical Society, Nov. 28, 1884, by Mr. G. H. Makins. Report taken in March, 1885.  
F.  37 years  
First symptom was pain in both sides of the small of the back, and soon after vomiting accompanied the pain. Four years ago she felt aching pains all down her left leg, which also felt as if it were dead. Attacks of vomiting have always occurred once in 3 weeks. Perforating ulcer under ball of right big toe. Pupils inactive to light and contract badly during accommodation. No anaesthesia; analgesia in left foot and leg, and in a less marked degree in right. No knee-jerks. Patient sways a little from side to side when she stands with feet together and eyes shut. The last symptom had been present for 4 years. Gait not ataxic.

Josiah Pickford.  
Shown at the Clinical Society, Nov. 29, 1884, by Mr. R. J. Godlee. Report taken in April, 1885.  
M.  60 years  
Lightning pains in lower limbs from onset of disease and soon afterwards in upper as well. Diplopia 15 years ago; paralysis of right external rectus 12 years ago. Occasional dribbling of urine for 4 years; sudden desire to micturate soon after having passed water. Sight weak; watch not heard at contact with right ear. Analgesia of lower half of left leg and foot. Knee-jerks absent.

Nine years ago patient fell down three steps and strained her left knee; her thigh and knee swelled up, but they were not painful.

Two years ago left knee gradually swelled and was rather painful; no accident to account for it. About 6 mos. late patient noticed cracking in her knee.

Twelve months ago the left big toe began to enlarge, and later of there appears to have been suppurative arthritis of the meta-
tarso-phalangeal joint.

Three and a half years ago left thigh swelled up painlessly; he had severe lightning pain at that time; he lay up for 3 weeks, and at the end of that time began to get about, but with difficulty.
Joints.—Left.—There is enlargement of the whole of the left lower limb, but most marked around the knee; bones entering into the joint are much enlarged and the patella is much broadened; there is marked looseness and lateral mobility of the tibia on the femur; patella is displaced outwards; coarse creaking can be felt in the knee when the joint is moved; movements are painless; no bony enlargements of ends of bones. Right.—Superficial tissues puffy; creaking sensation felt during movements; no looseness of joint.

Knees.—Left.—Much enlarged and puffy; condyles of femur and tuberosities of tibia much enlarged, and patella is broadened; the leg is rotated outwards, and there is some partial external displacement of the tibia; grating is produced by moving the patella on the condyles of the femur and by moving the joint; there is slight lateral mobility of the leg; no pain in joint except occasional dartings. No bony outgrowths of bone. Right.—Well-marked creaking on movement, but no enlargement of bones and no displacement of them.

Left big toe.—Shortened apparently due to wasting of head of metatarsal bone; there is some lateral mobility and grating in the joint.

Right big toe.—Skin over ball rather thicker than that over left; no sinus; considerable mobility in all directions and some grating, but no shortening.

Left hip is much enlarged, and the veins over it are big; the swelling is due to enlargement of the great trochanter; there is enlargement of the horizontal ramus of the pubes, and there is a spicule of bone which extends from the ilium just below the anterior superior spine or 6 inches down the front of the thigh. No loose bony fragments can be made out, and no bony outgrowth from the bones beyond those mentioned can be detected. Movements of the joints are attended with coarse grating.

Perforating ulcer under ball of right big toe 26 months ago; a piece of bone was discharged 17 months ago, and the ulcer healed 2 months ago. All children dead; first died at 16 months; miscarriage after this; third pregnancy terminated as a stillbirth; next child lived 18 months, and last 3½ years, and died of scarlet fever.

Pupils moderately contracted, but they react to light and in association with accommodation. No ataxic gait. Sexual functions not affected. Dupuytren's contraction of ring and little finger of both hands.
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<td>Frederick Waters. Shown at the Clinical Society, Nov. 28, 1884, by Mr. A. E. Barker. Case taken in April, 1885</td>
<td>M. 39</td>
<td>4½ years</td>
<td>Gastric crises, ataxy of the bladder and incontinence of urine marked the onset of the disease 4½ years ago. The gastric crises had been less frequent of late and the bladder trouble was not so marked as at onset of disease. Lightning pains in abdomen and legs during last 3 years. Two and a half years ago a perforating ulcer formed beneath the ball of the right little toe, and subsequently four others formed. From one of which on the big toe some pieces of bone were discharged. Heat and cold much confounded on inner side of left leg and left foot. All sexual desire and ability lost</td>
<td>Nine months ago left knee, thigh, and bladder, and in the course of 3 weeks the joint became disorganized. He had slight pains in the joint at the onset, and he suffered at the same time from attacks of sickness.</td>
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<tr>
<td>William Jermy. Shown at the Clinical Society, Nov. 27, 1884, by Sir Dyce Duckworth. Report taken Nov. 19, 1884</td>
<td>M. 45 mos.</td>
<td>Girdle pains occurred 18 months ago. Sharp intermittent &quot;rheumatic&quot; pains all over body, and especially in right knee, commenced 6 months ago. Occasional attacks of vomiting, without known cause, during the last 3 months. Patient staggered with his eyes shut. His gait is ataxic. Coordination of movements of upper limbs is impaired. Knee-jerks absent</td>
<td>Three years ago both elbow-joints became swollen suddenly and painlessly. There had been no previous injury to either joint. The affection of these joints reached its full development in 6 days.</td>
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<tr>
<td>Elizabeth Waterson. Under the care of Mr. John Hopkins, at the Central London Sick Asylum. Reported in Trans. Path. Soc., 1880, p. 194, by Dr. Buzzard. Report taken in April, 1885</td>
<td>F. 55</td>
<td>16 years</td>
<td>The first symptoms were well-marked gastric crises; the gastric pain and vomiting lasted for 2 or 3 weeks at a time. Soon after the onset of these attacks she suffered from shooting pains in her legs, and to a less extent in her arms; she could walk with difficulty 9 or 10 years ago, but she was very unsteady in the dark and staggered even in the light. Pupils are small and do not react to light, but they contract during accommodation. Impaired sensation in finger-tips both to touch and pain, and perception is delayed. Feels pain in left leg and sole of foot, but perception of it is delayed; pain not felt at all in the right side. No anesthesia on either side. Muscular sense impaired; knee-jerks absent; plantar reflex normal</td>
<td>Seven years ago she had a spontaneous fracture of her right thigh; and one year later the left hip joint suddenly swelled up.</td>
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</table>
### Description of Joints

**Left Knee.** — Ligaments lax and joint distended with fluid; considerable enlargement of upper end of tibia; slight enlargement of lower end of femur; no bossy bony outgrowths; tibia can be separated from femur for about ½ inch, and the two bones can be knocked together with an audible sound; lateral mobility of tibia; creaking in the joint; movement and percussion of the tibia against the femur do not cause pain. In June, 1885, there was an osteophytic growth from the external tuberosity of the tibia.

*Both elbow-joints* are greatly enlarged and contain fluid. The left is the most enlarged. The ulna is dislocated backwards. There is slight crepitus, but no pain on movement. There is an osseous mass in the capsule of the left joint.

**Left Hip Joint.** — Trochanter displaced upwards slightly, and is much enlarged and prominent.

**Right Hip Joint.** — Trochanter somewhat depressed.

There is a well-marked creaking in both joints, but particularly in the right.

### Remarks

Pupils react both to light and during accommodation. Left knee-jerk is normal and right is exaggerated; no ataxic gait. No anæsthesia. Patient has had perforating ulcers on right foot, which have healed. The patient died in February, 1886. No P.M.

No history of syphilis, rheumatism, or gout. Pupils react both to light and during accommodation. The thenar muscles and the interossei of both hands are wasted; there are several bad plantar callosities, and one with a perforation under right great toe. No vesical, rectal, or laryngeal crises. This patient had been lost sight of, and so a full account of his case could not be taken.

Three children died a day or two after birth; no miscarriages, and no stillbirths.
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<td>Henry Gigg.</td>
<td>M. 51</td>
<td>6 years</td>
<td>Illness dates from a fall down some stone steps 6 years ago, after which pains of a shooting character came on in the arms and later in the legs; these pains have continued and become much intensified. Since the fall his urine often passed involuntarily; patient was seized with a sudden desire to micturate and his urine at once passed from him. Sexual power and inclination increased before his accident, but since it he has gradually lost all sexual power. Numbness in toes of both feet since accident. Well-marked gastric crises commenced 3 years ago, and now recur with great severity about every month. Argyll-Robertson phenomenon; sensation normal in upper limbs. Anaesthesia of right leg and foot, and sensation is impaired in left; no complete analgesia, though a prick of a pin is indistinctly felt in right leg and foot. Heat and cold distinguished in right leg, but perception is delayed 3 seconds. No knee-jerk or plantar reflex on either side. Muscular sense in legs impaired so that he does not know the position of his legs unless he looks</td>
<td>Six years ago he fell down some stone steps and hurt his left elbow severely, and his right hip to a less extent; he walked a mile directly afterwards. His thigh turned black and blue for 6 weeks, and since that time he has had much difficulty in walking, and during the last 2 years he has been unable to walk at all</td>
</tr>
<tr>
<td>John Griffiths.</td>
<td>M. 43</td>
<td>7 years</td>
<td>Disease began with a feeling of &quot;pins and needles&quot; in his feet, and a year later he began to suffer from lightning pains of slight intensity in his legs, which continued till 2½ years ago; shooting pains in the shoulder 2 years ago. Pupils equal and contracted. Argyll-Robertson phenomenon; slight right. Tactile sensations normal in the lower extremities, but the prick of a pin does not cause a painful sensation in the lower half of the right leg or the lower third of left. Perception of sensations is slightly delayed. Plantar and abdominal reflexes exaggerated; knee-jerks absent. In early part of illness patient had increased sexual desire, and 1 year ago erection of the penis was caused by sudden exposure to cold air; urine was frequently passed involuntarily at that time and occasionally so now</td>
<td>Four and a half years ago his right ankle swelled suddenly, and a month later his right knee and whole leg began to swell. The swelling lasted 6 weeks and then disappeared except about the right knee, which has remained large ever since, and has continued to get more deformed and weak. About 2 years ago the patient noticed creaking in his left hip-joint and about a fortnight later his left thigh and leg began to swell; the general swelling soon disappeared, and a small swelling was noticed within the pelvis about the horizontal ramus of the pubes, and another on the ilium just below the anterior part of the iliace crest; this has been increasing in size since its first appearance</td>
</tr>
</tbody>
</table>
### Description of joints.

<table>
<thead>
<tr>
<th>Description of Joints</th>
<th>Remarks</th>
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</thead>
<tbody>
<tr>
<td>Right thigh is everted; trochanter is raised 1½ inches above Nclaton's line, and right leg 1½ inches shorter than the left. The soft tissues are swollen and puffy, but do not pit; there is creaking in the joint.</td>
<td>No history of syphilis.</td>
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<tr>
<td>Left olecranon process appears to have been broken and united by fibrous tissue.</td>
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<tr>
<td>The right knee is enormously enlarged; the skin over it is normal, but the superficial veins are dilated; there is very little fluid in the joint; the tibia is dislocated outwards and slightly backwards. The ligaments of the joint are relaxed, and when the patient stands up the tibia is still further displaced backwards; there is slight lateral mobility of the tibia. The lower end of the femur, both condyles and shaft, is much enlarged; the patella is enlarged and broadened; the upper end of the right tibia is much enlarged, the borders of the bones are devoid of bony projections except at the upper part of the external tuberosity, from which there is a bony outgrowth.</td>
<td>No ataxic gait. This patient poisoned himself 5 months after this report was taken. See the full report of the case for the P.M. appearances.</td>
</tr>
<tr>
<td>The left hip is less prominent than natural, and the trochanter has the normal shape and size, but it is at a higher level than natural. A large, hard, and apparently bony growth extends from the crest of the ilium to the trochanter major; the skin over it is normal, but the veins are dilated; on the surface of the horizontal ramus of the pubes there is another bony growth which extends outwards to near the anterior superior iliac spine, and inwards to near the symphysis pubis.</td>
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</tbody>
</table>
F. Stokes.
Under the care of Mr. Rickman J. Godlee at University College Hospital. Report taken in April, 1885

M. 42 years

Duration of disease.

Evidences of locomotor ataxy.

Aching pains in lower part of both forearms and hands 15 months; shooting pains in upper and lower limbs for 6 months; attacks of sudden sickness without apparent cause for 2 years. During the last year the patient had had sudden and uncontrollable desire to micturate. Right hand feels as though it were asleep, there is no anesthesia or analgesia on either side. Knee-jerk on left side very slight; on right it is exaggerated.

P.S.—In Sept., 1885, patient suddenly lost power of extending his fingers of right hand, and his right musculo-spiral nerve was found enlarged and tender. The loss of power lasted 3 or 4 days. On Feb. 3rd, 1886, the attacks of sickness were less marked. The whole of the right hand and the left thumb and forefinger fell numb. The coordination of movements of upper limbs was fair. The knee-jerk was brisk on the right side, but on the left it was very slight.

Fred. Weaver.
Under the care of Mr. Morrant Baker, at St. Bartholomew’s Hospital. Case taken May, 1885

M. 8 or 10 years

Lightning pains in legs commenced 8 or 10 years ago, and at first recurred about once a week, but they are less frequent now. Dimness of vision, with inability to stand with closed eyes or to run, during last 14 years. Pupils react well with accommodation, but only feebly to light. Numbness and tingling in sole of right foot. Ataxic gait. Knee-jerks absent. An attack of acute shooting pain in the pit of the stomach occurred whilst patient was in hospital.

John Austin.
Under the care of Mr. Morrant Baker, at St. Bartholomew’s Hospital. Case taken May, 1885

M. 12 years

Lightning pains in legs commenced 12 years ago. Left pupil contracted and right pupil dilated. No reaction to light or with accommodation.

James Rawlings.
Under the care of Mr. Morrant Baker, at St. Bartholomew’s Hospital. Case taken May, 1885

M. 42 years

The pupils do not contract to light. Slight double optic neuritis (?). Numbness of both feet, and lightning pains in both lower extremities. Knee-jerks absent.

Six and a half months ago right elbow began to be affected and gradually swelled up so that 6 weeks after onset it was larger than at present. There was no pain other than the shooting pain.

The whole of the right lower limb swelled up suddenly 18 months ago. Increased swelling of the knee followed injury to the joint 9 months later.

The right knee began to swell gradually and painlessly 18 months ago. Patient fell from a cart 3 months later, and further swelling of the knee followed.

Left leg twisted under patient 3½ years ago and was severely sprained. It swelled and was very painful. About the same time the right knee became enlarged and painful. Since then both knees have increased in size and become more painful.
Joint Disease in connection with Locomotor Ataxy. 327

**Description of joints.**

*The right elbow* is much swollen, due largely to fluid in the joints; the lower part of the humerus is much enlarged, and the forearm bones are partially dislocated inwards; the olecranon fossa of humerus is empty, and the olecranon can be felt at the inner side of the joint. Neither the head of the radius nor the olecranon process are enlarged. There is a bony nodule in the upper and inner part of the capsule. There is free lateral mobility at the joint and movements of the bones are quite painless.

*Right knee* much enlarged. Ligaments relaxed and much thickened. Free lateral mobility

*Left knee* most affected. Contiguous ends of femur and tibia enlarged by heaping up of new bone. Lateral movements increased in left knee; both knees are painful

**Remarks.**

No ataxic gait. Pupils react to light and also in association with accommodation; sexual functions normal; one year ago patient had a corn on the ball of the right great toe, under which suppuration took place, and an ulcer of the size of a sixpenny-piece resulted; no necrosis; ulcer healed in a little over a month. Had syphilis 21 years ago.

P.S.—On Feb. 3rd, 1886, the pupils reacted much better during accommodation than they did to light.

A perforating ulcer formed on the under surface of the right great toe 4 years ago. The skin over that toe now sweats excessively and is congested.

No history of syphilis or rheumatic fever. No perforating ulcers.

No history of syphilis. Rheumatic fever 12 years ago. No perforating ulcers.
<table>
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<tr>
<th>Name and reference</th>
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<th>Duration of disease</th>
<th>Evidences of locomotor ataxy</th>
<th>Duration and mode of onset of joint disease</th>
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<tr>
<td>E. Bewry. Under the care of Mr. B. Hill, at University College Hospital. Report taken in February, 1886</td>
<td>F. 48</td>
<td>14 or 15 years</td>
<td>Lightning pains in legs, arms, and trunk, commenced 14 or 15 years ago. Patient had diplopia a few years ago. Twelve months ago patient had a violent attack of sickness, which lasted for about a week. Much difficulty in walking in the dark during last year. Pupils equal and small. Argyll-Robertson phenomenon. Tactile sensation in feet and legs is normal. Painful stimuli not acutely felt on legs, and perception is delayed two seconds. Heat and cold are not readily distinguished on legs. Gait ataxic. Patient at once staggered when her eyes are shut. Knee-jerks absent. Plantar reflexes present.</td>
<td>Eight years ago, after a hard day’s work, right knee was found swollen, and felt as if it would give way. The leg, as far as the middle of the calf, was also much swollen. Patient’s doctor thought a blood-vessel had burst in the knee. No injury to join before onset. Patient whilst knee was swollen, but this passed off with the subsidence of swelling after a few days’ rest. Creaking in the joint of movement always noticed afterwards. The joint has often swelled up since first attack</td>
</tr>
<tr>
<td>Ziba Bickett. Under the care of Mr. R. J. Godlee, at University College Hospital. Report taken in June, 1886</td>
<td>M. 53</td>
<td>17 years</td>
<td>&quot;Rheumatic&quot; pains in legs for 17 years. These cannot be clearly separated from genuine lightning pains, which were most severe 11 years ago. Lightning pains began in left upper limbs about 2 years ago; they were confined to elbow, wrist, and hands till 4 or 5 months ago, when they attacked the left shoulder. Ataxy present in both legs for 10 or 12 years. None in upper limbs. Paralytic right internal strabismus for 10 or 12 years. Morning vomiting unattended with discomfort for last 20 years; only 5 attacks during last 18 months. Frequent calls to defecate and micturate for last 8 or 9 years. Pin’s point pupils. Argyll-Robertson phenomenon. Marked ataxy of lower limbs; knee-jerks absent. Sensation tactile and painful, impaired in feet and legs, and perception delayed 1½ seconds. No anaesthesia of upper limbs, but perception of sensation delayed ½ second. Sexual power totally lost.</td>
<td>Four or five months ago patient fell 4 feet, and struck his left shoulder, but no joint affection followed till the same shoulder was again struck 4 days ago. There was no immediate pain, but the same night the shoulder began suddenly to swell, and for three hours was the seat of intense pain. The pain then disappeared. P.S.—Ten months after the date of this report the right shoulder, upper arm, and right side of the chest swelled up and became discoloured. The patient suffered at the time from a severe attack of sickness and from lightning pains in all his limbs</td>
</tr>
<tr>
<td>Justine Smith. Under the care of Mr. Beck, at University College</td>
<td>F. 56</td>
<td>3 years</td>
<td>Gastric crises for 3 years; has had two well-marked crises whilst in hospital. Lightning pains in legs for 2 years. Numbness and “pins and needles” in right foot for about 3 years, and sensation of treading on soft things whilst walking. On admission hesitating gait, but no ataxia; unsteady, with</td>
<td>After a fall on right knee 14 months ago, the joint became painful for 14 days during that time it was gradually swelling, and has done so.</td>
</tr>
<tr>
<td>Description of joints.</td>
<td>Remarks.</td>
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<tr>
<td>Right knee is much swollen, and a bursa in popliteal space, which communicates with joint, is also distended with fluid. There are free lateral and antero-posterior sliding movements at the joint. The tibia is dislocated forward as patient lies in bed, but it can be easily reduced. The lower end of the femur is a little, and the upper end of the tibia is much enlarged. There are no large osteophytes, but there is a little irregularity along the border of the tibial articular surface. Creaking felt on moving joint.</td>
<td>No perforating ulcers of foot; no history of syphilis.</td>
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Left shoulder.—Four days after onset the shoulder and the whole of the upper arm were much swollen. No pitting of tissues; no pain on free manipulation. Movements a little impaired and attended by creaking. No displacement of bones or signs of fracture. P.S.—Six months after first observation all the swelling had subsided, and the head of the humerus was dislocated beneath the coracoid process; it could be reduced; coarse grating could be felt in the joint; grating could also be felt in the right shoulder, but there was no swelling and no dislocation. Four months later, and 4 days after the onset of acute disease in the right shoulder, the shoulder, upper arm, and the right side of the chest were enormously swollen, and the head of the humerus was dislocated beneath the coracoid process. | No perforating ulcers. No history of syphilis. |

Right knee is much swollen, due to the presence of much fluid in the joint. The lower end of the femur, the upper end of the tibia and patella are much enlarged. There is a rounded mass of new bone attached to the post. part of upper end of tibia. There is no marked wearing away of the articular ends of the bones. Coarse grating in the joint. Free lateral mobility at the joint. | No perforating ulcers of feet; no history of syphilis. |
<table>
<thead>
<tr>
<th>Name and reference</th>
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<th>Evidences of locomotor ataxy.</th>
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<tbody>
<tr>
<td>Hospital. Report taken in Dec., 1886</td>
<td>M. (?) 39 years</td>
<td></td>
<td>Eyes shut; pupils small, equal; Argyll-Robertson phenomenon; knee-jerks absent; plantar reflexes present; no anaesthesia or analgesia in legs; cold appreciated rapidly in legs; heat felt quickly as cold, and after a delay of 1½ seconds felt acutely as heat; sudden calls to micturate, and sometimes involuntary action of bladder</td>
<td>Up to time of admission, although the joint has been supported ever since the accident.</td>
</tr>
<tr>
<td>J. Taphouse. Under the care of Dr. T. D. Savill, at the Paddington Infirmary. Report taken in Dec., 1886</td>
<td></td>
<td></td>
<td>Pupils are equal. No contraction to light or in association with accommodation. No distinct gastric crises, but has often had &quot;attacks of the bile;&quot; knee-jerks absent 5 and 4 months ago—slight 3 months ago, but now present on both sides; tactile sensation fairly normal, but pins can be pricked into skin of legs and arms without causing pain; sudden calls to micturate, and occasional slight involuntary action of bladder</td>
<td>Two years and nine months ago, without injury or obvious cause, left knee swelled up, and reached large size in 3 days; there was some redness of skin, and slight pain first. No swelling of thigh or leg. Swelling of joint subsided in a few weeks, but it had returned from time to time since, and the joint had gradually been getting weaker.</td>
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<tr>
<td>Mary Winder. Under the care of Dr. S. H. Moore, at the Chelsea Infirmary. Report taken in Dec., 1886</td>
<td>F. 58 years</td>
<td></td>
<td>Lightning pains in both knees and both legs for six years; more marked on left side. Numbness in sole of left foot for 1 month. Pupils are of medium size and equal; they contract readily in association with accommodation, but react very slightly, if at all, to light. Sensation to tactile, and painful impressions, impaired on left leg and foot. Cold spoon not felt on left leg; hot spoon felt as a slight prick on left leg. The same temperature was readily distinguished, and fell as pain on right leg. Sensation not delayed. Gait ataxic; power of balance much impaired; no knee-jerks obtainable</td>
<td>Eight years ago left knee commenced swelling gradually, and soon after the right knee also swelled. Micturating pain in joints for years after onset, at first only of a darting character.</td>
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<tr>
<td>James Fare. Under the care of Dr. A. H. Robinson, at the Mile End Infirmary. Case taken Feb. 22nd, 1887</td>
<td>M. 46 years</td>
<td></td>
<td>Lightning pains in legs commenced 15 years ago. Ataxic gait and sensation of &quot;pins and needles&quot; in feet 10 years ago. Gastric crises began 9 years ago, and now recur about every 3 months. Loss of the power of appreciating the position of his legs for 5 or 6 years. Sensation impaired in hands for 5 years. Sexual power and desire lost for 4 or 5 years. Sudden and urgent calls to micturate during last 2 years. Pupils of medium size, equal, and react neither to light nor with accommodation. Tactile sensibility impaired in hands and forearms, and</td>
<td>Ten years ago, without any assignable cause, patient's left leg swelled up suddenly and painlessly. This swelling subsided in a week, and then his left arm swelled up suddenly and painlessly. Three months after the onset of the swelling the condition of the left knee...</td>
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</table>
Joint Disease in connection with Locomotor Ataxy.

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<thead>
<tr>
<th>Description of joints</th>
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<tr>
<td><strong>Left knee</strong> is enlarged; no fluid in the joint. Internal tuberosity of tibia enlarged; condyles of femur enlarged, and border of articular surface lipped; patellar not enlarged; coarse grating on moving joint. No bony growth in capsule. No dislocation, but well-marked lateral mobility in joint. No osteo-arthritis of any other joints.</td>
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<tr>
<td><strong>Left knee</strong> much enlarged, owing to great increase in size of bones and to fluid in the joint. Massive osteophytes attached to bones and others in the soft tissues of the joint. Free lateral mobility. No dislocation, but when patient stands the leg bends outwards, and forms an angle of 135°. with thigh on outer side of limb.</td>
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<tr>
<td><strong>Left elbow</strong> is much deformed by massive osteophytes attached to the lower end of the humerus; little or no fluid found in the joint; free unnatural mobility accompanied by coarse grating in the joint; no osteophytes in capsule.</td>
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<tr>
<td><strong>Left wrist</strong> enlarged by fluid, and osteophytic growths attached to the bones and the capsule; coarse grating on movement.</td>
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<tr>
<td><strong>Left shoulder</strong> much swollen from fluid in the joint; head of humerus dislocated beneath the spine of the scapula; free mobility at the joint. Masses of new bone attached to acromion and infraspinous fossa. Head of humerus much eroded; coarse grating produced by movement.</td>
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<tr>
<td>Perforating ulcers beneath both great toes, and on ball of right little toe; 3 years ago they had healed but they had broken open again from time to time since; they are now healed and the epithelium at the site of them is much thickened. The usual signs of locomotor ataxy are absent, with exception of the symptoms mentioned; no history of syphilis.</td>
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<tr>
<td>Bladder and rectum normal; no gastric crises; no perforating ulcers; skin of legs, especially the left, dry and scaly; no history of syphilis.</td>
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<td>Patient had a venereal sore 19 years ago, but no history of secondary syphilis could be obtained. Scratching the skin of the trunk and limbs is followed by the appearance of long-persisting wheals. No perforating ulcers.</td>
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<tr>
<td>Mary Ann Dowsett.</td>
<td>F. 50</td>
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<tr>
<td>Under the care of Dr. C. St. J. Wright, at the Islington Infirmary. Case taken March 21, 1887</td>
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<tr>
<td>Jane White.</td>
<td>F. 40</td>
</tr>
<tr>
<td>Under the care of Mr. J. R. Lunn, at the St. Marylebone Infirmary. Case taken April 21, 1887</td>
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<tr>
<td>Description of joints</td>
<td>Remarks</td>
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<tr>
<td>Left knee is enlarged, owing to hypertrophy of the bones and to osteophytic growths; the borders of the articular surface of the femur are &quot;lipped,&quot; as in typical osteoarthritis; there is much unnatural mobility. There is very little destruction of the articular ends of the bones; coarse grating on movement.</td>
<td>No history of syphilis. No perforating ulcers. Patient has been bedridden for nearly 6 years.</td>
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<tr>
<td>Right shoulder much swollen from fluid in the joint; head and tuberosities of humerus worn away; right humerus 1 inch shorter than its fellow, and its upper end can be displaced in all directions. No osteophytic growths.</td>
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<tr>
<td>Left shoulder grates on movement, but there is no deformity.</td>
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<tr>
<td>Tip-joints are both disorganised; the head and neck of each is absent; unnatural mobility at the joints; no osteophytic growths, and little or no fluid in the joints; coarse grating produced by movement.</td>
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<tr>
<td>Right knee is not swollen, but the ligaments are lax and allow of partial dislocation and unnatural mobility; grating on movement. No osteophytic growths</td>
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<tr>
<td>Left foot.—The anterior tarsal bones were enlarged, and caused much prominence on the dorsum; the sole of the foot was flattened; there was no pain; osteophytic growths projected from the astragalus and the scaphoid</td>
<td>Perforating ulcers formed on the soles of the feet 2 years ago, and again one year ago.</td>
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<tr>
<td>Both feet were similarly affected, they were much arched, and the bones of the tarsi in front of the astragalus stood out prominently on the dorsum. The joints were rigid but painless</td>
<td>Pupils contracted both to light and during accommodation. No gastric crises.</td>
</tr>
<tr>
<td>Name and reference</td>
<td>Sex and age</td>
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<td>—</td>
<td>M. 40</td>
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<tr>
<td>Under the care of Dr. Buzzard. Reported in the Lancet, Aug. 22, 1874, p. 261</td>
<td>F. 61</td>
</tr>
<tr>
<td>Hortense B. Under the care of Professor Charcot. Reported in Medical Times and Gazette, 1877, vol. i, p. 640</td>
<td>M. 30</td>
</tr>
<tr>
<td>Under the care of Dr. Buzzard. Reported in the Lancet, Jan. 18, 1879, p. 76</td>
<td>M. 60</td>
</tr>
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Joint Disease in connection with Locomotor Ataxy.

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<td>The right lower limb begins to swell a short distance below the groin. Opposite the knee the circumference is 5(\frac{1}{2}) inches more than on the opposite side. The swelling ceases suddenly 2 inches below the patella. Ligaments of knee relaxed, so that the leg forms an angle of 45° with the thigh on the outer side. Grating felt in the joint on movement.</td>
<td>Patient had been bedridden for 6 years (since the dislocation of the right hip).</td>
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</tbody>
</table>

Right hip not described.
Right knee not described.
Left shoulder much swollen, due to fluid effusion within the joint. The head of the humerus had disappeared, and the upper end of the shaft was very freely movable.
Right shoulder.—Cracklings felt on movement

Has had syphilis.

Right hip is much swollen from fluid in the joint; the head and neck of the femur are absent, and the trochanter is much raised. The movements at the joints are unnaturally free. Crepitus felt in the joint on movement. The limb is 2 inches shorter than its fellow in the recumbent, and 3 inches shorter in the erect posture. There is a spicule of bone 9 inches long apparently in the superior tendon of the rectus femoris

Left knee.—The joint was swollen, measuring 2\(\frac{1}{2}\) inches more in circumference than its fellow. Capsule thickened. Ligaments relaxed, allowing abduction of tibia to angle of 150° with femur; over-extension and free rotatory movements could be made. Grating felt on movement

No gastric crises.
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<tr>
<td>Henry H. Reported in the British Medical Journal, 1881, vol. i, p. 331, by Dr. Buzzard</td>
<td>M. 62 years</td>
<td>10 years</td>
<td>Gastric crises commenced 10 years ago; he had had 6 attacks in all; they lasted from 6 weeks to 3 months at a time, and obliged him to keep his bed. Lightening pains began in legs 3 or 4 years ago, and eventually extended to all parts of body. Pupils do not contract to light nor during accommodation. Tactile sensibility not impaired. Plantar reflexes excessive. Knee-jerks absent</td>
<td>Nearly 3 years at the left shoulder, whole of the upper arm and the adjacent part of the chest swelled. Pus was discharged near the elbow</td>
</tr>
<tr>
<td>Matilda H. Under the care of Dr. Radcliffe. Reported in the British Medical Journal, 1881, vol. i, p. 331, by Dr. Buzzard</td>
<td>F. 50 years</td>
<td>17 years</td>
<td>Gastric crises commenced 17 years ago, and had been more severe during last 2 years. &quot;Rheumatic pains&quot; in arms and legs for 15 years, and boring pains for 3 years; they had not affected her hip-joints. Gait atactic for 5 years. Pupils equal; they contract during accommodation, but not to light. Some loss of cutaneous sensibility in legs, and slight delay in appreciating painful stimuli. Marked hyperalgesia to heat in legs, but appreciation of the heat is delayed 3 seconds. Knee-jerks absent</td>
<td>Eighteen months at the left hip-joint went away with a snap, leaving without pain. Swelling at once disappeared in the groin. A few months later the left leg was shorter than the right</td>
</tr>
<tr>
<td>B. W. Under the care of Dr. Dreschfeld. Reported in the Lancet, July 10, 1880, p. 51</td>
<td>M. 50 years</td>
<td>15 years</td>
<td>Inability to walk in the dark and lightning-pains in the legs 15 years ago. Pupils contracted, react with accommodation but not to light. Marked atrophy of both optic discs. Upper limbs normal.Diminution of tactile sensibility in lower limbs, analgesia of certain spots, and retardation of sensibility. Muscular sense in lower limbs impaired. Gait atactic. Inability to walk with eyes shut</td>
<td>Seven years ago pains in the right thigh became more persistent. The patient remained in bed for a month, during which time the pain suddenly left him. and on trying to get up he found his right leg much shorter than the left. Three years ago the left knee began to give way without pain or swelling, and backward dislocation of the tibia gradually took place</td>
</tr>
<tr>
<td>— Shown at the International Medical Congress in London, Aug., 1881, by Mr. H. W. Page. Transactions, vol. i, p. 124</td>
<td>M. 44 1/2 years</td>
<td>30 years</td>
<td>Pains like &quot;jumping toothache&quot; in his legs 4 1/2 years ago. Gastric crises 2 years ago. Argyll-Robertson papillary symptoms. Doubtful anaesthesia of the sole of the right foot. Knee-jerks absent</td>
<td>Nine months ago right leg and foot swelled, and were at first pain, but the swelling continued to increase. Some months later the left foot was similarly affected, very rapidly and painlessly</td>
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### Description of joints.

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<tr>
<td><strong>Left shoulder</strong></td>
<td>The joint is enlarged, and apparently contains fluid. It is abnormally movable, and crepitus can be felt. The head of the humerus has apparently disappeared.</td>
</tr>
<tr>
<td><strong>Left hip</strong></td>
<td>The trochanter is raised and the limb shortened. The head of the femur is apparently absent. There is abnormal mobility at the joint.</td>
</tr>
<tr>
<td><strong>Right hip</strong></td>
<td>The head of the femur was dislocated on to the dorsum ili; it was freely movable and could easily be reduced. The head of the femur was not atrophied, and there was no deposit of new bone about the joint.</td>
</tr>
<tr>
<td><strong>Left knee</strong></td>
<td>The tibia was dislocated forwards, there was no atrophy of the articular surfaces, and no deposit of new bone. There was a mass of bone 1 inch long in the sheath of the sartorius muscle.</td>
</tr>
</tbody>
</table>

The cuboid, scaphoid, three cuneiform, and metatarsal bones of both feet are enlarged, and freely movable on one another in all directions. Handling the feet was painless.

The joint-affection probably began with a spontaneous fracture of the neck of the femur.

Gait not ataxic.

No history of syphilis. No gastric crises.

Gait not ataxic. A month after onset in right foot there were broken corns on the sole of the right foot, and an ulcer on the tip of the great toe. A month later several "gathered corns" had appeared on the left foot.
### Evidences of locomotor ataxy.

<table>
<thead>
<tr>
<th>Name and reference</th>
<th>Sex and age</th>
<th>Duration of disease</th>
<th>Evidences of locomotor ataxy.</th>
</tr>
</thead>
<tbody>
<tr>
<td>J. W. Shown at the International Medical Congress in London, Aug., 1881, by Mr. C. Macnamara. Transactions, vol. i., p. 125. Date of Report, May 18, 1881</td>
<td>M. 6 or 7 years 50 years</td>
<td>Severe lightning pains, dating from 6 or 7 years ago. Pupils contracted to the size of a pin’s head, inactive to light, but alter in size during accommodation. Sensibility of limbs normal. Gait ataxic. Knee-jerks absent. Optic discs pale.</td>
<td></td>
</tr>
<tr>
<td>J. P. Shown at the International Medical Congress in London, Aug., 1881, by Mr. C. Macnamara. Transactions, vol. i., p. 125. Date of Report, June 15, 1881</td>
<td>M. ? or 4 years 48 years</td>
<td>Had suffered for 12 years from rheumatic pains, which for 3 or 4 years had been sudden and very acute. Optic discs pale. No other symptoms observed.</td>
<td></td>
</tr>
<tr>
<td>Frederick G. Reported in the British Medical Journal, 1884, vol. i, p. 221, by Dr. Barrs</td>
<td>M. 2½ years 42 years</td>
<td>Two and a half years ago he began to notice difficulty in walking in the dark, and to suffer from intermittent shooting pains, called rheumatic, in both legs. The attacks of pain were always preceded by attacks of coughing, followed by epigastric pain and retching with vomiting of food. Both pupils were contracted, and the right more so than the left. They did not react to light, but they contracted during accommodation. Sensation much blunted from the soles of the feet to the middle of the calves. There was marked analgesia over the same area. There was no obvious ataxy. The plantar reflexes and the knee-jerks were absent.</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>Duration and mode of onset of joint disease.</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>Four months ago the shooting pains in his legs being very severe, his right hip and thigh became suddenly much swollen. There was no great pain in the part.</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>Three and a half months ago, without known cause, the right hip and thigh from groin nearly to knee swollen up suddenly with but little pain. The swelling gradually subsided, but the patient was left lame.</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>One year ago the right toe swelled up suddenly and painlessly and one week later the right hip and thigh swelled enormously. Two months later the joint was loose and painless; it grated during movement, and the limb was shortened. The left hip was attacked like the right 6 weeks ago and has passed through the same stages as it did.</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>Eighteen months ago the left thigh from the knee to the hip began to swell. Six months later the limb was still swollen, and the hip-joint was painful.</td>
<td></td>
</tr>
</tbody>
</table>
**Description of Joints.**

| Right hip.—Considerable swelling about the joints owing to displacement of trochanter upwards and backwards, and to much induration around the joints and within the pelvis. Right lower limb ½ inch shorter than left. It was neither inverted nor everted. The hip joint permitted a flail-like movement without causing pain. |
| Right hip.—There was much induration and swelling round the hip and within the brim of the pelvis. The trochanter was displaced upwards and backwards, and the limb was ½ inch shorter than its fellow. The joint could be moved in any direction without pain. |
| Right great toe-joints not described. |
| Hip-joints.—The great trochanter on each side is 1 inch or more higher than natural, and the heads of both femora are partially or entirely absorbed. |
| Left hip.—The whole thigh was swollen, but the upper and outer part, and the region of the joint, was occupied by a large fluid swelling beneath the muscles. There was unnaturally free mobility at the joint. Movements were quite painless and attended by dull grating. |

<p>| Remarks. |
| No gastric crises. No history of syphilis. |
| No history of syphilis. An ulcer formed at the site of a corn beneath the right great toe just before the affection of its joints. |
| No history of syphilis. |</p>
<table>
<thead>
<tr>
<th>Name and reference</th>
<th>Sex and age</th>
<th>Duration of disease</th>
<th>Evidences of locomotor ataxy</th>
<th>Duration and mode of onset of joint disease</th>
</tr>
</thead>
<tbody>
<tr>
<td>Reported by P. H. Kidd in the <em>Lancet</em>, July 12, 1884, p. 53</td>
<td>M. 42</td>
<td>1½ years</td>
<td>Lightning pains in legs for 1½ years. Incontinence of urine for 3 weeks. No difficulty in walking experienced by patient. Pupils small; contraction to light and during accommodation impaired, and particularly so to light. Atrophy of left optic disc. Tactile sensibility fairly good in soles of both feet, but perception of sensations is delayed. Analgesia of both feet. Unsteadiness when eyes are closed. Knee-jerks absent</td>
<td>Crackling in left hip-joint was noticed shortly after the onset of lightning pains. Swelling of left hip and upper half of thigh soon followed. Four months ago crackling commenced in right hip-joint, but there was no swelling</td>
</tr>
<tr>
<td>G. N. Reported in <em>Trans. Clin. Soc.</em>, 1884, p. 118, by Mr. Lucas</td>
<td>M. 30</td>
<td>4 or 5 years</td>
<td>Well-marked lightning pains, termed rheumatic, in upper and lower limbs, for 4 or 5 years. Occasional numbness in hands, especially the right, for 12 to 18 months. Giddiness in the morning for 9 months. Difficulty of walking in the dark for 6 or 7 months. Pupils unequal, inactive to light, and the left one contracts imperfectly during accommodation. Sensation in hands is slightly delayed. Sensation in feet is normal. Slight incoordination of movements of both upper and lower limbs. Knee-jerks absent. Sexual desire is impaired</td>
<td>About 2 years ago the right elbow enlarged without much pain and without superficial inflammation. Two months ago the right foot became swollen and painful</td>
</tr>
<tr>
<td>Eliz. C. Under the care of Mr. Jalland. Reported in the <em>Lancet</em>, Jan. 17, 1885, p. 106</td>
<td>F. 48</td>
<td>11 weeks</td>
<td>The first symptom noticed by the patient was the affection of her left hip-joint. Pupils dilated and inactive to light. Sensibility in the left lower limb is impaired. Knee-jerks and plantar reflexes on both sides are absent</td>
<td>Eleven weeks ago, whilst scrubbing a floor, the left hip became suddenly painful. Patient took to her bed at once. A fortnight later sudden pain and swelling attacked the left knee</td>
</tr>
<tr>
<td>W. C. Reported in the <em>Lancet</em> 1885, vol. i, p. 890, by Dr. Brooks</td>
<td>M. 40</td>
<td>2½ years</td>
<td>Gnawing pains in both legs at intervals between 2 and 3 years. Inability to walk steadily in the dark for less than 2 months. Pupils were unequal, and did not respond to light, but contracted during accommodation. Tactile sensibility was normal. Slight incoordination of movements of walking. Knee-jerks absent</td>
<td>Two months ago the left knee was found swollen, and it had since got gradually larger. The joint affection had been painless from the first</td>
</tr>
</tbody>
</table>
Joint Disease in connection with Locomotor Ataxy.

<table>
<thead>
<tr>
<th>Description of joints.</th>
<th>Remarks.</th>
</tr>
</thead>
<tbody>
<tr>
<td>Loud crackling sounds caused by movement at each hip-joint</td>
<td>No history of syphilis. No gas-tric crises. The onset of the disease of each hip was soon followed by wasting of the muscles of the corresponding thigh and leg.</td>
</tr>
<tr>
<td><strong>Right elbow</strong> measures 2½ inches more in circumference than the left. The swelling is due to massive osteo-phytic growths and bursal enlargements. The joint is painless during movement and manipulation. The osteo-phytic growths prevent pronation and supination, and limit extension. Right tarsus.—The dorsum of the foot is swollen, but it is not oedematous and not tender. There is aching pain beneath the head of the astragalus.</td>
<td>Patient had a chancre on his penis, but had no symptoms of secondary syphilis. No history of gastric crises. No perforating ulcers.</td>
</tr>
<tr>
<td><strong>Left hip.</strong>—About a month after onset of joint affection the head of the femur was dislocated on the dorsum illi. It could be easily reduced, with grating sensation and without pain.</td>
<td>There were neither lightning pains nor ataxic gait up to the time of the report. The evening temperature ranged between 101° F. and 102° F., whilst the knee-joint was much swollen.</td>
</tr>
<tr>
<td><strong>Left knee.</strong>—About a fortnight after onset, on removing splint, the tibia suddenly became partially dislocated backwards, without pain and with grating in the joint. There was a collar of osteophytic growths around the upper end of the tibia. Osteophytic growths felt in right iliac region and about upper third of linea aspera of right femur.</td>
<td>No gastric crises. No history of syphilis.</td>
</tr>
<tr>
<td><strong>Left knee</strong> was 2 inches larger in circumference than the right, owing to fluid effusion in the joint. There was semi-solid oedema of the upper third of the leg. The opposed ends of the femur and tibia were slightly enlarged, but there were no osteophytic growths. There was partial inward and backward dislocation at the joint. The ligaments were relaxed, and allowed of unnatural movements which were accompanied by loud crackling sounds.</td>
<td></td>
</tr>
<tr>
<td>Name and reference</td>
<td>Sex and age</td>
</tr>
<tr>
<td>-------------------</td>
<td>-------------</td>
</tr>
<tr>
<td>Reported by Dr. Buzzard, in his <em>Clinical Lectures on Diseases of the Nervous System</em>, 1882 (pp. 256—259), and by Dr. Hadden in <em>Trans. Path. Soc.</em>, 1886, p. 103. History and present state taken Nov., 1881. Abstract taken from Dr. Hadden's Report</td>
<td>M. 10 years</td>
</tr>
</tbody>
</table>
**Joint Disease in connection with Locomotor Ataxy.**

<table>
<thead>
<tr>
<th>Description of joints.</th>
<th>Remarks.</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Left knee</strong> much enlarged, joint cavity full of fluid; tibia partially dislocated backwards and inwards; joint flail like; grating on movement. Patella irregularly enlarged. Numerous bony outgrowths along the margins of the articular surfaces of femur and tibia, and one in the capsule. Moderate sclerosis of the articular ends of the bones. There was a bony outgrowth in the body of the semi-membranosus, extending upwards from the joint for several inches.</td>
<td>No history of syphilis. Perforating ulcer beneath right great toe. A black spot formed at this place 2 years ago, but the ulcer only appeared 6 weeks ago. The peri- and endo-neurium of the internal and external popliteal and the musculo-cutaneous nerves were thickened.</td>
</tr>
<tr>
<td><strong>Left shoulder</strong> much enlarged, distended with fluid, and quite disorganised. Movements very free and attended with grating. The glenoid cavity has disappeared, and the axillary border of the scapula runs up into the broadened coracoid process. The acromion process is much broader than natural. The capsule is strengthened by plates of bone, some of which, together with the acromion, coracoid process, and axillary border of the scapular are eroded. The head and tuberosities of the humerus are worn away, the exposed surface is only slightly eburnated. There are numerous loose bodies within the joint, and numerous polypoid growths, partly bony and partly cartilaginous, project into its cavity. The bursa beneath the infraspinatus communicates with the joint, and its inner surface is studded with growths like those in the joint. The appearances described above were observed post-mortem.</td>
<td>The motor cells in both the lumbar and cervical enlargements were less numerous on the left side than the right. No track of degeneration traceable from the sclerosed posterior or lateral columns to the diseased anterior cornua in either the lumbar or the cervical regions. The right elbow-joint and the right knee were diseased, but the description of them is scanty.</td>
</tr>
</tbody>
</table>
### Table II.—An Analysis of 66 Cases of Charcot's Joint Disease, in Mr. Morrant Baker's Paper, and 21 Cases described in Mr

<table>
<thead>
<tr>
<th>Name</th>
<th>Sex</th>
<th>Duration of locomotor ataxy at onset of joint affection</th>
<th>Nature of onset of joint affection</th>
<th>Condition of joints as regards pain</th>
<th>Gastric crises</th>
</tr>
</thead>
<tbody>
<tr>
<td>E. M.</td>
<td>F.</td>
<td>Both hip-joints, &quot;many years&quot;; right elbow, 10 years; right metatarsal-phalangetal joint of great toe</td>
<td>The disease of the right elbow followed a slight injury. The joint was found disorganised 12 months after onset</td>
<td>—</td>
<td>Present for 16 years before affection of elbow-joint</td>
</tr>
<tr>
<td>W. E.</td>
<td>M.</td>
<td>Right knee</td>
<td>Sudden, after a severe twist, accompanied by a crack in the joint</td>
<td>Not much pain after 3 weeks</td>
<td>Absent</td>
</tr>
<tr>
<td>J. G.</td>
<td>M.</td>
<td>Right knee, 1½ years</td>
<td>Sudden. The joint was painfully injured 6 mos. before onset. Joint disorganised 3 months after onset</td>
<td>Painless</td>
<td>Absent</td>
</tr>
</tbody>
</table>

**Patients exhibited a**

<table>
<thead>
<tr>
<th>Name</th>
<th>Sex</th>
<th>Duration of joint affection</th>
<th>Nature of onset of joint affection</th>
<th>Condition of joints as regards pain</th>
<th>Gastric crises</th>
</tr>
</thead>
<tbody>
<tr>
<td>Elizabeth</td>
<td>F.</td>
<td>Right knee, 22 years</td>
<td>Sudden. The joint was found disorganised 12 months after onset</td>
<td>Painless</td>
<td>Present for 4 years before onset of joint affection</td>
</tr>
<tr>
<td>Neale</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Christina</td>
<td>F.</td>
<td>Right knee, 10 years; left knee, 10 years; right shoulder, 1½ years</td>
<td>Gradual in left knee; sudden in both other joints. Rapid disorganisation of all the joints</td>
<td>Painless</td>
<td>Present for 10 years before onset of disease of knee-joints</td>
</tr>
<tr>
<td>Metcalf</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Janet</td>
<td>F.</td>
<td>Left knee, 6 years</td>
<td>Sudden after a fall</td>
<td>Painless</td>
<td>Began 8½ years after onset of joint affection</td>
</tr>
<tr>
<td>Saeger</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
Joint Disease in connection with Locomotor Ataxy.

Including all the cases contained in Table I, 3 cases described cases collected from foreign sources.

Morrant Baker's Paper.

<table>
<thead>
<tr>
<th>Lightning pains in limbs or not before joint affection.</th>
<th>Ataxy present or not in affected limbs before joint affection.</th>
<th>Perforating ulcers.</th>
<th>New bony formations.</th>
<th>Remarks.</th>
</tr>
</thead>
<tbody>
<tr>
<td>Present in legs from onset of disease. No mention with regard to arms</td>
<td>Present in lower limbs for 17 years before affection of elbow-joint</td>
<td>On right great toe</td>
<td>Hard nodules about elbow-joint, none about hips or great toe</td>
<td>—</td>
</tr>
<tr>
<td>Lightning pains in all parts of body; date of onset not stated</td>
<td>—</td>
<td>On right great toe</td>
<td>Massive osteophytes in the capsule and about the upper end of the tibia. No general enlargement of bones entering into joint</td>
<td>Feet always damp from perspiration. Tabes dorsalis verified post mortem. No neuritis of post. tibial nerve.</td>
</tr>
<tr>
<td>Shooting pains in right leg for 1½ years before onset of joint affection</td>
<td>—</td>
<td>Absent</td>
<td>Some osteophytic growths. Slight lipping of the articular surfaces of the femur and tibia</td>
<td>The left knee presented the ordinary appearance of early osteo-arthritis post-mortem.</td>
</tr>
</tbody>
</table>

The Clinical Society.

<table>
<thead>
<tr>
<th>Lightning pains in lower limbs for 22 years before</th>
<th>Present for 22 years before</th>
<th>Absent</th>
<th>Patella and condyles of femur slightly enlarged. No osteophytes.</th>
<th>—</th>
</tr>
</thead>
<tbody>
<tr>
<td>Present for 8 years before in lower limbs, and for a somewhat shorter time before in upper limbs</td>
<td>Present in legs for 8 years before; absent in arms</td>
<td>Absent</td>
<td>Slight and mainly in the capsule.</td>
<td>—</td>
</tr>
<tr>
<td>Present in legs for 6 years before</td>
<td>Present for one year before</td>
<td>Absent</td>
<td>The ends of the bones entering into the joints are enlarged; no osteophytes</td>
<td>There was some puffiness about the right knee, and the joint creaked on movement.</td>
</tr>
<tr>
<td>Name</td>
<td>Sex and age</td>
<td>Duration of locomotor ataxy at onset of joint affection</td>
<td>Nature of onset of joint affection</td>
<td>Condition of joints as regards pain</td>
</tr>
<tr>
<td>--------------</td>
<td>-------------</td>
<td>--------------------------------------------------------</td>
<td>-----------------------------------</td>
<td>-------------------------------------</td>
</tr>
<tr>
<td>Mary Hollis</td>
<td>F. 47</td>
<td>Left knee, 7 years</td>
<td>Gradual</td>
<td>Rather painful at onset, but quite painless at time of observation</td>
</tr>
<tr>
<td>— Frewing</td>
<td>F. 37</td>
<td>Left metatarsophalangeal joint of great toe, 5 years; right metatarsophalangeal joint of great toe commencing</td>
<td>Gradual</td>
<td>None at time of observation</td>
</tr>
<tr>
<td>Josiah Pickford</td>
<td>M. 60</td>
<td>Left hip, 12½ years</td>
<td>Sudden; the joint was disorganised in 3 weeks</td>
<td>Painless</td>
</tr>
<tr>
<td>Frederick Waters</td>
<td>M. 30</td>
<td>Left knee, 3½ years</td>
<td>Rapid; the joint was disorganised in 3 weeks</td>
<td>Rather painful at onset; quite painless at time of observation</td>
</tr>
<tr>
<td>William Jermy</td>
<td>M. 45</td>
<td>Both elbow joints. The joint affection began 1½ years before the symptoms of locomotor ataxy were noticed</td>
<td>Sudden. Dislocation probably occurred in a week after onset</td>
<td>Painless</td>
</tr>
<tr>
<td>Elizabeth Watson</td>
<td>F. 55</td>
<td>Left hip, 10 years</td>
<td>Sudden</td>
<td>Painless</td>
</tr>
</tbody>
</table>

*Other Cases observed:*
### Joint Disease in connection with Locomotor Ataxy

<table>
<thead>
<tr>
<th>Lightning pains in limbs or not before joint affection.</th>
<th>Ataxy present or not in affected limbs before joint affection.</th>
<th>Perforating ulcers.</th>
<th>New bony formations.</th>
<th>Remarks.</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Present in legs for 7 years before</strong> Began slightly 7 years before</td>
<td>Absent</td>
<td>The ends of the bones entering into the joint are much enlarged; no osteophytes</td>
<td>The right knee creaked on movement.</td>
<td></td>
</tr>
<tr>
<td><strong>Present in the left leg for 3 years before onset of joint affection</strong> Absent, but patient had had some difficulty in walking for 4 years before</td>
<td>Formed on right great toe 14 mos. before joint affection</td>
<td>None</td>
<td>Suppuration occurred in the left joint; analgesia more marked in left leg and foot than in right.</td>
<td></td>
</tr>
<tr>
<td><strong>Present in legs for 12½ years before</strong> Absent</td>
<td>Absent</td>
<td>Trochanter and horizontal ramus of pubes much enlarged. A spicule of bone 6 inches long, extends down thigh from front of iliac crest</td>
<td>Pupils react to light; gait not ataxic; analgesia of left leg and foot, but not of right.</td>
<td></td>
</tr>
<tr>
<td><strong>Present in legs for 2½ years before</strong> None up to time of observation</td>
<td>First formed 3½ years before onset of joint affection</td>
<td>Slight enlargement of lower end of femur; much enlargement of upper end of tibia; osteophytic growth from tibia</td>
<td>Commenced with atony of the bladder and incontinence of urine. Many of the usual symptoms were absent.</td>
<td></td>
</tr>
<tr>
<td>They did not occur till 18 months after</td>
<td>On right great toe</td>
<td>Osteophytic mass in capsule of left elbow-joint</td>
<td>This patient had well-marked locomotor ataxy at the time of observation. Pupils reacted to light.</td>
<td></td>
</tr>
</tbody>
</table>

**Remarks:**

- There was no anesthesia. Spontaneous fracture of the right thigh occurred 1 year before joint affection.

*This Committee.*

**Present for 10 years before** Present for 3 or 4 years before Absent | Left great trochanter much enlarged |
<table>
<thead>
<tr>
<th>Name</th>
<th>Sex and age</th>
<th>Duration of locomotor ataxy at onset of joint affection</th>
<th>Nature of onset of joint affection</th>
<th>Condition of joints as regards pain</th>
<th>Gastric complaint</th>
</tr>
</thead>
<tbody>
<tr>
<td>Henry Gigg</td>
<td>M. 51</td>
<td>Right hip. The symptoms of locomotor ataxy were observed at the time of onset of the joint affection</td>
<td>Gradual, after a fall</td>
<td>Painless</td>
<td>Began after 3 years after affection</td>
</tr>
<tr>
<td>John Griffiths</td>
<td>M. 43</td>
<td>Right knee, 2½ years; left hip 5 years</td>
<td>Sudden in knee; more gradual in hip</td>
<td>Painless</td>
<td>Absent</td>
</tr>
<tr>
<td>F. Stokes</td>
<td>M. 42</td>
<td>Right elbow, 1½ years</td>
<td>Gradual. The swelling was greatest 6 weeks after onset, and the joint was quite disorganised 6½ months after</td>
<td>Painless</td>
<td>Present for many years before joint affection</td>
</tr>
<tr>
<td>F. Weaver</td>
<td>M. 53</td>
<td>Right knee, 6½—8½ years</td>
<td>Sudden, with swelling of whole limb</td>
<td>—</td>
<td>Had an attack of acute gastric pain which occurred under observation</td>
</tr>
<tr>
<td>J. Austin</td>
<td>M. 63</td>
<td>Right knee, 10½ years</td>
<td>Gradual. Increased swelling after a fall 3 months after onset</td>
<td>Painless</td>
<td>—</td>
</tr>
<tr>
<td>James Rawlings</td>
<td>M. 42</td>
<td>Left knee (?) right knee (?)</td>
<td>Rapid in left knee after a fall</td>
<td>Painful at onset and since</td>
<td>—</td>
</tr>
<tr>
<td>E. Bewry</td>
<td>F. 48</td>
<td>Right knee, 6 years</td>
<td>Sudden after a hard day's work</td>
<td>Slight pain when much swollen at onset, but painless later on</td>
<td>—</td>
</tr>
</tbody>
</table>
### Joint Disease in connection with Locomotor Ataxy.

<table>
<thead>
<tr>
<th>Lightning pains in limbs or not before joint affection.</th>
<th>Ataxy present or not in affected limbs before joint affection.</th>
<th>Perforating ulcers.</th>
<th>New bony formations.</th>
<th>Remarks.</th>
</tr>
</thead>
<tbody>
<tr>
<td>They occurred simultaneously</td>
<td>Absent before onset of joint affection</td>
<td>Absent</td>
<td>None detected.</td>
<td></td>
</tr>
<tr>
<td>Present in legs for 1½ years before joint affection</td>
<td>Absent up to date of observation</td>
<td>Absent</td>
<td>Massive bony growths about hip. Enlargement of bones entering into knee-joint</td>
<td></td>
</tr>
<tr>
<td>Shooting pains before 1½ years before joint affection</td>
<td>Absent up to date of observation</td>
<td>On ball of right great toe 6 mos. before joint affection</td>
<td>Lower end of humerus irregularly enlarged. Bony nodule in capsule</td>
<td>The lesion of tabes dorsalis was not found post mortem.</td>
</tr>
<tr>
<td>Began 6½—8½ years before</td>
<td>Well marked at time of observation. Date of onset not known</td>
<td>On ball of right great toe 2½ years before joint affection</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Began 10½ years before</td>
<td></td>
<td>Absent</td>
<td>Mass of bone on inner side of tuberosity of tibia. Patella enlarged</td>
<td></td>
</tr>
<tr>
<td>Sent for 6 years before</td>
<td>Absent till 7 years afterwards</td>
<td>Absent</td>
<td>Contiguous extremities of femur and tibia enlarged.</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>Absent</td>
<td>Upper end of tibia much enlarged, and its upper margin is irregular.</td>
<td></td>
</tr>
<tr>
<td>Name</td>
<td>Sex and age</td>
<td>Duration of locomotor ataxy at onset of joint affection</td>
<td>Nature of onset of joint affection</td>
<td>Condition of joints as regards pain</td>
</tr>
<tr>
<td>---------------</td>
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</tr>
<tr>
<td>Ziba Bickett</td>
<td>M. 53</td>
<td>Left shoulder, 17 years; right shoulder, 18 years</td>
<td>Sudden in both joints, and after an injury in left. The left shoulder was found dislocated 6 mos. after onset, and the right one was dislocated and disorganised 4 days after onset</td>
<td>Severe pain (? lightning pains) in left shoulder for 3 hours, but none afterwards. Onset painless in right shoulder</td>
</tr>
<tr>
<td>Justine Smith</td>
<td>F. 56</td>
<td>Right knee, 2 years</td>
<td>Gradual after an injury. The joint was found disorganised 5 months after onset</td>
<td>Painful for 14 days after onset</td>
</tr>
<tr>
<td>J. Taphouse</td>
<td>M. 39</td>
<td>Left knee, (?) 3 months</td>
<td>Sudden. The joint was disorganised in a few weeks</td>
<td>Some pain at first. None at time of observation</td>
</tr>
<tr>
<td>Mary Winder</td>
<td>F. 58</td>
<td>Left knee, right knee</td>
<td>Gradual</td>
<td>Painless</td>
</tr>
<tr>
<td>James Fare</td>
<td>M. 45</td>
<td>Left knee, 5 years; left elbow, a week after knee; right shoulder, right wrist, phalangeal joints, 4 months after elbow</td>
<td>Sudden in left knee and elbow; gradual in the other joints; the left knee and elbow were disorganised in 3 months</td>
<td>Painless</td>
</tr>
<tr>
<td>M. A. Downsett</td>
<td>F. 50</td>
<td>Right hip, 3½ years; right shoulder, 7½ years; left hip, right knee, date of onset not known</td>
<td>Sudden in right hip and right shoulder; gradual in other joints; the right hip was disorganised in one month; the right shoulder was dislocated in a fortnight</td>
<td>The right hip was painful at onset, but painless later on; all the other joints were painless</td>
</tr>
<tr>
<td>Jane White</td>
<td>F. 40</td>
<td>Both tarsi, 2½ years</td>
<td>Gradual</td>
<td>Some pain at onset, but none at time of observation</td>
</tr>
<tr>
<td>J. Marshall</td>
<td>M. 39</td>
<td>Left tarsus, 3 years</td>
<td>Sudden</td>
<td>Absent</td>
</tr>
<tr>
<td>Lightning pains in limbs or not before joint affection</td>
<td>Ataxy present or not in affected limbs before joint affection</td>
<td>Perforating ulcers</td>
<td>New bony formations</td>
<td>Remarks</td>
</tr>
<tr>
<td>-------------------------------------------------------</td>
<td>-------------------------------------------------------------</td>
<td>------------------</td>
<td>---------------------</td>
<td>---------</td>
</tr>
<tr>
<td>Present in upper limbs for 2 years before; in lower limbs probably from onset of disease</td>
<td>Absent in upper limbs, but well marked in lower, which had been affected for 10 or 12 years</td>
<td>Absent</td>
<td>None</td>
<td>Suffered from frequent attacks of diarrhoea.</td>
</tr>
<tr>
<td>Present in legs for 1 year before</td>
<td>No obvious ataxy at date of report</td>
<td>Absent</td>
<td>The bones entering into the joint were enlarged. Osteophytic growth from tibia. Internal tuberosity of tibia enlarged. Condyles of femur enlarged and lipped. No bony growths in capsule</td>
<td>—</td>
</tr>
<tr>
<td>Absent up to date of observation</td>
<td>Absent up to date of observation</td>
<td>On both feet 3 or 4 mos. before onset of joint affection</td>
<td>—</td>
<td>—</td>
</tr>
<tr>
<td>Absent till 2 years after joint affection</td>
<td>Absent at onset of joint affection, but well marked at time of observation</td>
<td>Absent</td>
<td>Bones enlarged, massive osteophytes</td>
<td>The joint affections were the first symptoms noticed.</td>
</tr>
<tr>
<td>Present in legs for 5 years before affection of left knee, and in arms for 5 years before affection of upper limb joints</td>
<td>Appeared in legs at the same time as the affection of the left knee; no ataxy of upper limbs till 7 years after affection of upper limb joints</td>
<td>Absent</td>
<td>Massive bony growths about all the joints except the fingers.</td>
<td>—</td>
</tr>
<tr>
<td>Lightning pains in legs came on 6 months after affection of right hip, and in arms 6 months before affection of right shoulder shooting pains in legs 2½ years before</td>
<td>Appeared in legs 2 years before affection of right hip</td>
<td>Absent</td>
<td>Typical wasting of bone, without any osteophytic growths</td>
<td>The patient had been bedridden for 6 years, viz. ½ year after onset of disease in right hip.</td>
</tr>
<tr>
<td>Present in legs for 3 years before</td>
<td>No obvious ataxy at date of report</td>
<td>On both feet.</td>
<td>—</td>
<td>—</td>
</tr>
<tr>
<td>Present in legs for 3 years before</td>
<td>Probably began at same time as affection of foot</td>
<td>Absent</td>
<td>Osteophytic growths from astragalus and scaphoid.</td>
<td>—</td>
</tr>
</tbody>
</table>
## Cases recorded in London Journal

N.B. The reference to these Cases is given in Table I, where

<table>
<thead>
<tr>
<th>Name</th>
<th>Sex and age</th>
<th>Duration of locomotor ataxy at onset of joint affection</th>
<th>Nature of onset of joint affection</th>
<th>Condition of joints as regards pain</th>
<th>Gastric crises</th>
</tr>
</thead>
<tbody>
<tr>
<td>—</td>
<td>M. 40</td>
<td>Right knee, 5 years</td>
<td>The joint was found disorganised about 13 mos. after onset</td>
<td>Painless</td>
<td>There were no gastric crises, patient suffered from &quot;intestinal crises&quot; at earlier part of illness. Began in the interval between onset of hip and left shoulder.</td>
</tr>
<tr>
<td>Hortense B.</td>
<td>F. 61</td>
<td>Right hip, 7 years; right knee; left shoulder, 10 years; right shoulder</td>
<td>Sudden in hip and left shoulder, and rapid dislocation at both joints (in 1 month at shoulder)</td>
<td>—</td>
<td></td>
</tr>
<tr>
<td>W.</td>
<td>M. 30</td>
<td>Right hip, 4½ years</td>
<td>Sudden; the joint was disorganised in 3 months</td>
<td>Painful at onset, but painless later on</td>
<td>Absent</td>
</tr>
<tr>
<td>J. R. P.</td>
<td>M. 60</td>
<td>Left knee, 13 years</td>
<td>Gradual; the joint was disorganised in 8 months</td>
<td>—</td>
<td>Absent</td>
</tr>
<tr>
<td>Henry H.</td>
<td>M. 62</td>
<td>Left shoulder, 3 years</td>
<td>Sudden; the joint was found disorganised 3 years after onset</td>
<td>—</td>
<td>Began 7 years before joint affection</td>
</tr>
<tr>
<td>Matilda H.</td>
<td>F. 50</td>
<td>Left hip, 15½ years</td>
<td>Sudden, with a spontaneous fracture of the neck of the femur</td>
<td>Painless</td>
<td>Began 15½ years before joint affection</td>
</tr>
<tr>
<td>B. W.</td>
<td>M. 50</td>
<td>Right hip, 8 years; left knee, 12 years</td>
<td>Sudden in hip; more gradual in knee. The right hip was found dislocated one month after onset</td>
<td>Painless</td>
<td>Absent</td>
</tr>
<tr>
<td>—</td>
<td>M. 30</td>
<td>Both tarsi, 4 years</td>
<td>Sudden; the tarsal joints of both feet were disorganised in less than 9 months</td>
<td>Painful at first; but soon painless</td>
<td>An attack 1½ years before joint affection</td>
</tr>
<tr>
<td>J. W.</td>
<td>M. 50</td>
<td>Right hip, 6 years</td>
<td>Sudden; the joint was disorganised in 4 months</td>
<td>No great pain at onset, and painless at date of report</td>
<td>Absent</td>
</tr>
<tr>
<td>J. P.</td>
<td>M. 48</td>
<td>Right hip, 3½ years</td>
<td>Sudden; the joint was disorganised in 3½ mos.</td>
<td>But little pain at onset, and painless at date of report</td>
<td>Absent</td>
</tr>
</tbody>
</table>
Joint Disease in connection with Locomotor Ataxy.

they occur in the same order as in the following table.

<table>
<thead>
<tr>
<th>Lightning pains in limbs or not before joint affection.</th>
<th>Ataxy present or not in affected limbs before joint affection.</th>
<th>Perforating ulcers.</th>
<th>New bony formations.</th>
<th>Remarks.</th>
</tr>
</thead>
<tbody>
<tr>
<td>Present in legs for 5 years before</td>
<td>Present for 5 years before.</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Gait ataxic for 7 years before</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Present in legs for 4½ years before</td>
<td></td>
<td></td>
<td></td>
<td>Patient had been bedridden since affection of right hip.</td>
</tr>
<tr>
<td>Lightning pains in legs for 13 years before</td>
<td>Gait ataxic for 1 year before</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Absent</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>&quot;Rheumatic pains&quot; in legs for 13½ years, and boring pains for 6 months</td>
<td>Gait ataxic for 3½ years before</td>
<td></td>
<td>Spicule of bone, 9 in. long, in rectus femoris; not mentioned about joint. None.</td>
<td></td>
</tr>
<tr>
<td>Present in legs for 8 years before</td>
<td>Gait ataxic for 4 years before</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Lightning pains in legs for 6 years before</td>
<td>Gait ataxic for 4 months after onset; no record as to gait before</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Lightning pains in legs for about 3½ years before</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Absent</td>
<td>On both feet</td>
<td></td>
<td>The anterior tarsal and the metatarsal bones were enlarged.</td>
<td></td>
</tr>
<tr>
<td>Lightning pains in legs for 6 years before</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Lightning pains in legs for about 3½ years before</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

VOL. XX.
<table>
<thead>
<tr>
<th>Name</th>
<th>Sex and age</th>
<th>Duration of locomotor ataxy at onset of joint affection.</th>
<th>Nature of onset of joint affection.</th>
<th>Condition of joints as regards pain.</th>
<th>Gastric crises,</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>J. P.</td>
<td>M. 34</td>
<td>Right great toe, 12 years; right hip, 12 years; left hip, 18 years</td>
<td>Sudden; the right hip was disorganised in 2 months, and the left in 6 weeks</td>
<td>Painless</td>
<td>Absent, but intestinal crises had been present since onset of disease</td>
<td></td>
</tr>
<tr>
<td>Frederick G.</td>
<td>M. 42</td>
<td>Left hip, 1 year</td>
<td>Gradual; the joint was found disorganised 18 months after onset</td>
<td>Some pain 6 months after onset; none at time of observation</td>
<td></td>
<td></td>
</tr>
<tr>
<td>—</td>
<td>M. 42</td>
<td>Left hip, 0; right hip, 14 mos.</td>
<td>Sudden swelling of left hip; gradual onset in right hip</td>
<td></td>
<td>Absent</td>
<td></td>
</tr>
<tr>
<td>G. N.</td>
<td>M. 39</td>
<td>Right elbow, 2½ years; right tarsus, 4½ years</td>
<td>The right elbow was found disorganised 2 years after onset</td>
<td>Little pain in elbow; foot painful; elbow painless at date of report</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Elizabeth C.</td>
<td>F. 48</td>
<td>Left hip, 0; left knee, 14 days</td>
<td>Sudden, with swelling of both joints. The hip was found dislocated a month after onset, and the knee became dislocated a fortnight after</td>
<td>Painful</td>
<td></td>
<td></td>
</tr>
<tr>
<td>W. C.</td>
<td>M. 40</td>
<td>Left knee, 2½ years</td>
<td>Gradual; the joint was disorganised in little more than 2 months</td>
<td>Painless</td>
<td>Absent</td>
<td></td>
</tr>
<tr>
<td>J. A.</td>
<td>M. 52</td>
<td>Left knee, 0</td>
<td>Sudden swelling followed by recovery; gradual 6 months later</td>
<td>Painful at onset</td>
<td>Absent</td>
<td></td>
</tr>
<tr>
<td>—</td>
<td>M. 4 years</td>
<td>Left shoulder, 4 years; right elbow; right knee</td>
<td>Sudden in shoulder</td>
<td></td>
<td>Present for years before affection of shoulder</td>
<td></td>
</tr>
<tr>
<td>Lightening pains in limbs or not before joint affection.</td>
<td>Ataxy present or not in affected limbs before joint affection.</td>
<td>Perforating ulcers.</td>
<td>New bony formations.</td>
<td>Remarks.</td>
<td></td>
<td></td>
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<td></td>
<td></td>
</tr>
<tr>
<td>Pain called &quot;rheumatic&quot; present since onset of disease</td>
<td>—</td>
<td>On right great toe</td>
<td>None mentioned.</td>
<td>—</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Present in legs for one year before</td>
<td>No obvious ataxy at date of report</td>
<td>—</td>
<td>None mentioned.</td>
<td>—</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Began just before affection of left hip</td>
<td>Not noticed by patient</td>
<td>—</td>
<td>—</td>
<td>—</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Lightning pains in upper limbs for 2 or 3 years before affection of elbow, and in lower for 4 or 5 before affection of foot</td>
<td>No ataxic gait, but slight incoordination of movements of upper and lower limbs at date of report</td>
<td>Absent</td>
<td>Massive osteophytic growths about elbow-joint.</td>
<td>—</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Absent up to date of report</td>
<td>Absent up to date of report</td>
<td>—</td>
<td>Osteophytic growths around upper end of left tibia, and in right iliac region, and around right femur</td>
<td>The onset of the joint affection was soon followed by wasting of the muscles of the corresponding leg and thigh.</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Gnawing pains in legs for 2½ years before</td>
<td>Absent at onset of joint affection</td>
<td>Absent</td>
<td>Slight enlargement of opposed ends of tibia and femur; no osteophytes.</td>
<td>—</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Present in all limbs for 2 years before shoulder was affected</td>
<td>Absent in upper limbs</td>
<td>Present</td>
<td>Long spicules of bone in the semimembranosus; numerous osteophytic growths.</td>
<td>—</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

The evening temperature ranged between 101°—102° F., whilst the knee was much swollen.
Cases collected by M. Benjamin Ball (Gazette des Hôpitaux)
Athropathies survenant dans le cours d

<table>
<thead>
<tr>
<th>Name</th>
<th>Sex and age</th>
<th>Duration of locomotor ataxy at onset of joint affection</th>
<th>Nature of onset of joint affection</th>
<th>Condition of joints as regards pain</th>
<th>Gastric crises</th>
</tr>
</thead>
<tbody>
<tr>
<td>Jean P.</td>
<td>M. 38</td>
<td>Right knee, 1½ years; left knee, 3 years</td>
<td>—</td>
<td>Painless</td>
<td></td>
</tr>
<tr>
<td>Françoise</td>
<td>F. 49</td>
<td>Left shoulder, 10 years</td>
<td>Sudden; the joint was disorganised in 2½ mos.</td>
<td>Painless</td>
<td></td>
</tr>
<tr>
<td>Victorine</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Virginie P.</td>
<td>F. 56</td>
<td>Right shoulder, 3½ years</td>
<td>Sudden; the joint was found dislocated and disorganised 2 years after onset</td>
<td>Painless</td>
<td></td>
</tr>
<tr>
<td>Leon C.</td>
<td>M. 43</td>
<td>Right knee, 2 years; left knee, 3 years</td>
<td>Gradual</td>
<td>Painful</td>
<td></td>
</tr>
<tr>
<td>Eugène P.</td>
<td>M. 35</td>
<td>Left knee, 4 years</td>
<td>Sudden</td>
<td>Painless</td>
<td></td>
</tr>
<tr>
<td>B.</td>
<td>F. 56</td>
<td>Left elbow, 10 years</td>
<td>Sudden</td>
<td>Painless</td>
<td></td>
</tr>
<tr>
<td>X.</td>
<td>M. 35</td>
<td>Right knee, 3 years</td>
<td>Sudden</td>
<td>Painless</td>
<td></td>
</tr>
<tr>
<td>Louise L.</td>
<td>F. 56</td>
<td>Right knee, 5 years</td>
<td>Sudden</td>
<td>Painless</td>
<td></td>
</tr>
<tr>
<td>B.</td>
<td>M. 45</td>
<td>Left knee, 6 years</td>
<td>Sudden</td>
<td>Painless</td>
<td></td>
</tr>
<tr>
<td>Célestine G.</td>
<td>F. 42</td>
<td>Left knee, 5 years</td>
<td>Sudden</td>
<td>Painful</td>
<td></td>
</tr>
<tr>
<td>M. G.</td>
<td>M. 50</td>
<td>Left knee (?); right knee, 5 years</td>
<td>—</td>
<td>Painless</td>
<td></td>
</tr>
</tbody>
</table>
Joint Disease in connection with Locomotor Ataxy. 357

1868 and 1869), and by M. Joseph Michel (Étude sur les l'Ataxie Locomotrice Progressive).

<table>
<thead>
<tr>
<th>Lightning pains in limbs or not before joint affection.</th>
<th>Ataxy present or not in affected limbs before joint affection.</th>
<th>Perforating ulcers.</th>
<th>New bony formations.</th>
<th>Remarks.</th>
</tr>
</thead>
<tbody>
<tr>
<td>Present since onset of disease</td>
<td>Began shortly before affection of right knee</td>
<td>Absent.</td>
<td>—</td>
<td>—</td>
</tr>
<tr>
<td>Present for 6 years before, but upper limbs not specially mentioned</td>
<td>Present, but slight, in upper limbs for 2 mos. before</td>
<td>—</td>
<td>A few bony concretions in capsule</td>
<td>The postero-external group of cells in the anterior column of the spinal cord in the cervical region were atrophied.</td>
</tr>
<tr>
<td>Present since onset of disease</td>
<td>—</td>
<td>—</td>
<td>—</td>
<td>—</td>
</tr>
<tr>
<td>Present for 3 years before</td>
<td>Present a little less than 2 years before affection of right knee</td>
<td>On right foot</td>
<td>Hypertrophy of lower end of femur.</td>
<td>—</td>
</tr>
<tr>
<td>Present for 4 years before</td>
<td>Present at date of report; date of onset not mentioned.</td>
<td>—</td>
<td>—</td>
<td>—</td>
</tr>
<tr>
<td>Present for 3 years before</td>
<td>Not mentioned in upper limbs.</td>
<td>—</td>
<td>—</td>
<td>—</td>
</tr>
<tr>
<td>Present for 3 years before</td>
<td>Present for 4 months before</td>
<td>—</td>
<td>—</td>
<td>—</td>
</tr>
<tr>
<td>Present for 6 years before</td>
<td>Present for 6 years before</td>
<td>—</td>
<td>—</td>
<td>—</td>
</tr>
<tr>
<td>Present for 6 years before</td>
<td>Present for 6 years before</td>
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</tr>
<tr>
<td>Present for 6 years before</td>
<td>Present for 6 years before</td>
<td>—</td>
<td>—</td>
<td>—</td>
</tr>
<tr>
<td>Present for 5 years before</td>
<td>Present for 3½ years before</td>
<td>—</td>
<td>—</td>
<td>—</td>
</tr>
<tr>
<td>Present for 5 years before</td>
<td>Began just before</td>
<td>—</td>
<td>Hyperostosis of lower end of femur; upper end of tibia thickened.</td>
<td>—</td>
</tr>
<tr>
<td>Name</td>
<td>Sex and age</td>
<td>Duration of loco-motor ataxy at onset of joint affection</td>
<td>Nature of onset of joint affection</td>
<td>Condition of joints as regards pain</td>
</tr>
<tr>
<td>-------------------</td>
<td>-------------</td>
<td>----------------------------------------------------------</td>
<td>-----------------------------------</td>
<td>------------------------------------</td>
</tr>
<tr>
<td>Sylvain M.</td>
<td>M. 46</td>
<td>Left knee, 6 months.</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>Marie M.</td>
<td>F. 46</td>
<td>Right shoulder, 10 years</td>
<td>Sudden; the joint was dislocated and disorganised in 15 days</td>
<td>Painless</td>
</tr>
<tr>
<td>Anne P.</td>
<td>F. 63</td>
<td>Both knees</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>Rose Louise Leisier</td>
<td>F. 51</td>
<td>Right knee, 13 years; right shoulder, 13 yrs. There was an interval of 6 weeks between the affection of the two joints</td>
<td>Gradual at first in knee, Sudden swelling 3 weeks later, after a fall. Sudden in shoulder. The knee was completely disorganised in 2 months</td>
<td>Painful</td>
</tr>
<tr>
<td>Jules Lemaire</td>
<td>M. 37</td>
<td>Right hip, 12 years</td>
<td>Sudden, after a fall</td>
<td>-</td>
</tr>
<tr>
<td>Charlotte Pinaigre</td>
<td>F. 36</td>
<td>Right hip, 1 yr.; left hip, 1 yr.</td>
<td>Sudden</td>
<td>Painless</td>
</tr>
<tr>
<td>Léonore Delaunay</td>
<td>F. 55</td>
<td>Left shoulder, 6 years</td>
<td>Sudden. The joint was found dislocated 2 years later</td>
<td>Painless</td>
</tr>
<tr>
<td>Trevette</td>
<td>F. 37</td>
<td>Right knee, 1 year 8 months</td>
<td>Sudden</td>
<td>Painless</td>
</tr>
<tr>
<td>Honorine Janvier</td>
<td>F. 46</td>
<td>Left hip, 9 mos.</td>
<td>Sudden. The joint was disorganised and dislocated in 3 weeks</td>
<td>Painless</td>
</tr>
<tr>
<td>Galine Sydonie</td>
<td>F. 36</td>
<td>Left knee, 8 yrs.</td>
<td>Sudden, but preceded by creaking for 8 days</td>
<td>Painless</td>
</tr>
</tbody>
</table>
| Lightning pains in limbs or not before joint affection. | Ataxy present or not in affected limbs before joint affection. | Perforating ulcers. | New bony formations. | Remarks.
<table>
<thead>
<tr>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>Present for &quot;a long time&quot;</td>
<td>Present for a long time; at onset of joint affection patient was unable to walk</td>
<td></td>
<td>Bones entering into the knee-joints were thickened. No osteophytes.</td>
<td></td>
</tr>
<tr>
<td>Present</td>
<td>Present</td>
<td></td>
<td>Small exostosis on tibia.</td>
<td></td>
</tr>
<tr>
<td>Present</td>
<td>Present for 1 yr.</td>
<td></td>
<td>A few small osteophytes</td>
<td>The cells in the anterior columns of the cord were healthy.</td>
</tr>
<tr>
<td>Present in lower limbs for 6 years before. No mention of upper limbs</td>
<td>No mention of ataxy in upper limbs. The patient could only walk with crutches</td>
<td></td>
<td>None found on post-mortem examination</td>
<td>The cells in the anterior columns of the cord were healthy.</td>
</tr>
<tr>
<td>Present for 1 year 8 months before</td>
<td>Present for 1 yr. 8 mos. before</td>
<td></td>
<td>None found on post-mortem examination.</td>
<td></td>
</tr>
<tr>
<td>Doubtful</td>
<td>Absent</td>
<td></td>
<td>None found on post-mortem examination</td>
<td>Sensibility almost lost in left leg, and almost intact in right</td>
</tr>
<tr>
<td>Present for 8 years before</td>
<td>Present for 7 months before</td>
<td></td>
<td>Small osteophytes on the border of the articular surface of the tibia.</td>
<td></td>
</tr>
</tbody>
</table>
### Summary of Table II.

**Number of cases tabulated**

<table>
<thead>
<tr>
<th>Gender</th>
<th>Number</th>
</tr>
</thead>
<tbody>
<tr>
<td>Males</td>
<td>38</td>
</tr>
<tr>
<td>Females</td>
<td>28</td>
</tr>
</tbody>
</table>

**Joints affected.**

<table>
<thead>
<tr>
<th>Joint</th>
<th>Number</th>
</tr>
</thead>
<tbody>
<tr>
<td>Right knee</td>
<td>23</td>
</tr>
<tr>
<td>Left</td>
<td>22</td>
</tr>
<tr>
<td>Right hip</td>
<td>12</td>
</tr>
<tr>
<td>Left</td>
<td>12</td>
</tr>
<tr>
<td>Right shoulder</td>
<td>7</td>
</tr>
<tr>
<td>Left</td>
<td>6</td>
</tr>
<tr>
<td>Right elbow</td>
<td>4</td>
</tr>
<tr>
<td>Left</td>
<td>3</td>
</tr>
<tr>
<td>Right tarsus</td>
<td>3</td>
</tr>
<tr>
<td>Left</td>
<td>2</td>
</tr>
<tr>
<td>Right metatarso-phalangeal</td>
<td>3</td>
</tr>
<tr>
<td>Left</td>
<td>1</td>
</tr>
<tr>
<td>Right wrist</td>
<td>1</td>
</tr>
<tr>
<td>Phalanges of hand</td>
<td>1</td>
</tr>
</tbody>
</table>

**Total**

100

**No. of cases in which single joints were affected**

41

**more than one joint was affected**

25

<table>
<thead>
<tr>
<th>Cases</th>
<th>Number</th>
</tr>
</thead>
<tbody>
<tr>
<td>Both knees</td>
<td>6</td>
</tr>
<tr>
<td>Both hips</td>
<td>2</td>
</tr>
<tr>
<td>Both shoulders</td>
<td>1</td>
</tr>
<tr>
<td>Both elbows</td>
<td>1</td>
</tr>
<tr>
<td>Both tarsi</td>
<td>2</td>
</tr>
<tr>
<td>Both metatarso-phalangeal</td>
<td>1</td>
</tr>
<tr>
<td>Knee and hip</td>
<td>3</td>
</tr>
<tr>
<td>(a) Right elbow and right tarsus</td>
<td>1</td>
</tr>
<tr>
<td>(b) Right knee, and right shoulder</td>
<td>1</td>
</tr>
<tr>
<td>Both hips and right great toe</td>
<td>1</td>
</tr>
<tr>
<td>Both hips, right elbow, and right metatarso-phalangeal of great toe</td>
<td>1</td>
</tr>
<tr>
<td>(c) Both knees and right shoulder</td>
<td>1</td>
</tr>
<tr>
<td>(d) Both hips, right knee and right shoulder</td>
<td>1</td>
</tr>
<tr>
<td>(e) Left knee, left elbow, right shoulder, right wrist, and right phalangeal</td>
<td>1</td>
</tr>
<tr>
<td>(f) Right hip, right knee, and both shoulders</td>
<td>1</td>
</tr>
<tr>
<td>Left shoulder, right elbow, and right knee</td>
<td>1</td>
</tr>
</tbody>
</table>

(a) 2 years' interval.
(b) 6 weeks' interval.
(c) 1 year's interval.
(d) 4 years' interval between right hip and right shoulder.
(e) The knee was first and the others followed within 12 months.
(f) 3 years' interval between hip and shoulders.
Joint Disease in connection with Locomotor Ataxy.

Age of patient at onset of joint disease.

Between 20 and 30 years ........................................... 2
" 30 and 40 " .................................................. 11
" 40 and 50 " .................................................. 17
" 50 and 60 " .................................................. 11
" 60 and 70 " .................................................. 1

The youngest patient was aged 29 years and the oldest was aged 62 years.

Duration of locomotor ataxy at onset of joint affection.

Knee joints.

<table>
<thead>
<tr>
<th>Duration</th>
<th>Joints</th>
</tr>
</thead>
<tbody>
<tr>
<td>Under 1 year</td>
<td>3</td>
</tr>
<tr>
<td>1 year and under 2 years</td>
<td>3</td>
</tr>
<tr>
<td>2 years</td>
<td>8</td>
</tr>
<tr>
<td>5 &quot; 10 &quot;</td>
<td>11</td>
</tr>
<tr>
<td>10 &quot; 15 &quot;</td>
<td>6</td>
</tr>
<tr>
<td>22 &quot;</td>
<td>1</td>
</tr>
</tbody>
</table>

The earliest period at which the joint disease began was (in 1 case) at the same time as the patient noticed symptoms of locomotor ataxy.

Hip joints.

<table>
<thead>
<tr>
<th>Duration</th>
<th>Joints</th>
</tr>
</thead>
<tbody>
<tr>
<td>Under 1 year</td>
<td>3</td>
</tr>
<tr>
<td>1 year and under 2 years</td>
<td>4</td>
</tr>
<tr>
<td>2 years</td>
<td>3</td>
</tr>
<tr>
<td>5 &quot; 10 &quot;</td>
<td>3</td>
</tr>
<tr>
<td>10 &quot; 15 &quot;</td>
<td>5</td>
</tr>
<tr>
<td>15½ &quot;</td>
<td>1</td>
</tr>
</tbody>
</table>

The earliest period at which the joint disease began was (in 2 cases) at the same time as the patient noticed symptoms of locomotor ataxy.

Shoulder joints.

<table>
<thead>
<tr>
<th>Duration</th>
<th>Joints</th>
</tr>
</thead>
<tbody>
<tr>
<td>3 years and under 5 years</td>
<td>2</td>
</tr>
<tr>
<td>5 &quot; 10 &quot;</td>
<td>3</td>
</tr>
<tr>
<td>10 &quot; 15 &quot;</td>
<td>5</td>
</tr>
<tr>
<td>15 &quot; 20 &quot;</td>
<td>2</td>
</tr>
<tr>
<td>3½ &quot;</td>
<td>1</td>
</tr>
</tbody>
</table>

Elbow joints.

<table>
<thead>
<tr>
<th>Duration</th>
<th>Joints</th>
</tr>
</thead>
<tbody>
<tr>
<td>1½ years</td>
<td>1 joint</td>
</tr>
<tr>
<td>2½ &quot;</td>
<td>1</td>
</tr>
<tr>
<td>5 &quot;</td>
<td>1</td>
</tr>
<tr>
<td>10 &quot;</td>
<td>1</td>
</tr>
<tr>
<td>19 &quot;</td>
<td>1</td>
</tr>
</tbody>
</table>

In one case both joints became affected 1½ years before symptoms of locomotor ataxy were observed by the patient, but at the time of observation (3 years after the onset of the joint affection) the symptoms were well marked, and included ataxy of both upper and lower limbs.
Report of the Committee on

Tarsus.

<table>
<thead>
<tr>
<th>Duration</th>
<th>Number of Joints</th>
</tr>
</thead>
<tbody>
<tr>
<td>2½ years after onset</td>
<td>2 joints</td>
</tr>
<tr>
<td>3 ½</td>
<td>1</td>
</tr>
<tr>
<td>4 ½</td>
<td>2</td>
</tr>
<tr>
<td>4 ½</td>
<td>1</td>
</tr>
</tbody>
</table>

Metatarso-phalangeal joints.

<table>
<thead>
<tr>
<th>Duration</th>
<th>Number of Joints</th>
</tr>
</thead>
<tbody>
<tr>
<td>5 years after onset</td>
<td>2 joints</td>
</tr>
<tr>
<td>12 ½</td>
<td>1</td>
</tr>
</tbody>
</table>

Wrist joint.

<table>
<thead>
<tr>
<th>Duration</th>
<th>Number of Joints</th>
</tr>
</thead>
<tbody>
<tr>
<td>5 years after onset</td>
<td>1 joint</td>
</tr>
</tbody>
</table>

Phalangeal joints of fingers.

<table>
<thead>
<tr>
<th>Duration</th>
<th>Number of Cases</th>
</tr>
</thead>
<tbody>
<tr>
<td>5 years after onset</td>
<td>1 case</td>
</tr>
</tbody>
</table>

Nature of onset of joint affection.

<table>
<thead>
<tr>
<th>Nature of Onset</th>
<th>Number</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sudden</td>
<td>56</td>
</tr>
<tr>
<td>Gradual</td>
<td>23</td>
</tr>
<tr>
<td>Not mentioned</td>
<td>21</td>
</tr>
</tbody>
</table>

An injury preceding the onset of the joint affection is mentioned in 9 cases.

Rapidity of disorganisation of joints.

Sudden onset.

<table>
<thead>
<tr>
<th>Duration</th>
<th>Number of Joints</th>
</tr>
</thead>
<tbody>
<tr>
<td>4 days after onset</td>
<td>1 joint</td>
</tr>
<tr>
<td>1 week</td>
<td>2</td>
</tr>
<tr>
<td>2 weeks</td>
<td>3</td>
</tr>
<tr>
<td>3</td>
<td>3</td>
</tr>
<tr>
<td>4</td>
<td>5</td>
</tr>
<tr>
<td>6</td>
<td>1</td>
</tr>
<tr>
<td>2 months</td>
<td>2</td>
</tr>
<tr>
<td>3</td>
<td>4</td>
</tr>
<tr>
<td>3½</td>
<td>1</td>
</tr>
</tbody>
</table>

Gradual onset.

<table>
<thead>
<tr>
<th>Duration</th>
<th>Number of Joints</th>
</tr>
</thead>
<tbody>
<tr>
<td>4 months after onset</td>
<td>1 joint</td>
</tr>
<tr>
<td>5½</td>
<td>1</td>
</tr>
<tr>
<td>9</td>
<td>2</td>
</tr>
</tbody>
</table>

Joints disorganised when first observed:

<table>
<thead>
<tr>
<th>Duration</th>
<th>Number of Joints</th>
</tr>
</thead>
<tbody>
<tr>
<td>6 months after onset</td>
<td>1 joint</td>
</tr>
<tr>
<td>12 ½</td>
<td>1</td>
</tr>
<tr>
<td>2 years</td>
<td>2</td>
</tr>
<tr>
<td>3</td>
<td>1</td>
</tr>
</tbody>
</table>

In two joints continuously observed disorganisation was complete in 2 and 8 months after the onset, and in three joints it was found at 5, 6½, and 18 months after the onset.

Mode of onset not stated.

In five joints disorganisation was found at 6 and 12 mouths, 2, 2, and 3 years after the onset.
Joint Disease in connection with Locomotor Ataxy.

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Condition of joints as regards pain.

Painful at onset in 20 cases.

Painless .... 34

In all the cases but two the pain very rapidly subsided, and the joints continued painless, in all but two cases, up to the time of observation.

Gastric and intestinal crises.

These symptoms are referred to in connection with 42 out of 45 English cases. In all except two cases the crises were gastric.

<table>
<thead>
<tr>
<th>Symptom</th>
<th>Cases</th>
</tr>
</thead>
<tbody>
<tr>
<td>Present before onset</td>
<td>16</td>
</tr>
<tr>
<td>Present after onset</td>
<td>6</td>
</tr>
<tr>
<td>Absent</td>
<td>17</td>
</tr>
<tr>
<td>Doubtful</td>
<td>3</td>
</tr>
<tr>
<td>Not mentioned</td>
<td>3</td>
</tr>
<tr>
<td>Total</td>
<td>22</td>
</tr>
</tbody>
</table>

If both the doubtful cases and those in which the symptom is not mentioned be reckoned amongst those in which the symptom was absent, there still remain, as nearly as possible, 50 per cent. of cases in which gastric crises were present.

Ataxy present or not in the affected limbs before the onset of the joint disease.

In the upper limbs the symptom is mentioned in 10 cases, and in 9 of these it was absent at the time the joint disease began. In one case it had been present for 2 months. In the lower limbs the symptom is mentioned in 37 cases, and in 20 cases it was absent at the onset of the joint disease. In 17 cases it was present. In 9 cases of the latter series it had been present for periods ranging from just before the onset of the joint disease up to 8 years before. In one case it had been present for 22 years before the onset of the joint affection.

Perforating ulcers.

This symptom is very rarely referred to in the cases we have collected from Journals and Transactions of Societies, so we have analysed those cases only which we have observed ourselves. In 27 cases we have investigated the matter, and we find that in 18 cases there were no perforating ulcers, and that in 9 cases, or one third of the total number, they were present.
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</tr>
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</tr>
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<td>29</td>
</tr>
</tbody>
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</thead>
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</tr>
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</tr>
<tr>
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</tr>
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</tr>
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